# RISK PROFILE:
# CIGUATOXINS IN SEAFOOD

Prepared as part of a New Zealand Food Safety Authority
contract for scientific services

by

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March 2007

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**ACKNOWLEDGEMENTS**

The authors would like to thank the following for information, comments and valuable discussions:
- Dr Penny Truman, ESR Keneperu Science Centre
- Ruth Pirie, ESR Keneperu Science Centre
- Dr Hoe Chang, National Institute of Water and Atmospheric Research (NIWA)
- Dr Greg Simmons, Medical Officer of Health, Auckland Regional Public Health

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## SUMMARY

Ciguatera fish poisoning is solely caused by the consumption of seafood contaminated with toxins produced by dinoflagellate microalgae, particularly *Gambierdiscus toxicus*. While there are isolated reports of ciguatera fish poisoning resulting from consumption of non-finfish species, the disease is usually caused by the consumption of large finfish species from circumtropical regions.

While there have been reports of potentially ciguatoxic dinoflagellate species in northern New Zealand waters, no cases of ciguatera fish poisoning have been associated with fish from these waters and, at least under New Zealand conditions, *Ostreopsis* and *Coolia* dinoflagellates appear to produce palytoxins, rather than ciguatoxins. All reported cases of ciguatera fish poisoning in New Zealand have been due to consumption of risk fish species imported from the Pacific Islands, principally Fiji, or consumed in the Pacific Islands. In all but one documented case of fish importation, the fish were imported by individuals for their own consumption.

In New Zealand approximately six cases per year come to the attention of the public health system. Internationally, it has been suggested that notified cases may only represent 2-20% of total cases. This would equate to an average number of annual cases in New Zealand of 30-300. As no diagnostic test for ciguatera is currently available and New Zealand physicians would generally be unaccustomed to diagnosing the disease, a considerable degree of under-reporting would be expected.

The risk of ciguatera fish poisoning amongst the general New Zealand population is likely to be very low and will mainly be associated with travel-acquired disease. The risk for the Pacific Island population will be considerably higher due to the practice of private importation of potentially ciguatoxic fish for personal consumption. There is currently little control on the private importation of seafood into New Zealand. With the information currently available, it is not possible to further categorise the level of risk within the Pacific Island community.

The major data gaps identified in this document are:

- Frequency of importation and consumption of risk fish species; and
- Frequency of ciguatoxin contamination in risk fish species.

# 1 INTRODUCTION

The purpose of a Risk Profile is to provide contextual and background information relevant to a food/hazard combination so that risk managers can make decisions and, if necessary, take further action. The place of a risk profile in the risk management process is described in "Food Administration in New Zealand: A Risk Management Framework for Food Safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000). Figure 1 outlines the risk management process.

**Figure 1: Risk Management Framework**

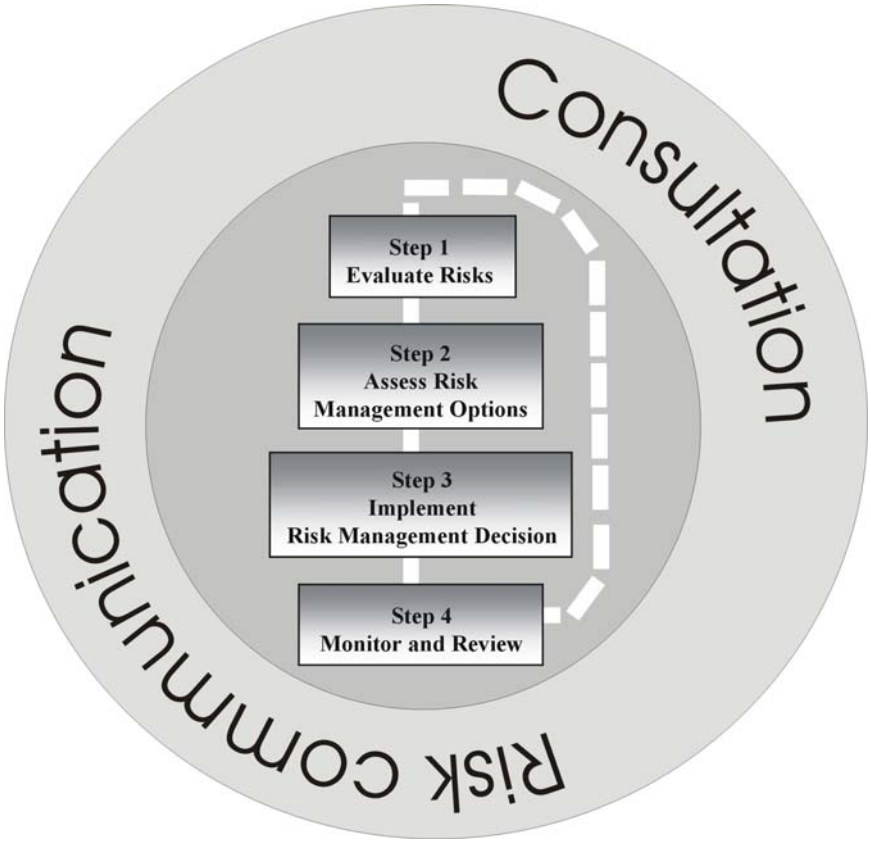



Figure reproduced from "Food Administration in New Zealand. A risk management framework for food safety" (Ministry of Health/Ministry of Agriculture and Forestry, 2000).

In more detail, the four step process is:

*1. Risk evaluation*

- Identification of the food safety issue
- **Establishment of a risk profile**
- Ranking of the food safety issue for risk management
- Establishment of risk assessment policy
- Commissioning of a risk assessment
- Consideration of the results of risk assessment

2. *Risk management option assessment*
    - Identification of available risk management options
    - Selection of preferred risk management option
    - Final risk management decision

3. *Implementation of the risk management decision*

4. *Monitoring and review.*

The risk profile informs the overall process, and provides an input to ranking the food safety issue for risk management.

This Risk Profile concerns ciguatoxins in seafood, formed through the affected fish feeding on certain dinoflagellate microalgae, and the subsequent occurrence of ciguatera fish poisoning (CFP) in humans.

The sections in this Risk Profile are organised as much as possible as they would be for a conventional qualitative risk assessment, as defined by Codex (Codex, 1999).

*Hazard identification, including:*
- A description of the organism or toxin
- A description of the food group

*Hazard characterisation, including:*
- A description of the adverse health effects caused by the organism or toxin
- Dose-response information for the organism or toxin in humans, where available

*Exposure assessment, including:*
- Data on the occurrence of the hazard in the New Zealand food supply.
- Data on the consumption of the food group by New Zealanders.
- Qualitative estimate of exposure to the organism or toxin (if possible).
- Overseas data relevant to dietary exposure to the organism or toxin

*Risk characterisation:*
- Information on the number of cases of adverse health effects resulting from exposure to the organism or toxin with particular reference to the identified food (based on surveillance data).
- Qualitative estimate of risk, including categorisation of the level of risk associated with the organism or toxin in the food (categories are described in Appendix 1).

*Risk management information:*
- A description of the food industry sector, and relevant food safety controls.
- Information about risk management options.

*Conclusions and recommendations for further action*

# 2 HAZARD IDENTIFICATION: THE ORGANISM AND THE TOXINS

## 2.1 *Gambierdiscus toxicus*

While marine algae had been hypothesized to be the source of ciguatera fish poisoning (CFP), it was not until 1977 that toxins were isolated from detritus collected from the surface of dead coral from the Gambier Islands in French Polynesia (Yasumoto *et al.*, 1977). The toxins isolated were compared to reference ciguatoxin from the liver of moray eel (P-CTX-1) and one of them was judged to be identical or closely related.

The toxin was shown to be associated with the cells of a dinoflagellate micro alga present in the detritus that was renamed *Gambierdiscus toxicus* (Adachi and Fukuyo, 1979). *G. toxicus* is a photosynthetic species that normally grows as an epiphyte and has a diameter of approximately 80 µm (Lehane, 1999). Although *G. toxicus* can swim if disturbed it is usually found attached to certain macroalgae (Lehane, 1999). The macroalgae constitute a food source for some herbivorous fish. *G. toxicus* is distributed circumtropically between latitudes 32°N and 32°S (FAO, 2004).

While the wide range of symptoms observed with CFP have lead to conjecture that a range of toxins from different dinoflagellates may be involved (Juranovic and Park, 1991), *G. toxicus* is generally considered to be the principal cause of the disease in the Pacific. Another dinoflagellate, *Ostreopsis lenticularis*, has also been associated with ciguatoxic fish in the Caribbean (Tosteson *et al.*, 1986; Tosteson, 2004). *Coolia monotis*, another dinoflagellate species closely related to *Ostreopsis* that is present in New Zealand coastal waters (Rhodes *et al.*, 2000) has also been implicated in CFP in the Caribbean (Pottier *et al.*, 2001).Toxin production varies between different strains of *G. toxicus* and not all strains are toxin-producing (Holmes *et al.*, 1991).

Increases in incidence of CFP have been shown to follow periods of *G. toxicus* proliferation, with a lag time of approximately three months between peak *G. toxicus* densities and peak human cases (Chateau-Degat *et al.*, 2005). Growth is favoured by water temperatures of about 30°C, water depths of 1-4 metres and approximately 11% of full sunlight (FAO, 2004). There appears to be a lag time of 13-16 months between seawater peak temperatures and maximum *G. toxicus* densities (Chateau-Degat *et al.*, 2005). While increases in ciguatera cases are associated with increases in *G. toxicus* densities, the organism does not produce 'blooms' such as those sometimes referred to as 'red tide' and fish kills have not been reported in association with *G. toxicus*.

