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EPIZOOTIC PODOKNEMIDOKOPTIASIS IN AMERICAN ROBINS

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ABSTRACT: Epizootics of scaly leg disease caused by infection with the submacroscopic mite *Knemidokoptes jamaicensis* (Acari: Knemidokoptidae) in migratory American robins (*Turdus migratorius*) from a residential area of Tulsa (Oklahoma, USA) are documented during the winters (December through February) of 1993–94 and 1994–95. Estimates of 60 to >80% of the birds in several different flights arriving in the area had lesions consistent with knemidokoptic mange. Epizootic occurrence of *K. jamaicensis* also is confirmed incidentally in American robins from Georgia (USA) in 1995 and 1998 and in Florida (USA) in 1991. These are the first confirmed epizootics of scaly leg attributed to infections with mites specifically identified as *K. jamaicensis* in North America. Severity of observed lesions in American robins ranged from scaly hyperkeratosis of the feet and legs to extensive proliferative lesions with loss of digits or the entire foot in some birds. Histologically, there was severe diffuse hyperkeratosis of the epidermis which contained numerous mites and multifocal aggregates of degranulating to degenerating eosinophilic heterophils; there was mild to severe superficial dermatitis with aggregates of eosinophilic heterophils and some mononuclear cells. Based on limited data from affected captive birds in Florida, we questioned the efficacy of ivermectin as an effective acaricide for knemidokoptiasis and propose that conditions associated with captivity may exacerbate transmission of this mite among caged birds. While knemidokoptic mange apparently can result in substantial host morbidity and possibly mortality, the ultimate impact of these epizootics on American robin populations presently is unknown.

Key words: American robin, epizootic, *Knemidokoptes jamaicensis,* knemidokoptic mange, podoacariasis, scaly leg, *Turdus migratorius.*

INTRODUCTION

Knemidokoptic mange in wild and domestic birds results from infections with any of several species of barely macroscopic mites (Acari: Knemidokoptidae) that cause feather loss (depluming), cutaneous lesions around the beak and on the face (scaly face), or lesions on the feet and legs (podoacariasis, scaly leg, tassel foot) (Fain and Elsen, 1967; Urquhart et al., 1996). The prevalence of subclinical infections of knemidokoptids is largely undocumented in domestic and free-ranging birds. In wild birds and mammals infected with related taxa of sarcoptoid mange mites, the appearance of a greater number of clinical cases than the usual random isolated case appearing in the population probably denotes an epizootic event (Pence and Windberg, 1994). Their propensity to cause localized epizootics is well documented in knemidokoptid species that infect domestic poultry (Urquhart et al., 1996), domestic caged birds (Fain and Elsen, 1967), and caged wild birds (Poulsen, 1964). Herein, we presume the appearance of multiple clinical cases of knemidokoptiasis within a particular wild bird population represents an epizootic.

Apparent epizootics of podoknemidokoptiasis caused by *Knemidokoptes jamaicensis* are reported in migrating chaffinches (*Fringilla coelebs*) from the Black-Caspian Sea isthmus of southern Russia (Voinov et al., 1978) and probably this mite species in the same host from England (UK) (Macdonald and Gush, 1975). Mason and Fain (1988) found 8% of forest ravens (*Corvus tasmanicus*) in Tasmania (Australia) with skin lesions around the tibiotarsal

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joints caused by *Knemidokoptes intermedius*.

Epizootic knemidokoptic acariasis in wild free-ranging avian populations in North America is not well documented. Kirmse (1966) found 4 to 18% prevalence of foot and leg lesions attributed to knemidokoptic mange during trapping and banding of red-winged blackbirds (*Agelaius phoenicus*), common grackles (*Quiscalus quiscula*), and brown-headed cowbirds (*Molothrus ater*) in Ontario (Canada). Also, Stewart (1963) found <1% prevalence of scaly leg during banding of brown-headed cowbirds in Alabama (USA). Carothers et al. (1974) reported a 7 to 25% prevalence of scaly leg knemidokoptiasis in an establishing population of evening grosbeaks (*Coccothraustes vespertinus*) in Arizona (USA). Mites causing these infections were not specifically identified. Herein, we describe presumed epizootics of podoknemidokoptiasis caused by *K. jamaicensis* in migratory American robins (*Turdus migratorius*) in Oklahoma (USA) and confirm cases involving this species from the same host in Georgia (USA) and Florida (USA).

