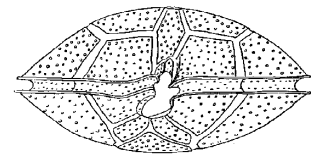




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# CIGUATERA

INFORMATION BULLETIN

Number 2 - July 1992

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## NOTE FROM THE CO-ORDINATOR

This issue of the Ciguatera Information Bulletin contains timely updates on the global spread of toxic dinoflagellates, the origin of ciguatera and the treatment of ciguatera with mannitol. Research on ciguatera at the Queensland University of Technology over the last 8 years is outlined. The ciguatera problem in the Cook Islands, Fiji and the Solomon Islands is summarised. The potential for reef disturbance to exacerbate ciguatera is highlighted in an article from the Republic of Kiribati.

The recent Ciguatera Conference in Papeete, Tahiti was a success. Paul Dalzell gives a summary of highlights of this meeting. A Ciguatera Management Workshop, to be held in Brisbane in April 1993, is announced in this issue.

Articles related to ciguatera are now requested for the next issue of this bulletin. Please forward these either to myself or to the Fisheries Information Project, SPC, before the end of February 1993.

**Richard Lewis**

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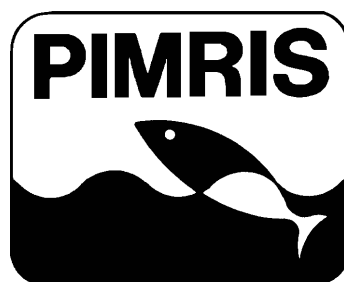
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PIMRIS is a joint project of 4 international organisations concerned with fisheries and marine resource development in the Pacific Islands region. The project is executed by the South Pacific Commission (SPC), the South Pacific Forum Fisheries Agency (FFA), the University of the South Pacific's Pacific Information Centre (USP-PIC), and the South Pacific Applied Geoscience Commission (SOPAC). Funding is provided by the International Centre for Ocean Development (ICOD) and the Government of France. This bulletin is produced by SPC as part of its



Pacific Islands Marine Resources Information System

commitment to PIMRIS. The aim of PIMRIS is to improve the availability of information on marine resources to users in the region, so as to support their rational development and management. PIMRIS activities include: the active collection, cataloguing and archiving of technical documents, especially ephemera ('grey literature'); evaluation, repackaging and dissemination of information; provision of literature searches, question-and-answer services and bibliographic support; and assistance with the development of in-country reference collections and databases on marine resources.

## On the global spreading of toxic dinoflagellates

by Gustaaf M. Hallegraeff,  
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Toxic dinoflagellate blooms, in a strict sense, are completely natural phenomena which have occurred throughout recorded history. For example, Captain Vancouver lost one of his crew in British Columbia in 1793 after eating contaminated mussels (paralytic shellfish poisoning), and Captain Cook is claimed to have suffered from ciguatera fish poisoning when visiting New Caledonia in 1774.

However, in the past two decades the public health and economic impacts of such events appear to have increased in frequency, intensity and geographic distribution. One example, the increased global distribution of paralytic shellfish poisoning (PSP), is illustrated in Figure 1.

Until 1970, toxic dinoflagellate blooms of *Alexandrium (Gonyaulax) tamarensis* and *Alexandrium (Gonyaulax) catenella* were only known from temperate waters of Europe, North America and Japan. By 1990, this phenomenon was well documented from throughout the Southern Hemisphere, in South Africa, Australia, India, Thailand, Brunei, Sabah, the Philippines and Papua New Guinea.

Other species of the dinoflagellate genus *Alexandrium*, such as *A. minutum*, as well as the unrelated dinoflagellates *Gymnodinium catenatum* and *Pyrodinium bahamense* var. *compressum* (all species not previously known to be toxic) have now also been implicated\*. To some extent, this increased global distribution of PSP simply reflects our increased awareness of toxic species and the enormous expansion in aquaculture efforts.

Evidence is accumulating, however, that human activities contribute significantly to this increase through the stimulation of dinoflagellate blooms by cultural eutrophication and by the spreading of nuisance organisms in ships' ballast water. Cargo vessel ballast water was first suggested as a vector in the dispersal of non-indigenous marine plankton some 90 years ago. The diatom *Odontella (Biddulphia) sinensis*, well known from the tropical and subtropical coasts of the Indo-Pacific, had not been reported in European waters until 1903 when it produced dense plankton blooms in the North Sea. Since it appeared unlikely that this large diatom could have been overlooked previously and impossible that it could have been carried by currents from distant oceans, Ostenfeld (1908) suggested that this species was introduced via the

water or sediment contained in ships' ballast tanks. Subsequently, Hallegraeff and co-workers (1990) confirmed this possibility by culturing the related diatom species *Odontella aurita* from a ballast water sample collected at the end of a voyage from Japan to Australia. Whereas the introduction of *O. sinensis* was apparently without harmful effects, the more recent introduction into the North Sea of the diatom *Coscinodiscus wailesii* has caused problems due to the clogging of fishing nets by extensive diatom mucous production.

The issue of ballast water transport of plankton species gained considerable interest in recent years, when Hallegraeff and co-workers (1988, 1991, 1992) brought forward evidence that non-indigenous toxic dinoflagellate species had been introduced into Australian waters in sensitive aquaculture areas, with disastrous consequences for commercial shellfish farm operations.

Paralytic shellfish poisoning was unknown from the Australian region until the 1980s, when the first outbreaks appeared in the ports of Hobart (*Gymnodinium catenatum*), Melbourne (*Alexandrium catenella*) and Adelaide (*Alexandrium minutum*). While the plankton stages of diatoms and dinoflagellates show only limited survival during the voyage in dark ballast tanks, their resistant resting spores are well suited to survive these conditions. One single ballast tank thus was estimated to contain more than 300 million toxic dinoflagellate cysts which could be germinated into confirmed toxic cultures (Hallegraeff and Bolch, 1991).

In Hobart, Tasmania, an examination of historic plankton samples, cyst surveys in sediment depth cores and genetic studies using enzyme electrophoresis and sexual compatibility experiments have provided strong circumstantial evidence that the toxic dinoflagellate *G. catenatum* was introduced in the last 10 to 20 years. Resting spores of this species have been confirmed in 4 ballast water samples entering Australian ports from either Korea or Japan. This organism has now been well-established in southern Tasmania and dense blooms in 1986, 1987 and 1991 necessitated the closure of up to 15 shellfish farms for periods up to 6 months.

Similarly, the toxic dinoflagellate *Alexandrium catenella*, which has caused the closure of shellfish farms in Port Phillip Bay, Melbourne, was not known

\* Note from the editor: a case of paralytic shellfish poisoning in 1972 near Port Moresby, Papua New Guinea, caused by *P. bahamense*, is cited by J.L. McLean (1974), SPC *Information Circular* 54.