For islands in eastern Polynesia (Tuvalu, Rarotonga, Kiribati, Western Samoa, French Polynesia), which experience a local warming during El Niño events, a positive association has been reported between El Niño events, elevated surface sea temperatures (SST) and reported incidence of ciguatera fish poisoning (Hales *et al.*, 1999), while for islands in western Polynesia (Fiji, Vanuatu, New Caledonia), which experience a local cooling during El Niño events, there is a negative association between El Niño events and SST and a weak negative association between SST and reported incidence of ciguatera fish poisoning. The reason for these differences in the pattern of association between climatic conditions of ciguatera fish poisoning is currently unknown.

*G. toxicus* is commonly found growing epiphytically on macroalgae that colonise damaged coral reefs, following either natural (tidal waves, earthquakes, hurricanes) or anthropogenic events (military and tourist developments) (FAO, 2004).

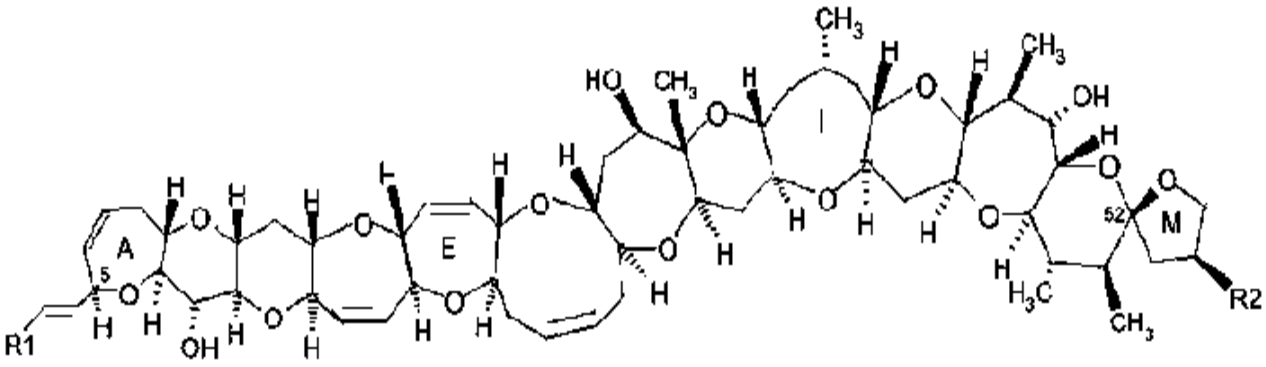
## 2.2 Ciguatoxins and their Precursors

*G. toxicus* produces lipid-soluble ciguatoxins (CTX) and water-soluble maitotoxins (MTX) (de Fouw *et al.*, 2001). While all strains produce maitotoxins, only some produce ciguatoxins (Holmes *et al.*, 1991). The ciguatoxins isolated from *G. toxicus* are less polar than those isolated from ciguateric fish and are usually referred to as gambiertoxins (Lehane, 1999).

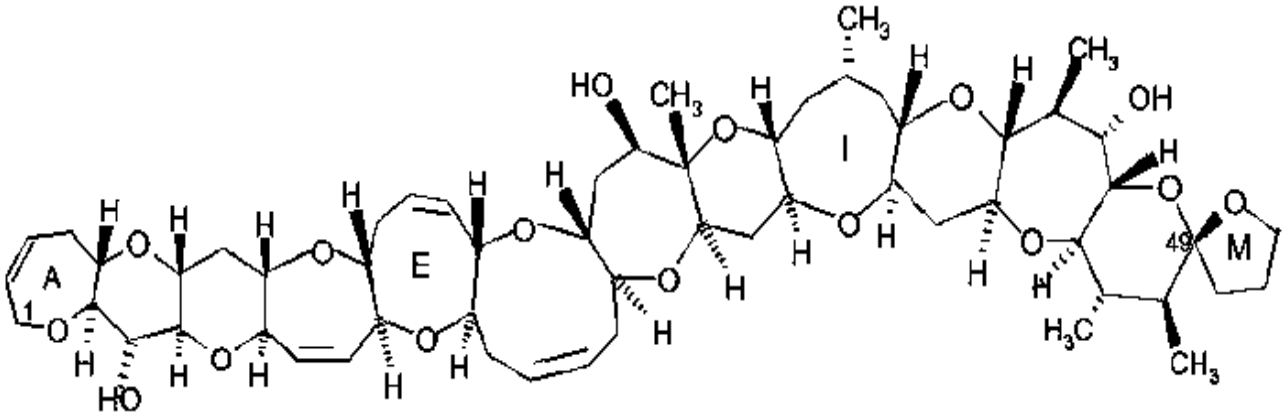
### 2.2.1 Structure and Nomeclature

While ciguatoxins from different geographical areas are similar in structure, differences have been determined and a prefix is used to distinguish toxins from the Pacific (P-CTX), the Indian Ocean (I-CTX) and the Caribbean (C-CTX). Ciguatoxins are lipid-soluble polyether compounds consisting of 13 to 14 rings fused by ether linkages into ladder-like structure (FAO, 2004). They are heat-stable and remain toxic after cooking and exposure to mild acidic and alkaline conditions. Ciguatoxins arise from biotransformation of precursor gambiertoxins in the fish. As they progress through the environmental web, from dinoflagellate to herbivorous fish to carnivorous fish, they undergo oxidative modification and become increasingly polar and increasingly toxic.

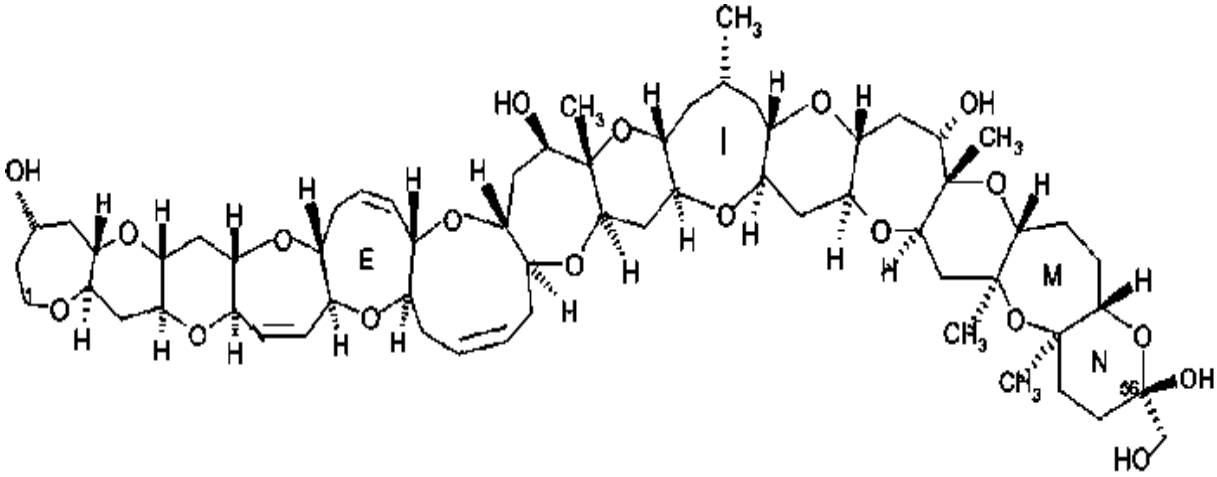
**Figure 2: Structure of Pacific (P) and Caribbean (C) ciguatoxins (CTXs)**




P-CTX-1: R1 = $^{1}CH_2OHCHOH$, R2 = OH
P-CTX-2 and P-CXT-3 (isomers): R1 = $^{1}CH_2OHCHOH$, R2 = H
P-CTX-4B and P-CTX-4A (isomers): R1 = $^{1}CH_2CH$, R2 = H




P-CTX-3C




C-CTX-1 (C-CTX-2)

*Reproduced from FAO (2004)*

P-CTX-1 is the principal and most toxic ciguatoxin isolated from ciguatoxic carnivorous fish in the Pacific and is believed to be the main cause of CFP. It has been suggested that P-CTX-1 is formed from P-CTX-4A, produced by *G. toxicus*, by acid-catalysed spiroisomerisation and oxidative modification in the liver of carnivorous fish (Nicholson and Lewis, 2006). It is uncertain whether the structural differences seen in Caribbean ciguatoxins are due to differences in the precursors or differences in metabolism by Caribbean fish species.

### 2.2.2 Toxicity

Table 1 lists the major ciguatoxins that have been characterized and their toxicity, as measured by intraperitoneal $LD_{50}$ in the mouse.

