lowed by the reintroduction in Perak of the original strain from bats and its subsequent southward movement in infected pigs, would explain observed strain differences. Models suggest that evolution of the virus within pig populations would result in lower death rates but prolonged illness. Although the pig-adapted virus strain may have circulated on both northern and southern farms, sampling biases in favor of the more virulent strain would be expected in areas of high death rates, which would explain the observed genetic relationships between sequenced isolates.

We suggest that pigs be experimentally infected with the Perak strain of Nipah virus to determine whether differences exist in illness and death caused by this virus. Further sequencing of virus from archived pig samples will clarify with greater confidence whether multiple strains circulated in both regions.

Acknowledgments

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In response: Pulliam et al. (1) presented a model to help explain the observed Nipah virus (NV) strain differences reported earlier by AbuBakar et al. (2). The model is built around an assumption that NV was endemic in several pig farms in the north of Malaysia and that a subsequent reintroduction of the original NV caused the fatal encephalitis outbreak in 1998.

While the model is plausible, that NV infection was endemic among pigs before the 1998 outbreak is difficult to imagine in the absence of verifiable evidence. As with any virus that crosses species, NV would likely have caused severe infection, and what happened in 1998 is a classic example. Before NV could have evolved, become less virulent, and subsequently become endemic, it would have been first introduced to pigs. This initial introduction would have caused an outbreak, but no such outbreaks were reported before 1998. Furthermore, the life span of pigs reared in farms is relatively short before they are sent to the markets, which limits the time in which NV evolution could take place. Slaughtering these pigs would also

have caused infection among abattoir workers and pork handlers. At present, the finding of 2 different NV strains from 2 different outbreak foci favors the suggestion that 2 possibly overlapping NV outbreaks occurred in Malaysia in 1998. Further investigation of NV archived materials would shed further light into the possible origin of NV in the 1998 Malaysia outbreaks.

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Trichinellosis Outbreak

To the Editor: Trichinellosis is a zoonotic disease caused by the nematode *Trichinella*. Although now uncommon as a result of public health control measures, trichinellosis outbreaks have been reported in the United States (1), Europe (2,3), Mexico (4), Thailand (5), Canada (6), Lebanon (7–10), and elsewhere.

In Israel, the disease is rare because most Jewish and Muslim citizens avoid eating pork. Until 1997, only 6 small outbreaks were reported in humans; they occurred mostly in

the Christian Arab population. However, from 1998 to 2004, 10 similar trichinellosis outbreaks involving 200 Thai migrant agricultural workers occurred. The workers all took part in festive meals whose main dish was uninspected wild boar, hunted in the Upper Galilee in northern Israel, near the Lebanese border. Wild boar was also the source of several large outbreaks that were reported from 1975 to 1997 in southern Lebanon (7–10).

We report an outbreak among a group of 47 male Thai workers (mean age 32 years). The workers participated in a festive meal where the implicated wild boar meat was served. Two weeks later, 26 of them had symptoms of trichinellosis. Serologic tests were performed on all 47 workers 2–4 weeks after they ate the infected meat (first time point), 6 and 8 weeks later (second time point), or both. The specimens were tested for immunoglobulin G antibodies to *Trichinella spiralis* with the LMD Elisa kit lot 9910231 (Alexon-Trend, Ramsey, MN, USA). According to the kit insert, absorbance readings ≥ 0.3 optical density (OD) units are positive.

A case-patient was defined as a worker who had ≥ 1 of the following symptoms of trichinellosis: muscle soreness, edema of upper eyelids, fever, ocular symptoms, gastrointestinal symptoms, maculopapular rash, or pulmonary symptoms. Workers with no clinical symptoms were divided into 2 subgroups. Asymptomatic case-patients were workers with ≥ 1 positive serologic test result with or without elevated absolute eosinophil count. Nonpatients were workers whose serologic results remained negative during the 2 months of study, with normal absolute eosinophil count.

At the onset of symptoms, 2 weeks after the meal, 26 patients arrived at the emergency room of Barzilai Hospital, Ashkelon, with abdominal pain with various degrees of myalgia (23 [88%]), fever (3 [11%]), periorbital edema (11 [42%]), headache (12

[46%]), rash (9 [34%]), and cough (1 [4%]). Only 1 patient did not seroconvert during the 2-month study.

Of 18 symptomatic patients, 13 (72%) were positive at the first time point (mean ± standard deviation [SD] OD 0.87 ± 0.80; in another 4 patients, seroconversion was observed at the second time point. At this second time point, 21 persons were tested, and 20 (95%) were positive (OD 2.89 ± 1.16). Five patients showed moderate eosinophilia ($1.0–5.0 \times 10^9$ cells/L), and 4 patients had marked eosinophilia ($>5.0 \times 10^9$ cells/L). No direct correlation was observed between severity of symptoms, degree of eosinophilia, and antibody levels (OD).

Of the 21 asymptomatic workers, 7 did not have cases of trichinellosis, and 14 (67%) had ≥ 1 positive sample. At the first time point, 12 workers were tested; 7 (58%) were positive (OD 0.64 ± 0.91). At the second time point, seroconversion was observed in 4 other workers. At this time, 14 persons were tested; 10 (71%) were positive (OD 1.76 ± 1.62). In this group, 1 person had moderate eosinophilia, and 2 had marked eosinophilia.

All the persons who ate the infected meat were treated with mebendazole, 5 mg/kg twice a day for 5 days. All symptomatic patients recovered.