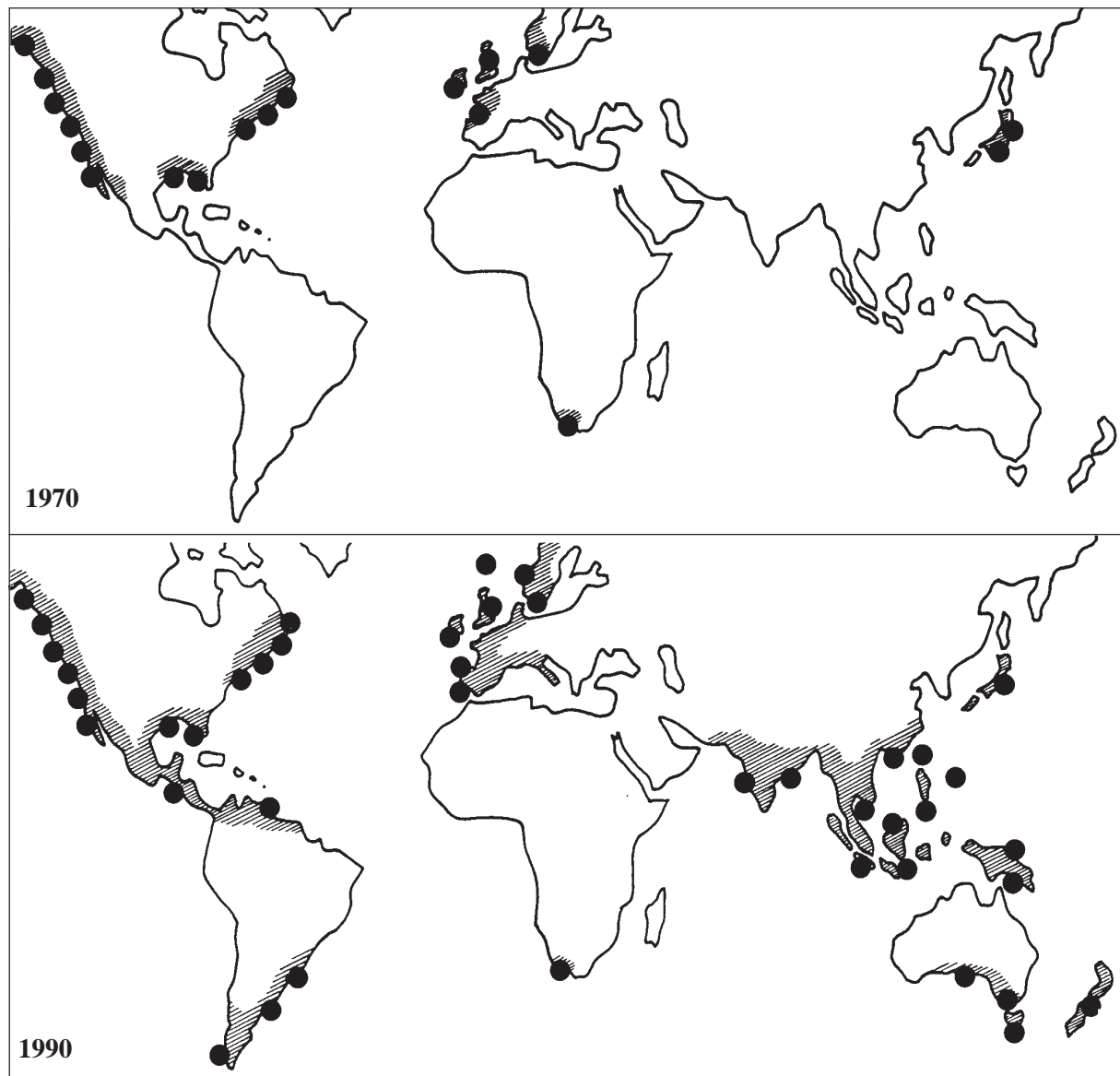


Figure 1: Global distribution of paralytic shellfish poisoning 1970 (above) and 1990 (below)

from the area before 1986. Viable resting spores of this species have been detected in ballast water being discharged into this port, and rRNA sequencing has indicated a remarkable match between ballast water and harbour water cultures of this dinoflagellate (Scholin and Anderson, 1991). Finally, the toxic dinoflagellate *Alexandrium minutum* appeared in the Port River, Adelaide, in 1986 in an area where sediment surveys carried out in 1983 failed to detect resting spores in sediments. The port of Adelaide has a shipping link with the Mediterranean, which has the only other known global population of this dinoflagellate, and rRNA sequencing has indicated a remarkable match between Australian and Spanish cultures of this species.

Another vector for the dispersal of toxic dinoflagellates (especially their resting spores) is shellfish stocks transferred from one area to another, as the faeces and digestive tracts of bivalves can at

times be loaded with viable dinoflagellate cells. The Japanese seaweeds *Sargassum muticum*, *Undaria pinnatifida* and *Laminaria japonica* thus are thought to have been introduced into European waters via sporophyte stages associated with introduced Japanese oyster spat. While benthic dinoflagellates such as *Gambierdiscus toxicus* are not known to produce resistant resting spores, these species are well capable to survive dispersal as epiphytes attached to drifting macroalgae ('rafting').

Bomber and co-workers (1988) observed *G. toxicus* cells among 30 out of 198 drift algal samples collected in the Florida straits and Bahamian waters. Translocation of toxic strains of this species by ships' ballast water or as epiphytes on seaweeds fouling the hulls of ships are other vectors for the introduction of *G. toxicus* into tropical regions which may previously have been free of ciguatera (as may have occurred, for example, at Hao Atoll).

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## Ciguatera research at the Queensland University of Technology

by Michael F Capra,  
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An active ciguatera research group has existed at the Queensland University of Technology (QUT) since 1985. This group has worked on various aspects of ciguatera research including the effects of ciguatoxin on vertebrate nerves, the symptomatology of ciguatoxin in humans and the response of fish to ciguatoxin. The group has been led by myself (M. Capra) and a medical colleague (J. Cameron) from the Princess Alexandra Hospital in Brisbane.

Since 1985 three students have completed and been awarded higher degrees for work on ciguatera research (A. Flowers and C. Blanton - Masters degrees; S. Hahn - Ph.D. degree). Currently another student (C. Purcell) is completing a Ph.D. research program. Aspects of our work on ciguatera at QUT are briefly reviewed below.

### The effects of ciguatoxin on nerves

Although there have been many clinical reports describing the neurological signs and symptoms of ciguatera, very little has been documented as to the electrophysiological disturbance ciguatoxin causes in the peripheral nervous system.

The initial electrophysiological studies undertaken at QUT were on nerves in anaesthetised rats. The nerve chosen for study was the ventral coccygeal nerve of the rat tail. This nerve was electrically

stimulated by subcutaneous needle electrodes and the elicited compound nerve action potentials were recorded by a second set of subcutaneous needle electrodes placed proximally to the stimulating electrodes. This rat tail preparation has been used to gain some insight into the mode of action of ciguatoxin on peripheral nerves.

A number of nerve conduction parameters were measured, the most useful of which were nerve conduction velocity, the duration of the refractory periods and the magnitude and duration of the supernormal period.

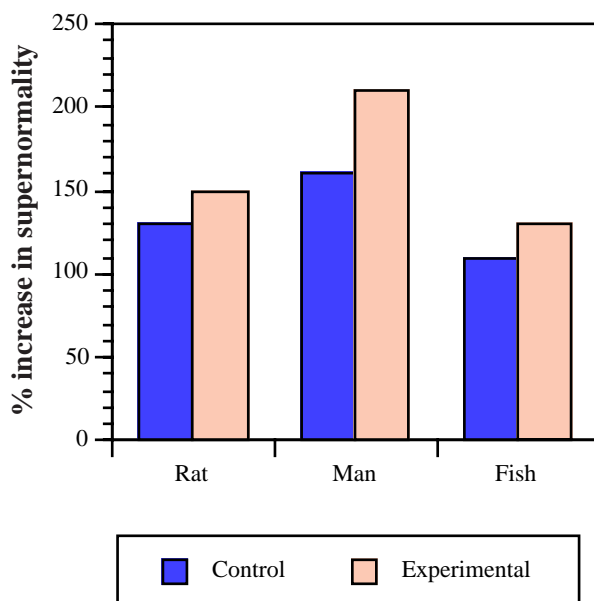
The refractory periods and the supernormal period give some indication of fundamental ionic and molecular processes that occur during nervous transmission. When a nerve carries an impulse there is a brief period after that impulse in which the nerve is refractory (0.5 - 4 msec). In the first part of this period (absolute refractory period) the nerve cannot carry a second impulse while in the latter part of the period (relative refractory period) a greater stimulus will elicit a second impulse.

The refractory period is related to the physiological processes controlling the movement of sodium ions (Na<sup>+</sup>) across the nerve membrane. The regulated movement of Na<sup>+</sup> is the basis of normal nerve function. After the refractory periods, the supernormal period (6 - 30 msec), occurs in which

the nerve becomes more excitable and can be more easily stimulated to carry another impulse. During the supernormal period the movement of  $\text{Na}^+$  through the  $\text{Na}^+$  channels of the nerve membrane becomes easier.

