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Case Report

Cutaneous Dermatophilosis in a Meadow Jumping Mouse (*Zapus hudsonius*)

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A laboratory-housed, wild-caught, subadult, male meadow jumping mouse (*Zapus hudsonius*) presented with extensive scaling of the face, limbs, and tail and severe edema of the paws. Postmortem examination revealed marked distal limb edema with focal digital hematomas and white scales, scabs, and crusts affecting the majority of nonhaired skin. Histopathologic analysis revealed severe, multifocal, chronic-active exudative and proliferative dermatitis characterized by multilaminated crusts covering the epidermis. The epidermis was expanded by hyperkeratosis, acanthosis, and hyperplasia. The superficial dermis contained moderate edema, hemorrhage, and pigmentary incontinence, and was infiltrated by granulocytes and mononuclear cells. The laminated crusts contained numerous branching filaments of gram-positive coccoid bodies arranged in parallel rows, consistent with cutaneous *Dermatophilus congolensis* infection. This diagnosis was confirmed through bacterial culture and 16S rRNA PCR analysis. In the presented case, factors that might have contributed to disease progression include climatic conditions at the capture site and stress associated with trapping and laboratory housing.

Meadow jumping mice (*Zapus hudsonius*) are a small North American rodent that reliably prepare for hibernation in response to changes in photoperiod.[38,39,42] The use of these rodents in hibernation studies represents an attractive alternative to more traditional models because meadow jumping mice are small, docile, and can easily be manipulated into and out of hibernation.

Dermatophilus congolensis is a facultative anaerobic (although the bacteria grow well under aerobic conditions), gram-positive, branching filamentous, actinomycete bacterium that divides both longitudinally and transversely within mature filaments to form stacked, parallel rows of coccoid bodies, resulting in a characteristic 'train track' or 'stacked coin' morphology.[46] Under wet conditions, the dormant coccoid bodies are activated to motile zoospores that infiltrate the skin of hosts, causing an acute, subacute, or chronic disease known as dermatophilosis or cutaneous streptothricosis.[27,46] Disease distribution is worldwide, and the condition has been reported to occur in animals and (less frequently) in humans. *D. congolensis* is considered a zoonotic agent, given that the majority of reported human cases cite contact with animals prior to clinical presentation.[13,18] Animal infection is most frequently documented in cattle, sheep, and goats, where morbidity and mortality—as well as damage to wool, pelts, and leather— can result in considerable economic loss.[1,14,28,31,41,43,61,62] In addition, dermatophilosis is frequently diagnosed in horses, with fewer reports in other domestic animal species including pigs, cats, and dogs.[9,10,14,30,44] Furthermore, cutaneous dermatophilosis has been documented in a broad range of wildlife species, including camels, cottontail rabbits, deer, buffalo, antelope, bears, ground squirrels, raccoons, woodchucks, skunks, seals, and various reptiles.[8,20,22,24,28,37,41,43,44,51-53,55,58,62,63] Spontaneous infection has been reported in NHP including an orangutan (although not confirmed by bacterial culture), a woolly monkey, a titi monkey, and owl monkeys.[11,21,34,36] Experimental infections have occurred in mice, guinea pigs, rabbits, rats, rhesus macaques, cynomolgus macaques, and squirrel monkeys.[2,3,4,8,12,16,17,26,32-34,49,50,64]

D. congolensis is considered a normal component of the cutaneous microflora and likely requires a compromised skin barrier as a precursor to active infection.[12,46,67] Lesions are generally conserved across species and may consist of proliferative and exudative dermatitis with crusting in early stages and dermal scarring, points of dermal hemorrhage, and alopecia in advanced infections.[12,46] Mats of exudate admixed with hair often separate from the raw dermis below, forming classic 'paintbrush'-type lesions. Palisading laminar hyperkeratosis, intraepidermal pustules, and a localized inflammatory response often characterize cutaneous infection.

Herein we describe the first reported case of cutaneous dermatophilosis in a meadow jumping mouse. In the presented case, lesions were severe, diffuse, and rapidly progressive.

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The affected jumping mouse was part of an IACUC-approved effort to establish a breeding colony of meadow jumping mice. All jumping mice were housed in an AAALAC-accredited facility at the Massachusetts Institute of Technology (Cambridge, MA). Meadow jumping mice were collected within the Bolton Flats Wildlife Management Area (Bolton, MA), as permitted by the Massachusetts Division of Fisheries and Wildlife. Animals were captured by using live traps (LFA Folding Trap, HB Sherman, Tallahassee, FL) according to approved practices.[56] Traps were baited with rolled oats and peanut butter, set before dusk, checked at dawn, and left closed during the day.

