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Authors: Bertelsen, Mads F., Østergaard, Kristine, Monrad, Jesper, Brøndum, Emil T., and Baandrup, Ulrik

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***Monodontella giraffae* Infection in Wild-caught Southern Giraffes (*Giraffa camelopardalis giraffa*)**

Mads F. Bertelsen,^{1,6} Kristine Østergaard,² Jesper Monrad,³ Emil T. Brøndum,⁴ and Ulrik Baandrup⁵

¹ Centre for Zoo and Wild Animal Health, Copenhagen Zoo, Roskildevej 38, Frederiksberg C, Denmark; ² Zoophysiology, Department of Biological Sciences, University of Aarhus, Aarhus C, Denmark; ³ Danish Centre for Experimental Parasitology, Faculty of Life Sciences, University of Copenhagen, Dyrøgevej 100, Frederiksberg C, Denmark; ⁴ Institute of Physiology and Biophysics, University of Aarhus, Aarhus C, Denmark; ⁵ Department of Pathology, Vendsyssel Hospital, Hjørring, Denmark; ⁶ Corresponding author (email: mfb@zoo.dk)

ABSTRACT: Postmortem examination of seven wild-caught southern giraffes (*Giraffa camelopardalis giraffa*) from Namibia demonstrated focal discoloration, biliary thickening, and peribiliary fibrosis affecting mainly the left liver lobe. The giraffes were infected with *Monodontella giraffae*, previously associated with lethal infections in captive okapis (*Okapia johnstoni*) and giraffes. Contrary to this, all seven giraffes investigated in the present study were clinically healthy. Based on these findings, it is suggested that the nematode *M. giraffae* may not be an unusual parasite of the giraffe and that it does not necessarily cause detrimental liver disease.

Key words: Bile duct, *Giraffa camelopardalis giraffa*, giraffe, liver, *Monodontella giraffae*, parasitology, pathology.

The nematode *Monodontella giraffae*, first identified as *Uncinaria smithi* by Weidman (1918) and later described by Yorke and Maplestone (1926), is an oviparous nematode parasite hosted by the giraffe (*Giraffa camelopardalis*) and the okapi (*Okapia johnstoni*). The adult worm is found in the biliary tracts of the liver. Little is known about *M. giraffae*, but several case reports from the first and middle part of the 20th century describe debilitating disease in individual zoo animals (Leiper, 1935; Kreis, 1950; Scheidegger, 1950; Teuscher, 1955; Frank et al., 1963). To our knowledge, no systematic studies have been published on *M. giraffae*, and notably, there are no accounts of this parasite in free-ranging or newly captured giraffes. Here we describe seven cases of *M. giraffae* infection in wild-caught giraffes from Namibia.

Seven southern giraffes (one female and six males) with a body mass of 399 ± 75 kg (mean $\pm$ SD) and an estimated age of 2–3 yr were captured near Kalkfeld in Namibia ($20^{\circ}53'14.90''$ S, $16^{\circ}10'37.88''$ E) in July 2006. They were held in bomas near the capture site in Namibia for 6 wk (18×6 m, gravel flooring) and moved to South Africa, where they were held in small pens (6×4 m, concrete flooring) for an additional 2 wk. Animals were treated with doramectin (0.2 mg/kg sc, Dectomax, Pfizer Animal Health, Exton, Pennsylvania, USA) immediately after capture and again 6 wk later, as part of a routine deworming plan. In September 2006, animals were euthanized after a physiologic study and immediately underwent a thorough postmortem examination. Selected tissues were fixed in formalin and processed routinely for histologic examination, mounted, and stained using hematoxylin-eosin (HE) and Periodic Acid Schiff (PAS). Nematodes were preserved in 70% ethanol.

