

4 simultaneous Markov chains over 2 million generations, sampled every 100 generations, and ended with a calculation of a 50% majority rule consensus tree. On the phylogenetic tree, sequences of Estonian isolate group together with those of other *E. multilocularis* isolates from different countries and were clearly separated from those of all other species (data not shown). The results of genetic analysis confirmed morphologic identification of *E. multilocularis*.

This study reports a new location of *E. multilocularis* in Europe. Estonia is the northernmost country on the mainland of the continent where *E. multilocularis* has been described. Because no studies have been published on the occurrence of *E. multilocularis* in Estonia in either foxes or rodents, whether this report identifies a stable endemic area or whether the parasite has expanded its range recently cannot be determined. Although a limited number of foxes were examined, the occurrence of *E. multilocularis* appears to be frequent and widespread in Estonia, which poses a risk for putatively parasite-free adjacent countries in Fennoscandia.

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Influenza Virus Infection in Racing Greyhounds

To the Editor: Influenza is globally the most economically important respiratory disease in humans, pigs, horses, and fowl (1). Influenza virus is known for its continuous genetic and antigenic changes, which impeded effective influenza control (1,2). More importantly, emergence of a new subtype by genetic reassortment or interspecies transmission is of great concern for preventing influenza epidemics and pandemics (1). Recently, influenza outbreaks have occurred in species (feline and canine) that historically do not carry influenza virus (3,4), which alerted both regulatory and scientific communities to expansion of the host range of influenza virus. We report an outbreak of respiratory disease by influenza virus infection in Iowa racing greyhounds after influenza outbreaks in Florida in 2004.

In mid-April, an influx of racing greyhounds into Iowa greyhound tracks resulted in outbreaks of respiratory disease within the track compounds. The disease was characterized by rapid onset of fever and cough, rapid respiration, and hemorrhagic nasal discharge. The illness rate was almost 100% in both race-track compounds, although the death rate was <5%. Most affected dogs recovered, yet many died of hemorrhagic pneumonia. Therapeutic administration of broad-spectrum antimicrobial drugs reduced the severity of the disease but could not control it.

Tissue samples from 4 animals that died of severe pneumonia were submitted to the Iowa State University Veterinary Diagnostic Laboratory. The animals represented 2 different racing tracks located in eastern and western Iowa. On gross examination, lungs exhibited extensive red to red-

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black discoloration with moderate to marked palpable firmness. Mild fibrinous pleuritis was also noted. Microscopically, lung sections were characterized by severe hemorrhagic interstitial to bronchointerstitial pneumonia. Patchy interstitial change with alveolar septal thickening, coagula of debris in alveoli, and associated atelectasis were evident. Focally extensive pyogranulomatous bronchointerstitial pneumonia with dilatation of airways by degenerate cells and debris was observed. Scattered vasculitis and vascular thrombi were apparent.

Microbiologic testing for conventional viral and bacterial agents did not show any important pathogens except *Streptococcus equi* subsp. *zoepidemicus* from lung tissues of all animals examined. Two of the 4 lung samples were positive for influenza A virus by a real-time reverse transcription-polymerase chain reaction (RT-PCR) (5). Viral pneumonic lesions of both lungs were positive for immunohistochemistry (IHC) with monoclonal antibody specific for the nucleoprotein of influenza A virus (6) and with antigen-capturing enzyme-linked immunosorbent assay (*Directigen Flu A*, Becton-Dickinson, Sparks, MD, USA). Bronchioalveolar lavage samples from the 2 positive lungs were also positive by RT-PCR for influenza A virus.

Virus isolation was attempted; the influenza virus in canine lungs was unexpected since no influenza virus infection in dogs had been reported, except a recent communication at a meeting of veterinary diagnosticians (4). A virus that was able to agglutinate rooster erythrocytes was isolated in Madin-Darby canine kidney cells from lung and bronchioalveolar lavage fluid of 1 of the 2 animals in which influenza virus was detected by IHC and RT-PCR. Isolates were determined by RT-PCR to be influenza A virus of H3 subtype. The US Department of Agriculture National

Veterinary Services Laboratory (Ames, IA) subtyped the virus isolates (A/Canine/Iowa/13628/2005) as H3N8 by using hemagglutination-inhibition assay and neuraminidase-inhibition assay.

Sequencing hemagglutinin (HA) and neuraminidase (NA) genes of both isolates showed 100% and 99.8% identity, respectively, between the 2 isolates. Phylogenetically, the HA gene (GenBank accession no. DQ146419) of the isolates was genetically close (96%–98% nucleotide homology) to the HA gene of recent H3N8 equine influenza viruses (7). The NA gene (DQ146420) of the isolates also showed 96%–98% homology with the NA gene of recent H3N8 equine influenza viruses. Internal genes remain to be sequenced.

