### Acknowledgment

We thank Heather Schnur for her editorial help.

## Esther Marva,\* Alex Markovics,† Michael Gdalevich,\* Nehama Asor,\* Chantal Sadik,\* and Alex Leventhal\*

\*Ministry of Health, Jerusalem, Israel; and †Kimron Veterinary Institute, Beit-Dagan, Israel H2

### References

- Roy SL, Lopez AS, Schantz PM. Trichinellosis surveillance—United States, 1997–2001. MMWR Morb Mortal Wkly Rep. 2003;52:SS1–6.
- 2. Desenclos JC. Pork related trichinellosis: emergence of a new mode of transmission? Euro Surveill. 1999;4:77.
- Djordjevic M, Bacic M, Petricevic M, Cuperlovic K, Malakauskas A, Kapel CM, et al. Social, political and economic factors responsible for the reemergence of trichinellosis in Serbia: a case study. J Parasitol. 2003;89:226–31.
- Zamora-Chavez A, O-Cavazos ME, Bernal-Rendono RM, Berrones Espericaeta D, Vazquez Antona C. Acute trichinosis in children. Intrafamilial epidemic outbreaks in Mexico City. Bol Med Hosp Infant Mex. 1990;47:395–400.
- Khamboonruang C, Nateewatana N. Trichinosis: a recent outbreak in northern Thailand. Southeast Asian J Trop Med Public Health. 1975;6:74–8.
- Schellenberg RS, Tan BJ, Irvine JD, Stockdale DR, Gajadhar AA, Serhir B, et al. An outbreak of trichinellosis due to consumption of bear meat infected with *Trichinella nativa* in 2 northern Saskatchewan communities. J Infect Dis. 2003:188:835–43.
- Haim M, Efrat M, Wilson M, Schantz PM, Cohen D, Shemer J. An outbreak of *Trichinella spiralis* infection in southern Lebanon. Epidemiol Infect. 1997;119: 357–62.
- Olaison L, Ljungstrom I. An outbreak of trichinosis in Lebanon. Trans R Soc Trop Med Hyg. 1992;86:658–60.
- Blondheim DS, Klein R, Ben-Dror G, Schick G. Trichinosis in southern Lebanon. Isr J Med Sci. 1984;20:141–4.
- Matossian RM, Rebeiz J, Stephan E. Serodiagnosis by fluorescent antibody staining of an outbreak of trichinosis in Lebanon. Ann Trop Med Parasitol. 1975; 69:387–92.

Address for correspondence: Esther Marva, Ministry of Health – Central Laboratories, POB 34410, Jerusalem, Israel 91342; fax: 972-2-655-1866; email: esther.marva@eliav.health. gov.il

# Ciguatera Fish Poisoning, Canary Islands

To the Editor: Ciguatera outbreaks usually occur in the area between  $35^{\circ}$  north and  $35^{\circ}$  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

## LETTERS



Figure. Worldwide distribution of ciguatera. Gray indicates coral reef regions located between 35° north and 35° south latitudes; darker gray indicates disease-endemic areas of ciguatera; black circle indicates Canary Islands (latitude 28°06' north, longitude 15°24' west. Source: refs. 4 and 5.

substances in fish tissues. Results were positive.

A 150-g sample of the fish was delivered to the US Food and Drug Organization's Gulf Coast Seafood Laboratory, Dauphin Island, Alabama, USA, for sodium channel-specific in vitro assay (8) and liquid chromatography-mass spectrometry (LC/MS/ MS) analysis. Assay results were positive and the ciguatoxin content of the fish sample was estimated to be 1.0 ppb (ng/g). Caribbean ciguatoxin (CCTX-1: MH+ m/z 1141.6) was confirmed by LC/MS/MS by using multiple reaction monitoring (9). The amount of ciguatoxin in the fish tissue estimated by in vitro assay was low, and close to the limit the LC/MS/MS method can detect. At least 2 additional toxins were detected in the fish sample by in vitro assay of liquid chromatography fractions. We cannot rule out the possibility that these toxins represent new ciguatoxinlike structures unique to the eastern Atlantic. Further studies are necessary to elucidate all toxins implicated in this outbreak.

Classic symptoms of ciguatera developed in our patients after eating a fish they captured in the Canary

Islands, which are not in the ciguatera-endemic zone (Figure). The preliminary results of this outbreak investigation suggest the presence of ciguatoxins or ciguatoxinlike structures in fish from temperate waters of the eastern Atlantic. Ciguatera poisoning is a matter of public health concern and residents of coastal West Africa and the regional island archipelagos could be a new community at risk for this seafood intoxication syndrome. We emphasize that ciguatera poisoning is a debilitating disease, and therapeutic intervention strategies are very limited (10).

#### Acknowledgments

We thank Ana Gago, for her collaboration in analytical procedures and Isaías. Naranjo for his editorial assistance in preparing the manuscript.

Jose-Luis Pérez-Arellano,\*† Octavio P. Luzardo,\* Ana Pérez Brito,‡ Michele Hernández Cabrera,\*† Manuel Zumbado,\* Cristina Carranza,\*† Alfonso Angel-Moreno,\*† Robert W. Dickey,§ and Luis D. Boada\* \*University of Las Palmas de Gran Canaria, Las Palmas de Gran Canaria, Spain; †Hospital Universitario Insular de Gran Canaria (Canary Health Service), Las Palmas de Gran Canaria, Spain; ‡Hospital de Fuerteventura, Puerto del Rosario, Spain; and §Gulf Coast Seafood Laboratory (Food and Drug Administration), Dauphin Island, Alabama, USA

### References

- Ting JY, Brown AF. Ciguatera poisoning: a global issue with common management problems. Eur J Emerg Med. 2001;8: 295–300.
- 2. Caplan CE. Ciguatera fish poisoning. CMAJ. 1998;159:1394.
- Pearn J. Neurology of ciguatera. J Neurol Neurosurg Psychiatr. 2001;70:4–8.
- Juranovic LR, Park DL. Foodborne toxins of marine origin: ciguatera. Rev Environ Contam Toxicol. 1991;117:51–94.
- 5. Lewis RJ. The changing face of ciguatera. Toxicon. 2001;39:97–106.
- Puente S, Lago M, Subirats M, González Lahoz JM. Ciguatera: un caso importado. Med Clin (Barc). 1995;104:357.
- Glaziou Ph, Legrand AM. The epidemiology of ciguatera fish poisoning. Toxicon. 1994;32:863–73.
- Dickey R, Jester E, Granade R, Mowdy D, Moncreiff C, Rebarchik D, et al. Monitoring brevetoxins during a *Gymnodinium breve* red tide: comparison of sodium channel specific cytotoxicity assay and mouse bioassay for determination of neurotoxic shellfish toxins in shellfish extracts. Nat Toxins. 1999;7:157–65.
- Pottier I, Hamilton B, Jones A, Lewis RJ, Vernoux JP. Identification of slow and fastacting toxins in a highly ciguatoxic barracuda (*Sphyraena barracuda*) by HPLC/MS and radiolabelled ligand binding. Toxicon. 2003;42:663–72.
- Pérez CM, Vásquez PA, Perret CF. Treatment of ciguatera poisoning with gabapentin. N Engl J Med. 2001;344: 692–3.

Address for correspondence: José-Luis Pérez-Arellano, Infectious Diseases and Tropical Medicine Service, Department of Medical and Surgical Sciences, Health Sciences Faculty, University of Las Palmas de Gran Canaria, PO Box 550, 35080 Las Palmas de Gran Canaria, Spain; fax: 34-928-45-1413; email: jlperez@ dcmq.ulpgc.es

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.