MATERIALS AND METHODS

On 10 January 1994 and again on 13 December 1994 one and two specimens, respectively, of adult American robins with proliferative and scabby lesions on their feet and legs were delivered to the office of the U.S. Fish and Wildlife Service (USFWS; Tulsa, Oklahoma) by M. Shoup, a local resident and member of the Audubon Society, who lived in a residential area of Tulsa (36°04′N, 95°35′W). Members of the Tulsa Audubon Society observed, with and without use of binoculars, numerous other cases of affected legs and feet in birds from flocks of robins migrating through the area during the winters of 1993–94 and 1994–95; M. Shoup maintained a written log in which she recorded dates of arrival and departure of flocks, subjectively estimated numbers of birds migrating through the area, and estimated numbers of birds that were affected as a percentage of all birds observed (M. Shoup, pers. comm.). The affected birds submitted to USFWS were killed by cervical dislocation, frozen, and forwarded to the National Wildlife Health Center (NWHC; Madison, Wisconsin, USA) for evaluation. Mites collected from the lesions were tentatively identified as *Knemidokoptes* sp. Scrapings, tissue sections, paraffin blocks containing tissue, photographs, and entire legs of the three affected birds were forwarded to Texas Tech University Health Sciences Center (TTUHSC; Lubbock, Texas, USA) where extracted mites were examined and identified.

Between 10 and 14 February 1995, six American robins found dead of a suspected organophosphate pesticide toxicosis in a neighborhood of Warner Robins (Houston County, Georgia; 32°21′N, 83°22′W) were obtained by the Georgia Department of Natural Resources (Wildlife Resources Division, Atlanta, Georgia) and submitted to the Southeastern Cooperative Wildlife Disease Study (SCWDS; University of Georgia, Athens, Georgia) for evaluation. Chlorpyrifos toxicosis subsequently was confirmed as the cause of death in all these birds. Two of these six robins had extensive dry scaly lesions on both legs. On microscopic examination of scrapings of these lesions, mites tentatively identified as *Knemidokoptes* sp. were observed. Specimens were forwarded to the National Veterinary Services Laboratory (Ames, Iowa, USA) where they were identified as *K. jamaicensis* (J. W. Mertins, pers. comm.). Also, skin scrapings and tissue sections of these lesions were subsequently examined at TTUHSC.

More recently, during mist netting of passerines for an ongoing study on mycoplasmosis by SCWDS, three robins with lesions consistent with scaly leg were captured on 4 February 1998 in Clarke County (Georgia; 33°34′N, 83°14′W). Skin scrapings from the legs of two of the three robins were forwarded to TTUHSC for examination.

On 6 February 1991 three of four American robins captured during late winter (29 January to 12 March 1991) by mist nets at the U.S. Department of Agriculture, National Wildlife Research Center, Florida Field Station (USDA DWRC FFS; Gainesville, Florida; 29°22′N, 82°13′W) presented with hyperkeratotic lesions on the legs and feet. Representative tissue specimens and scrapings from legs of infected robins were forwarded to G. Kollias (College of Veterinary Medicine, University of Florida, Gainesville, Florida) who identified the infection as knemidokoptiasis and suggested oral dosing of the infected birds with 0.2 mg/kg ivermectin (AGVET, Merck Frost, Rahway, New Jersey, USA) (G. Kollias, pers. comm.). On 8 February 1991 two infected birds also were forwarded to E. C. Greiner (College of Veterinary Medicine, University of Florida) for diagnosis. Mites tentatively identified as *Knemidokoptes mutans* were isolated from the le-

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sions (E. C. Greiner, pers. comm.). Subsequently, mites from one of these birds were reexamined at TTUHSC.

Death resulted from cervical dislocation in all birds examined from Oklahoma, Georgia in 1998, and Florida in 1991. From all the above sources, tissue for histopathologic study was excised from skin lesions on the legs and feet and fixed in 10% buffered formalin for at least 24 hr; fixed tissue was then processed and embedded in paraffin; and sections were cut at 4 to 6 μm, stained with hematoxylin and eosin, and mounted on glass slides. Additionally, skin scrapings and/or the entire lower leg and foot from each bird were preserved in 70% ethanol. Skin scrapings were examined microscopically for mites by placing a drop of the suspended material in one drop of 10% KOH solution on a glass slide. Representative specimens from skin scrapings and/or extracted from tissue in embedded paraffin blocks dissolved in xylol were prepared as whole mounts in Hoyer's medium (Krantz, 1978) for determination of species.

