The life cycle of *M. laryngeus* is not completely known, but it is assumed to be similar to *S. trachea*, which penetrates the intestinal wall and migrates through the body of the animal to the tracheolaryngeal region (8). Eggs produced are deposited in the tracheal mucosa, swallowed, and pass in the feces.

Chronic cough and fever are the major symptoms associated with *M. laryngeus* in humans, with occasional reports of hemoptysis when the worms are in the bronchus. Worms in the larynx may cause irritation and a crawling or scratching sensation. Symptoms of asthma have been reported, and leukocytosis and eosinophilia may occur. Our patient had respiratory symptoms, persistent cough, and hemoptysis, without leukocytosis or eosinophilia.

The diagnosis of parasitosis is usually made by finding expectorated worms or visualizing by bronchoscopy and removal by forceps. Eggs may be found in sputum or feces. In our case, eggs were not found in sputum or feces.

The worms are coughed up by the patient or removed with forceps during bronchoscopy. When anthelmintics such as mebendazole and albendazole have been used, patients may have reported improvement.

Although mammomonogamiasis may not be considered an emerging parasitosis, physicians should be aware of the condition especially in patients with pulmonary symptoms who visited disease-endemic areas.

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**Human Angiostrongylus cantonensis, Jamaica**

To the Editor: Angiostrongylus cantonensis is the most common cause of eosinophilic meningoencephalitis worldwide (1). The parasite’s presence has been well documented in Jamaica in rats (definitive host) and a variety of mollusks (intermediate hosts); infections occur in humans sporadically on the island. However, the mode of transmission of infections to humans in Jamaica, where raw or undercooked mollusks are not usually eaten, is not well understood (2).

An outbreak of *A. cantonensis* occurred among American medical students vacationing in Jamaica in 2000. An epidemiologic investigation identified the probable source of infection (Caesar salad), but no biologic contaminant was determined (2). During a field investigation of *A. cantonensis*, we spoke with local farmers and vendors to identify possible routes of food contamination. While our observations were preliminary and anecdotal in nature, our findings provide valuable insight into local transmission and control of this parasite.

Humans can become infected by eating the intermediate hosts, slugs and snails, of *A. cantonensis*. Freshwater shrimp serve as paratenic hosts and reservoirs of infection for humans, both naturally and experimentally (3,4). Most reports of Jamaican eating practices indicate that terrestrial snails and slugs are not eaten and that shrimp and other meats are always eaten well cooked (5). However, during interviews with a farmer near Mavis Bank, a rural area outside of Kingston, and fishermen at the Coronation Market, Jamaica’s largest fresh produce market, we discovered that freshwater and saltwater shrimp, as well as mussels (paratenic hosts), are occasionally eaten raw. Freshwater shrimp or mussels are eaten, particularly by men, directly from rivers and streams, and freshwater and saltwater bait shrimp are eaten by fishermen.

In Jamaica, molluscicides are routinely applied to growing vegetables such as cabbage, lettuce, and bok choy to keep snails and slugs away.
ősorption of raw, infected shrimp may
be a source of sporadic angiostrongylus
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Nipah Virus Strain Variation

To the Editor: AbuBakar et al. described strain variation in Nipah virus during the 1998–1999 outbreak in Malaysia (1). They found an isolate from pigs in Perak, as well as from a flying fox, that differed markedly from pig and human isolates from the main epidemic in southern Malaysia. AbuBakar et al. proposed that this finding indicates 2 separate spillover events from bats to pigs occurred, the first in Perak in 1998 and the second in southern Malaysia in 1999. However, investigations at the time of the outbreak showed that many pigs were moved from Perak onto southern farms in early 1999. We suggest that successive spillovers of the pig population in the north can also explain the observed strain differences between northern and southern isolates.

A model from experimental studies and active farm data demonstrate that Nipah virus may have circulated repeatedly and become endemic within 1 or several large pig farms in Perak (J.R.C. Pulliam, unpub. data), which is consistent with the occurrence of human cases in Perak before the 1998–1999 outbreak. Evolution of the virus population in pigs, fol-