**Table 1: Source, molecular mass and toxicity of major ciguatoxins***

| Ciguatoxin | Source# | Molecular mass (Da) | $LD_{50}$ (μg/kg ip) |
|---|---|---|---|
| P-CTX-1 | Carnivore | 1110 | 0.25 |
| P-CTX-2 | Carnivore | 1094 | 2.3 |
| P-CTX-3 | Carnivore | 1094 | 0.9 |
| P-CTX-3C | *G. toxicus* | 1044 | ND |
| 2,3-Dihydroxy-P-CTX-3C | Carnivore | 1056 | 1.8 |
| 51-hyrdoxy-P-CTX-3C | Carnivore | 1038 | 0.27 |
| CTX-4A | *G. toxicus*, Herbivore | 1060 | 2 |
| CTX-4B | *G. toxicus*, Herbivore | 1060 | 4 |
| C-CTX-1 | Carnivore | 1140 | 3.6 |
| C-CTX-2 | Carnivore | 1140 | 1 |
| I-CTX-1 | Carnivore | 1140 | ~0.5 |
| I-CTX-2 | Carnivore | 1140 | ~0.5 |

** From Nicholson and Lewis (2006). For reference to original toxicological studies see this reference*
ip = intra-peritoneal
# Carnivore = toxin found in carnivorous fish, Herbivore = toxin found in herbivorous fish, *G. toxicus* = toxin found in benthic detritus containing the dinoflagellate, *G. toxicus*

In general, the toxins become more oxidized, more polar and more toxic as they move from dinoflagellate to herbivorous fish to carnivorous fish.

Ciguatoxins are ichthyotoxic (toxic to fish) at high levels, with symptoms including behavioural and morphological changes preceding death (Lewis, 1992). The lethality of ciguatoxins to fish is likely to impose an upper limit to the concentration in fish flesh consumed by humans – this may contribute to the relatively low incidence of human fatalities resulting from ciguatera (Lewis, 1992).

### 2.2.3 Mechanism of toxicity

Excitable membranes are critical to the function of nerves and muscles. Their function depends on the normal activity of ion channels and membrane ion pumps. Sodium ion channels are transmembrane proteins involved in intercellular communication (Lehane, 1999). The proteins form pores in the plasma membrane, the opening and closing of which are controlled by gating systems.

Ciguatoxin has been shown to bind to a receptor site on the sodium channel, leading to prolonged opening of the sodium channel and excessive influx of sodium ions into the cell (Lombet *et al.*, 1987). Lombet *et al*. (1987) demonstrated that ciguatoxin binds to the same receptor site as the shellfish biotoxin, brevetoxin, but with an affinity 20-50 times higher. This has been shown to cause nodal swelling in nerve fibres (Benoit *et al.*, 1996) and alteration of nerve function in humans (Cameron *et al.*, 1991).

The increased movement of sodium ions into cells also causes the cells to exude sodium and take up calcium, which acts as a trigger for muscle contraction (Swift and Swift, 1993).

### 2.2.4 Methods of analysis for ciguatoxins

#### *2.2.4.1 Animal bioassays*

Animal bioassays have utilized cat, mongoose, chicken, mouse, mosquito and fish (Hokama and Yoshikawa-Ebesu, 2001). The mongoose has some advantages as an oral feeding model, as it will retain fish fed to it, rather than regurgitating, and exhibits a range of symptoms analogous to those observed in humans (Banner *et al.*, 1960; Hamilton *et al.*, 2002b).

A mosquito bioassay has been used extensively in French Polynesia (Pompon *et al.*, 1984). The method involves injection of a crude fish extract into the intrathoric cavity of mosquitoes (*Aedes aegyptii*) and determination of the $LD_{50}$. The method shows good correlation with toxicity in mouse, cat and humans.

The most commonly used animal bioassay has been the intraperitoneal injection of fish extracts into the mouse (Banner *et al.*, 1960). The assay has been standardized (IP injection of 20 mg of ether extract of fish muscle) to define a mouse unit (MU) for ciguatoxin toxicity based on the formula:

$$\text{Log (MU)} = 2.3 \log (1 + 1/T)$$

Where T is time to death in hours. One MU is the concentration of toxin which kills a 20 gram mouse in 24 hours and is equivalent to a dose of approximately 5 ng P-CTX-1 (Lehane, 1999). The mouse bioassay has been shown to detect ciguatoxins in 71% of fish implicated in cases of ciguatera fish poisoning in Queensland (Lehane, 1999). While the mouse assay is very effective for the detection of ciguatoxic fish the method is expensive, time consuming and ethically questionable. However, the mouse bioassay is not reliable for the detection of low-toxicity ciguatoxic fish (Lehane, 1999). These drivers have lead to the development of a range of alternative methods for the detection of ciguatoxins or ciguatoxicity.

#### *2.2.4.2 Cell-based assays*

Cell-based assays offer increased sensitivity and require minimal test material compared to animal bioassays. Cytotoxicity to sodium channels has been measured in mouse neuroblastoma cells (Manger *et al.*, 1995). The neuroblastoma assay is significantly more sensitive than the mouse bioassay and is able to detect ciguatoxin activity at levels of approximately $10^{-4}$ MU. Detection limits are between 0.25 and 1 pg CTX-1. The assay can also be used to detect saxitoxin, the main toxin involved in paralytic shellfish poisoning (PSP) and brevetoxin, the main toxin involved in neurotoxic shellfish poisoning (NSP) (Manger *et al.*, 1995). The neuroblastoma assay is currently established at ESR for the detection of saxitoxin and brevetoxin (Truman and Lake, 1996; Truman *et al.*, 2002). Recent improvements have been made to this method, involving fluorimetric detection of changes in membrane potential instead of changes in specific enzyme activities (Louzao *et al.*, 2004)

Cell-based assays utilizing HeLa and fibroblastic mammalian kidney cells (Swiss mouse) have also been used to detect ciguatoxins (Hokama and Yoshikawa-Ebesu, 2001). However, little information is available on the performance characteristics of these assays.

Cell-based assays, particularly the neuroblastoma assay, are highly sensitive, relatively simple to perform and correlate well with results from the mouse bioassay in its ability to rank samples in terms of ciguatoxicity, as they are directly measuring the major toxic effect

due to the fish extracts. The neuroblastoma assay is currently being trialed for the detection of ciguatoxin activity in human blood (Matta *et al.*, 2002).

#### *2.2.4.3 Immunoassays*

Several assays have been developed based on the interaction between ciguatoxins and specific antibodies raised against ciguatoxins.

The first such assay involved a radioimmunoassay with sheep polyclonal antibodies raised against purified moray eel ciguatoxin (Hokama *et al.*, 1977). The sheep antibody was coupled to iodine-125. This method was used to screen over 5,000 fish samples for ciguatoxin during 1979-1981 (Kimura *et al.*, 1982). The screening programme was effective in identifying potentially ciguatoxic fish and preventing ciguatera fish poisoning due to the monitored fish species during the monitoring period.

Subsequently, enzyme immunoassays were developed, firstly with sheep polyclonal anti-CTX coupled to horseradish peroxidase, followed by replacement of the polyclonal antibodies by monoclonal IgG antibodies (Hokama *et al.*, 1998; Hokama and Yoshikawa-Ebesu, 2001). These test principles have been used to develop a commercial product (Cigua-Check; http://cigua.oceanit.com/) that can be used to test very small quantities of fish flesh for the presence of ciguatoxins. Cigua-Check will also detect other polyether marine toxins, including okadaic acid and brevetoxin. Cigua-Check is currently registered on the AOAC International website with status "Seeking peer-verified method status" (http://www.aoac.org/testkits/kits-toxins.htm). However, the status of this kit does not appear to have progressed in some years.

It should be noted that immunoassays detect ciguatoxin and other toxins on the basis of structure and a greater response in the immunoassay does not necessarily relate to greater toxicity of the associated fish sample (Lehane, 1999).

#### *2.2.4.4 Chemical methods*

Analytical methods based on high-performance liquid chromatography (HPLC) have been used to detect and quantify ciguatoxins from fish samples to sub-parts per billion levels (Lewis *et al.*, 1999). These techniques allow the ciguatoxin mixtures present in sample to be elucidated (Hamilton *et al.*, 2002a; Pottier *et al.*, 2002; Vernoux and Lewis, 1997). As with the immunoassays, chemical methods detect ciguatoxins on the basis of their chemical structure. However, if coupled with existing knowledge on the toxicity of different ciguatoxin congeners, such methods can indicate the likely toxicity of fish samples.

Chemical methods for the detection and quantification of ciguatoxins are hampered by the lack of pure, commercially available standard materials and most investigative studies are dependent on either in-house purification of the compound(s) from a fish source, or donation of purified material from other research groups.

#### *2.2.4.5 Summary*

Methods for the detection of ciguatoxins can be broadly grouped into two types:

- Methods that detect a biological response (animal bioassays and cell-based assays)

- Methods that detect a chemical or chemical structural element (immunoassays and chemical methods)

Assessment of the relative merits of these methods is hampered by incomplete understanding of the aetiology of the ciguatera fish poisoning. While it is believed that CTX-1 is the major determinant of ciguatoxicity, it is not the only ciguatoxic compound and there have been suggestions that toxins other than ciguatoxins may play a role in the disease. These uncertainties will also contribute to difficulties in determining a threshold dose for ciguatoxicity, below which a ciguatoxic fish may be considered safe to eat. For example, in the mouse bioassay, one mouse unit (MU) is reported to equate to approximately 0.005 μg P-CTX-1 while it has been estimated that the lower limit for human toxicity is approximately 0.05 μg P-CTX-1. Despite the method appearing to have sufficient sensitivity to detect potential sources of human intoxication, the method was only able to detect ciguatoxicity in 71% of fish associated with ciguatera fish poisoning cases (Lehane, 1999).

Recent investigations of suspect ciguatera fish poisoning cases in the US have used a combination of cell-based assays and chemical methods, to detect sodium ion channel toxicity and to confirm the presence of ciguatoxins as the likely putative agents (Quilliam, 2001).