In our studies of rat nerves it has been found that when rats are given a sublethal dose of ciguatoxin the conduction velocity is decreased, the refractory period is extended and the magnitude (Figure 1) and duration of the supernormal period are both increased. These studies on alterations in nerve conduction parameters, especially the changes in the supernormal period, indicate that ciguatoxin is acting on the  $\text{Na}^+$  channels in the peripheral nerves and producing an increase in the ease of  $\text{Na}^+$  channel opening or in the time course of  $\text{Na}^+$  channel opening.

In 1987 two major outbreaks of ciguatera poisoning occurred in Australia – one in Sydney, New South Wales and the other in Maryborough, Queensland. Conduction velocity, refractory period and supernormal period (Figure 1) studies were performed on the Sural nerve of 15 of these victims who were showing acute signs and symptoms of ciguatera poisoning. Control studies were performed on 15 age-matched non-poisoned individuals with no obvious neurological dysfunction. In the 15 people who had ingested toxic fish there was a significant decrease in conduction velocity, increase in the refractory period and increase in the magnitude and duration of the supernormal period. The nerves of the human



**Figure 1: The effects of ciguatoxin on the magnitude of the supernormal period at an interstimulus separation of 10 ms in laboratory rats, humans and coral trout.**

victims of ciguatera poisoning were affected in an identical manner to the nerves of rats exposed to ciguatoxin under controlled conditions. Hence, the rat nerve preparation has become a very useful model to study potential therapies for ciguatera poisoning and is currently being used by our group to assess potential therapeutic benefits of mannitol on peripheral nerve conduction.

Concurrently with studies on rats and humans the QUT group has examined the effects of ciguatoxin on fish nerves. The fact that a fish can carry sufficient ciguatoxin to poison several humans yet show no overt signs of intoxication has always intrigued our group. The electrophysiological studies performed on fish nerves have shown that these nerves respond in the same way to ciguatoxin as the nerves of rats and humans. Coral trout, *Plectropomus* sp., nerves respond to ciguatoxin with a decrease in conduction velocity, an increase in the duration of the refractory and supernormal periods and an increase in magnitude of supernormality (Figure 1).

Studies on the flux of  $\text{Na}^+$  across fish nerves have also indicated that ciguatoxin acts on fish nerves and increases the opening of  $\text{Na}^+$  channels. The actions of ciguatoxin on fish nerves indicate that fish nerves are susceptible to this toxin and have led us to speculate that some protective partitioning mechanism must exist in fish so that exposure of targets within the nervous system is minimalised.

### Symptomatology of ciguatera poisoning

Data have been collected on a number of ciguatera victims over the past eight years. A number of patients have had prolonged expression of signs and symptoms and these are currently being followed by our group. In 1987 Australia's largest single outbreak of ciguatera poisoning, in which 63 people were poisoned, occurred in Sydney. Sydney is well below the latitude at which ciguatoxic fish are captured and the offending fish was caught in a ciguatera-endemic region of Hervey Bay in Queensland.

The victims of the Sydney outbreak were followed for six months after ingestion of toxic fish and the mean duration of symptoms was determined (Table1). Even six months after the ingestion of toxic fish several victims suffered from one or more symptoms (Table2). One woman who consumed 1kg of the toxic fish in two 500 g portions over a three-day period had eight symptoms and was debilitated to the extent that she was unable to resume employment six months after her initial poisoning. Currently the victims of the Sydney outbreak are being contacted to document any persistent or recurrent symptoms.

**Table 1: Type and duration of symptoms in 40 of the 63 victims of the ciguatera poisoning outbreak in 1987 in Sydney**

Symptom	% with symptom	Duration of symptom (days mean $\pm$ SE)
Nausea	83	17 $\pm$ 7
Vomiting	50	11 $\pm$ 9
Abdominal pain	68	9 $\pm$ 2
Diarrhea	78	5 $\pm$ 1
Headache	85	32 $\pm$ 9
Vertigo	65	24 $\pm$ 9
Memory disturbance	43	121 $\pm$ 18
Anxiety	60	60 $\pm$ 13
Depression	63	54 $\pm$ 11
Joint pain	83	59 $\pm$ 10
Paresthesia, hands	88	50 $\pm$ 8
Paresthesia, lips	78	35 $\pm$ 8
Temperature perception reversal	80	45 $\pm$ 7
Muscle pain	93	40 $\pm$ 8
Loss of energy	83	49 $\pm$ 8
Shortness of breath	38	16 $\pm$ 6
Sweating	48	12 $\pm$ 4
Salivation	15	12 $\pm$ 4
Pruritus	88	36 $\pm$ 7
Skin rash	25	17 $\pm$ 3

**Table 2: Persisting symptoms in the victims of the 1987 ciguatera outbreak in Sydney 6 months after ingestion of toxic fish**

Number of persistent symptoms	Number of victims with symptoms
1	11
2	5
3	1
4	2
8	1

### Response of fish to ciguatoxin

A series of toxicological experiments was undertaken with two species of small Pomacentrid fish, *Chromis nitida* and *Pomacentrus wardi*. These fish (1 to 8 g) were anaesthetised in MS222, then microinjected in the peritoneal cavity with varying doses of ciguatoxin. The fish were allowed to recover from anaesthesia and monitored for several days for signs of toxicity. Each species of fish was susceptible to ciguatoxin but at much higher doses than those for mammals. Interestingly, *C. nitida*, which is a planktivore, was significantly more susceptible to ciguatoxin than was *P. wardi*, a browser, that feeds in areas where *G. toxicus* is found.

The results of this study suggest that the feeding niche of tropical fish may influence their susceptibility to intoxication. Fish that naturally come into contact with *G. toxicus* may have evolved

partitioning strategies to reduce the likelihood of ciguatoxin acting on binding sites in nerve fibres. Histopathological studies have also been performed on pomacentrids injected with ciguatoxin. Cytological changes have been observed in the gut, gills and livers of fish injected with ciguatoxin. Studies are continuing on the resistance of fish to ciguatoxin with particular emphasis on partitioning mechanisms.

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**Ciguatera fish poisoning and reef disturbance in South Tarawa, Kiribati**

by Temakei Tebano,  
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Samples of several fish species from each side (east and west) of the blasted channel (Nei Tebaa) on the ocean side of the Dai Nippon Causeway were collected in early 1989 (Figures 1 and 2). This large causeway links the islet of Betio and Bairiki on the Southern portion of Tarawa Atoll. Our purpose was to set up a database on fish toxicity levels in the area, in an effort to safeguard the general public from risks associated with ciguatera fish poisoning. Prior to the construction of the causeway fish in this area were considered safe to eat by local fishermen.

Bioassay results (i.p. in mice) provided by the Southern Fisheries Centre in Brisbane (see *Ciguatera Information Bulletin #1*) show that the eels caught east and west of the channel, as well as those caught off the neighbouring reefs, were very toxic. Parrot fish caught east of the channel were found to be only slightly toxic, while those from the western side were not. The surgeon fish, *Ctenochaetus striatus*, caught east of the channel were found to be toxic while those west of the channel were not.

One of the two specimens of the blue spot coral trout, *Cephalopholis argus*, from the eastern side was slightly toxic; the other was not. Other fish species, including parrot fish, wrasse, surgeonfish and emperors tested only slightly toxic to mice.

It is apparent that toxicity levels may vary from species to species and from location to location. The

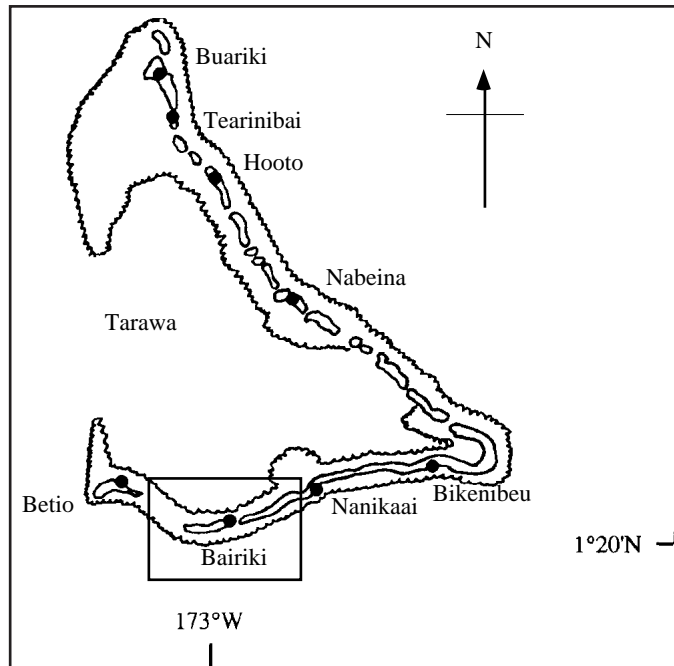


Figure 1. Map of Tarawa, Republic of Kiribati

toxicity of moray eel appeared to be highly variable, with some individuals being extremely toxic. In general, the eastern side of the channel is more toxic than the western side .

