

## **Sarcoptic Mange in Raccoons in Michigan**

Authors: Fitzgerald, Scott D., Cooley, Thomas M., Murphy, Alice, Cosgrove, Melinda K., and King, Betty A.

Source: Journal of Wildlife Diseases, 40(2) : 347-350

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-40.2.347>

---

BioOne Complete ([complete.BioOne.org](https://complete.BioOne.org)) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at [www.bioone.org/terms-of-use](https://www.bioone.org/terms-of-use).

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

---

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

## Sarcoptic Mange in Raccoons in Michigan

Scott D. Fitzgerald,<sup>1,2,5</sup> Thomas M. Cooley,<sup>3</sup> Alice Murphy,<sup>1</sup> Melinda K. Cosgrove,<sup>3</sup> and Betty A. King<sup>4</sup>

<sup>1</sup> Diagnostic Center for Population and Animal Health, Michigan State University, East Lansing, Michigan 48824, USA; <sup>2</sup> Department of Pathobiology and Diagnostic Investigation, College of Veterinary Medicine, East Lansing, Michigan 48824, USA; <sup>3</sup> Rose Lake Wildlife Disease Laboratory, Wildlife Division, Michigan Department of Natural Resources, East Lansing, Michigan 48823-9454, USA; <sup>4</sup> Southeast Michigan Wildlife Rehabilitation, Gross Ile, Michigan 48138, USA; <sup>5</sup> Corresponding author (email: Fitzgerald@dcpah.msu.edu)

**ABSTRACT:** Sarcoptic mange is a cause of pruritic skin disease in domestic dogs and a wide range of wildlife species. We describe sarcoptic mange in free-ranging raccoons (*Procyon lotor*). Three adult raccoons from upper Wayne County, Michigan (USA), were captured, killed, and submitted for diagnostic evaluation. The animals were intensely pruritic, and two had advanced alopecic and crusting lesions over their dorsum and hind limbs. Skin scrapings and skin biopsies revealed crusting and hyperkeratotic dermatitis with high numbers of *Sarcoptes scabiei* adults, larvae, nymphs, and eggs. These raccoons were not otherwise debilitated, with minimal internal parasites, good body condition, and no evidence of infectious bacterial or viral diseases. Because sarcoptic mange is highly contagious and affects many species, including humans, transiently, it is important that wildlife biologists and rehabilitators include sarcoptic mange in their differential list for raccoons exhibiting pruritus and alopecia.

**Key words:** Parasites, pathology, *Procyon lotor*, raccoon, *Sarcoptes scabiei*, sarcoptic mange.

Sarcoptic mange due to infestation with *Sarcoptes scabiei* is characterized by severe pruritic dermatitis. Sarcoptic mange occurs frequently in domestic dogs and transiently affects cats and humans (Scott et al., 2001). The list of wild species reported naturally infected by *Sarcoptes* is long and includes animals from many orders and families (Bornstein et al., 2001). In the present report, we provide the first description of raccoons (*Procyon lotor*) naturally infected with *S. scabiei*.

During 2002 and 2003, approximately 175 and 125 raccoons, respectively, were handled by animal control personnel and rehabilitators in Wayne County, Michigan (USA). Approximately 75% of these raccoons exhibited skin lesions suggestive of mange. We describe findings from three

adult raccoons, two male and one female, weighing 2.8–4.7 kg, captured in Trenton, Michigan (Wayne County, 42°08.337'N, 83°10.552'W) by Trenton Animal Control during spring 2003. These raccoons all exhibited severe pruritis, with widespread alopecia and crusting dermatitis. Because of the severity of the skin condition, the animals were humanely killed by injection and submitted for further diagnostic evaluation.

Two raccoons had severe dermal lesions extending along the dorsum from the head to the tail base and laterally down both sides of the thorax and abdomen. There was marked alopecia in affected areas and thick (up to 7 mm), fissured, brown-black crusts covering much of the affected skin (Fig. 1A). The third raccoon had less severe skin lesions, with patchy alopecia around the tail base and all four limbs, and limited crust formation. Affected skin was scraped with a scalpel blade to obtain hairs and crusts for parasitologic examination. Full-thickness skin sections were resected and fixed in 10% buffered formalin for paraffin embedding and routine histopathologic testing.

