Ciguatera[1]

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Ciguatera is form of food poisoning caused by toxins produced by dinoflagellates, a large group of protists that occur in marine and fresh water habitats. The toxins accumulate in the tissues of marine fish and invertebrates that eat the dinoflagellates and produce the poisoning when they, in turn, are consumed by humans and other animals.

Background

Historical

There are various versions for the origin of the word ciguatera, including a "lost in translation" version that the term came when in the 1800s an Englishman in Cuba explained that he contracted the illness after eating "seawater" fish. Actually, the term originates from the word *cigua,* the Spanish common name of a gastropod (*Cittarium* (*Livonia*) *pica*), which is commonly consumed troughout the Caribbean, particularly in cebiche, and which has been linked to ciguatera. This mollusk is sometimes called *siwa* in the English speaking Caribbean. The word was used in Havana in 1787 by the biologist Antonio Parra to describe an intoxication with *L. pica*, and became common after the Cuban naturalist, Felipe Poey, used it to describe similar occurrences.

Early reference to ciguatera-like illnesses are made in the 1555 Chronicle of the Indies by Pedro Martyr de Anglería, but earlier references to what could be ciguatera include Homer's Odyssey (800 BC), and an outbreak in China in 600 BC. In the times of Alexander the Great (323-356 BC), soldiers were prohibited from consuming fish to avoid illness during the conquests. More definite reports occur in 1601 (Indian Ocean), in 1770 (South Pacific), in 1774 by Captain James Cook, and in 1792 from French Polynesia.

Significant advances in our knowledge of ciguatera poisoning have been made since Randall hypothesized in 1959 that the toxin was introduced into the food chain by herbivorous fish that consumed toxic microalgae and were, in turn, consumed by larger predatory fish (Randall 1959). Notable among these are the identification and isolation of ciguatoxin in 1967 (Scheuer 1967), the discovery by Yamamoto an colleagues of a dinoflagellate that produced the ciguatoxin (Yamamoto *et al.* 1977), and the identification of the structure of a major ciguatoxin and its precursor in *G. toxicus* (Murata *et al.* 1989).

Dinoflagellates

The dinoflagellates form a large and diverse group of microscopic, usually unicellular organisms that are classified as protists (cellular organisms that cannot be classified as fungi, animals, or plants). They generally have two whip-like body projections (the flagellae) of unequal size that are used for locomotion and which result in a characteristic spiral swimming motion. Some dinoflagellates are photosynthetic and free-living, others are autotrophic and symbiotic with marine animals and protozoa, some are predators on protozoa, and a few forms are parasitic. Some species produce neurotoxins which can kill fish and accumulate in organisms that can then be eaten by humans and other animals.

Large blooms of dinoflagellates often impart a reddish tinge to the water and are known as "red tides". The "Florida red

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tide" is usually caused by the dinoflagellate *Karenia brevis,* whereas in the northern East Coast of North America the major culprit is another dinoflagellate known as *Alexandrium fundyense.*

The species most commonly associated with ciguatera is the photosynthetic dinoflagellate *Gambierdiscus toxicus* (Figure 1). This species normally grows as an epiphyte in other large algae or on the surface of dead coral. It can often disperse to new regions on pieces of floating algae but it is not associated with red tides.

Figure 1. The dinoflagellate *Gambierdiscus toxicus.*

Credits: Florida Fish and Wildlife Conservation Commission.

Ciguatera Poisoning

The ciguatera chain starts when herbivorous animals consume the dinoflagellates and their toxins, concentrate and transform the toxins in their tissues, and pass them up the food chain, usually with further accumulation and concentration accompanying each step. More than 400 marine species in 60 different families have been found to accumulate ciguatoxins (Brusle 1997). Among the most important because of their seafood value are barracuda; some snapper (for example red, dog, blackfin, and cubera); amberjack, kingfish; some grouper (for example red, Nassau, snowy, yellowedge, and speckled hind); and hogfish; (Figure 2). Rarely, some primary consumers including herbivorous fish and invertebrates also cause ciguatera poisoning.

The risk of ciguatoxicity can vary widely, even within species, depending upon the ubiquitness of ciguatoxins in the individual's diet; the rate of consumption; the ability to absorb, metabolize, and excrete the toxins; and the individual growth rate. Within species, the percent of poisonous individuals tends to increase with size because older individuals have more time to consume and accumulate toxins in their tissues, but there are no hard and fast rules regarding organism size and risk of ciguatera.