A comparison between toxicity level in fishes collected before and after the causeway construction was also made. It appears that toxicity levels of fishes may have risen after the completion of the causeway. Reef disturbance may have contributed to such an increase.

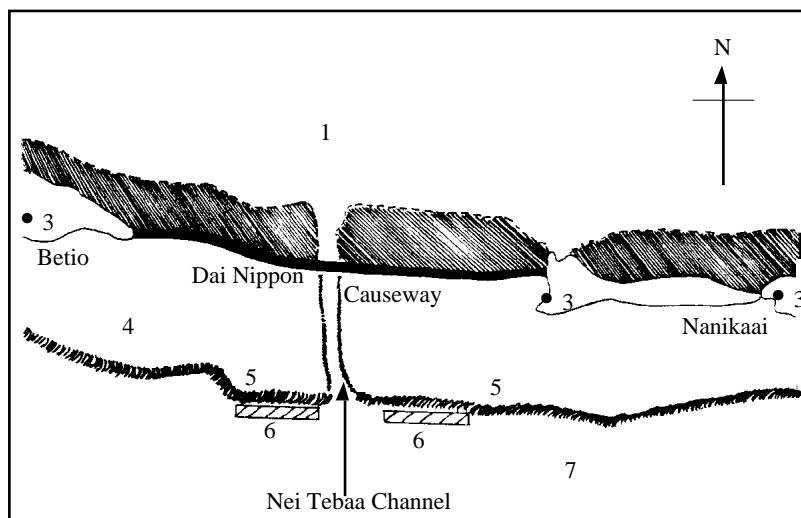


Figure 2. A map of the Dai Nippon Causeway and the Nei Tebaa Channel. The cross-hatched areas indicate the fish collection sites.

## The origin of ciguatera — an update

by Mike Holmes,  
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Ciguatera is caused by a class of polyether toxins called ciguatoxins. The structures of the three major ciguatoxins (ciguatoxin-1, -2 and -3) found in ciguateric fish have been recently determined (Murata et al., 1990; Lewis et al., 1991a). Ciguatoxin-1, the most potent of these toxins, is thought to originate from the metabolism (oxidation) of ciguatoxin-3. This metabolism probably occurs in fishes. Ciguatoxins-2 and -3 probably originate from toxic precursors called gambiertoxins which are produced by the benthic dinoflagellate *Gambier-discus toxicus* (Murata et al., 1990; Holmes et al., 1991). The structural differences between ciguatoxins-2 and -3 indicate that they probably have different gambiertoxin precursors.

Our group has recently isolated two chromatographically different types of gambiertoxins from a cultured clone of *G. toxicus* (Holmes and Lewis, 1991). The bioassay signs displayed by mice injected with either of these gambiertoxins were identical to those displayed by mice injected with ciguatoxin-2 or -3, and included hind-limb paralysis. This sign in mice clearly differentiated these gambiertoxins from ciguatoxin-1, which does not induce hind-limb paralysis.

We therefore believe that the two gambiertoxins we have isolated may be precursors of ciguatoxin-2 and -3 (the less polar gambiertoxin being the precursor of ciguatoxin-3). The two gambiertoxins were extracted from a clonal culture (a culture started from a single cell of *G. toxicus*) which indicates that ciguateric strains of *G. toxicus* may produce the precursors of all the ciguatoxins found in ciguateric fishes. If this result applies for all gambiertoxin-producing strains of *G. toxicus*, it would mean that any differences in the type of ciguatoxins found between ciguateric fishes would result from differences in the metabolism and excretion of gambiertoxins/ciguatoxins between fish species (Lewis et al., 1991b).

Not all strains of *G. toxicus* produce gambiertoxins. Gillespie et al., (1985) and Holmes et al., (1990) have previously reported wild and cultured strains of *G. toxicus* which only produce the water-soluble toxin, maitotoxin. There is no evidence for the bioaccumulation of maitotoxins in the flesh of fishes to cause ciguatera. Recently, we have found that only two out of thirteen cultured strains of *G. toxicus* produced gambiertoxins (Holmes et al., 1991). Ciguatera may therefore only occur when strains of *G. toxicus* genetically capable of producing gambier-toxins are eaten by fishes. This may explain why ciguatera incidence correlates with *G. toxicus* numbers in some areas but not in others.

If only certain strains of *G. toxicus* can produce gambiertoxins, then the translocation of these strains may account for the introduction of ciguatera into areas previously free of ciguatera. Translocation of toxic dinoflagellates led to the introduction of paralytic shellfish poison into areas in Australia previously free of this disease (see article on p. 2). It has been shown that *G. toxicus* can survive on drift algae and therefore could be translocated by this mechanism (Bomber et al., 1988).

The concentration of gambiertoxins that have been extracted from *G. toxicus* cultures is considerably less than has been extracted from some wild *G. toxicus* (Holmes et al., 1991). The cell concentrations of gambiertoxins can change over the time that *G. toxicus* strains are maintained in culture (Holmes and Lewis, 1991). Therefore environmental conditions may lead to either increases or decreases in the production of gambiertoxins in wild *G. toxicus*, thereby increasing or decreasing the risk of ciguatera over time. These environmental parameters may be separate from those that promote growth of *G. toxicus* in the wild. The determination of the environmental parameters which control growth and gambiertoxin production in the wild presents a formidable challenge for future researchers.

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**Mannitol: the treatment of choice in the acute phase of ciguatera**

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 Southern Fisheries Centre, Q.D.P.I.,  
 Deception Bay, Australia

Until the introduction of intravenous mannitol therapy, treatment for ciguatera was only symptomatic and supportive. Mannitol was first used in the Marshall Islands (Palafox et al., 1988) and soon after in Australia (Pearn et al., 1989). These studies both reported dramatic and sustained improvement in many of the victims of ciguatera. Specifically mannitol appeared to reduce the duration and severity of ciguatera. Several recent experiences with mannitol and ciguatera lend further support to these earlier studies. Mannitol is now the treatment of choice in the acute phase of ciguatera in Australia (Figure 1). Mannitol is most effective at reversing the neurological disturbances, particularly in the more severe cases of ciguatera.

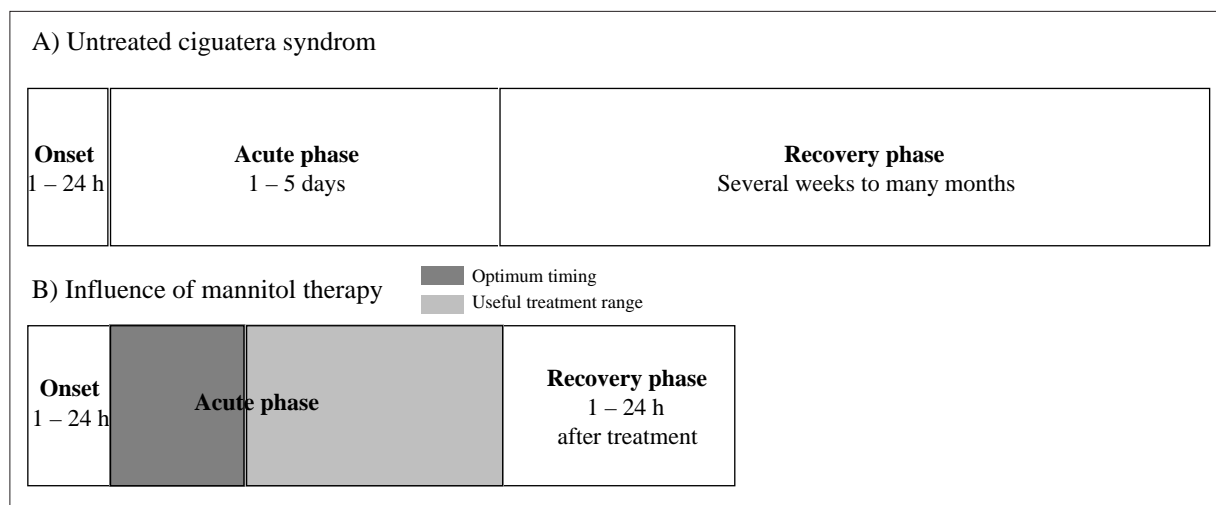
indication to the use of mannitol is dehydration. Patients must be adequately hydrated prior to the infusion of mannitol. The response of patients to mannitol infusion is often dramatic, with some symptoms abating during the infusion.