Until the state of knowledge on ciguatera fish poisoning develops further none of the available detection methods will be able to distinguish between a positive test result likely to result in disease and a positive test result unlikely to result in disease.

## 3 HAZARD IDENTIFICATION: THE FOOD

While the topic of the current risk profile relates to ciguatoxins in all seafood, few reports have been found of ciguatoxins in species other than finned fish. Ciguatoxin has been detected in the viscera of a marine snail, the turban shell (*Turbo argyrostoma*), which has occasionally caused ciguatera-like intoxication in humans (WHO, 1984). Jellyfish consumption was also implicated as the source of one case of ciguatera fish poisoning of a 12-year-old Tongan girl residence in the USA, although no samples of the implicated jellyfish could be obtained for testing (Zlotnick *et al.*, 1995). The jellyfish was carried into the country from American Samoa. There is some evidence to suggest that marine shrimps may act as a vector for transfer of gambiertoxins to fish species, but there is no evidence to suggest that crustacea are able to biotransform gambiertoxins to ciguatoxins (de Fouw *et al.*, 2001).

The foods included in the category of seafood include fish (or finfish), molluscan shellfish and crustacea. The 24-hour dietary recall records from the 1997 National Nutrition Survey (Russell *et al.*, 1999) indicate that the seafood consumption of New Zealanders is principally fish (83%), followed by mollusca (11%), and crustacea (6%). The mode of transmission of ciguatoxin (from dinoflagellate to herbivorous fish to carnivorous fish) and the epidemiology of CFP mean that subsequent discussion will focus on finfish.

### 3.1 Relevant Characteristics of the Food

The risk of ciguatoxin contamination in finfish is related to their geographical location and their position in the food web, rather than compositional aspects of the food.

While hundreds of fish species have been implicated in ciguatera fish poisoning worldwide, the predominant species and their geographical distribution are summarised in Table 2.

**Table 2: Major fish species associated with ciguatera fish poisoning**

| Fish Family | Fish species | Geographical distribution |
|---|---|---|
| Acanthuridae | Lined surgeonfish (*Acanthurus lineatus*) | Indo-Pacific |
| Albulidae | Bonefish (*Albula vulpes*) | Worldwide in warm waters |
| Balistidae | Gray triggerfish (*Balistes carolinensis*) | Atlantic, Gulf of Mexico |
| Carangidae | Horse-eye jack (*Caranx latus*) | Atlantic |
| | Lesser amberjack (*Seriola fasciata*) | Western Atlantic |
| Carcharhinidae | Whitetip shark (*Carcharhinus longimanus*) | Worldwide |
| Labridae | Humphead wrasse (*Cheilinus undulates*) | Indo-Pacific |
| | Hogfish (*Lachnolaimus maximus*) | Western Atlantic |
| Lutjanidae | Northern red snapper (*Lutjanus campechanus*) | Western Atlantic, Gulf of Mexico |
| | Yellowtail snapper (*Ocyurus chrysurus)* | Western Atlantic |
| | Chinamanfish (*Symphorus nematophorus*) | Western Pacific |
| Megalopidae | Tarpon (*Megalops atlanticus*) | Eastern Atlantic |
| Mugilidae | Narrowhead gray mullet (*Mugil capurrii*) | East Central Atlantic |
| Muraenidae | Giant moray (*Gymnothorax javanicus*) | Indo-Pacific |
| Scaridae | Heavybeak parrotfish (*Chlorurus gibbus*) | Indo-Pacific |
| | Blue parrotfish (*Scarus coeruleus*) | Western Atlantic |

| Fish Family | Fish species | Geographical distribution |
|---|---|---|
| Scombridae | Spanish mackerel (*Scomberomorus maculatus*) | Western Atlantic |
| Serranidae | Red grouper ((*Ephinephelus morio*) | Western Atlantic |
| | Spotted coral grouper (*Plectropomus maculatus*) | Western Pacific |
| Sparidae | Saucereye porgy (*Calamus calamus*) | Western Atlantic |
| Sphyraenidae | Great barracuda (*Sphyraena barracuda*) | Indo-Pacific, Western Atlantic |
| Xyphidae | Swordfish (*Xiphias gladius*) | Atlantic, Indo-Pacific, Mediterranean |

*Adapted from (Farstad and Chow, 2001)*

While the fish species listed in Table 2 are the most important contributors to ciguatera fish poisoning worldwide, there is considerable regional variability. Table 3 summarises results of several studies that have looked at the proportions of ciguatera cases due to various families of fish in various regions.

**Table 3: Fish families contributing to ciguatera fish poisoning in different geographical areas**

| Fish family | Percentage of ciguatera cases due to fish family (%) | | | |
|---|---|---|---|---|
| | Guadeloupe 1993-1994 | Florida 1954-1992 | French Polynesia 1964-1977 | New Caledonia 1993* |
| Carangidae | 21 | 8 | 4 | 3 |
| Lutjanidae | 16 | 11 | 4 | 11 |
| Sphyraenidae | 2 | 48 | 1 | 0 |
| Scombridae | 5 | 1 | 1 | 13 |
| Serranidae | 16 | 19 | 9 | 43 |
| Scaridae | 0 | 0 | 5 | 6 |
| Labridae | 0 | 8 | 0 | 0 |
| Acanthuridae | 0 | 0 | 65 | 0 |
| Other | 40 | 5 | 11 | 16 |

*Adapted from (Pottier et al., 2001)*

* Statistics are presented here as they were in the originally publication (Amade, 1993). It should be noted that the percentages presented here to not add to 100%.

In Fiji, the most common fish species implicated in ciguatera have been reported as Lutjanidae (two-spot red snapper, blubberlip snapper), Serranidae (brown-marbled grouper, coronation cod), Muraenidae (undulating moray), Sphyraenidae (great barracuda) and Lethrininae (sweetlip emperor) (Singh, 1992).

While fish species associated with cases of ciguatera fish poisoning in New Zealand are often identified, popular and often non-specific names have been used. Species identified include moray eel (family Muraenidae), kawakawa, reef cod and coral trout. Kawakawa is a type of tuna (*Euthynnus affinis*, family Scombridae), while reef cod and coral trout are almost certainly types of reef grouper (family Serranidae).

### 3.2 The Fishing Industry in New Zealand

(Information mainly from the New Zealand Seafood Industry website: http://www.seafood.co.nz/)

In 1978 New Zealand extended its Exclusive Economic Zone (EEZ) from 12 to 200 nautical miles. Since then, the fishing industry has expanded to include harvesting mid and deep-water species from within this EEZ. Initially, this involved joint ventures with overseas companies experienced in this type of fishing, but now most boats are New Zealand owned. Although New Zealand's EEZ is the fourth largest in the world, covering 1.3 million square nautical miles, 65% percent of that is too deep for commercial fishing. New Zealand's EEZ extends from 56ºS to 26ºS and overlaps the latitudes in which ciguatera fish poisoning is prevalent.

By the early 1980s fishing pressure had reduced the size of a number of New Zealand's major fisheries, particularly the inshore fisheries. New Zealand regulatory authorities responded to this situation by introducing the Quota Management System (QMS) in 1986 with the aims of conserving major fisheries stocks and making the fishing industry more sustainable. QMS involves the industry and government agencies working together to continually assess stock levels of all quota-managed species. From these assessments, the Ministry of Fisheries (MFish) sets a yearly Total Allowable Commercial Catch (TACC) for each species concerned. The TACC is divided into a number of Individual Transferable Quotas (ITQs). When a species is first brought under the Quota Management System, Maori are given 20% of the total quota. The remainder is distributed amongst those people who hold commercial fishing permits for that species - based on how much they caught over previous years.

Since 1986, restructuring in the fishing industry has resulted in more quota being held by fewer individuals or companies. The leading New Zealand fishing companies and quota holders are currently Sealord Products Ltd, Sanford Limited, Talley/AMALTAL, Vela Ltd, Moana Pacific Ltd and the member companies of the Seafood Industry Consortium.

There are now 50 species or species groups controlled by the quota system. The system covers most major fisheries within New Zealand's EEZ, and will eventually cover all our commercially harvested species. Species currently under quota management are not generally risk species for ciguatera, although some species from the families Scombridae and Carcharinidae are included in the quota system.

New Zealand's total seafood harvest is made up of mid and deep water species (80% of total), pelagic (12%) and inshore species (10%). The Food and Agriculture Organization of the United Nations (FAO) consolidates information on production and consumption of commodities through their FAOSTAT databases (http://faostat.fao.org/). For the most recent reported year (2004), New Zealand harvested 517,000 tonnes of finfish, with domestic consumption of 73,000 tonnes.

New Zealand's domestic market for fish is estimated to be about $NZ150 million per year and the seafood sector is predominantly an export industry. With exports in the region of NZ$1.43 billion in 2000 (representing 323,000 tonnes of produce), the seafood sector ranks

amongst the top five export sectors in the New Zealand economy, with Japan, the USA and the European Union being the largest markets.