Epidemiologic investigation indicated that 1 large piece of meat was put in boiling water for just a few minutes before being eaten. The meat that remained from the meal was examined microscopically, and encysted *Trichinella* larvae were identified (Figure).

The attack rate in this outbreak was higher (85%) than that in other published outbreaks. One explanation for this high rate could be that our case definition was broader and included any exposed person who had a positive serologic result during the 2-month study period. Moreover, all those who ate the investigated meal gave at least 1 blood sample. In other outbreaks, only samples from acute symptomatic patients were taken (8), the follow-up was incomplete because some patients did not return for convalescent-phase serologic testing (8), or not all the affected persons were studied (7).

This outbreak demonstrates the need to increase awareness and knowledge of trichinellosis and its epidemiologic features among medical personnel, public health teams, and workers. Health education and promotion are important for migrant workers, who should be reached and informed about how to prevent trichinellosis.
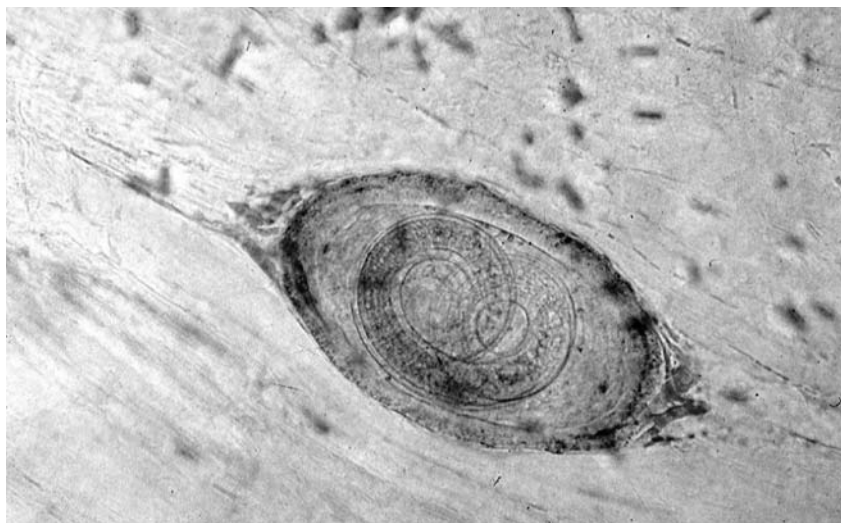

Figure. Trichinella larvae in a sample of infected meat (light microscopy, ×100).

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Ciguatera Fish Poisoning, Canary Islands

To the Editor: Ciguatera outbreaks usually occur in the area between 35° north and 35° south latitude, mainly in the Caribbean, Indo-Pacific islands, and the Indian Ocean (1–5) (Figure). Occasionally, ciguatera poisoning has been reported outside disease endemic areas, such as the Bahamas, Canada, or Chile, but no case had been described in the West African region until now. European and Spanish cases have been rarely described and are mainly associated with seafood imported from disease-endemic regions (6).

Ciguatera fish poisoning is a clinical syndrome caused by eating contaminated fish (1). The causative toxins of its clinical manifestations are ciguatoxins (7). These toxins are transmitted by dinoflagellates of the species *Gambierdiscus toxicus*, which lives adhered to damaged coral reefs in tropical seas (2). Herbivorous fish species accumulate toxins in their musculature, liver, and viscera after ingesting dinoflagellates. Larger marine carnivores eat contaminated fish and concentrate ciguatoxins (1,2).

More than 425 species of fish are associated with ciguatera poisoning in humans. The most commonly implicated fish are barracuda, red snapper, grouper, amberjack, sea bass, surgeonfish, and moray (eel) (2,3). In January 2004, 2 fishermen captured a 26-kg amberjack (local name: Medregal Negro; scientific name: *Seriola Rivoliana*) while scuba diving along the coast of the Canary Islands, Spain. The fishermen filleted the fish and stored fillets in a household freezer. Within a few days, one of the fishermen and 4 family members consumed some fish, and neurologic and gastrointestinal symptoms developed within 30 minutes to 28 hours. The 5 family members sought treatment at the emergency room of Hospital de Fuerteventura and the Outpatient Clinic of Infectious Diseases and Tropical Medicine Service of Hospital Insular de Las Palmas.

The 5 family members exhibited a combination of gastrointestinal (diarrhea [4 persons], nausea/vomiting [3 persons], metallic taste [1 person]), cardiologic (heart rhythm disturbances [2 persons]), systemic (fatigue [5 persons], itching [3 persons], dizziness [1 person]), and neurologic manifestations (myalgia [3 persons], peripheral paresthesia [3 persons], perioral numbness [2 persons], and reversal of hot and cold sensations [3 persons], which is pathognomonic of ciguatera poisoning). These clinical observations and laboratory data were collected from a prospective questionnaire filled in by physicians at the patients' first visits. No hematologic or biochemical abnormalities were detected in any patient. Based upon the symptomatic profiles, relationships of the patients, and their common dietary histories, ciguatera intoxication was diagnosed in all. None of the patients required hospitalization. The neurologic and gastrointestinal symptoms resolved over several weeks, but intermittent recurrence of some symptoms, at lower intensities, was noted for several months.

A portion of the implicated fish was recovered from freezer storage at the fisherman's home. A solid-phase membrane immunobead assay with a monoclonal antibody directed against Pacific ciguatoxins and related polyether toxins was used to detect ciguatoxins or other antigenically related