The mannitol treatment regimen for ciguatera is shown in Table 1. Ciguatera is diagnosed when symptoms typical of ciguatera appear within 24 hr of eating a suspect fish (see *Ciguatera Information Bulletin* no. 1 for details). The most important contra-

**Table 1. Mannitol therapy for confirmed ciguatera**

- Intravenous infusion of 20% mannitol solution over 30
- 1 g mannitol given per kg body weight (i.e. 5ml/kg)
- Adequate hydration must be established prior to manni
- Mannitol is most useful during the acute phase of cigu

Other patients report slower improvement, with some having a relapse about a day after treatment. In these latter cases, a second infusion of mannitol often has further beneficial effects. In Australia, not all ciguatera sufferers (<25%) respond to the mannitol treatment. The reasons for poor response of some people have not been determined. However,



**Figure 1. Mannitol therapy for ciguatera. (A) shows the normal course of the disease and (B) the influence of intravenous mannitol. The apparent optimum timing for the treatment is early in the acute phase.**

the Australian experience indicates that mannitol is most useful when given early in the acute phase of the illness (Fig.1), and may be of less benefit when given during the normal recovery phase of ciguatera. A double-blind clinical trial of the mannitol therapy has not been reported to date.

The mechanism of mannitol's beneficial action in cases of ciguatera has been studied by the Ciguatera Research Group at the Southern Fisheries Centre, Deception Bay. These studies indicate that mannitol does not displace ciguatoxin from its site of binding, nor does it chelate ciguatoxin. The appearance of an oedema (swelling) of the cells (Schwann cells) surrounding myelinated peripheral nerves (and possibly similar cells in the central nervous system) in cases of ciguatera may provide the explanation. Hyperosmotic mannitol, through its water drawing action would reduce this cell swelling and thereby effectively reverse the course of this often distressing and debilitating disease.

To explain the long term effectiveness of mannitol, I hypothesise that (i) mannitol prevents long-term nerve damage (eg. lesions, anoxic zones) that probably develops from the oedema (ii) ciguatoxin normally remains bound to its receptors in the body

only for a few days (not weeks), and the longer-term effects of ciguatera relate to nerve damage.

These hypotheses also explain why mannitol would be most effective when given early in the disease. Interestingly, it has been repeatedly observed in Australia that the diuresis that would normally follow an infusion of mannitol does not appear when mannitol is used to treat ciguatera (all patients were adequately hydrated). The explanation for this observation is not apparent, but it does suggest that mannitol does not act to flush ciguatoxin from the body by increasing urine output. Further studies are required to define more precisely why mannitol is useful in the treatment of ciguatera.

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## Fourth International Conference on Ciguatera Fish Poisoning in Tahiti

by P. Dalzell,  
South Pacific Commission,  
Noumea, New Caledonia

Researchers studying ciguatera presented the latest research developments and exchanged information at the Fourth International Ciguatera Conference in Papeete from 4 to 7 May. Inshore Fisheries Scientist Paul Dalzell represented SPC.

Topics ranged from general country statements about ciguatera and the ecology of the dinoflagellate, *Gambierdiscus toxicus*, to the results of highly specialised physiological research on the mechanism by which ciguatoxins affect nerve and muscle cells.

Paul Dalzell and Richard Lewis of the Queensland Department of Primary Industry jointly chaired the session on the socio-economic impact of ciguatera. Paul presented summaries of data on fish landings in the Pacific Islands and incidence of ciguatera. He also introduced the new SPC Fisheries Programme/Health Programme Ciguatera Database and gave some preliminary results. Participants were also given a chance to see the new commercially produced ciguatera kits produced by Hawaii Chemtect International. These are based on the monoclonal antibody test devised by Dr Y. Hokama of the Hawaii University Medical School.

Two forms of test kit were demonstrated to the meeting: a small disposable kit containing a single test, and a larger more elaborate kit containing equipment and re-agents for multiple testing. The single test comes in card form, contains all the re-agents, and is designed to be used to test one fish. The larger kit, which can be used for up to 50 tests, is designed for multiple testing on one fish or testing several fish.

About 100 persons attended the meeting. The Pacific Islands were well represented, with people attending from the Cook Islands, the Federated States of Micronesia, Fiji, French Polynesia, Guam, Kiribati, New Caledonia and Solomon Islands. Participants also came from Australia, France, Germany, Japan, Mayotte, Martinique, Puerto Rico, Réunion and the United States.

The Inshore Fisheries Research Project supported the attendance of Edwin Oreihaka from the Solomon Islands Fisheries Division and Ahser Edwards from the Community College of Micronesia in the Federated States of Micronesia.

The conference proceedings, containing the papers presented at the meeting, will be published later this year. The venue for the next conference has yet

to be decided. However, Dr Lewis is organising a ciguatera management workshop to be held in Queensland, Australia, in May 1993.

*Note: Below are summaries of the country statements from Solomon Islands, Fiji and the Cook Islands which were presented at the conference.*

## Ciguatera in the Solomon Islands

*by E. Oreihaka,  
Fisheries Division,  
Solomon Islands*

Ciguatera fish poisoning receives very little recognition in Solomon Islands. There is no organised research or monitoring of ciguatera fish poisoning carried out in Solomon Islands as yet, so as to determine the current status of the problem and there are other major health problems such as malaria to worry about.

Though there have not been any confirmed cases of ciguatera fish poisoning in the Solomon Islands as yet, from traditional knowledge and anecdotal information on case histories, fish poisoning which was probably ciguatera has occurred in certain areas. These are restricted to reefs, atolls and small islands. No cases of ciguatera fish poisoning appear to have occurred on any of the major islands in the Solomon Islands.

Fish species which are considered ciguatoxic in the Solomon Islands include;

- *Lutjanus bohar*
- *Lutjanus sebae*
- *Sphyraena barracuda*
- *Symphorichthys spirilus\**
- *Platax teira*.

\* Note from the editor: it could be *Symphorus nematophorus*

It is believed that some people have traditional medicine for treating ciguatoxin-intoxicated patients.

Apart from the regulation imposed by the Provincial Government of Temotu Province, which prohibits sale of fish species considered ciguatoxic in the province, there is no law or regulation concerning ciguatera poisoning in Solomon Islands.

Ciguatera fish poisoning is as yet not a major health problem in Solomon Islands. It is therefore not clear at this stage as who should take responsibility for dealing with ciguatera issues. It does, however, threaten coastal fisheries development and thus perhaps should principally be regarded as a fisheries problem. For further information on ciguatera in the Solomons please contact the Permanent Secretary, Ministry of Natural Resources, Fisheries Division, P.O. Box G24, Honiara, Solomon Islands.

## Status of ciguatera in Fiji

*by Parmanand Singh,  
Fisheries Centre of Labasa,  
Vanua Levu, Fiji*

In Fiji, cases of ciguatera have increased significantly in the last decade. In 1989, 1990 and 1991 the number of cases attended by government medical authorities were 683, 787 and 1,012 respectively. Some people call these figures alarming because innocent people are victims of intoxication and the inability of fisheries science to address the problem which has occurred for quite some time. Ciguatera poisoning is seen as an unnecessary obstacle in coastal fisheries development.