Jumping mice were treated with a topical pyrethrin-based ectoparasiticide prior to facility entry and orally with ivermectin (10 µg/mL in drinking water) for 4 wk, beginning on arrival.

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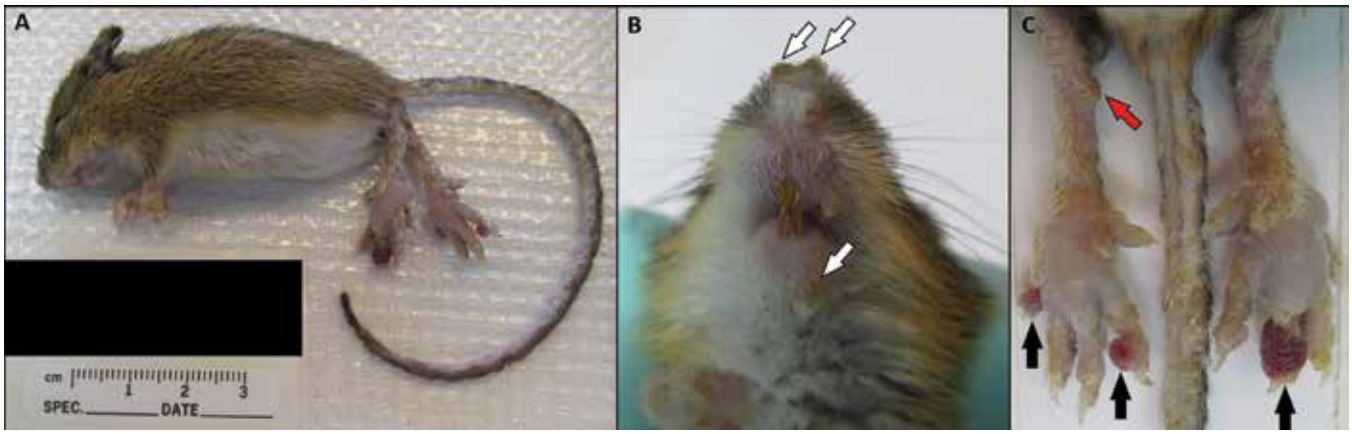

Figure 1. Meadow jumping mouse (*Zapus hudsonius*), external lesions. (A) White flakes, thick crusts, and scabbing on nonhaired skin of the feet and tail. (B) Small, round, pale-tan papules (diameter, <1 mm) on the face (white arrows). (C) Severe edema of the distal hindlimbs. Hematomas on multiple digits (black arrows). Papules similar to those on the face were present on the hindlimbs (red arrow) and forelimbs (not pictured).

Due to conspecific aggression, jumping mice were singly housed in static polycarbonate microisolation cages (Allentown Caging, Allentown, NJ) with corncob bedding (1/4-in. Bed-o Cobs, The Andersons Lab Bedding Products, Maumee, OH). Each animal was provided a small, translucent, amber-colored hut (Mouse Igloo, BioServ, Flemington, NJ) and a small amount of paper nesting material (Enviro-Dri, Shepard Specialty Papers, Kalamazoo, MI). The housing room was temperature- (20 ±1 °C) and humidity- (30% to 70%) controlled, with the light cycle set to 16 h light, 8 h dark to promote breeding and to delay entry into hibernation. Pelleted rodent chow (LabDiet RMH3000, PMI, St Louis, MO) and water were available without restriction. Cages were changed weekly, and jumping mice were visually screened for health status at least once daily.

Individual mice were screened for disease on facility entry. The presented mouse was negative for excluded agents including murine parvovirus, murine norovirus, mouse hepatitis virus, mouse rotavirus, lymphocytic choriomeningitis virus (LCMV), mouse adenovirus types 1 and 2, ectromelia virus, pneumonia virus of mice, reovirus, Sendai virus, Theiler murine encephalomyelitis virus, β-hemolytic *Streptococcus* spp. (groups A and B), *Citrobacter rodentium*, *Clostridium piliforme*, *Corynebacterium kutscheri*, *Mycoplasma pulmonis*, *Pasteurella pneumotropica*, *Salmonella* spp., *Streptococcus moniliformis*, *Streptococcus pneumoniae*, *Cryptosporidium* spp., *Entamoeba* spp., *Syphacia* spp., *Aspiculuris* spp., *Spironucleus muris*, *Helicobacter bilis*, *H. hepaticus*, *H. mastomyrinus*, *H. rodentium*, and *H. typhlonius* according to fecal PCR testing (Mouse FELASA Complete PRIA, Charles River Laboratories, Wilmington, MA) as well as *Myobia musculi*, *Myocoptes musculinus*, *Radfordia affinis*, *R. ensifera*, *Leptospira* spp., and all New-World hantaviruses by PCR-specific assays (Charles River Laboratories).