### 3.2.1 Imported food

According to import statistics for the year ending September 2005 New Zealand imports approximately 11,000 tonnes of seafood and seafood products. Imports from within the ciguatoxin risk zone include:

- Fish from the Pacific Islands; Cook Islands (approximately 11 tonnes), Fiji (202 tonnes), New Caledonia (2.5 tonnes), Niue (0.25 tonnes), Samoa (27 tonnes), Tonga (0.5 tonnes).
- Fish from other countries fully or partially in the circumtropical zone (32º North to 32º South); Australia (878 tonnes), China (552 tonnes), Hong Kong (4.5 tonnes), India (20 tonnes), Indonesia (68 tonnes), Malaysia (26 tonnes), Namibia (3.4 tonnes), Papua New Guinea (19 tonnes), Peru (46 tonnes), Philippines (6.2 tonnes), Singapore (98 tonnes), South Africa (93 tonnes), Sri Lanka (2.1 tonnes), Taiwan (66 tonnes), Thailand (1,632 tonnes), USA (71 tonnes), Vietnam (22 tonnes)

Finfish imports from countries within the ciguatoxic zone (3,850 tonnes) equates to approximately 5% of domestic consumption. Crustacea and mollusks are also imported from many of these countries.

The data above concern the New Zealand population as a whole. Some information on consumption of imported seafood by Pacific Island people has also been reported (Thornton *et al.*, 2002). Data collected by the 2001 Pacific Island Food Safety Campaign indicated that 56% (95% confidence interval 47-65%) of 124 Samoan, Tongan and Cook Island respondents aged 15 years and over reported consuming seafood brought back by their families and friends from the Pacific Islands in the preceding 12 months. However, these frequency data do not allow determination of the volume of personally imported fish.

# 4 HAZARD CHARACTERISATION: ADVERSE HEALTH EFFECTS

Consumption of fish contaminated with ciguatoxins may result in ciguatera fish poisoning (CFP) or ciguatera seafood poisoning (CSP). The characteristics of the disease are variable and depend on the type and amount of toxin present and on the individual's susceptibility (Lehane and Lewis, 2000). There is currently no diagnostic test for CFP/CSP and diagnosis is by the presentation of characteristic symptoms and a history of recent consumption of potential ciguatoxic fish.

## 4.1 Symptoms

Time to onset of symptoms: 1-70 hours, mean 2-6 hours.

Symptoms: Initial symptoms are intense vomiting, diarrhoea and abdominal pain within hours of fish ingestion, generally lasting 24-36 hours. This is followed, usually within 12-14 hours of onset, by development of neurological disturbance, including paraesthesia (tingling, crawling or burning sensation of the skin) and dysaesthesia (reversal of temperature perception), arthralgia, myalgia, muscle cramping and weakness. Pruritis (itching) and sweating are also commonly experienced during this stage of the illness.

Other symptoms that may occur in a proportion of cases include hallucinations, transient paralysis, dysphasia (difficulty in speaking), aching joints, palpitations, dry mouth, disturbed vasomotor regulation including deranged blood pressure control, brachycardia or tachycardia.

Severe cases may result in paralysis, coma and death, although this is rare.

Condition: Ciguatera

Toxins: See section 2.2 for a full description of the causative toxins.

People Affected: The whole population is susceptible to intoxication, although in ciguatera endemic areas susceptibility may increase with age due to accumulation of ciguatoxin in the body as a result of previous exposures.

Long Term Effects: The neurological disturbance characteristic of ciguatera usually resolves within weeks of onset, but in some cases may persist for months or even years. The toxin may be stored in adipose tissue for several years and symptoms may recur during periods of stress, such as exercise, weight loss or excessive alcohol consumption (Barton *et al.*, 1995).

Some cases exhibit an allergy-like syndrome that can persist for several years, in which symptoms typical of ciguatera are brought on by consumption of non-toxic fish or, occasionally, chicken or pork (Lehane and Lewis, 2000).

Sensitivity to alcohol can persist for several years and in some cases alcohol consumption may cause recurrence of ciguatera symptoms (Gillespie *et al.*, 1986).

Ciguatera has also been associated with subsequent development of polymyositis (a chronic inflammatory muscular disease) (Stommel *et al.*, 1991) and chronic fatigue syndrome (Barton *et al.*, 1995).

Treatment: Ciguatera treatment is mainly supportive, although intravenous mannitol has been shown to provide benefit in severe cases. Injection of the anaesthetic lignocaine into the peritoneum has also been used to reverse the major ciguatoxin-induced changes in nerve conduction.

### 4.2 Dose Response

Ciguatoxins found in Pacific fish (P-CTX) differ in chemical structure and toxicity to those found in Caribbean fish (C-CTX) (Vernoux and Lewis, 1997). Based on mouse intraperitoneal $LD_{50}$ values C-CTX possesses about 10% of the potency of P-CTX.

Most cases of ciguatera studied in the Pacific involve consumption of fish containing 0.1-5 µg P-CTX-1/kg. Based on an expected fish meal size of 500 g, Lehane and Lewis (2000) estimated that mild ciguatera could be expected from ingestion of a dose of 0.05 µg P-CTX-1, while a dose of 0.5 µg P-CTX-1 would be expected to be toxic to most people. Caribbean ciguatoxins appear to be less toxic than Pacific ciguatoxins and it has been estimated that C-CTX-1 levels of greater than 0.25 µg/kg would be required to elicit adverse reactions in humans (Lewis *et al.*, 1999).

Information from National Nutrition Surveys conducted in New Zealand (Ministry of Health, 2003; Russell *et al.*, 1999) indicate that a normal fish serving for New Zealanders is considerably less than 500 g (approximately 40-400 g). No information is available on the serving sizes for fish consumed after personal importation.

# 5 EXPOSURE ASSESSMENT

## 5.1 The Hazard in the New Zealand Food Supply: Ciguatoxin in Seafood

### 5.1.1 Ciguatoxic dinoflagellates in New Zealand waters

*Gambierdiscus toxicus* is the primary dinoflagellate believed to be responsible for production of the toxins that result in ciguatera fish poisoning. It has been reported that *G. toxicus* has been recorded once in a sample collected in the north of New Zealand (Chang *et al.*, 2000), however, no primary reference for this detection was given.

*Ostreopsis* and *Coolia* species of dinoflagellates are closely related to *Gambierdiscus*, and have been implicated in ciguatera fish poisoning in the Caribbean (Tosteson, 2004). A survey of dinoflagellates in upper North Island coastal waters found *Ostreopsis lenticularis*, *O. siamensis* and *O. ovata* to be widespread, with cell concentrations greatest during the Summer at times of peak sea surface temperatures (approximately 21ºC) (Chang *et al.*, 2000). *O. siamensis* and *Coolia monotis* isolated from New Zealand waters have been shown to produce lipid soluble toxins (Rhodes *et al.*, 2000), which have been tentatively identified as palytoxins or 'palytoxin-like' (Rhodes *et al.*, 2002). While palytoxins are also sodium channel toxins, they differ from ciguatoxins/brevetoxins in their mechanism of action (Truman *et al.*, 2005). Palytoxins also appear to be significantly less stable than ciguatoxins and there is no evidence that they accumulate up the food chain (Dr Penny Truman, ESR, personal communication).

### 5.1.2 Ciguatoxic fish in New Zealand

No information was located on surveillance of ciguatoxins in fish from New Zealand waters.

The ESR Suspect Food Poisoning database contains details of six outbreaks that were investigated for the involvement of ciguatoxins, on the basis of case symptoms. As no ciguatoxin-specific assay was available, food samples associated with these outbreaks were analysed using a neuroblastoma assay, developed for analysis of the shellfish biotoxin, brevetoxin (Louzao *et al.*, 2004; Manger *et al.*, 1995). Brevetoxin has a similar mechanism of toxicity to ciguatoxin, acting via the sodium ion channels of the cell membrane and sharing a common receptor site on the sodium channel with ciguatoxin (Lombet *et al.*, 1987; Nicholson and Lewis, 2006). In all six outbreaks fish samples were implicated and in all cases the fish samples were found to have sodium ion channel dependent toxicity of 3.6-1000 µg brevetoxin equivalents/100 g fish flesh. Control (non-ciguatoxic) fish analysed at the same time were negative in this assay.

## 5.2 Food Consumption: Seafood

According to Food Balance Sheets held by FAO (http://apps.fao.org) New Zealanders have available for consumption approximately 24.3 kg/year/capita or approximately 67 g/person/day of seafood. This is made up of fish (88%), crustacea (5.5%), cephalopods (octopus, squid and cuttlefish; 5.5%) and mollusca (1%).

The WHO regional diets (see http://www.who.int/fsf/GEMS/index.htm) give lower consumption figures for fish and seafood, ranging from 13 g/person/day (Middle Eastern

diet) to 46.3 g/person/day (European diet). The New Zealand diet is usually considered to be closely aligned with the European diet.

The 1997 National Nutrition Survey (Russell *et al.*, 1999) gives a much lower figure of 23.9 g/person/day of which fish (83%) makes up the majority of the seafood consumed, followed by mollusca (11%), and crustacea (6%) (ANZFA, 2001). The simulated typical diets derived for the 1997/98 New Zealand Total Diet Survey (Brinsdon *et al.*, 1999) arrived at a near identical level of seafood consumption (25 g/person/day, averaged across adult males and females).

These figures are also similar to estimates of seafood consumption by the Australian population of 25.7 g/person/day (Australian Bureau of Statistics, 1999), the US population of 14.3 g/person/day (fish and shellfish, EPA, http://cfpub.epa.gov/ncea/cfm/exposfac.cfm?ActType=default), the UK population of 22.3 g/person/day (http://statistics.defra.gov.uk/esg/statnot/efsuk.pdf) and the Canadian population of 25.5 g/person/day (http://www.statcan.ca/english/Pgdb/People/Families/famil1102d.htm).