In all seven giraffes, postmortem examination demonstrated focal pale discoloration, affecting mainly the lateral margin of the left liver lobe. On the surface of the visceral face of the liver, several elongate, raised yellow-white tracks were visible (Fig. 1). On cross section, there was marked thickening of the walls of the biliary system with surrounding fibrosis. Multiple thin (1 mm), 15–25-mm-long nematodes were visible within the biliary tracts. The total number of worms in each

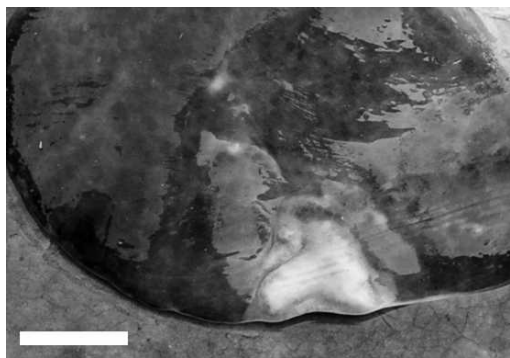


FIGURE 1. Lateral margin of the left liver lobe of a giraffe affected by *M. giraffae*. Note fibrosis along the major bile ducts. Bar=5 cm. Photo courtesy of M. Hasenkam.

animal was not quantified, but was estimated to range between 100 and 500. Body condition of the giraffes was medium to good, and no other significant pathologic findings were seen. The single female examined did not appear more or less affected than the six males. Standard biochemical blood parameters were within normal limits (ISIS 1998).

Microscopically, the lobular architecture of the liver was intact. Portal tracts of the macroscopically involved areas were widened because of fibrosis and proliferation of bile ducts. The fibrosis was of a periductal nature, with sparse inflammatory infiltrates, dominated by lymphocytes and eosinophils. Most of the bile ducts examined had normal cuboidal epithelium, but in the larger collecting ducts, focal areas of very hyperplastic, papillomatous epithelium with excessive mucin production were prominent (Fig. 2).

The worms were identified as *M. giraffae* based on their characteristic hookworm appearance observed in both sexes (Yorke and Maplestone, 1926; Frank, 1963): the anterior end was bent dorsally, the buccal cavity was deep containing a prominent dorsal cone and with cutting plates on the ventral oral margin; the male bursa was asymmetrical, and the slender spicules were equal (length about 900 μm), alated, and fused distally with barbed tips (Figs. 3 and 4). All worms were either adult males or gravid females.

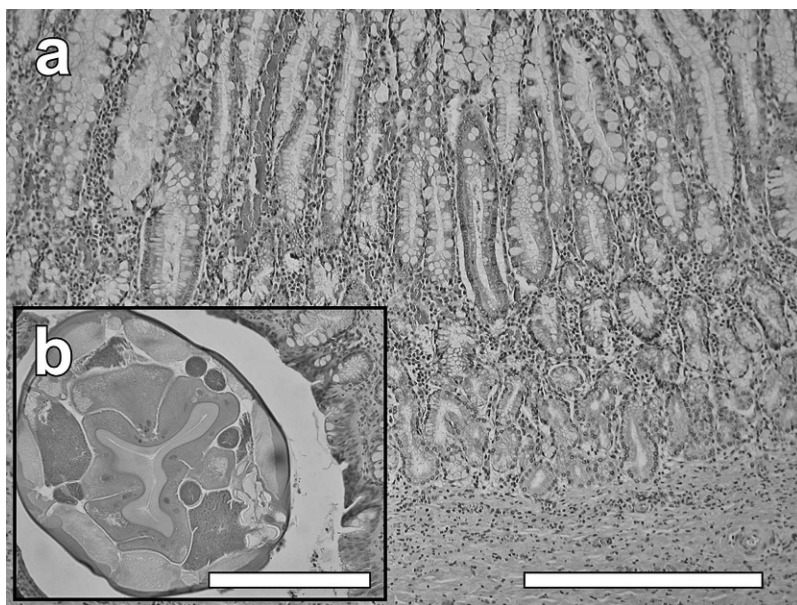


FIGURE 2. a. Photomicrograph showing periductal fibrosis, and hyperplastic, papillomatous epithelium with excessive mucin production within bile ducts. Bar=500 μm . b. Cross section of *M. giraffae* within a bile duct. Bar=500 μm .

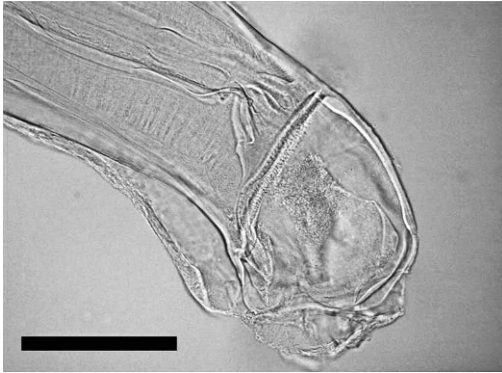


FIGURE 3. *M. giraffae*. Characteristics of anterior end: dorsally bent with a deep buccal cavity containing a prominent dorsal cone and with cutting plates on the ventral oral margin. Bar=500 μ m.