The affected male jumping mouse was captured on 24 August 2015 and was noted to be a subadult that had not yet attained full body size. In Massachusetts, the breeding season for this species begins in May or June, and the capture date and stage of growth imply that the affected mouse was in its first year of life and thus not likely more than 2 to 3 mo old.[5] The mouse had been in captivity for 1 wk prior to presentation.

On presentation, the mouse was bright, alert, and responsive, with a weight of 14.09 g. The mouse appeared adequately hydrated as evidenced by subjectively normal intrascapular skin elasticity. The animal had extensive alopecia, erythema, papules, raised plaques, and nodules covered by yellow to light brown crusts and scabs and displayed an abnormal gait. Due to the aggressive nature of these lesions (in part suggestive of ectromelia virus infection) and poor prognosis, CO_2 euthanasia was performed, followed by necropsy.

Gross pathology. Postmortem examination revealed edematous enlargement of the hindlimbs (Figure 1 A and C). Tan to yellow crusts covered the skin of the feet and tail. The muzzle and nose had 3 small, round, pale-tan papules (diameter, <1 mm; Figure 1 B). Similar nodules were present on the limbs. Digits 2 and 5 of the right hindpaw and digit 3 of the left hindpaw had 2- to 4-mm nodules that contained red-brown translucent fluid (hematomas; Figure 1 C). All other tissues and organs were grossly unremarkable.

All lesions and sections of unaffected skin, muscle, bone, joint, heart, lungs, liver, spleen, kidney, and gastrointestinal tract were collected and fixed in 10% neutral buffered formalin. Bone samples were decalcified (Cal-Rite, Richard–Allan Scientific, Kalamazoo, MI) for 24 to 48 h. After fixation, all tissues were processed, trimmed, embedded, sectioned, and stained with hematoxylin and eosin for histopathologic analysis. Samples of major organs and lesions were removed aseptically and retained at –80 °C in both sterile cryotubes and in bacteriologic freezing media (*Brucella* broth [Becton Dickinson, Franklin Lakes, NJ] containing 20% glycerol [Macron Chemicals, Center Valley, PA]) for analysis.

Histopathology. Multilaminated crusts (Figure 2 A) composed of abundant keratin, cellular and karyorrhectic debris, and degenerate neutrophils, admixed with numerous 1- to 2-µm, branching filaments consisting of stacked coccoid bodies, expanded the stratum corneum of affected skin (Figure 2 D). Hyperkeratosis, acanthosis, epidermal hyperplasia, and low numbers of neutrophils expanded the dermis. Laminated orthokeratotic to parakeratotic hyperkeratosis, hypergranulosis (Figure 2 B), and multiple coalescing intracorneal (Figure 2 A) and subcorneal (Figure 2 C) pustules composed of low numbers of granulocytes affected the superficial layers of the epidermis. Acanthosis, spongiosis, and vacuolar degeneration characterized by keratinocytes with intracytoplasmic vacuoles expanded the suprabasal layers of epidermis (Figure 2 C). The epidermal hyperplasia was irregular, with prominent rete ridges (Figure 2 A and B) and numerous mitotic figures in keratinocytes in the stratum basale (Figure 2 B and C). The superficial dermis had moderate edema, hemorrhage, and low numbers of granulocytes, mononuclear cells, and melanomacrophages (pigmentary incontinence; Figure 2 C). Occasionally, erosions and ulcerations disrupted the epidermis. The morphologic diagnosis was