Mites were identified to species level according to the descriptions in Fain and Elsen (1967). Representative specimens of *K. jamaicensis* from the robins collected at the above localities are deposited in the U.S. National Museum and USDA Acari Collection (Systematic Entomology Laboratory, U.S. Department of Agriculture, Agriculture Research Service, Beltsville, Maryland, USA).

RESULTS

All mites obtained on infected birds from the 1993–94 and 1994–95 epizootics in Oklahoma, from the birds in Georgia during 1994 and 1998, and from the 1991 epizootic in Florida and examined at TTUHSC were identified as *K. jamaicensis.* Moreover, the pathology of lesions in all these hosts was consistent with that of scaly leg disease produced by this species (see Fain and Elsen, 1967; Pence, 1970). While some of these robins were slightly emaciated and had little body fat, there were no other significant visceral lesions except those related to the trauma of cervical dislocation.

In the two birds from the 1994–95 epizootic in Oklahoma the skin of the unfeathered parts of the legs and feet in one, and only the feet of the other, of these robins was markedly thickened and very rough, gray-white, desiccated, and fractured. The claw had sloughed from the first digit of one foot in the bird with the most extensive infection. Under a stereomicroscope numerous 0.10 mm black orifices were seen on the surface of the skin, suggestive of sites of invasion by the mites. Removal of the superficial proliferating epidermal layer revealed the frequently described (Pence, 1970) "honeycomb pattern" with large numbers of mites in the stratum corneum. American robins examined from Georgia and Florida had very similar lesions on the legs and feet, and occasionally loss of digits.

As previously described in other hosts (Pence, 1970), the histopathologic lesion was severe diffuse hyperkeratosis. The hyperkeratotic layer contained numerous mites and some aggregates of degenerating and/or degranulating eosinophilic heterophils and a few mononuclear cells in the epidermal scale and within some areas of the underlying superficial dermis.

In the Oklahoma epizootic, numerous American robins were observed with leg and foot lesions ranging from a white powdery substance on the legs to scab formation and proliferative growths with complete loss of the digits or entire feet in some birds (M. Shoup, pers. comm.). Although the exact prevalence of podoacariasis among American robins migrating through the Tulsa area was undetermined, numerous birds in several flocks were observed with leg and foot lesions consistent with knemidokoptiasis during the winters of 1993–94 and 1994–95. The following accounts are excerpted from the notes of M. Shoup (pers. comm.).

Flights with affected birds first were seen in November 1993 and again in January 1994. There were hundreds of individuals in each flight and about 60% or an estimated 300 to 500 birds had gray scaly lesions on their legs and feet. A single specimen was obtained and submitted to the NWHC in January 1994. A diagnosis of podoknemidokoptiasis caused by *Knemidokoptes* sp. was obtained.

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The following winter three flights with affected robins were seen. On 23 November 1994 ≥400 robins appeared in the area. An estimated 60 to 70% had lesions on the feet and legs consistent with less advanced cases of knemidokoptiasis. This flock of robins left the area after approximately 2 wk. On 9 December 1994 a flock of approximately 1,000 robins appeared in which about 80% had moderate to advanced lesions on the feet and legs. They stayed in the area for 4 wk and by the end of that period all the birds that remained appeared to have lesions consistent with scaly leg. Two birds from this flock were captured and sent to the NWHC in December 1994 where they were subsequently diagnosed as infected with *Knemidokoptes* sp. Finally, on 1 February 1995 a flight of ≥200 birds appeared. Approximately 70% of these birds had advanced lesions, and many had lost digits from one or both feet and/or an entire foot. As in the other flocks observed during this and the preceding year, the more severely affected robins had difficulty in perching and walking, many of them appeared to be lethargic, and most did not attempt to feed. Carcasses of dead birds were not observed. The origin or destination of the above flocks was undetermined, and it is unknown if any or all of these flights were part of the same or different migratory populations. There has been no followup on the status of mange in these flocks in subsequent years (1995–98).

Prevalence of knemidokoptiasis in the free-ranging wild population of American robins in Florida during the 1991 epizootic was not estimated. The American robins captured by mist nets were held in captivity for a feeding behavior study. During the course of the USDA DWRC FFS studies, nine of 66 (14%) American robins had evidence of scaly leg at the time of capture. These birds were maintained in captivity for periods of 1 to 48 days ($\bar{x} \pm SE = 25 \pm 2$) days; they were housed in close proximity to each other in individual wire cages in covered outdoor aviaries. Caged robins suffered traumatic foot lesions from contact with the wire cages. Twenty-six of 60 (43%) surviving robins had clinical signs of podoacariasis at the time of their release.