The data above concern the New Zealand population as a whole. Some information on consumption of imported seafood by Pacific Island people has also been reported (Thornton *et al.*, 2002). Data collected by the 2001 Pacific Island Food Safety Campaign indicated that 56% (95% confidence interval 47-65%) of 124 Samoan, Tongan and Cook Island respondents aged 15 years and over reported consuming seafood brought back by their families and friends from the Pacific Islands in the preceding 12 months.

## 5.3 Qualitative Estimate of Exposure

### 5.3.1 Number servings and serving sizes

While information is available on general consumption of seafood by New Zealanders, there is little information on the consumption of risk material for ciguatera fish poisoning.

### 5.3.2 Frequency of contamination

Unknown. Testing of fish for ciguatoxicity in New Zealand has only been carried out in response to reported cases of suspect food poisoning.

### 5.3.3 Predicted contamination level at retail

Only one reputed ciguatera fish poisoning incident in New Zealand was associated with fish purchased at retail. The fish at retail was imported.

### 5.3.4 Growth rate during storage and most likely storage time

Not relevant. While the toxin appears to be stable in the fish, no new toxin product will occur in fish during storage.

### 5.3.5 Heat treatment

Ciguatoxins are highly heat stable.

## 5.4 Overseas Context

### 5.4.1 Ciguatoxin in seafood

There are significant difficulties in trying to define the prevalence of ciguatoxins in fish, largely due to the sporadic nature of *G. toxicus* blooms. Prevalence may be defined for a particular fish species at a particular geographical location at a particular time, although even this approach presents difficulties as fish may move from location to location and the place where they are tested may not be the place where they acquired the ciguatoxins (Lehane, 1999).

Few studies have been carried out to determine either the prevalence of ciguateric fish in particular environments or the level of the toxins in particular fish.

During 1979-1981, the United Fishing Agency (UFA) and the National Marine Fisheries Service (NMFS) in Hawaii carried out screening of commercial catches of *Seriola dumerili* (amberjack) for the presence of ciguatoxin by radioimmunoassay (RIA) (Kimura *et al.*, 1982). Of 5529 fish tested, 824 (15%) were rejected due to positive or borderline RIA results and were not release for sale. No cases of ciguatera linked to consumption of *Seriola dumerili* were reported by local health authorities during the course of the monitoring programme. No significant temporal trends were observed in the prevalence of ciguatoxin-positive *Seriola dumerili*.

HPLC-MS analysis of ciguatoxic fish from Queensland coastal waters determined the relative levels of CTX-1, CTX-2 and CTX-3 (Lewis and Sellin, 1992). The respective amounts found were 0.19, 0.09 and 0.02 μg/kg in the flesh of narrow-barred Spanish mackerel (*Scomberomorus commersoni*), 0.08, 0.09 and 0.07 μg/kg in the flesh of grouper (*Plectropomus* spp.) and 0.67, 0.61 and 0.06 μg/kg in the flesh of blotched javelin fish (*Pomadasys maculates*).

Using an HPLC-MS-MS method and purified P-CTX-1 as an internal standard, analyses were carried out on 30 Caribbean fish extracts (Lewis *et al.*, 1999). The fish from which the extracts had been taken had previously been tested for ciguatoxicity by the mouse bioassay. All eight fish identified as toxic by mouse bioassay contained 0.5-2.0 ppb of C-CTX-1 equivalents. Of 12 fish classified as borderline, 11 contained C-CTX-1 in the range 0.1-0.5 ppb, while two of 10 fish classified as non-toxic contained C-CTX-1 at approximately 0.1 ppb.

Analysis of three fish implicated in ciguatera poisoning by LC-MS detected C-CTX-1 in the flesh at levels in the range 0.24-13.8 μg/kg (Pottier *et al.*, 2002). The fish were a grey snapper *Lutjanus griseus*), a black jack (*Caranx lugubris*) and a grouper (*Serranidae*). Other congeners were detected, but not quantified.

# 6 RISK CHARACTERISATION

## 6.1 Adverse Health Effects in New Zealand

### 6.1.1 Incidence

Ciguatera seafood poisoning is not specifically a notifiable disease in New Zealand and, hence, systematic incidence data on cases of intoxication are not available. However, ciguatera cases and outbreaks are notified under the disease category 'acute gastroenteritis' (http://www.moh.govt.nz/moh.nsf/0/A38E98064984642BCC257045007EC9CA/$File/notifiablediseases.pdf).

### 6.1.2 Outbreaks

Since 1998, ten outbreaks and one sporadic case of ciguatera fish poisoning have been reported in the Episurv database. Relevant details of these incidents are summarised in Table 4.

**Table 4: Outbreaks or cases of ciguatera fish poisoning in New Zealand 1998-2005**

| Year | Cases | Age range (years) | Time to onset range (hours) | Implicated Food | Country of origin of fish |
|---|---|---|---|---|---|
| 1998 | 6 | 15-45 | NS | Kawakawa fish | Fiji |
| 1999 | 1 | 48 | NS | Raw fish | Fiji |
| 1999 | 7 | 16-66 | 4-12 | Fish | Fiji |
| 1999 | 2 | 57-65 | 3 | Moray eel | Samoa |
| 2001 | 4 | 30-60 | 4-11 | Reef cod | Fiji |
| 2001 | 4 | 47-86 | 9-64 | Coral reef trout | Fiji |
| 2002 | 7 | 5-43 | 5-36 | Kawakawa fish | Fiji |
| 2002 | 2 | 45-46 | 5 | Kawakawa fish | Fiji |
| 2003 | 5 | 7-50 | 6.5-24 | Reef fish | Fiji |
| 2003 | 2 | 43-56 | 1 | Moray eel | Samoa |
| 2005 | 3 | 33-61 | 11.5-64.5 | Coral trout | Fiji |

All but one (AK2002130) of the references in Table 4 appear to relate to incidents of personal importation of reef fish. In the remaining incident the imported fish was sold through a discount retail outlet.

Simmons presented outbreak reports for four of the outbreaks in Table 4, covering 12 cases (Simmons, 2005).

#### *6.1.2.1 AK2001101*

A four-person suspected food poisoning outbreak involved an Indian family that privately imported and consumed fish from Fiji, was reported during June 2001. All four cases experienced gastrointestinal (diarrhea, vomiting, nausea), neurological (paraesthesia of the lips and hands, temperature perception reversal, itchy skin) and other (chills, shortness of breath, difficulty walking, muscle pain, loss of energy, joint pain and numbness in the legs) symptoms. Duration of symptoms ranged from half a day, for some gastrointestinal symptoms, to more than eight days for some neurological and other symptoms. Leftover fish

was found to contain sodium channel-dependent toxicity, estimated at approximately 22 μg brevetoxin equivalents/100 g.

#### *6.1.2.2 AK2003006*

A three person suspected food poisoning outbreak was reported during January 2003. Two further cases were identified during the investigation. The cases were all members of the same family (mother, father, two sons, aunt). The group had consumed a meal of fish privately imported from Fiji. The incubation period from consumption to first symptoms ranged from 6.5 to 24 hours. The most common symptoms (reported in all five cases) were diarrhea, chills, difficulty walking, itchy skin, muscle pain, temperature perception reversal and loss of energy. Other symptoms reported by three or more cases were abdominal pain, nausea, shortness of breath, skin rash, headache, joint pain and paraesthesia of the tongue. Skin rash and itchy skin were the most long lasting symptoms with median durations of 30 and 35 days respectively. Sample of the fish curry were tested and exhibited sodium channel-dependent toxicity at an approximate level of 1 mg brevetoxin equivalents/100 g.

#### *6.1.2.3 AK2003183*

A two person suspected food poisoning outbreak was notified during September 2003. A 5-8 kg moray eel privately imported from Samoa had been divided between three families. Both cases reported symptoms of diarrhoea, abdominal pain, chills, vomiting, vertigo, difficulty walking, loss of energy, and paraesthesia of the hands. Other symptoms (headache, paraesthesia of the lips, numbness in hands, numbness in legs, depression, joint pain, visual defects and short-term memory loss) were experienced by one or other, but not both, of the cases. Symptoms lasted for from less than one day up to 25 days, with loss of energy being the most long-lasting symptom. The eel contain sodium channel-dependent toxicity of 0.6 mg brevetoxin equivalents/100 g.

#### *6.1.2.4 AK2005015*

A three person suspected food poisoning outbreak was reported during February 2005. The implicated meal included a portion of a 2-3 kg coral trout personally imported from Fiji, after being given as a gift. All cases experienced symptoms of shortness of breath, dental pain, paraesthesia of the lips, numbness in the legs, muscle pain, loss of energy and joint pain. Other symptoms (chills, skin rash, nausea, vertigo, difficulty walking, itchy skin, neck stiffness, temperature perception reversal, paraesthesia of the hands and paraesthesia of the toes) were experienced by one or two of the cases. Symptoms lasted from less than one day up to 42 days (joint pain). No fish was available for testing.

### 6.1.3 Clinical consequences of ciguatoxin fish poisoning

Of six outbreak-related cases and one sporadic case report in the Episurv database, none of the cases were reported as being hospitalised or resulting in fatality, although information is lacking in some cases. Of the outbreaks studied in detail by Simmons, none of the cases were reported as being hospitalised or dying (Simmons, 2005).

Isolated cases of hospitalisation due to ciguatera fish poisoning have been reported in New Zealand (Anonymous, 1995; Crump *et al.*, 1999).