All seven giraffes hosted the hepatic nematode, *M. giraffae*. This is a somewhat surprising finding, because previous reports suggest that *Monodontella* is a very pathogenic parasite. Several early case reports describe lethal *M. giraffae* infection in okapis (Leiper, 1935; Van den Berghe, 1937; Schneidegger, 1950) and giraffes (Teuscher, 1955; Frank et al., 1963). The giraffes investigated here were immature (~ 3 yr), but although the hepatic alterations described in this paper could worsen over time, it appears unlikely that infection should inevitably lead to devastating disease and death. Indeed, considering the high

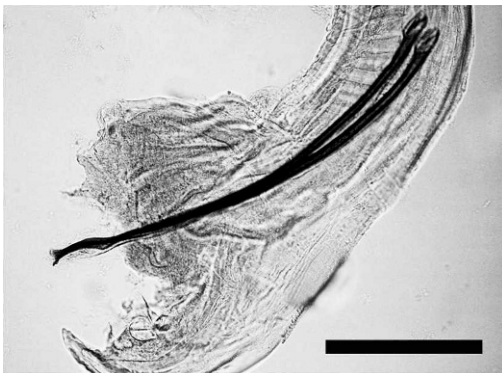


FIGURE 4. *M. giraffae*. Characteristic features of male posterior end: The male bursa is asymmetric, and the slender spicules of equal length (about 900 μ m), alated, and fused distally with barbed tips. Bar=500 μ m.

prevalence (100%) in this small study, it appears plausible that the true prevalence in the population is considerable, and likely that mortality is low.

The lesions described here are similar to those produced in domestic ruminants by mature intrabiliary trematodes such as *Fasciola* spp. (Kelly, 1993), with the notable exception that mineralization was not a feature of *M. giraffae* infection. Unlike in *Fasciola* infections, there were no signs of migratory stages, nor of previous larval migration. The lifecycle of *M. giraffae* is unknown; attempts to experimentally infect various laboratory animals (mice, rats, guinea pigs, sheep, rabbits) via the transcutaneous route were unsuccessful (Frank et al., 1963). *Monodontella giraffae* is closely related to the ruminant hookworms *Bunostomum phlebotomum* and *Bunostomum trigonocephalum*, which are known to infect percutaneously as well as orally. However, as for *M. giraffae*, the life cycles of other closely related members of the subfamily *Bunostominae* are also unknown, for example, *Grammocephalus* spp., the bile duct hookworms of elephants and rhinos (Soulsby, 1982).

In all the investigated animals, the left liver lobe was most severely affected. This is similar to what is usually seen in trematode-induced cholangiohepatitis in domestic ruminants (Kelly, 1993), but in contrast to the predilection for the right liver described in the case report by Frank et al. (1963).

Severe proliferative changes paralleling cholangioadenomas were reported in an okapi (Scheidegger, 1950) and a giraffe (Frank et al., 1963) with *M. giraffae*. In humans, cholangiocarcinomas have been associated with liver fluke infection (Schwartz, 1986). In the present cases, the changes to the ductular epithelium were those of reactive, irritative mucous metaplasia, rather than of adenomatous neoplasia. Future studies of mature giraffes from the same area might shed light on the possible carcinogenicity of monodontellosis.

Assuming that the prepatent period of *M. giraffae* is similar to that of other members of the *Bunostominae*, for instance, *Bunostomum* spp. (49–59 days; Eckert et al., 2005) and *Gaigeria pachyscelis* (70 days; Soulsby, 1982), it is considered unlikely that the animals in the present investigation became infected during the 60 days they were held in captivity. The fact that only adult worms were recovered from these cases further support this theory, making it most likely that the infection reflects the situation in the natural habitat. If anything, the antiparasitic treatment (doramectin) may have reduced the number of parasites retrieved.

This paper demonstrates *M. giraffae* to be a prevalent parasite in the biliary tract of young southern giraffes in central Namibia. Studies of adult free-ranging animals are highly encouraged.

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