Ciguatera in Fiji causes many symptoms in humans, depending on the dosage received. These include gastrointestinal (diarrhoea, pain, nausea),

neurological and cardiovascular disturbance. Symptoms begin a few hours after eating the fish and can last for days and months. After recovering from a bout of ciguatera poisoning, the symptoms can be brought on again after eating more fish, even if those fish would not harm another person.

There is no cure for ciguatera poisoning yet and treatment is symptomatic only. Aspirins and Panadol tablets are used as painkillers, Phagon tablets and Stemetil injections for vomiting, and patients with diarrhoea, vomiting and dehydration are treated with intravenous fluids (only patients with these serious symptoms are admitted to hos-

pital). Doctors claim ciguatera poisoning is not fatal and the disease is not morbid. As a result the problem of ciguatera is left in the hands of fisheries scientists.

It is difficult to advise people in Fiji how to avoid ciguatera poisoning because it is so sporadic. Some fish are more prone to cause ciguatera than others, especially at certain times of the year or in certain areas. However, Island peoples have known about ciguatera poisoning for centuries and local communities usually know which fish to avoid at which time of the year. The best way to avoid ciguatera poisoning is to seek the advice of the people living in the area. Ciguatera intoxication has increased recently due to movement of fish to larger urban centres where people are not able to use local knowledge for guidance.

There is a sudden increase in ciguatera during the months of October and November, when the balolo rises. It is not yet known whether balolo is directly responsible or if it is the associated factors that are responsible for this increase.

It is very difficult to screen for proneness ciguatera unless a reliable test is developed. At the moment the only real way of ascertaining such areas is

through cases of poisoning. Many cases are not reported to the medical authorities and mild cases are often misdiagnosed.

Ciguatera fish poisoning in Fiji is confined to carnivorous reef fish. The larger specimens are more likely to be toxic, probably through accumulation of ciguatoxin over a long period or having taken in a large amount at one time ie. at the time of the balolo spawning.

Most common species of fish implicated in ciguatera are:

Fijian name	Scientific name
Bati damu	<i>Lutjanus bohar</i>
Dokonivudi	<i>Lethrinus miniatus</i>
Ogo	<i>Sphyraena barracuda</i>
Dabea	<i>Gymnothorax undutus</i>
Donu	<i>Variola louti</i>
Regua	<i>Lutjanus rivulatus</i>
Delabulewa	<i>Epinephelus fuscoguttatus</i>
Sumusumu	<i>Arothron stellatus*</i>

(\*Note from the editor: in this case, it's tetraodontid poisoning)

## Ciguatera fish poisoning in the Cook Islands

by Dr Wolfgang Losacker,  
Rarotonga Hospital,  
Cook Islands

The Cook Islands are 15 islands situated about 3,000 miles north-east of New Zealand. The Polynesian inhabitants number about 18,000 and are closely related to the Tahitians.

The Cook Islands are scattered over 2 million square kilometres of the Pacific Ocean. There are two distinct groups, the northern group made up of Penrhyn, Pukapuka, Manihiki, Rakahanga, Nassau and Suvarrow, which are all atoll islands with the exception of Nassau. The southern group consists of two atolls, Palmerston and Manuae, and 7 islands, Rarotonga, Aitutaki, Atiu, Mauke, Mangaia, Mitiaro and Takutea.

The traditional basic food in the islands consists of fish and taro, although on the main island of Rarotonga these foods are mixed with additional locally grown vegetables and fruit as well as imported foods.

There are no large ocean-going fishing boats belonging to the Cook Islands that could catch fish on long lines or with nets. Only small amounts of fish are caught in the southern group, which has tiny lagoons and virtually no continental shelf,

while in the northern group, with less population, there are still plenty of ocean fish as well as reef and lagoon fish available.

Ciguatera fish poisoning is hardly any problem in the northern group of the Cook Islands. There have been only a few cases reported. However, the people are cautious and do not eat the Napoleon wrasse (**Maratea**) in Penrhyn. Two serious ciguatera poisoning cases occurred in Rarotonga from a large red snapper (**Anga-mea**) that was brought over from Penrhyn.

In the northern group very few vegetables are grown. Only on the atolls are fish still foremost in the diet. Most of the people are good fishermen and like to paddle out with small outrigger canoes. They will put 3 or 4 lines down to about 150 m or go trolling outside the reef with small aluminium boats and outboard engines. They mainly catch, tuna, bonito, barracuda, wahoo and dolphin fish. There has been no ciguatera from these fish in the Cook Islands to-date. In the northern group few people go net fishing in the reef passages or in the lagoon.

This pattern is different in the southern group of the Cook Islands. The most cases of ciguatera fish poisoning have been in Rarotonga, the main island with about 9,000 inhabitants, Atiu with about 980 inhabitants, Aitutaki with 2,500 inhabitants, and Mitiaro which has a population of 250.

In 1989, 158 cases of fish poisoning were reported in the Cook Islands: statistics only mention this common term, but nearly all of these were ciguatera intoxications. In 1990, there were 109 cases and 1991 reports show 81 cases: 35 on Rarotonga, 24 on Atiu, 15 in Aitutaki and 7 on Mitiaro. During the first 3 months of 1992, 39 cases were reported, 19 in January, 15 in February, and 5 in March. But it has been estimated that only 10-20% of fish poisoning cases are actually reported. Severe cases that need medical attention are mostly reported, while many mild cases remain unrecorded.

### **Fish species causing ciguatera fish poisoning**

In the Cook Islands most poisoning were caused by eating: black surgeon fish (**Maito**), unicorn fish (**Ume**), snake mackerel (**Manga**) brown moray (**A'a pata**), red snapper (**Anga-mea**) and different cod species such as the peacock cod (**Roi**).

A survey by Mrs. Dawn Turner and her geography students on the island of Atiu in 1989 interviewed 953 people about ciguatera fish poisoning. The results showed that 183 people (19%) have had ciguatera poisoning symptoms in the past: 67 people in 1989, 52 in 1988 and 64 in earlier years. The main fish species causing the poisoning included surgeon fish, parrot fish, reef shark, unicorn fish, moray eel, red snapper and groupers. 64 people had severe ciguatera and 55 of these were admitted to hospital.

In the Cook Islands, most of the ciguatera intoxication cases reported that they had eaten fish caught on the reefs in the northern side of the islands, where most of the hurricane damage is to be seen, or surrounding recent ships' wreckage, or where rubbish had been dumped, e.g. in some parts of Aitutaki lagoon. Ciguatera dates back to the end of the last world war when the Americans dumped surplus supplies. Most people in the Cook Islands still avoid catching fish in these areas, especially those species that have previously caused ciguatera. On some smaller islands like Atiu and Mitiaro there is little possibility of avoiding potentially ciguateric fish because there is only a small amount of fish and there is a limited supply of other food available.

There seems to be a peak during the hotter months when cases of ciguatera increase through sudden outbreaks, although poisoning cases can be also seen at other times of the year.

### **Report of cases**

A 50-year-old male was admitted to Rarotonga Hospital in 1985 about two hours after having eaten parts of a large red snapper (**Anga-mea**), with heavy diarrhoea, vomiting, general weakness, joint pains, reversal of temperature sensations and severe muscle pain.

Under the treatment of intravenous fluids, antiemetics, antihistamines, calcium, and a very light diet consisting of toast, rice and water, he slowly improved and was discharged after three weeks, but joint pain in both knees and elbows as well as tingling skin sensations lasted for more than a year.

All his pigs died after eating the inner organs of that fish when he cleaned it and his cat was paralysed for about a week but survived. All his chickens had to be killed as they were running around with their guts hanging out at their back and of his neighbour's two dogs died shortly after eating discarded parts out of the rubbish drum.

In 1984, 15 people suffered from ciguatera fish poisoning after having eaten a large snake mackerel (**Aamanga**) at the same function. They all suffered from nausea or even vomiting and diarrhea with some also having abdominal distension with symptoms of a paralytic ileus.