Hospital discharge records show a slightly different picture, with one case in 2002, three cases in 2003, five cases in 2004 and one case in 2005 reported as having ciguatera fish poisoning (Ruth Pirie, ESR, personal communication). Of these 10 hospitalisations only two appear to relate to outbreaks or cases listed in Table 4. Some uncertainty exists with respect to these hospital discharge data as, while the latest international disease coding system (ICD 10) includes a specific code for ciguatera (T61.0), the previous system (ICD 9) did not. Some of these records will have been translated from the ICD 9 code 9880 (toxic effect of noxious substances eaten as food – fish and shellfish) and may include cases hospitalised due to shellfish biotoxin or other intoxication.

Quod and Turquet (1996) reported that 10% of cases on Réunion Island (Indian Ocean) were hospitalised. A study carried out in the Cook islands found that 55 of 183 (30%) ciguatera fish poisoning cases required hospitalization (Losacker, 1992).

Mortality from CFP is generally be less than 1% and most likely to occur when the most toxic parts of the fish (liver, roe) are consumed (Lehane, 1999). In a study of 3009 CFP cases in French Polynesia, the case mortality rate was 0.1%, with approximately one third of cases being confined to bed due to their illness (Bagnis *et al.*, 1979).

A case mortality rate of approximately 20% (98 out of 500 cases) was reported for an outbreak in Madagascar (Boisier *et al.*, 1995). The outbreak appears to have been caused by consumption of a shark.

#### 6.1.4 Case control studies and risk factors

No case control studies for New Zealand were identified.

### 6.2 Adverse Health Effects Overseas

#### 6.2.1 Incidence

Ciguatera fish poisoning is not generally a notifiable disease and estimates of its incidence usually come from isolated studies or dedicated surveillance programmes. Incidence estimates for ciguatera fish poisoning are generally only available for at-risk populations – small island nations within tropical and sub-tropical waters with a high level of fish consumption. Available estimates are summarized in Table 5. It has been estimated that the global incidence of ciguatera fish poisoning is in the range 25,000 to 500,000 cases per annum (Quod and Turquet, 1996).

**Table 5: Incidence data for ciguatera fish poisoning overseas**

| Country | Incidence (cases/100,000) | Year | Reference |
|---|---|---|---|
| Australia (Queensland) | 1.6 | | (Ruff and Lewis, 1994) |
| Dade county (Miami), USA | 50 | 1974-1976 | (Lawrence *et al.*, 1980) |
| French Polynesia | 363 | 1992-2001 | (Chateau-Degat, 2005) |
| Hawaii, USA | 8.7 | 1984-1988 | (Gollop and Pon, 1992) |
| Puerto Rico | 900 | 1980-1982 | (Pottier *et al.*, 2001) |

| Country | Incidence (cases/100,000) | Year | Reference |
|---|---|---|---|
| Réunion Island (Indian Ocean) | 7.8 | 1986-1994 | (Quod and Turquet, 1996) |
| South Pacific: | | 1992 | (SPEHIS, 1993) |
| American Samoa | 0.0 | | |
| Cook Islands | 870 | | |
| Fiji | 160 | | |
| French Polynesia | 440 | | |
| Federated States of Micronesia | 10 | | |
| Guam | 0.0 | | |
| Kiribati | 1730 | | |
| Marshall Islands | 570 | | |
| Nauru | 0.0 | | |
| New Caledonia | 90 | | |
| Niue | 40 | | |
| Northern Mariana Islands | 140 | | |
| Palau | 0.0 | | |
| Pitcairn Island | 0.0 | | |
| Papua New Guinea | 0.0 | | |
| Solomon Islands | 0.0 | | |
| Tokelau Islands | 810 | | |
| Tonga | 10 | | |
| Tuvalu | 1980 | | |
| Vanuatu | 700 | | |
| Wallis and Fortuna | 0.0 | | |
| Western Samoa | 80 | | |
| US Virgin Islands | 730 | 1980 | (Morris Jr *et al.*, 1982) |
| Vanuatu | 460 | 1988 | (Goodman *et al.*, 2003) |

It is generally agreed that these incidence figures probably only account for 10-20% of actual cases (de Fouw *et al.*, 2001).

Three epidemiological patterns have been described for ciguatera fish poisoning (Lehane, 1999):

- Endemic areas, where cases occur year-round;
- Epidemic areas, where only outbreaks are observed; and
- Intermediate areas, where outbreaks occur, but cases are also seen between outbreaks.

Outbreaks in Fiji, Florida and Hawaii have been reported to be seasonal, occurring mainly in Spring and early Summer (Lehane, 1999). The occurrence of ciguatera outbreaks and cases in New Zealand is fairly consistent with this observation, with over 60% of the incidents reported in Table 4 being reported between August and December.

### 6.2.2 Contributions to outbreaks and incidents

Of Australian foodborne disease outbreaks reported during the period 1995-2000, 11% of outbreaks and 2% of cases were due to ciguatera (Dalton *et al.*, 2004). No deaths were reported due to ciguatera fish poisoning. The median number of cases per outbreak was five with a range from 2 to 33.

A review of foodborne disease outbreaks in the USA in the period 1983-1987 identified 2,397 outbreak, representing 91,678 cases (Bean *et al.*, 1990). A total of 26% of outbreaks and 2% of cases were due to chemical agents, of which ciguatoxins and scombrotoxin accounted for 73% of the outbreaks. From 1988-1992, ciguatera accounted for 42 of 2,423 outbreaks (1.7%) and 176 of 77,373 cases (0.2%) (Bean *et al.*, 1996). No deaths were associated with ciguatera outbreaks. For the period 1993-1997, 2,751 outbreaks were identified, affecting 86,058 cases (Olsen *et al.*, 2000). Of these, ciguatera accounted for 60 (2.2%) of outbreaks and 205 (0.2%) of cases, with no deaths resulting.

Within the USA there is considerable state-to-state variation in the contribution of ciguatera to total outbreaks and outbreak-associated cases. For the period 1978-1987, ciguatera accounted for 80% of outbreaks in Hawaii (71% of cases), 56% of outbreaks on Guam (48% of cases) and 5% of outbreaks in Florida (4.4% of cases), while in Washington ciguatera only accounted for 0.6% of outbreaks and 0.3% of cases (Institute of Medicine, 1991).

In Canada, between 1975 and 1984, seafood (fish and shellfish) accounted for approximately 7% of all foodborne outbreaks and 4% of cases (Todd, 1997). Seafood toxins accounted for about 2% of all outbreaks, although the proportion of these due to ciguatera was not reported.

In Cuba, from an average of 269 foodborne disease outbreaks per annum, during the period 1984-1988, 3.2% were estimated to be due to ciguatera (Todd, 1996).

Finfish may be the cause of several types of intoxications including scombroid (histamine) poisoning, tetrodotoxin poisoning and ciguatera fish poisoning. Quod and Turquet (1996) reviewed 159 outbreaks involving 477 cases that occurred on Réunion Island during 1986-1994 in which disease was due to finfish consumption. Of the total cases, 78.6% were due to ciguatera fish poisoning, 15.5% due to scombroid poisoning, with the remaining 5.8% due to 'hallucinatory' poisoning, tetrodotoxin poisoning or undetermined toxins.

### 6.2.3 Clinical consequences of ciguatera fish poisoning

A wide range of gastrointestinal, neurological and cardiovascular symptoms have been reported in cases suffering from ciguatera fish poisoning. The frequency of various symptoms is believed to vary geographically and to be related to toxicological differences between the toxins and the mix of toxins exhibited in different regions. Reported data, mainly from the Western Pacific, are summarised in Table 6.

**Table 6: Frequency of symptoms associated with ciguatera fish poisoning**

| | Percent of cases | | | | | |
|---|---|---|---|---|---|---|
| **Region** | French Polynesia | Queensland, Australia | Fiji | Réunion Island | Vanuatu | Victoria, Australia |
| **Number of cases** | 3009 | 219 | 792 | 167 | 95 | 30 |
| **Study reference** | (Bagnis *et al.*, 1979) | (Ting *et al.*, 1998) | (Ting *et al.*, 1998) | (Quod and Turquet, 1996) | (Goodman *et al.*, 2003) | (Ng and Gregory, 2000) |
| Gastrointestinal | | | | | | |
| Diarrhoea | 70.6 | 64.2 | 51.2 | 49 | 67.4 | 67 |
| Vomiting | 37.5 | 35.0 | 29.8 | 50 | 62.1 | 17 |
| Abdominal pain | 46.5 | 52.0 | 58.9 | 29 | 32.6 | 47 |
| Nausea | 42.9 | 54.9 | | 50 | | 30 |
| Neurological | | | | | | |
| Paraesthesia<br>-of extremities<br>-circumoral | <br>89.2<br>89.1 | <br>63.5-71.2<br>65.8 | <br><br>51.7 | <br>82<br> | 7.4<br><br> | <br>77-87<br>57 |
| Temperature reversal | 87.6 | 76.1 | 55.3 | 65 | 2.1 | 63 |
| Ataxia | 37.7 | 54.0 | | | | 37 |
| Tremor | 26.8 | 30.5 | | | | 23 |
| Dental pain | 24.8 | 37.2 | | 5 | 2.1 | 13 |
| Cardiovascular | | | | | | |
| Hypotension | 12.2 | | | 25 | 43.0 | |
| Brachycardia | | | | 14 | 46.1 | |
| Other | | | | | | |
| Arthralgia | 85.7 | 79.1 | 69.3 | 29 | 15.7 | 57 |
| Myalgia | 81.5 | 83.3 | | 38 | | 77 |
| Pruritis | 44.9 | 76.3 | 35.1 | 37 | 4.2 | 27 |
| Vertigo | 42.3 | 44.9 | 37.6 | 32 | | |
| Headache | 59.2 | 62.2 | | 22 | | |
| Chills | 59.0 | 49.2 | 42.2 | | | 70 |
| Perspiration | 36.7 | 42.6 | 34.0 | 19 | | |
| Neck stiffness | 24.2 | 26.7 | | | | 33 |
| Watery eyes | 22.4 | 41.1 | | 11 | | |
| Skin rash | 20.5 | 25.9 | 2.4 | | | |
| Dysuria | 18.7 | 22.0 | | 8 | 7.4 | 10 |
| Salivation | 18.7 | 9.9 | 10.0 | 10 | | |
| Dyspnoea | 16.1 | 28.3 | 8.5 | 5 | | 13 |
| Paresis | 10.5 | 26.5 | | | | |
| Asthenia | 60.0 | 90.3 | 70 | | | 70 |
| Hallucinations | | | | 16 | | |

It should be noted that different studies vary in their classification of symptoms as 'neurological' and the classification used in Table 6 is based on that of Ng and Gregory (2000).