Samples from each raccoon were processed separately for parasitologic testing. Skin scrapings, 100–200 mg each, were placed in a beaker with 30 ml of 10% potassium hydroxide and heated, without boiling, on a stir plate until all hair was dissolved (Bowman, 1999). The suspended material was poured into a centrifuge tube and centrifuged for 5 min at 700 × G, and the supernatant was discarded. Pellets were resuspended in saturated sucrose solution, specific gravity 1.22, and centrifuged again for 5 min at 700 × G. Each tube was filled with sucrose to form a

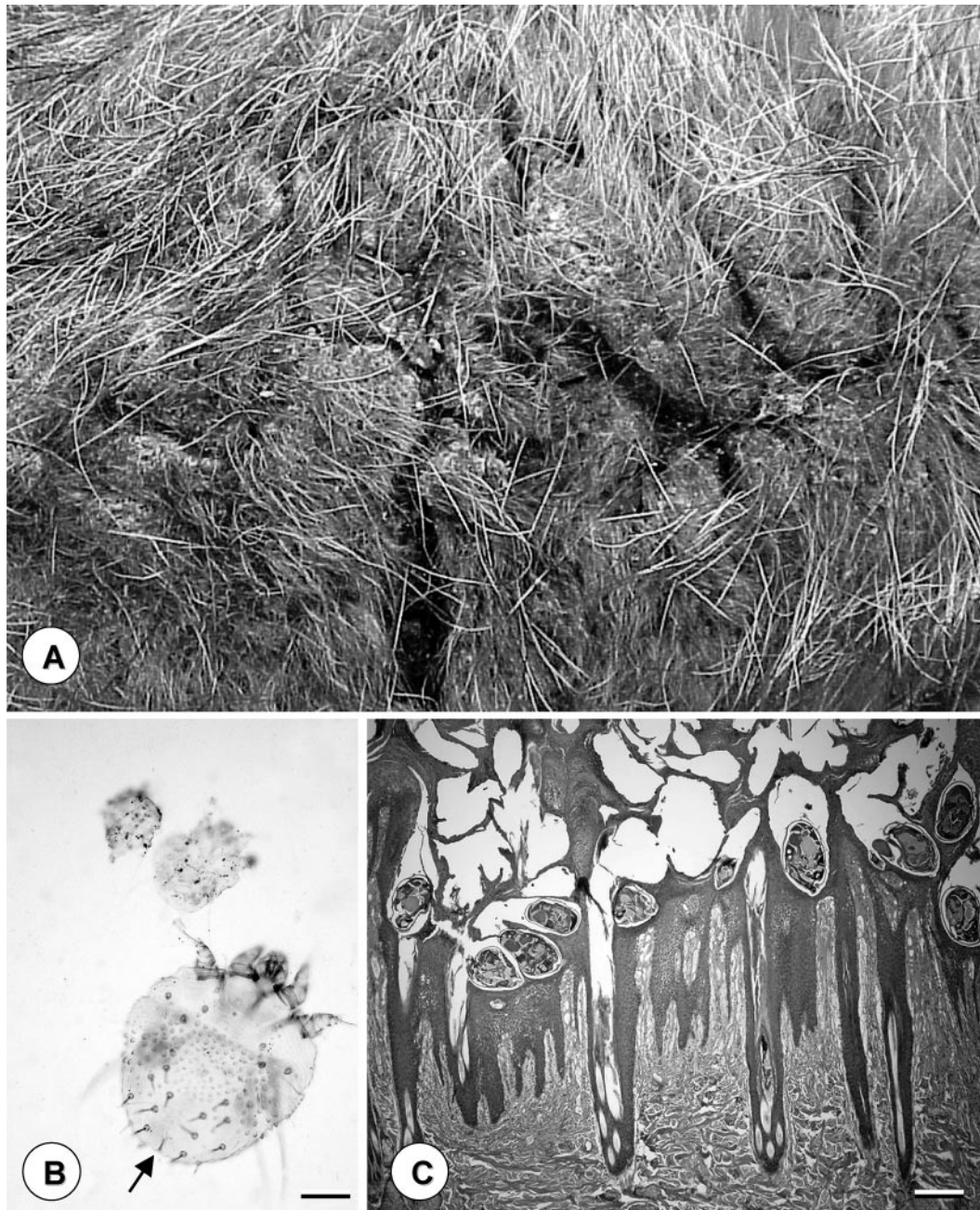


FIGURE 1. A. Photograph of skin from the back of an adult raccoon exhibiting severe crusts with fissures and moderate alopecia. B. Photomicrograph of an adult *Sarcoptes scabiei* mite; note two pairs of anterior legs, and the terminal anus (arrow). Adjacent are two smaller nymphs. Bar = 100  $\mu$ m. C. Photomicrograph of sarcoptic mange infested skin. Note the marked hyperkeratosis with intracorneal tunnels and numerous mites, and the hyperplastic epidermis with rete ridges. H&E. Bar = 250  $\mu$ m.



slight meniscus at the top, then a coverslip was placed in contact with the sucrose for 5–10 min. Each coverslip was carefully removed, placed on a glass slide, and microscopically examined for the presence of mites and eggs. Numerous mites, of all stages from eggs to adults, were present in each preparation from all three raccoons. An estimate of the total number of mites and eggs recovered from 1 cm<sup>2</sup> of skin from one of the severely affected raccoons and treated by the above procedure was 1,179 eggs per cm<sup>2</sup>, 641 larvae per cm<sup>2</sup>, 433 nymphs per cm<sup>2</sup>, and 312 adults per cm<sup>2</sup> (mean of three counts). Adult mites were roughly circular in shape, with eight legs with long, unsegmented pedicels ending in bell-shaped suckers on both anterior pairs of legs, whereas both pairs of posterior legs did not protrude beyond the margin of the body. The dorsal body surface had grooves, triangular scales, and paired spines and hairs. Adult male mites were 169×238 µm (mean of 10 mites measured), whereas adult females were 269×369 µm (mean of 10 mites measured); these measurements fall within published ranges for *S. scabiei* (Bowman, 1999). Larval and nymph forms were obviously smaller than adults and were similar in appearance, except that larvae had only six legs; nymphs had eight legs. Representative parasites have been deposited in the A. J. Cook Arthropod Collection, Natural Science Building, Michigan State University (East Lansing, Michigan; accession number VC200301). The principal feature which differentiates *Sarcoptes* mites from *Notoedres* mites is a terminal anus (Fig. 1B); the anus is located dorsally in *Notoedres* (McDaniel, 1979; Bowman, 1999).