General weakness, headaches, prickling skin sensations and severe arthralgia in the main joints were common in most cases. With symptomatic treatment, all patients were able to be discharged from hospital after 5 days. In one case, heavy watery eye irritation occurred, which rapidly improved under intravenous hydrocortisone application. Several rats, dogs and cats died after having eaten some parts of the same fish that had been dumped.

### **Clinical symptoms seen with ciguatera fish poisoning**

The first symptoms occur mostly within one hour. Paraesthesia and prickling round the lips and inside the mouth and throat were specified, followed by headaches, diarrhea, nausea or vomiting and often accompanied by abdominal cramps. Soon after, typical sensitivity disturbances appear with cold water feeling hot and hot water cold. General weakness with muscle pain and numbness occurred, especially in the legs. With severe intoxication arthralgia was localised mainly in the large joints like the knee and elbow. Only in a few cases were hypotension and bradycardia seen.

## Treatment

Until 1988 severe cases were treated symptomatically with intravenous (i.v.) fluids, mostly saline infusions, i.v. antiemetics, i.v. antihistamines, i.v. calcium, i.v. B6 and B12 vitamins, pain relief and a very light diet beginning after 24 hours of only i.v. fluid applications. Hospitalisation usually lasted about one week, but symptoms like general weakness and sensitive skin disturbances were specified for many months after. The longest ongoing symptom seemed to be the arthralgia that in some cases lasted up to three to four years. Repeated cases usually showed more severe symptoms.

Since 1988, when the successful treatment of ciguatera fish poisoning with intravenous Mannitol was reported by Palafox et al., this treatment has been used in the Cook Islands and in nearly all cases all symptoms disappeared within 24 to 48 hours. The earlier the treatment can be given after intoxication (especially when the patient has not had ciguatera poisoning before) the more effective the treatment is.

## Precautions to avoid ciguatera fish poisoning

Especially in the outer islands of the Cook Islands, the saying is that fish which no flies land on must be toxic. There are no specific tests for ciguatera available in the Cook Islands. The other precautions known are feeding parts of the fish to cats, dogs or chickens, and then observing them for clinical symptoms of intoxication for two to three hours. Some brave men sometimes taste the fish themselves

first to protect their family from intoxication, to see whether anaesthesia with pricking around the lips and mouth occurs as the very earliest symptom.

The Health staff on all of the Cook Islands are aware of the dangers of ciguatera. Improvement of case reports is encouraged and people are warned not to fish in places where ciguatera intoxications have occurred previously.

On the other hand, the introduction of i.v. mannitol has certainly seen a great improvement in the management as well as in shortening of time required for the treatment of this disease.

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## Improved ciguatoxin test kit being developed

*Source: Hawaii Fishing News, October 1991*

The ciguatera-toxin test kit to be made commercially available this year will be different from the test kit with which Hawaii fishermen have become familiar.

Although the commercial kit is still in the design and development phase, it already appears that it will include a plastic stick with a membrane rather than the wooden stick coated with white correction fluid that is now being used.

Robert Goldsmith, president of Hawaii Chemtect International (HCI), which owns the ciguatera test-kit patent, said he expected the kit to be available in the first quarter of 1992 and to retail in the 70-cent range, depending on the market.

Goldsmith also said that the kit will be better than the presently used kit because it does not register false positives, that is it does not read positive for fish that are not infected with ciguatera.

The present test has registered false positives, but several sources suggest that this could be due to oversensitivity of the test and a need to recalibrate it.

The improved kit is being developed and researched at the University of Arizona, Nutrition and Food Service Department, with funds from HCI. Dr Douglas Parks is the primary investigator and is being assisted by research assistant Sam Rua. In a phone interview, Rua clarified some technical aspects of the test kit improvement.

The kit presently distributed contains an antigen and enzyme combination, which develops coloration when combined with ciguatera toxin. The improved kit does not use the enzyme, so you get a direct reading of the antibody that is bound to the toxin that is on the membrane, Rua said. The membrane-based system has better control and more conformity of performance, he added.

Although there is no evidence of false positives to date, an inter-laboratory collaborative study on the improved test is being planned with the Food and Drug Administration to validate the improved kit

Dr George Hoskin, associate director of the FDA Office of Seafood, said that the FDA has purchased some test kits from Chemtect to become familiar with them and plans to do a mini-collaboration study once the kit is more fully developed.

That collaborative study would involve the FDA's Office of Physical Sciences, Center of Food Safety and Applied Nutrition. Dr Sam Page, who is in this office, said that they are very anxious to work with anyone on ciguatera as they consider it a "very significant problem". Noting a case of ciguatera in Cincinnati, Ohio, from fish caught in the Caribbean, Page said "It is not just a tropical problem".

A preliminary experiment to validate the improved test could be completed within a year, but full-blown studies may take more than a year, Page said. The source of the fish to be used in the test is yet to be defined, he added. As to when the experiment might begin, Page said it depends on when the company finalises the format of the test kit.

In the meantime the company can still market the kit, but the claims would have to be fairly careful, Hoskin said. However, for federal regulatory use, the government would not use the kit without it being tested thoroughly, he added.

Some fishermen have indicated that they wished the test kit presently being used in Hawaii would be quicker and have more of a gradient in the colour chart measuring toxicity levels. Rua said that they are experimenting with both factors in the improved kit.

He also said that the improved kit in its marketable format would allow fishermen to test more than one fish at a time.



## Ciguatera fish poisoning

Source: *Hawaii Fishing News*,  
October 1991

Public response from the briefings held throughout the Islands of Hawaii indicate a great demand for information on ciguatera poisoning and test kits. The public wants a better understanding of the fish poisoning situation and the kits to determine if their catch is safe for consumption.

A total of 2,000 persons attended briefings held at Wai'anae, O'ahu, in June; Hilo and Kona on the island of Hawaii in July; Wailuku, Maui, in September; and Lihue, Kaua'i, in October. Approximately 1,600 test kits were distributed.

At each of these functions, the following two questions were repeatedly asked:

1. Where can we get more kits?
2. What are commercial fishermen supposed to do?

The University of Hawaii has sold the patent for commercial kit production to Hawaii Chemtect International (HCI). HCI indicated that kits would be commercially available in January 1992, with two versions of the kit planned, one to test a single catch and one to test large batches. These two versions will therefore service both recreational and commercial fishermen.

In the interim, the University of Hawaii will be continuing its efforts to research the phenomena of ciguatera poisoning. The University's share of the

royalties generated from the patent will be directed to Dr Hokama to continue the field testing programme and to support public informational briefings. The research program envisions the statewide distribution of another 5,000 kits.

The role of the University has been to research the phenomena of ciguatera fish poisoning and to disseminate the resultant information. Though 5,000 test kits will not meet the total needs of recreational fishermen, the primary purpose will be and always has been to obtain data on the location and types of fish tainted with ciguatera. Analysis of the data could lead to the determination of factors that cause outbreaks of ciguatera, as well as geographical locations that present ciguatera poison hazards.

For the 4,600 test kits distributed, only 400 of the data sheets have been returned. The University requests that fishermen who have obtained kits mail in the data sheets as the data is essential to its efforts in minimising ciguatera toxicity in Hawaii's fish and poisoning risk to the people.

An concern identified from the data already obtained is the level of toxicity in fish that test borderline or positive with the publicly distributed Solid Phase Immunoassay (SPIA) test kit using two sticks.

Since 1981, the University has also been using another technique called the Stick Enzyme Immunoassay (SEIA) test, or, more commonly, the Six Step Stick Test to test for ciguatera toxicity.

The two-step stick test kit is used for public distribution instead of the six-step stick test for several reasons. The SEIA test procedures uses an enzyme that is extremely sensitive to light and should be used in a controlled laboratory environment. The SEIA test also uses a number of other solutions requiring controlled conditions and is more complicated due to additional step procedures.

Because the six-step stick SEIA method provides the means to characterise the level of toxicity, fish testing positive or borderline by the two-stick test kit should undergo further characterisation by this test.