Although ivermectin was used to treat some of the affected birds captured at USDA DWRS, a drug treatment regimen was not established for treatment of infected birds. Only 12 of 67 robins were treated with ivermectin. Of these, four severely affected robins were treated at the above recommended dosage and released in only 1 to 4 days. Two additional birds infected at the time of capture were given ivermectin just prior to release after 9 to 15 days of captivity. Four robins without clinical signs of scaly leg at the time of capture were treated and never developed lesions during the subsequent 11 to 16 days of captivity. However, two other treated robins without clinical signs of podoacariasis at the time of capture subsequently developed the characteristic lesions during the following 29 days of captivity.

The twice documented clinical cases of knemidokoptiasis in the small sample of American robins from Georgia were incidental findings secondary to other studies. However, multiple cases in both instances indicate possible epizootic status here as well.

DISCUSSION

Gross lesions of the feet and legs of the American robins were similar to those of podoacariasis described in other passerines infected with *Knemidokoptes* sp. in North America (Kirmse, 1966; Pence, 1970; Carothers et al., 1974) and elsewhere (Kutzer, 1964; Fain and Elsen, 1967). While the advanced chronic lesions of podoacariasis are fairly unique and do not resemble those seen in any other common conditions, the early acute lesions of scaly leg may resemble, or be indistinguishable from, the wartlike proliferative skin lesions of avian pox in passerines. However, most reports of avian pox in these birds indicate a mild, self-limiting infection (Stallknecht,

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1997); lesions usually are few in number and appear as innocuous warty growths on one or two toes (Karstad, 1971). Certainly, avian pox is a common epizootic viral disease of many species of passerine birds in North America (Stallknecht, 1997), it may reach epizootic proportions in certain dove and passerine populations using outdoor feeders in winter (Hansen, 1987), and foot lesions have been seen in isolated cases from wild American robins (Karstad, 1971). However, we saw no evidence of avian pox in any birds actually examined from the epizootics reported herein. Moreover, the advanced lesions with extensive scaly hyperkeratosis often with loss of digits or the entire foot observed in robins from Oklahoma and Florida were more typical of those seen in knemidokoptic mange than lesions of avian pox. This, along with the confirmatory diagnosis of *K. jamaicensis* in small but random samples of robins from all the time periods and localities of this study is strong evidence that this mite is the cause of these epizootics.

The observations on the epizootic of knemidokoptiasis as an incidental finding secondary to a captive feeding behavior study of American robins in Florida are limited. However, these data do provide some basis for speculation on treatment and effects of captivity on knemidokoptic mange in this host.

Ivermectin may not be an effective acaricide for these mite infections; two asymptomatic birds that were treated with ivermectin at the time of capture subsequently developed clinical cases of knemidokoptiasis prior to release 4 wk later. However, because there was not a consistent treatment regimen or control group, the efficacy of ivermectin cannot be definitively determined for American robins from this study. Ivermectin has proven highly efficacious for other avian species with knemidokoptiasis (Bowman and Lynn, 1995).

While only nine of 66 (14%) robins had lesions indicative of knemidokoptiasis at the time of capture, 24 of 66 (36%) birds presented with clinical signs of scaly leg at the time of release 1 to 6 wk later. Conditions associated with captivity may have exacerbated the transmission of this mite infection since birds were held in close proximity to each other and the captive birds often suffered foot lesions from mechanical trauma. Also, the increased incidence of podoacariasis in these robins may have resulted from lowered resistance in birds with subclinical infections subjected to the stresses of captivity.

Originally described from specimens collected on a golden thrush (*Turdus aurantius*) from Jamaica by Turk (1950) and subsequently reported in a second thrush (*T. nudigenis*) from Trinidad by Fain and Elsen (1967), ours is the first report of *K. jamaicensis* from a species (*T. migratorius*) of Turdidae in continental North America. However, there are verified records of *K. jamaicensis* by Fain and Elsen (1967) in the host families Icteridae including the red-winged blackbird from Ohio (USA) and New York (USA), common grackle from Ontario, and Brewer's blackbird (*Euphagus cyanocephalus*) from California (USA), and the Mimidae in a catbird (*Dumatella carolinensis*) from Washington (USA); and by Pence (1970, 1972) in the host families Fringillidae and Corvidae, consisting of a rufous-sided towhee (*Pipilo erythrophthalmus*) and a common crow (*Corvus brachyrhynchos*), respectively, both from Louisiana (USA). Other suspected, but unconfirmed, records for *K. jamaicensis* in North America may include any or all of the following: red-winged blackbirds from Ontario (Kirmse, 1966) and Alabama (Stedman, 1981); common grackles, a crested flycatcher (*Myiarchus crinitus*), and a black-capped chickadee (*Parus atricapillus*) from Ontario (Kirmse, 1966); brown-headed cowbirds from Ontario (Kirmse, 1966) and Florida (Stewart, 1963); evening grosbeaks, a Cassin's finch (*Carpodacus cassinii*), and a house sparrow (*Passer domesticus*) from Arizona (Carothers et al., 1974); and common grackles, boat-tailed grackles (*Quiscalus major*), and red-winged blackbirds from Florida

