

Ciguatera Poisoning

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INTRODUCTION

The acutely toxic effects of ciguatera poisoning have been recorded for centuries and were associated with the consumption of warm water marine fish caught at specific locations. However, the mechanism responsible for triggering outbreaks of ciguatera toxicity has been understood for only a few decades. The current name of the affliction is derived from “cigua” a Spanish term for a Caribbean marine turban shell, *Turbo pica*, which can cause a similar toxic effect after ingestion (Lewis and King 1996). In 1774 Captain Cook and his crew suffered from ciguatera fish poisoning on two occasions while in the New Hebrides region. The species thought to be responsible was red bass, *Lutjanus bohar* (Lehane 1999).

Ciguatera poisoning is known to occur throughout the tropics and in some subtropical waters. It is usually associated with coral reef habitats and is particularly prevalent in areas that have experienced some form of ecosystem disruption. Some examples of this may be pollution from industry, agricultural and human effluent; reef damage from cyclones and coral bleaching triggered by rising water temperatures through the insidious effects of climate change. However, not all damaged reef environments exhibit outbreaks of ciguatoxic fish (Lewis and King 1996).

Outbreaks of ciguatera are sporadic and unpredictable, affecting up to 50,000 people a year throughout the world. The spectre of ciguatera contamination causes considerable economic loss in the Pacific and the Caribbean countries, including the USA where it is the most common nonbacterial form of food poisoning associated with fish consumption (Juranovic and Park 1991).

The presence of ciguatera in the Northern Territory is not extensive and is mostly confined to the north east Arnhem region and Groote Eylandt (refer to Map). Ciguatera has also been reported from waters adjacent to Coburg Peninsula however, ciguatoxic fish from this area are rare. The coastal areas east of Darwin are relatively sparsely populated and this may contribute to the generally low rates of ciguatera poisoning in the Northern Territory.

WHAT CAUSES CIGUATERA TOXICITY?

As early as 1808 the idea that a marine alga may be implicated in ciguatera poisoning was proposed by Chisholm. Then in 1958, Randall further advanced the idea that a toxic benthic (bottom living) organism proliferating on newly disturbed sea beds may be finding its way into the food chain via small bottom feeding fish (Lehane 1999). In 1977 a Japanese research team headed by Dr Yasumoto discovered ciguatoxin in a motile photosynthetic dinoflagellate called *Diplopsalis*. The organism was later renamed *Gambierdiscus toxicus* after the Gambier Islands in French Polynesia where the initial discovery was made. There are about 30 species of dinoflagellates which produce bioactive compounds and some of these compounds rank as the most powerful non-proteinaceous poisons known (Brusle 1997).

The gambier-toxins, which are precursors to ciguatoxins, are produced only by certain strains of *Gambierdiscus toxicus* that live on a variety of macroscopic algae species that are eaten by small

herbivorous fish (Lewis and King 1996). The gambier-toxins are bio-converted to ciguatoxins which accumulate in the flesh and organs of small fish. Such small fish are then consumed by larger predatory fish that further concentrate the toxins in their tissues. Humans then become the final link in the food chain when they consume the larger fish.

WHAT SPECIES OF FISH CAN CAUSE CIGUATERA POISONING?

The precise number of fish species that are susceptible to ciguatoxin accumulation is not known but it is thought to range between 300 to possibly 400 globally. Some species have been found to have higher concentrations than others, which may relate to the degree of tolerance to the toxin that a particular species may have. It was previously thought that the ciguatera toxin did not harm the fish that carried it; however, it is now understood that excessive concentrations will eventually kill the fish, although they are still able to survive with high levels of the toxin without displaying any symptoms or pathology. It has been found that ciguatera is lethal to freshwater fish (Lewis 1992).

The fact that the human death rate from ciguatera poisoning is very low (less than 1%) tends to support the idea that high levels of ciguatoxin kill off many carriers before they reach the human end of the food chain. The species found to have the highest ciguatoxin levels is the moray eel (*Gymnothorax* spp.); however, the highest human mortality rate from ciguatera poisoning (20%) is attributed to the consumption of a shark (*Carcharhinus amboinensis*) on the east coast of Madagascar (Lehane 1999). In that incident 500 unfortunate seafood enthusiasts were poisoned and 98 subsequently died. Death from ciguatera poisoning has often been associated with the eating of the most toxic parts of the affected fish, which is the liver and the roe. According to Helfrich et al. (1968) the amount of toxin in the liver may be up to 50 times greater than an equivalent amount of muscle.