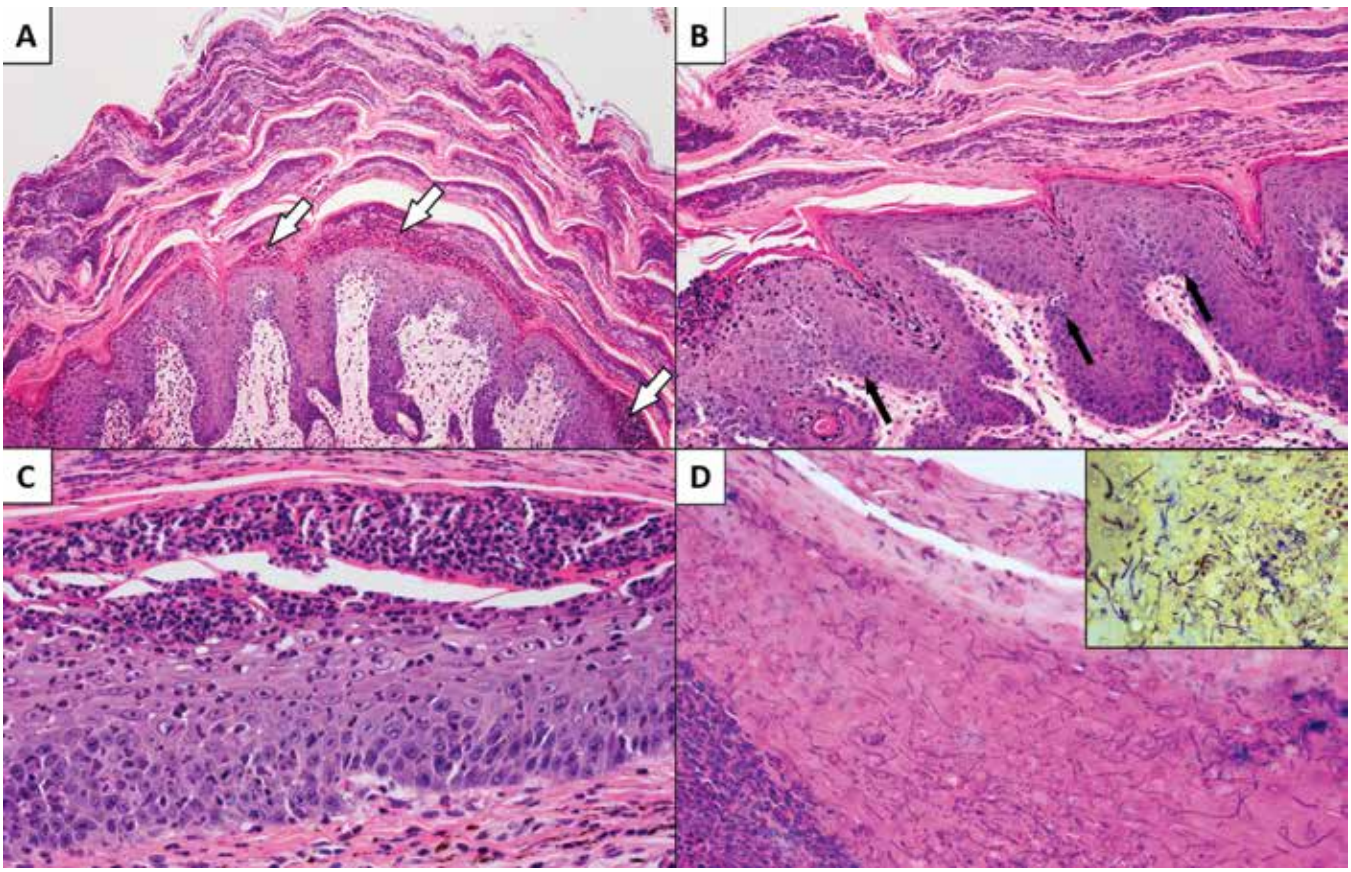

Figure 2. Meadow jumping mouse (*Zapus hudsonius*), affected skin. (A) Multilaminated crusts, resulting from extensive orthokeratotic and parakeratotic hyperkeratosis and intracorneal pustules (white arrows), expand the dermal architecture. Hematoxylin and eosin stain; magnification, 40×. (B) Hyperplasia characterized by prominent branching rete ridges, and frequent mitotic figures in the stratum basale (black arrows). Hematoxylin and eosin stain; magnification, 200×. (C) Intracorneal pustules characterized by low to moderate numbers of granulocytes. Hematoxylin and eosin stain; magnification, 400×. (D) The epidermal surface contained crusts composed of cellular debris and degenerate granulocytes admixed with numerous long branching filaments of coccoid organisms. Hematoxylin and eosin stain; magnification, 600×. Insert: affected epidermal surface; Modified Gram stain; magnification, 600×.

severe, multifocal, chronic, exudative, and proliferative dermatitis with superficial cocci and filamentous bacteria, consistent with cutaneous dermatophilosis.