In conclusion, recent outbreaks of hemorrhagic pneumonia and associated deaths in Iowa racing greyhounds were primarily due to infection by an H3N8 influenza virus genetically and antigenically similar to equine influenza viruses. This conclusion can be supported by a previous report of fatal hemorrhagic pneumonia by H3N8 virus infection in racing greyhounds in Florida (4). The fact that greyhounds in 2 different racetracks, which are in geographically remote sites in Iowa, simultaneously died of the disease without the involvement of sick horses suggests that the influenza virus isolate is likely a canine-adapted strain and able to perpetuate and spread among dogs. While influenza virus infection was likely responsible for the disease outbreaks, the contribution that *S. zoepidemicus* might have made to the disease and the severity of clinical manifestations remains to be further evaluated since the bacterium has been implicated in respiratory disease and septicemia-associated problems in many different animal species (8,9).

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Syngamoniasis in Tourist

To the Editor: *Mammomonogamus laryngeus* (*Syngamus laryngeus*) is a nematode parasite found in the larynx of tropical mammals (1), especially cattle and cats and occasionally humans (2). We report a case in a 65-year-old Caucasian man who visited Brazil from July 20 to September 9, 2004. The patient stayed in Rio de Janeiro and Ilhéus in northern Brazil. He ate local food, including salads, raw vegetables and fruits, and drank what he assumed was safe water.

Upon return to Portugal in September 2004, the patient experienced a cough and fever. He was seen in an emergency service and chest radiograph indicated infiltration in the left inferior lobe, the right basal hilum, and right apex. A complete blood count revealed a leukocyte count of 9,700/mm³, 81% polymorphonuclear leukocytes and 2.1% eosinophils. He was treated with antimicrobial drugs; a week later a radiograph showed bronchovascular markings. The patient failed to follow recommendations and in mid-October, he returned to the hospital

with a persistent cough and expectoration.

In late November the patient had a persistent cough with hemoptysis. He was given antimicrobial drugs; a computed tomographic scan showed an infiltration, a sequela to pneumonia, localized in the left superior lobe. Symptoms persisted, and bronchofibrosopic examination in January 2005 showed thickening of the bilateral bronchovascular bundles and discrete diffuse inflammation in the bronchial mucosa. A Y-shaped worm, moving and wrapped in viscous, bloody mucus, was seen around the right medial bronchus. A worm was seen in the left main bronchus and, upon closer examination, a male and female worm in copula were seen. The worms removed with forceps and identified as *M. laryngeus* (Figure). Eggs from the female were characteristic of the species.

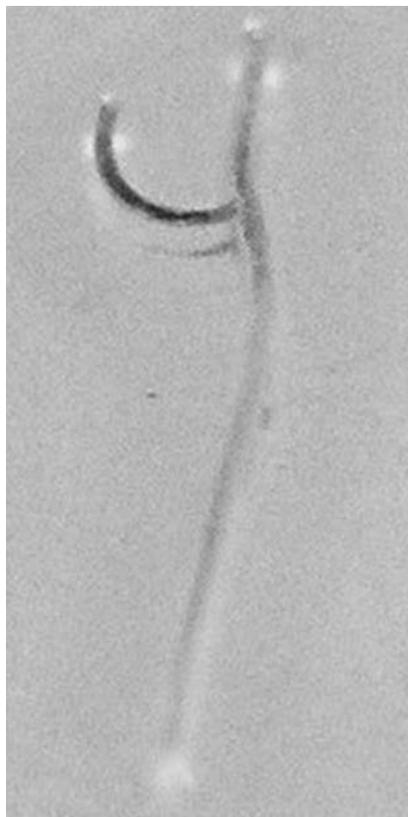


Figure. Male and female *Mammomonogamus laryngeus* recovered from the bronchial mucosa.

The patient was treated with albendazole 200 mg, 3×/day for 3 days, followed by mebendazole 100 mg, 3×/day for 3 days. The cough and hemoptysis clinically improved and abated by early February.

The genus *Mammomonogamus* consists of 2 major species, *M. laryngeus* and *M. nasicola*. The former is a parasite of the laryngotracheal region of bovids and felines, and the latter is found in the nasal fossa of bovids. *M. laryngeus* and *M. nasicola* belong to the family *Syngamidae* that contains the gapeworm of birds, *S. trachea*.

Possibly 100 human infections (3), most caused by *M. laryngeus*, have been reported from the Caribbean Islands and South America, especially Brazil, with other reports from Australia, Canada, the United States, France, United Kingdom (4), the Philippines (2), Thailand (5), and Korea (6). Many of the cases reported outside of the Caribbean and South America were usually acquired while the patient was visiting areas where *M. laryngeus* was endemic. Naturally infected ruminant host are found in tropical America, India, Africa, Malaysia, the Philippines, and Vietnam (7).

M. laryngeus is blood red; the males are joined permanently to the female and are characteristically Y shaped (Figure). The males are ≈3 mm and the females are ≈10 mm in length. The mouth opening is wide, and the buccal capsule is cup-shaped with 8–10 small teeth. The worms attach to the mucosa of the larynx in animals and cause bronchitis and cough.

The means of transmission of *M. laryngeus* is unknown but it is assumed to be similar to that of *S. trachea*, which is acquired by ingesting an embryonated egg, hatched larvae, or a paratenic host such as earthworms, snails, or arthropods. The patient in our case could have been infected by eating contaminated raw vegetation or drinking contaminated water while traveling through Brazil.