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during the 1991 epizootic in robins (K. E. Brugger, unpubl. data). Finally, two cases of scaly leg caused by *K. jamaicensis* in a single evening grosbeak from New Mexico (USA) and Montana (USA), respectively, submitted to NWHL were recently diagnosed (D. B. Pence, unpubl. data).

In the Palearctic and Paleotropical regions, *K. jamaicensis* has been definitively diagnosed in cases of scaly leg from many hosts including: the family Fringillidae in feral and domestic canaries (*Serinus canaria*) from the Republic of South Africa by Fain and Elsen (1967), wild chaffinches from Russia (Voinov et al., 1978) and England (Fain and Elsen, 1967), a wild siskin (*Carduelis spinus*) and goldfinch (*Carduelis carduelis*) from Austria (Kutzer, 1964), and a caged canary, an aviary goldfinch, and a wild greenfinch (*Carduelis chloris*) from Australia (Domrow, 1992); the family Motacillidae in a pipit (*Anthus* sp.) from Sri Lanka (Fain and Elsen, 1967); the family Bombycillidae in the Bohemian waxwing (*Bombycilla garrulus*) from Italy (Ballarini, 1967); the family Muscicapidae in the sedge warbler (*Acrocephalus schoenobaenus*) from Spain (Estrada Pena et al., 1984) and Nigeria (Fry et al., 1969); and the family Meropidae in the bee-eater (*Merops apiaster*) from Nigeria (Fry et al., 1969).

These numerous and geographically diverse records are evidence for an almost ubiquitous distribution of *K. jamaicensis* in passerine birds. However, most records are of isolated cases and only the multiple cases reported in chaffinches from Russia (Voinov et al., 1978) and the American robins described herein are regarded as epizootics specifically attributable to this mite species. While *K. jamaicensis* is suspected in the epizootics reported in blackbirds from Canada (Kirmse, 1966) and in evening grosbeaks from Arizona (Carothers et al., 1974), the identity of the specific causative agent can not be confirmed. Thus, considering the observed and estimated morbidity associated with knemidokoptiasis in American robins and because other epizootics may have occurred, we emphasize the importance of documenting future epizootics and establishing the specific identification of the knemidokoptid species involved.

MANAGEMENT IMPLICATIONS

We suspect that *K. jamaicensis* infection in wild birds is similar to other forms of knemidokoptic mange in domestic and caged birds; usually, these are progressive chronic infections not undergoing remission without treatment (Poulsen, 1964; Fain and Elsen, 1967; Urquhart et al., 1996). Also, among the robins in the Oklahoma epizootics, there was a high prevalence of lethargic individuals with debilitating lesions which interfered with feeding and probably increased their potential as prey of domestic and wild predators. Thus, an increased mortality rate among infected robins was suspected, at least as a short-term impact. However, what effect this had at the population level was not determined. Moreover, lack of quantitative data on host population dynamics and on the transmission rate, infectivity, pathogenicity, virulence, duration and outcome of infection, remission or recovery rate, and other essential epizootiological aspects of *K. jamaicensis* in infected wild birds precludes meaningful speculation on the ultimate impact of the above epizootics among robins. To the casual or inexperienced observer, diseases with a high visual impact in terms of potential for morbidity and mortality such as these knemidokoptic mange epizootics in American robins may appear to have devastating consequences to a species. However, Pence and Windberg (1994) showed that over the long-term, a sarcoptic mange epizootic had an insignificant or compensatory effect at the host population level among coyotes (*Canis latrans*) which experienced nearly 70% mortality from the disease. Thus, prior to initiation of any management plans to intercede in the control, prevention, or eradication of this, or other epizootic diseases in natural popu-

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lations, an understanding of the host, spatial, temporal, and other factors affecting the dynamics of the epizootics at the population level is essential.

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