Bacterial culture and molecular diagnostics. Bacterial colonies were cultivated from a crust sample taken from the affected skin at the time of necropsy. Colonies were isolated onto tryptic soy agar plates containing 5% sheep blood (Blood Agar Plate, Remel, Lenexa, KS), which subsequently were incubated for 24 to 48 h at 37 °C under aerobic conditions. Resulting bacterial colonies were gray to white, raised, rough, irregularly shaped, and β-hemolytic (Figure 3 A). Consistent with histopathologic findings, Gram staining revealed a homogeneous population of gram-positive, branching filaments of stacked coccoid bodies (Figure 3 B).

DNA was extracted from bacterial colonies (High Pure PCR Template Preparation Kit, Roche Diagnostics, Indianapolis, IN). The conserved bacterial primers 9F (5′ GAG TTT GAT YCT GGC TCA G 3′) and 1541R (5′ AAG GAG GTG WTC CAR CC 3′) from 16S rRNA genes were used to amplify 1.43-kb PCR products by using established methods.[54] DNA nucleotide sequencing was performed according to the Sanger method (Quintara Biosciences, South San Francisco, CA). Nucleotide sequence alignment by using the National Center for Biotechnology Information (NCBI) Basic Local Alignments Search Tool (BLAST) revealed 99% similarity to *D. congolensis* strain NBRC 105199 (DSM 44180; GenBank accession number, AB550800.1) based on 100% coverage.

Discussion

Among the environmental factors associated with *D. congolensis* infection, increased rain and humidity are thought to be of primary importance.[12,67] Increased saturation of the hair and skin have been linked to increased prevalence of dermatophilosis, and lesion distribution in some species tends to be concentrated in body regions prone to direct rain exposure.[12,15,46] Likewise, high humidity and intense or sustained periods of precipitation occur routinely in regions and countries where dermatophilosis is most prevalent.[67] Moisture also promotes *D. congolensis* infection by causing the release of infective zoospores from affected scabs and by compromising the skin barrier through maceration and dilution of normal antimicrobial components.[1,29,47,48] In addition, water can act as a medium for autoinfection by promoting the transfer of organisms from body site to body site and can serve as a vehicle for both direct horizontal and mechanical vector transmission.[7,67] Some laboratory studies have suggested that fulminant experimental infection requires continual moistening of infective zoospores.[19] Stress, climatic conditions, and dietary deficiencies may further increase disease susceptibility.[67]

In the wild, meadow jumping mice often inhabit moist environments and favor riparian areas or grasslands and meadows near water; jumping mice have been observed feeding in vegetation suspended over standing water, and they are strong swimmers that can take to the water to escape predators.[23,35,45,61,65,68] The affected animal was captured in a meadow dominated by