Histologically, the two severely affected raccoons had extensive parakeratotic and orthokeratotic hyperkeratosis, with numerous intracorneal tunnels containing large numbers of adults, larvae, nymphs, and eggs of mites (Fig. 1C). By counting the number of mites per 10× objective field under the microscope and averaging five

different fields, we estimated that the two severely affected raccoons had approximately 10 times as many mites present as the less severely affected raccoon. In addition, there were numerous aggregates of neutrophilic debris, mixed bacterial colonies, and lakes of serum. The epidermis was hyperplastic with rete ridge formation. Vessels in the superficial dermis were congested, and there were mild mononuclear leukocyte infiltrates around vessels; eosinophils were rarely seen. Lesions in the less severely affected raccoon were much milder. Crusts and hyperkeratosis were more limited and were associated with fewer, more widely scattered mites. The epidermis was mildly hyperplastic, and the underlying dermis had mild to minimal perivascular infiltrates.

This is the first report of *S. scabiei* in raccoons, which is surprising given how common raccoons are throughout much of their range in North America and how frequently they interact with humans and their pets. A recent review of sarcoptic mange in wild species lists >100 species affected, including primates, canids, felids, mustelids, bears, cattle, horses, rodents, rabbits, and marsupials (Bornstein et al., 2001). Several other procyonids have been reportedly infected with *Sarcoptes*, including red pandas (*Ailurus fulgens*) and coatis (*Nasua nasua*). We have observed sarcoptic mange in wild red foxes (*Vulpes vulpes*), coyotes (*Canis latrans*), gray wolves (*Canis lupus*), black bears (*Ursus americanus*), porcupines (*Erethizon dorsatum*), and fox squirrels (*Sciurus niger*), and the gross appearance was similar to that exhibited in these raccoons. The principal differential diagnosis for the dermal lesions in these raccoons was other forms of mange; both demodectic and notoedric mange have been reported in raccoons (Hamir et al., 1993; Ninomiya and Ogata, 2002). However, *Notoedres cati* infestation in raccoons causes a much milder dermal lesion that consists of alopecia and erythema limited to the limbs and base of the tail, whereas *Demodex* mites are limited to

hair follicles and appear very different microscopically. Therefore, both the gross lesions and specific anatomic features of the mites examined microscopically were useful in differentiating among these mange conditions.

