Fatal Enteritis Caused by Sphaeridiotrema globulus (Trematoda: Psilostomidae) in a Whistling Swan 1

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M. pertinax was originally reported to cause atrophy of mucosa at attachment sites (Blair et al., 1979, op. cit.), but in heavier infections with a hundred or so parasites, a hyperplastic response is seen (Speare et al., 1983, op. cit.). In fecal samples it is possible to differentiate the eggs of the two species by size, shape, and state of development. The egg of *G. wallabicola* is larger than that of *M. pertinax*.

Length of 139 μm (133–144 μm) and width of 80 μm (77–83 μm) given in the original description of *M. pertinax* are slightly larger than in the present study. The width of eggs of *G. wallabicola* in this report are larger than in the original description (82–90 μm), while lengths are similar, the lengths in the original report ranging from 145 to 160 μm. The original measurements for *M. pertinax* were on fresh material, while those for *G. wallabicola* were from fixed parasites. The measurements in this study and in previous descriptions, however, agree fairly closely. In the present study the dimensions of the eggs of each species were significantly different ($P < 0.001$; Student's t-test) with no overlap of ranges. If the previously reported dimensions are also taken into account, however, some eggs of *M. pertinax* from the upper end of its range may have the same dimensions as eggs of *G. wallabicola* from the lower end of its range. The simplest feature for differentiation is the presence of an active miracidium in the egg of *M. pertinax*. Additionally, the polar thickening is more prominent and knob-like in the egg of *G. wallabicola*.

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Fatal Enteritis Caused by *Sphaeridiotrema globulus* (Trematoda: Psilostomidae) in a Whistling Swan[1]

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Sphaeridiotrema globulus infections have been reported to cause mortality in American coots (*Fulica americana*) (Trainer and Fischer, 1963, J. Wildl. Manage. 27: 483–486), lesser scaup (*Aythya affinis*) (Price, 1934, Proc. Helminthol. Soc. Wash. 1: 31–34), canvasbacks (*Aythya valisineria*) (Cornwell and Cowan, 1963, Trans. N. Am. Wildl. Nat. Resour. Conf. 23: 173–199), oldsquaw (*Clangula hyemalis*) (Sileo, pers. comm.), Muscovy ducks (*Cairina moschata*) (Campbell and Jackson, 1977, Aust. Vet. J. 53: 29–31), and a mute swan (*Cygnus olor*) (Speckman et al., 1972, J. Wildl. Dis. 8: 1–2). Infections of mute swans have been enzootic at Lake Musconetcong in northern New Jersey since 1970 (Roscoe and Huffman, 1982, Avian Dis. 26: 214–224). The swans contracted parasites presumably from ingesting the intermediate host snail *Goniobasis virginica* which was the only species of snail in the lake found to harbor infective metacercaria of *S. globulus* (Huffman and Fried, 1983, J. Parasitol. 69: 49).

On December 3, 1981 three adult and two 7-mo-old whistling swans (*Olor columbianus*) were observed on Lake Musconetcong in Netcong, New Jersey. The following day one of the juveniles was observed attempting to climb onto skim ice. It exhibited signs of weakness which included "limber neck" and "wing droop." The bird was found dead on December 5, 1981.

The bird was immediately necropsied. Blood smears were stained with Diff-Quik (Dade Diagnostics, Inc., Aguada, Puerto Rico 00602, USA). Trematodes and cestodes were fixed in hot AFA and stained with Gower's Carmine (Gower, 1939, Stain Technol. 14: 31–32). Portions of brain, liver, lung, spleen, heart, kidney, proventriculus, femoral marrow, cecum and in-

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testine were collected in 10% buffered formalin for histologic processing and staining with hematoxylin and eosin. The liver and spleen sections were also stained using Pinkerton's method for rickettsia (Luna, 1968, Manual of Histologic Staining Methods of the AFIP, McGraw-Hill Book Co., New York, 258 pp.). The stained tissue sections were examined under light microscopy.

The swan was in good flesh with a body weight of 22 kg. Gross examination revealed anemia characterized by watery thin blood, a pale flaccid myocardium and pale pink lungs. The lower small intestine was distended with a bloody fibrin core which adhered to the mucosa in segments with hemorrhaging ulcers. Small trematodes, 1 mm diameter, in groups of three to 22 were associated with each ulcer. A total of 107 were recovered.

The parasites were identified as *S. globulus* based on morphologic characteristics (Price, 1934, op. cit.). A representative specimen (USNM Helm. Coll. No. 77485) was deposited at the U.S. National Parasite Collection, Beltsville, Maryland. Another species of trematode (*Ribeiroia* sp.) and a species of cestode (*Drepanidotaenia* sp.) was found in the lower small intestine but did not appear to induce any significant lesions.

Histologic examination of the intestine revealed trematodes adhering to the mucosa by large acetabula. The trematodes were present in ulcers which penetrated the muscularis mucosae into the circular muscle layer. This layer was edematous, necrotic and infiltrated with lymphocytes and a few eosinophils on the perimeter of the ulcer. The muscular acetabulum of the trematodes adhered to the muscular layer of the intestine. No reaction suggestive of encapsulation or encystment was evident. The intestinal villi adjacent to the ulcer and overlying the inflamed muscular layer were infiltrated with lymphocytes and a few eosinophils. Hemorrhage was pronounced in these peripheral areas. The spleen was reactive with many large lymphocytes and blast forms filling the sinuses. The femoral bone marrow contained primarily adipose tissue with very few hemopoietic cells. Perivascular hepatitis, consisting primarily of a lymphocytic infiltration, was observed. Extramedullary erythropoiesis was most prominent in the liver although a few foci were noted in the kidney. No rickettsia were demonstrated in the Pinkerton's stained liver section. No significant lesions were seen in the brain, lung, heart, proventriculus and cecum. Polychromatophilic erythrocytes, binucleate cells, blast cells in mitosis in heart blood smears, and extramedullary erythropoiesis were indicative of the inadequate compensatory response to the blood loss.

The perivascular hepatitis was apparently not the result of a rickettsial infection. Previous attempts by Macy (1964, Proc. 1st Int. Congr. Parasitol. Rome, pp. 537–538) to isolate rickettsia from cecae of ducks infected with *S. globulus* were unsuccessful. Therefore, it seems unlikely there is a hyperparasitism of *S. globulus* with a rickettsia as occurs with salmon poisoning disease (Knapp and Millemann, 1970, *In* Infectious Diseases of Wild Mammals, Iowa State Univ. Press, Ames, Iowa, pp. 332–342). The perivascular hepatitis may be a response to an enterotoxemia or parasitic secretion (Roscoe and Huffman, 1982, op. cit.).

Whistling swans have the same food habits as the mute swans and the bird in question apparently consumed *G. virginica*, infected with metacercaria of *S. globulus*, at Lake Musconetcong. The signs and lesions of a *S. globulus* infection in the whistling swan are similar to those described in the mute swan. This report represents a new host record for *S. globulus*.