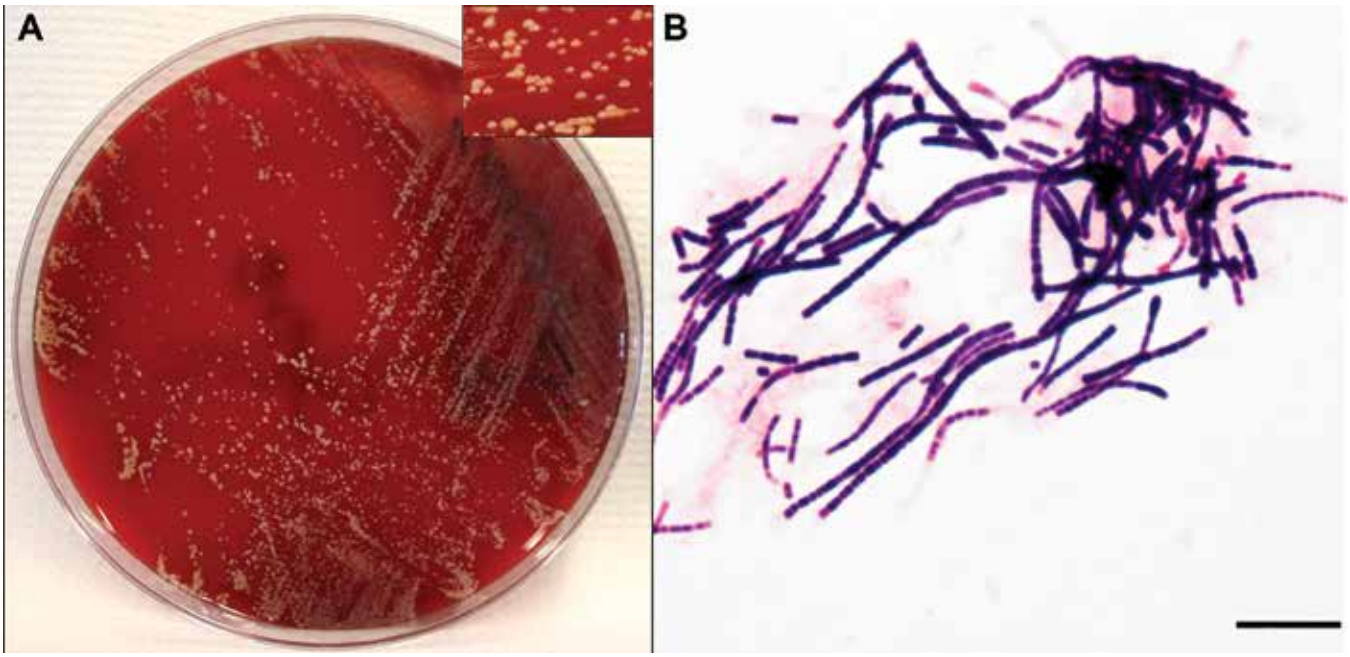

Figure 3. *Dermatophilus congolensis*, morphologic characteristics of bacterial colonies cultivated from affected skin. (A) Colonies were gray to white, raised, irregularly shaped, and β-hemolytic, with a rough surface. (B) Organisms were gram-positive with coccoid morphology and formed large aggregates of branching filaments. Gram stain; scale bar, 10 µm.

sedges, grasses, and herbaceous vegetation and bordered by trees, wetlands, and 2 slow-flowing rivers. Although traps were never set in or near standing water and even though rainfall in the region during the month of capture was lower than normal (4.88 cm compared with a 31-y-average of 9.70 cm), extensive overnight accumulation of dew on all vegetative and ground surfaces was observed each morning during the collection period of these animals.[60] This mouse was not noted to be excessively wet on capture, but its moist biologic niche may have provided conditions favorable for the spread of *D. congolensis.*

Asymptomatic colonization may precede fulminant infection, and stressors may initiate disease progression.[27] Likewise, glucocorticoid administration has been reported to increase the severity of *D. congolensis* lesions in mice.[2] *D. congolensis* is thought to be transmitted mainly by direct contact or by means of mechanical vectors, including various fly and tick species, and might be spread in a more limited fashion through contact with contaminated scabs or other organic matter.[12,46] Individual housing of jumping mice within the facility coupled with modern sanitation practices make direct and vector-associated spread unlikely. In addition, in the time between entry into the facility and emergence of clinical signs, no events occurred that would have created abnormally wet or humid conditions (for example, cage flood) in this mouse's cage. Given this situation, we consider it likely that this mouse was colonized prior to capture and that the stress of capture and adjustment to the animal facility promoted disease progression.

Although *D. congolensis* is generally susceptible to a wide range of therapies, treatment may still present a challenge, with environmental conditions as well as host and bacterial factors affecting outcome. Successful interventions range across species, although to our knowledge, successful treatment of rodent dermatophilosis has not yet been reported. For the treatment of mild infections in livestock and horses, moisture control, daily cleansing (using an antimicrobial shampoo or application of topical chlorhexidine preparations), and topical application of agents including lime sulfur, chloramine, potassium permanganate, and various iodine compounds have led to favorable outcomes.[57,59] Topical preparations made from plant species including *Senna alata*, *Lantana camara*, *Mitracarups scaber*, *Allium sativum*, *Lavandula angustifolia*, and *Thymus vulgaris* have also been used.[6,66] For more severe infections, antibiotics are routinely used. No single drug is considered a 'gold standard' in treatment, but oxytetracycline, streptomycin, penicillin, enrofloxacin, and gentamycin have been used successfully in various species.[1,25,30,9,40,50,59] Although no additional cases of cutaneous dermatophilosis have been identified in this colony, future cases could be treated with interventional regimens adapted from those used in other mammalian species.

In this report, we describe classic gross and histologic *D. congolensis* lesions in a nontraditional laboratory rodent, a meadow jumping mouse, with *D. congolensis* infection confirmed by bacterial isolation and PCR analysis. The cutaneous lesions are consistent with published reports in many other species. This condition does not affect laboratory animals frequently, and this case demonstrates the value of a comparative diagnostic approach when identifying illness in a wild-caught species whose range of disease is not well established.

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