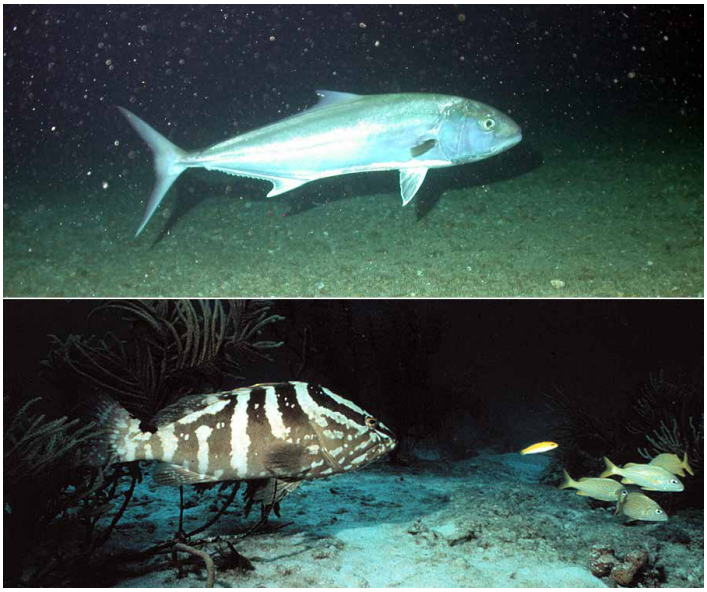

Figure 2. Jacks (top) and grouper (bottom) are sometimes ciguatoxic.

There are different forms of dinoflagellate toxins which can vary depending upon geographic location. Some, such as the *maitiotoxins* are excreted and cause problems only if the intestines of infected fish are consumed. *Ciguatoxins* (Figure 3), on the other hand, tend to accumulate in various tissues including muscle and internal organs. They are extremely powerful and resistant to heat and cold, so cooking or freezing the seafood does not eliminate the poison.

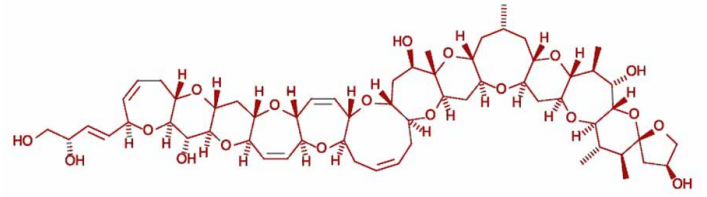

Figure 3. Chemical structure of one form of ciguatoxin.

Distribution and Prevalence

Ciguatera poisoning is associated with marine tropical environments and occurs within 35°N and 35°S latitude. In the Atlantic, it is common in Florida and the Bahamas, throughout the Caribbean, particularly, Cuba, Dominican Republic, Haiti, Puerto Rico and in the Leeward Islands, including The Virgin Islands. In the Pacific, it is found in French Polynesia, the Philippines, Fiji, Samoa, Tonga, Vanuatu, Hawaii, the Cook and Marshall Islands, New Caledonia and Australia. In the Indian Ocean, ciguatera commonly occurs in Reunión, Madagascar, Mauritius and the Seychelles and has also been reported from Sri Lanka,

the Maldives and the Comoro and Chagos Archipelagoes. Note that ciguatoxic seafood imported from these regions can be consumed and cause poisoning in any area of the world.

Estimates of the prevalence of ciguatera poisonings vary widely, partly because many cases are misdiagnosed or not reported at all. For example, CDC and others estimate that only 2-10% of ciguatera cases are actually reported in the United States. Shepherd (2007) estimates of annual ciguatera cases worldwide to be at least 50,000.

In the United States, there were 60 outbreaks and 205 cases reported between 1993 and 1997; Hawaii averaged 8 outbreaks and 29 reported cases per year, between 1990 and 1998, whereas Florida averaged 5.4 outbreaks and 21 cases per year between 1994 and 2003 (Nesheim and Yatkine 2007).

Testing and Diagnosis

There are tests available for detecting ciguatera in fish, the most common being the mouse bioassay, but they are complicated and can take more than four days to obtain results. Several simpler and quicker field tests for the toxin have been developed, but they either remain untried and untested or have had unacceptably high rates of erroneous results. Several tests that could yield results quicker are under development by government and private entities including one by the the Food and Drug Administration's Seafood Products Research Center. A major obstacle in the development of rapid testing is the sampling problem created by the normally large variation in toxin content within and between individuals.

Likewise, there are no accurate diagnostic tests for ciguatera poisoning. Currently, diagnosis of the condition is done based upon symptoms and upon the patient's immediate food consumption history.

Symptoms

Gastrointestinal symptoms, including diarrhea and vomiting, are the first to appear and can quickly lead to dehydration and shock. Other related symptoms can include excessive salivation and abdominal pain. Neurological symptoms follow the gastrointestinal ones and include hypersensitivity to cold, inversion of cold and hot sensations (hot feels cold and cold feels hot), numbness or "pins and needles" feeling in the limbs and lips, dizziness, ataxia, tremors, muscular stiffness, hypersensitivity of the nipples, depression, itching, and general muscle pain. Patients sometimes also complain about overall weakness, dry mouth, and blurred vision. Sometimes, cardiac symptoms such as slow heart beat (bradycardia) and hypertension are also observed, especially in older or overweight patients.