In Australia almost all ciguatera poisoning has occurred from fish caught in Queensland or the Northern Territory; however, the most extensive ciguatera poisoning incident in Australia took place in Sydney in 1987, when 63 people became affected following the consumption of Spanish mackerel from the Hervey Bay region of Queensland (Capra 1997). It should be noted that while Spanish mackerel are incriminated in some ciguatera poisoning events, they are a highly regarded fish among Australian consumers and are unlikely to be affected unless caught at specific habitats where ciguatera outbreaks are known to occur.

Fish species implicated in ciguatera outbreaks in the Northern Territory and Queensland, including outbreaks in other parts of Australia that have imported fish from these regions mostly include:

Barracuda	<i>Sphyraena jello</i>
Chinaman fish	<i>Symphorus nematophorus</i>
Cobia	<i>Rachycentron canadus</i>
Coral cod	<i>Cephalopholis miniata</i>
Coral trout	<i>Plectropomus</i> spp.
Flowery cod	<i>Epinephelus fuscoguttatus</i>
Groper	<i>Epinephelus lanceolatus</i>
Paddle tail	<i>Lutjanus gibbus</i>
Queenfish	<i>Scomberoides commersonianus</i>
Red bass	<i>Lutjanus bohar</i>
Red emperor	<i>Lutjanus sebae</i>
Spanish mackerel	<i>Scomberomorus commerson</i>
Spotted mackerel	<i>Scomberomorus munroi</i>
Sweetlip emperor	<i>Lethrinus miniatus</i>
Trevally	<i>Caranx</i> spp.

WHAT ARE THE SYMPTOMS OF CIGUATERA POISONING?

The symptoms of ciguatera poisoning are a complex combination of impacts on three systems in the body, usually commencing with the digestive system, followed by the cardiovascular system and then at various levels of neurological pathology (Ragelis 1984). Some of the neurological symptoms can persist for months or many years. Symptoms that usually present within 1 to 24 hours after the ingestion of affected fish include:

Digestive	Nausea Vomiting Diarrhoea Abdominal cramps
Cardiovascular	A slow pulse rate of between 40 to 50 beats/min Or, an erratic or elevated pulse rate of between 100 and 200 beats/min Reduced blood pressure
Neurological	Headache, including fatigue, vertigo and fainting Intense itching Temperature reversal (burning sensation to skin on contact with cold water) Audio and visual hallucinations Pain in joints Muscle pain Muscular paralysis and possible breathing difficulties Convulsions Sweating Painful urination Numbness, tingling, 'pins and needles'

It is understood that ciguatoxins can penetrate mucous membranes and skin, and have been known to cause tingling in the hands of those who have cleaned and filleted ciguateric fish. It has been found that ciguatoxins can be transferred to babies via the mother's breast milk, and are also capable of crossing the placenta to the foetus. The localised pain experienced by partners of affected people following sexual intercourse suggests that ciguatoxins are also excreted in other body fluids (Lehane 1999).

Clinical evidence indicates that ciguatoxins accumulate in the human body and are stored in fat cells and possibly proteins, as is the case in fish muscle. These storage reservoirs of the toxins can assist in future triggering of clinical symptoms from time to time. The body develops no immunity to the toxin and in fact subsequent attacks tend to be worse. The reoccurrence of the symptoms can be brought on by a number of influences including stress, exercise, rapid weight loss, alcohol consumption and eating other fish that are not necessarily ciguateric, including cold water species. The excretion of the toxin is a slow and incomplete process that occurs in part via the urinary system (Adams 1993).

TREATMENT

Following the onset of any of the aforementioned symptoms, immediate medical treatment should be sought. The most effective treatment at present is an intravenous injection of mannitol which in most cases provides a fairly rapid improvement to most symptoms as long as it is started early enough. Mannitol functions as an osmotic diuretic that essentially flushes the body's cells of the toxin (Lehane 1999).