In conjunction with the Department of Health and other State agencies, the Office of Technology Transfer and Economic Development (OTTED) is investigating the feasibility of establishing testing sites utilising the six-stick method on each major island (Kaua'i, Maui and Hawaii) to further characterise toxicity level in fish testing positive or borderline with the two-stick method.

If feasible, fish that test borderline or positive with the two-stick test kit, would be frozen and 1-inch cubes of flesh from the head, stomach and tail of the fish would be sent to a designated lab for further testing with the six-stick method.

## Identifying ciguatera-infected reefs

*Source: Hawaii Fishing News, October 1991*

The ideal situation would be to know the cause of ciguatera outbreaks and therefore be able to prevent them. A stepping stone to that goal is being able to identify ciguatera-infected reefs. Once these reefs are identified, activity around them can be monitored in order to pinpoint the cause of dinoflagellate blooms. These microscopic algae produce the ciguatoxin. The toxin enters the food chain when fish eat the dinoflagellates. People suffer from ciguatera when they eat the infected fish.

Because fish move about one cannot assume that the reef where an infected fish was caught is the reef that infected the fish. Because dinoflagellates are so ephemeral, they are hard to locate. How then does one locate the infected reef?

A scientist from Moss Landing Marine Laboratory of the California State University who has been

doing research in Hawaii may be on the way to discovering the answer.

Rikk Kvitek has been involved in looking for ciguatoxin in invertebrates such as sea urchin, sea cucumbers, snails and clams.

He decided he wanted to collect invertebrates in areas of the Kona Coast that had a history of ciguatoxin in the past couple of years – such as Mauna Lani, Puako, Kawaihae and Miloli'i – and compare them with invertebrate species collected on the Hamakua Coast, where no ciguatera cases have been reported.

Kvitek predicted that snails, urchins and other invertebrates that feed along the bottom would be accumulating toxins from the dinoflagellates and that there would be a difference in the ciguatoxin levels found in the same species on the two coasts.



Using the improved ciguatera-toxin test-kit being developed at the University of Arizona for Hawaii Chemtect International, Kvitek was able to identify one species that appeared to be toxic on the Kona coast but not on the Hamakua coast. It is a marine snail and common cowrie, *Cypraea maculifera*, or the reticulated cowrie.

Because snails are usually located within a few metres of the area where they eat, they would be a better marker or indicator of toxicity in the reef than fish or dinoflagellates, Kvitek said.

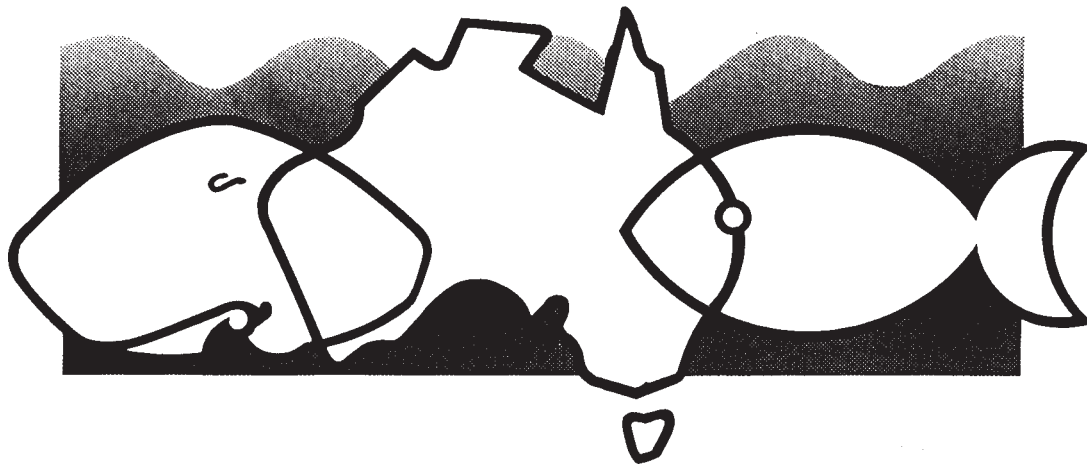
But, he stressed, the results are only preliminary and a lot of work needs to be done.

Kvitek is in the process of writing a scientific paper with Dr Douglas Parks of the University of Arizona, Nutrition and Food Science Department, that will summarise the research.

They also may seek funding to continue their research in Hawaii by proposing a reef survey and monitoring programme.

## Upcoming workshop

### Ciguatera Management Workshop Brisbane, April 1993



The four-day international Workshop will focus on current research that has implications for the management of ciguatera, and will include sessions on:

- chemical and immunological aspects of detection;
- pharmacology and treatment;
- origin;
- clinical aspects and epidemiology

The Workshop format will comprise talks by invited speakers, workshop sessions and poster presentations.

**Workshop Secretariat**  
**Southern Fisheries Centre**  
**P.O.Box 76, Deception Bay, Queensland 4508**  
**AUSTRALIA**  
**tel.: (617) 203 1444**  
**fax: (617) 203 3517**

Major sponsors: Fishing Industry Research and Development Council, and the Queensland Department of Primary Industries

## New references in SPC library

by J.P. Gaudechoux,  
South Pacific Commission,  
Noumea, New Caledonia

We received recently two new references (listed below) to be added to the Fisheries Information Project's ciguatera bibliographic database. They will be held in the SPC library and will be available on request.

If there are documents that you feel should be added to the database, please send us a copy, or, if

this not possible, a photocopy of the cover page. Documents do not need to be formal publications — many of those held in the database are not — and we are keen to archive as much 'grey literature' (internal reports, correspondence, unpublished data, etc...) as possible.

Thanks in advance for your help.

### New references

Lewis, R.J., M. Sellin, M.A. Poli, R.S. Norton, J.K. MacLeod and M.M. Sheil (1991). Purification and characterisation of ciguatoxins from moray eel (*Lycodontis javanicus*, Muraenidae). *Toxicon* Vol. 29 (9): 1115-1127.

Gonzalez i Anadon, G. (1992). Contribution à la connaissance des processus ciguatériques: Etude anatomo-physiologique et histocytologique des effets de(s) toxine(s) de *Gambierdiscus toxicus* chez *Serranus cabrilla*. Etude hépatique de poissons ciguatérigènes. Thèse de doctorat de l'Université de Perpignan, 335 pp. (Abstract in English is shown below.)

*The liposoluble toxins produced by the etiological agents of ciguatera, such as Gambierdiscus toxicus, are accumulated by many species of reef fish through the food chain, culminating in humans. Although fish abnormalities have never been proved nor pathological effects identified, recent data from literature indicate that some macroscopical changes occur in fish following ingestion of G. toxicus. A series of experiments on*

*Serranus cabrilla fed with the toxic alga showed us (mouse test) that most of the toxicity from the dinoflagellates crosses the gastro-intestinal barrier of the cabrilla sea bass.*

*Monitoring of thirty variables (morphometric, haematological and enzymological), as well as the histocytological study of the liver, revealed physiological disturbances (diminution of growth, metabolic and macrophagic activation and inactivity of detoxification mechanisms) and tissue lesions in the liver (dilatation and sinusoidal fibrosis, lipid degeneration and necrosis), all corresponding to a reliable pathology. An analysis of discriminant variables allowed 80% of the fish to be correctly classified.*

*An histocytological and histopathological study of the liver of some reef species made it possible to confirm the existence of histological characteristics (association with pancreatic tissue, lipofucsin granules and lipid reserves) in relation to the metabolism of lipids and/or of liposoluble toxins and the description of non-specific lesions (sub-acute inflammation, haemorrhagia, lipid degeneration and necrosis) that can be associated with the intoxications or parasites, as shown by the literature.*

## Welcome to new members

by J.P. Gaudechoux,  
South Pacific Commission,  
Noumea, New Caledonia

The Ciguatera Special Interest Group is growing. We have received additional completed questionnaires from the individuals listed below. The previous list of members is available in the first issue of *SPC Ciguatera Information Bulletin*.

If you are on the list and your name and address are wrong, please send us a correction. If you are not on the list and would like to be, fill in the form enclosed with the *Bulletin* or write to us for a new one.

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