Onset of symptoms is usually quick (1-3 hours after ingestion of contaminated fish), but symptoms can appear as soon as 20 minutes after ingestion of the toxins or can be delayed for 12 hours or longer. Normally symptoms last two or three weeks, but some can become chronic and last for more than a year.

Treatment

There is no specific treatment for ciguatera poisoning. Normally patients are given supportive treatment, and therapies for amelioration of symptoms. Decontamination of the gastrointestinal tract with activated charcoal may be valuable if done within 3-4 hours of ingestion, and use of antiemetics may help control vomiting. Volume replacement therapy is often essential to counteract fluid loss due to vomiting and diarrhea, and use of atropine is recommended for treatment of bradycardia. Analgesics to control pain, and antihistamines to reduce itching are also commonly administered to ciguatera patients. The use of mannitol has become common in the treatment of ciguatera poisoning after early reports of significant improvement of symptoms after intravenous administration (Palafox et al. 1988), but its mode of action is still unknown.

Prevention

Although the risk of contracting ciguatera poisoning is usually small, the only sure way to prevent ciguatera is to avoid eating tropical reef fish. This, however is often not practical or possible. The risk of contracting ciguatera poisoning can be decreased by refraining from eating the internal organs where the toxins often accumulate.

Myths and Folklore

There are many **untrue** beliefs regarding ciguatera poisoning including:

- Your lips will tingle if you touch them with a ciguatoxic fish.
- Cats will not touch fish with ciguatera.
- Fish contaminated with ciguatoxins swim erratically.
- Ants and turtles will refuse to eat ciguatoxic fish.
- A silver spoon will tarnish if placed in a pot cooking with ciguatoxic fish.
- Grated coconut turns green if cooked with ciguatoxic fish.

- A fish slice held to up to the sun is contaminated with the toxin if it doesn't show a rainbow effect.
- If flies are attracted to the fish, then it is OK to eat.

Resources

In the United States, The American Association for Poison Control Centers (http://www.aapcc.org/) provide free, confidential advice on poisoning related issues 24 hours a day, 7 days a week through its help line (1-800-222-1222). The World Health Organization provides a listing and contact information for international poison centers at its website: http://www.who.int/gho/phe/chemical_safety/poisons_centres/en/index.html.

References

Bruslé J. 1997. Ciguatera fish poisoning: A review. Sanitary and economic aspects. Les Editions INSERM, Paris.

Kelly, AM, CC Kohler, and DR Tindall. 1992. Are crustaceans linked to the ciguatera food chain? Environmental Biology of Fishes 33: 275-286.

Lange WR. 1987. Ciguatera Toxicity. Am. Fam. Phy. 35:177-82.

Palafox NA, LG Jain, AZ Pinano, TM Gulick, *et al.* 1988. Successful treatment of ciguatera fish poisoning with intravenous mannitol. JAMA 259:2740-42.

Murata, M., MA Legrand, Y Ishibashi, and T Yasumoto. 1989. Structure of ciguatoxin and its congener. J. Amer. Chem. Soc. 111: 8929-8931.

Nesheim, MC and and AL. Yaktine (editors). 2007. Seafood Choices: Balancing Benefits and Risks. National Academies of Science, Committee on Nutrient Relationships in Seafood: Selections to Balance Benefits and Risks. The National Academies Press, Washington, DC.

Quod, JP. and J Turquet. 1996. Ciguatera in Reunion Island (SW Indian Ocean): Epidemiology and clinical patterns. Toxicon 34(7):779–85.

Ragelis EP. 1984 Ciguatera seafood poisoning. Pp. 25-36 In: Seafood Toxins, EP Ragelis (ed). Washington, DC: (ACS Symp. Ser. 262) American Chemical Society.

Randal JE. 1958. A review of ciguatera tropical fish poisoning with a tentative explanation of its cause. Bull. Mar. Sci. Gulf Carib. 8: 236-67.

Sheperd, SM. 2007. Infectious and Toxic Illness from Fish and Shellfish Ingestion Part II. Passport Health 10:1 - http://www.passporthealthusa.com/news/vol_10_no_1.asp

Scheuer PS, W Takahashi, J Tsutsumi, and T Yoshida. 1967. Ciguatoxin: isolation and chemical nature. Science 155:1267-68.

Yasumoto T, I Nakajima, R Bagnis, *et al.* 1977. Finding a dinoflagellate as a likely culprit of ciguatera. Bull. Jpn. Soc. Sci. Fish. 1977; 43: 1021-26.