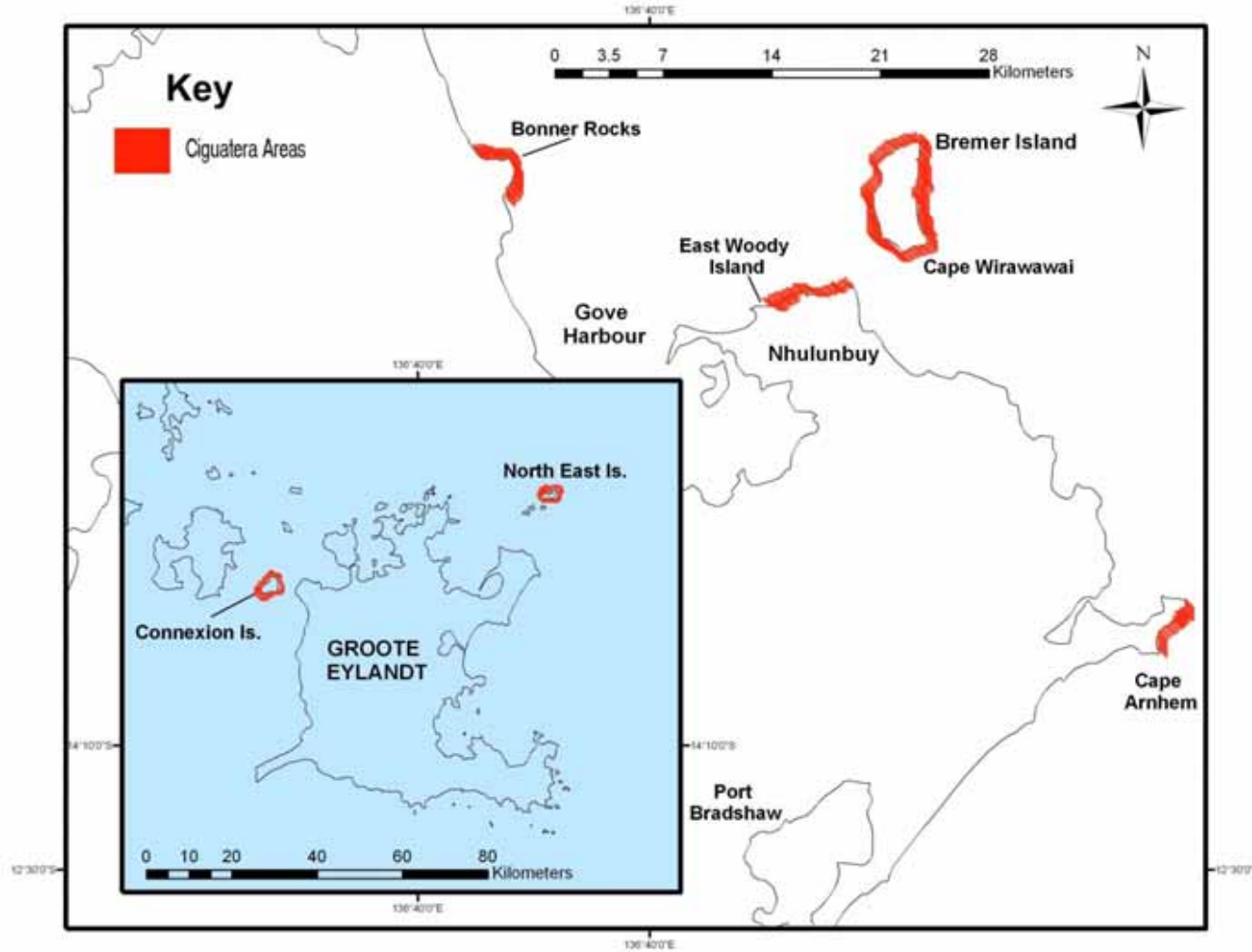
DETECTION OF CIGUATERA

The detection of ciguatera is problematic outside of a laboratory environment (where bioassays, immunoassays and chromatographic techniques can be applied) as the toxins are tasteless and odourless. In some regions, portions of suspect fish have been fed to cats or mongoose. The negative aspects of such a test are that it takes time and is inhumane (Lehane 1999). Test kits have recently been developed; however, they are not yet regarded as effective in all situations.

HOW TO AVOID CIGUATERA POSONING?

- Clearly the most obvious step that can be taken is to avoid eating fish that are caught at known ciguatera locations, particularly if it is known that a recent outbreak of ciguatera has occurred there.
- If the catch location of a fish is unknown but it is suspected of being unsafe, eat only small portions (50-100 g) of any individual fish and if necessary make up the shortfall with similarly small portions from other fish of the same species.
- If possible eat smaller (less than 2-3 kg) rather than larger reef and pelagic fish that fall within the risk group.
- Avoid the liver, roe, head and viscera of any tropical marine fish.
- Avoid fish from areas that have undergone habitat disturbance, whether natural or human induced.
- Avoid fish harvested from the windward side of an oceanic island.
- Avoid eating moray eels.
- Avoid excessively large carnivorous fish.

It should be noted that while ciguatoxin levels in fish generally increase over time, it may not necessarily follow that a relentless escalation towards lethal toxin levels will eventuate. Excretion of the toxins occurs as a slow and constant process and if the cause of ciguatoxin input into the food chain diminishes then the fish at that location may gradually detoxify. It is known that ciguatoxin levels in fish decrease to around 50% after between 90 to 300 days (Lewis and Holmes 1993).



The above maps indicate the areas in the Northern Territory from where fish have caused ciguatera poisoning. While not all fish from these areas are likely to be ciguateric, caution should be applied when catching and consuming fish species from the risk group listed on page 2.

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