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#### Short communication

# Sarcocystis neurona retinochoroiditis in a sea otter (Enhydra lutris kenyoni)

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#### ABSTRACT

Sarcocystis neurona is an important cause of fatal disease in sea otters in the USA. Encephalitis is the predominant lesion and parasites are confined to the central nervous system and muscles. Here we report retinochoroiditis in a sea otter (Enhydra lutris kenyoni) found dead on Copalis Beach, WA, USA. Salient lesions were confined to the brain and eye. Multifocal nonsuppurative meningoencephalitis was present in the cerebrum and cerebellum associated with S. neurona schizonts. The retina of one eye had a focus of inflammation that contained numerous S. neurona schizonts and merozoites. The focus extended from the retinal pigment epithelium inward through all layers of the retina, but inflammation was most concentrated at the inner surface of the tapetum and the outer retina. The inner and outer nuclear layers of the retina were disorganized and irregular at the site of inflammation. There was severe congestion and mild hemorrhage in the choroid, and mild hemorrhage into the vitreous body. Immunohistochemistry with S. neurona-specific polyclonal rabbit antibodies stained schizonts and merozoites. To our knowledge this is the first report of S. neurona-associated retinochoroiditis in any naturally infected animal.

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#### 1. Introduction

Sarcocystis neurona is an important cause of a fatal disease, initially named equine protozoal myeloencephalitis (EPM), of horses in the Americas (Dubey et al., 2001a). An EPM -like disease has been reported in many hosts including marine mammals, dogs, cats, raccoons, and mink. Of all its natural hosts, EPM is probably most common and most severe in sea otters (Lindsay et al., 2000; Kreuder et al., 2003; Thomas et al., 2007; Miller et al., 2010). Recently, S. neurona infection was associated with a large scale die off in sea otters in California (Miller et al., 2010). In April 2004, 40 southern sea otters (Enhydra lutris neresis) were

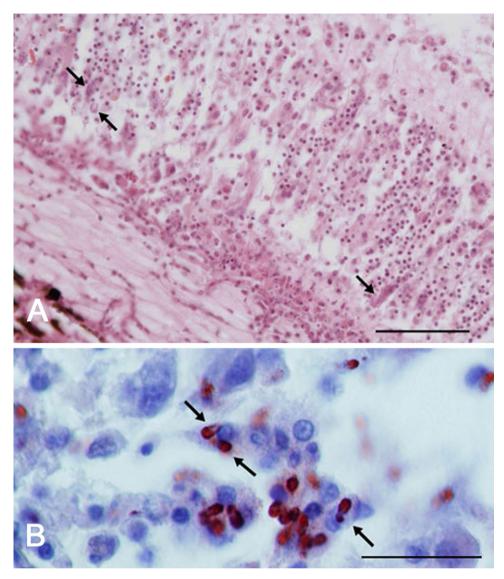
found beached on the coast line near Morro Bay, California. Sixteen of these 40 otters were investigated in detail for causes of mortality (Miller et al., 2010). Protozoal meningoencephalitis was the predominant lesion and *S. neurona* was identified in 15 of these 16 otters; there was no mention of lesions in eyes. Here, we report the first case of *S. neurona*-associated retinochoroiditis in any naturally infected animal.

#### 2. Materials and methods

#### 2.1. Necropsy and histological examination

A female subadult sea otter (115 cm long, tail 30 cm) was found dead 29 April 2010 on the north end of Copalis Beach, WA, USA. A necropsy was performed the next day. Samples of brain, pituitary gland, trigeminal nerve, heart,

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**Fig. 1.** (A) Histological section of one eye of the sea otter. Note the inflammatory focus with *S. neurona* (arrows) barely visible at this magnification. HE stain, bar =  $100 \, \mu m$ . (B) Several intracellular *S. neurona* merozoites (arrows) in retinal cells of the same eye. Immunohistochemical reaction with *S. neurona*-specific polyclonal rabbit antibodies, bar =  $35 \, \mu m$ .

lung, tracheobronchial lymph node, thymus, thyroid gland, 2 inguinal lymph nodes, liver, kidney, spleen, adrenal gland, pancreas, bile duct, stomach, 4 intestinal sections, skeletal muscle including tongue, diaphragm and abdominal wall, and 1 eye were fixed in 10% buffered formalin. The second eye was fixed in Bouin's fixative. Paraffin-embedded sections were examined after staining with hematoxylin and eosin (HE). Selected sections were examined immunohistochemically with polyclonal rabbit antibodies specific to *Toxoplasma gondii* and *S. neurona* as described (Thomas et al., 2007).

#### 3. Results

The animal was in good body condition and had no significant gross lesions.

Salient lesions were confined to the brain, heart, skeletal muscle, and one eye. Neural lesions consisted of moderate to severe multifocal nonsuppurative meningoencephalitis in the cerebrum and cerebellum associated with immature and mature protozoal schizonts, and extracellular merozoites. Immunohistochemistry for canine distemper virus was negative in brain. Protozoa reacted positively with *S. neurona* antibodies and were negative for *T. gondii*.

A moderate number of *Sarcocystis* tissue cysts (sarcocysts) were present within myofibers in all skeletal muscle sections. Sarcocysts contained metrocytes and bradyzoites and were morphologically similar to *S. neurona* sarcocysts. A moderate number of acutely necrotic or degenerate myofibers were accompained by mild mononuclear cell accumulations.

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