The severe crusting, large body surfaces involved in the lesion, and massive numbers of mites present are similar to the canine form of sarcoptic mange known as crusted scabies. This condition, which was first described in humans and is known as Norwegian scabies, is characterized by much larger numbers of mites and is commonly associated with compromised immune systems (e.g., canine distemper infection or corticosteroid therapy; Gross et al., 1992). However, no evidence of immunocompromise was found in these raccoons. All three raccoons were in good body condition, with plentiful fat stores. A composite fecal examination showed only low numbers of *Capillaria* spp. eggs. A full necropsy examination failed to reveal any underlying infectious diseases such as canine distemper or parvoviral enteritis. Whether raccoons are simply highly susceptible to *S. scabiei* and routinely develop the crusted form of the disease will require the review of additional cases.

The dermal lesions associated with sarcoptic mange may be caused by direct damage incited by the mites and irritating effects of their secretions or by a hypersensitivity response directed against the mites and their products. In animal hosts that have a well-developed allergic response to sarcoptic mange, the lesions are principally caused by soft-tissue trauma in response to pruritus, and very few mites are generally found in these animals (Pence and Ueckermann, 2002). The two severely affected raccoons in the present report had numerous mites present within the hyperkeratotic epidermis, few if any eosinophils present within the inflammatory infiltrates, and little or no fibroplastic response. All of these histologic features are consistent with a minimal or absent hypersensitivity response in these raccoon

hosts (Pence and Ueckermann, 2002). Mammalian hosts may fail to respond with a hypersensitivity response because of immunologic compromise, lack of previous exposure to mites, or anergy.

Because sarcoptic mange has such a wide host range, is highly contagious, and may even infect humans transiently, wildlife rehabilitators, wildlife biologists, and others who commonly come in contact with raccoons should be aware that raccoons are susceptible to this disease. When personnel are presented with raccoons exhibiting pruritis, alopecia, and dermal crusting, these animals should be quarantined, handled wearing gloves to prevent mange transmission, and steps taken to diagnose whether these animals have *S. scabiei* infestation.

#### LITERATURE CITED

- BORNSTEIN, S., T. MORNER, AND W. M. SAMUEL. 2001. *Sarcoptes scabiei* and sarcoptic mange. In *Parasitic diseases of wild mammals*, 2nd Edition, W. M. Samuel, M. J. Pybus, and A. A. Kocan (eds.). Iowa State University Press, Ames, Iowa, pp. 107–119.
- BOWMAN, D. D. 1999. Georgis' parasitology for veterinarians, 7th Edition. W. B. Saunders Company, Philadelphia, Pennsylvania, pp. 63–66.
- GROSS, T. L., P. J. IHRKE, AND E. J. WALDER. 1992. Veterinary dermatopathology, a macroscopic and microscopic evaluation of canine and feline skin disease. Mosby Year Book, St. Louis, Missouri, pp. 123–125.
- HAMIR, A. N., D. E. SNYDER, AND C. A. HANLON. 1993. First report of a *Demodex* sp. in raccoons (*Procyon lotor*). *Journal of Wildlife Diseases* 29: 139–141.
- MCDANIEL, B. 1979. How to know the mites and ticks, the pictured key nature series. W. C. Brown Company, Dubuque, Iowa, pp. 270–271.
- NINOMIYA, H., AND M. OGATA. 2002. Notoedric mange in two free-ranging North American raccoons (*Procyon lotor*) in Japan. *Veterinary Dermatology* 13: 119–121.
- PENCE, D., AND E. UECKERMAN. 2002. Sarcoptic mange in wildlife. *OIE Scientific and Technical Review* 21: 385–398.
- SCOTT, D. W., W. H. MILLER, AND C. E. GRIFFIN. 2001. Parasitic skin diseases. In *Muller & Kirk's small animal dermatology*, 6th Edition. W. B. Saunders Company, Philadelphia, Pennsylvania, pp. 476–484.

*Received for publication 13 June 2003.*