It has been reported that gastrointestinal symptoms are more common in Caribbean ciguatera fish poisoning, while neurological symptoms are more prominent in Pacific cases (Pottier *et al.*, 2001).

### 6.2.4 Case control studies

No case control studies were identified, possibly because the link between ciguatera fish poisoning and seafood is so well established.

### 6.2.5 Risk assessments and other activity overseas

A semi-quantitative comparative risk assessment was carried out for a range of hazard/product combinations of significance to the Australian seafood industry (Sumner and Ross, 2002). Each hazard/product combination was assigned a risk ranking score using the Risk Ranger risk assessment tool (Ross and Sumner, 2002). Seafood associated risk fell in a score range from 31 to 72, with the score of 72 relating to the risk to recreational gatherers due to algal biotoxins during an algal bloom. The risk to recreational fishers in Queensland due to ciguatera in reef fish was scored at 60 (third highest of 19 risks assessed) and the risk to the general Australian population due to ciguatera in reef fish was scored at 45 (sixth equal of 19 risks assessed).

The Risk Ranger software was also used to perform a scenario-based risk assessment for ciguatera fish poisoning in a hypothetical Pacific atoll group where reef fish is consumed locally (16% of harvest, consumed weekly by the entire population) or exported to New Zealand (84% of harvest, consumed a few times per year by 25% of the population) (Sumner *et al.*, 2004). Based on an assumed contamination rate of one fish per 1000, Risk Ranger predicted 520 island-based cases of ciguatera in a population of 10,000 and 3,000 New Zealand cases in a population of 4,000,000. However, it should be stressed that this was a hypothetical exercise only.

## 6.3 Estimate of Risk for New Zealand

No data are available on the prevalence of ciguatoxins in seafood available for consumption in New Zealand. There is very limited evidence of the occurrence of the causative dinoflagellate species in New Zealand waters. The normal temperatures of New Zealand coastal waters are likely to be less than optimal for these organisms, and no cases of ciguatera fish poisoning associated with fish from New Zealand waters have been reported.

The small number of recent outbreaks of ciguatera fish poisoning in New Zealand have mainly related to consumption of seafood privately imported from Pacific islands, such as Fiji and Samoa. The importance of this food source means that most of the risk from this food/hazard combination will occur within the Pacific Island community. Sporadic cases also occur in the non-Pacific Island population due to consumption of ciguatoxic fish while visiting the Pacific Islands (Crump *et al.*, 1999).

The current risk of ciguatera fish poisoning to the general New Zealand population is likely to be very low, as there is a low probability of exposure to ciguatoxins. The current risk of ciguatera fish poisoning to the Pacific Island community is likely to be considerably higher than for the general population. Consumption of potentially ciguatoxic fish amongst this population occurs regularly, although there is no information on the prevalence of ciguatoxicity in privately imported fish.

## 6.4 Risk Categorisation

The rationale for categorisation of food/hazard combinations is presented in Appendix 1.

While data are incomplete, it appears that hospitalisation due to ciguatera fish poisoning is not common (<10%), although in one outbreak both cases were hospitalised (100%). No fatalities have been reported from this cause in New Zealand. The case fatality rate for ciguatera fish poisoning has been reported to be less than 0.1%.

Information in Table 4 suggests that approximately six to seven cases of ciguatera fish poisoning are notified in New Zealand each year, giving a crude incidence rate of 0.2 per 100,000. It is recognised that ciguatera fish poisoning is usually unreported by a factor of 10-50. Given the relative inexperience of local physicians with this disease, the level of under-reporting is likely to be high.

The incidence rate will be higher amongst Pacific Island groups. If an under-reporting factor of 50 were applied and it was assumed that cases would be predominantly within the Pacific Island community (approximately 287,000 at the last census; http://www.stats.govt.nz/census/2006-census-data/classification-counts/about-people/ethnic-group.htm), the crude incidence rate for the Pacific Island community could be of the order of 100/100,000 population. An incidence rate of this order would be viewed as high.

## 6.5 Risk Summary

| Food/hazard combination | Severity | Incidence | Trade importance | Other considerations |
|---|---|---|---|---|
| Ciguatoxins in seafood | 2 (0.5-5% serious outcomes) | 3 (1-10 per 100,000) | | Seafood imported from the Pacific Islands is a major risk factor |

# 7 RISK MANAGEMENT INFORMATION

Limited risk management activities for control of ciguatera fish poisoning are in place worldwide (FAO, 2004). Where controls are in place they usually take the form of bans on the taking and sale of high-risk fish from known ciguatoxic locations. Additional risk management activities include public education, particularly of high risk groups, such as recreational fishermen.

## 7.1 Relevant Food Controls: New Zealand

### 7.1.1 Regulatory control of commercial seafood importation

The NZFSA's legislative vehicle for imported foods is the New Zealand (Prescribed Foods) Standard 2002. The standard prescribes "finfish in waters from tropics world-wide, the extreme southeastern US (including south Florida) and the Bahamian region, Barracuda, amber jack, horseye jack, black jack, other large species of jack, king mackerel, large groupers and snappers and mackerel and barracuda in waters from mid and north eastern Australia" for the condition "the presence of ciguatoxin contamination". The Standard is given force through the application of Standard Management Rules (SMRs), defining detention, documentation and testing requirement for release of product onto the New Zealand market. The foods prescribed due to potential ciguatoxin contamination do not currently have an associated SMR (Hilary Eade, NZFSA, personal communication).

Finfish related SMRs cover:

- Smoked or vacuum packed fish (salt and aerobic plate count)
- Manufactured fish products (*Listeria monocytogenes*)
- Histamine susceptible fish species (tuna, bonito, herring, mackerel, kingfish) (histamine)
- Fish susceptible to mercury contamination and microbiological spoilage (Dogfish and shark) (mercury and total volatile nitrogen)

While these SMRs may lead to inspection of some potential ciguateric fish species, they are unlikely to provide effective regulatory control on importation of ciguateric fish.

### 7.1.2 Private importation of seafood

Privately imported seafood, particularly reef fish from the Pacific Islands is a significant risk factor for ciguatera fish poisoning. According to the Biosecurity NZ (http://www.biosecurity.govt.nz/files/border/travel/travellers-brochure.pdf) this practice is not illegal and does not require a permit, although a requirement exists to declare these foods to officers of the Ministry of Agriculture and Forestry (MAF) and receive a biosecurity clearance. Biosecurity NZ guidelines on private importation of fish do not set a limit on quantity, although NZFSA have a limit of 20 kg below which no permit is required.

A ban on private importation of seafood from the Pacific Islands was suggested by Auckland District Health Board's Public Health Unit but has not been implemented. The unit has issued advice to consumers on ciguatera fish poisoning through their Food Safety Advice publication

(http://www.arphs.govt.nz/publications/Advice_Publications/FS/2002/FS_Dec02.pdf). The advice given was:
How to prevent ciguatera poisoning:

- Do not eat or sell tropical reef fish that weigh more than 2.5 kilograms
- Avoid importing, selling or eating certain species of large fish; grouper, sea bass, barracuda, snapper, mackerel and coral trout
- Clean the fish well
- Eat only small portions of large reef fish
- Avoid eating the roe (eggs), liver, head or guts of the fish where the toxin accumulates.

What to do if you suspect you have ciguatera poisoning:

- Contact your doctor immediately – give full details of your illness including symptoms and food items eaten
- Where possible save any leftover fish for testing
- Contact a Health Protection Officer at Public Health Office.

## 7.2 Relevant Food Controls: Overseas

### 7.2.1 Australia

A range of control measures are in place in Australia, including legislation, Codes of Practice (COP) and commercial purchasing specifications.

Under the Queensland Fisheries Regulations 1995 it is prohibited to take or possess specified high ciguatera risk species from the Platypus Bay area of Fraser Island (http://www.legislation.qld.gov.au/LEGISLTN/CURRENT/F/FisherR95.pdf).

The industry Code of Practice for charter fishing tourism, produced by the Queensland Charter Vessels Association (QCVA) (http://www.qcva.com.au/pdf/ACVA_INDUSTRY_CODE_OF_PRACTICE.pdf). The Code contains a description of ciguatera and lists:

- Species prohibited to be taken under the Queensland Fisheries Act 1994;
- Recommendations from the Queensland Seafood Marketers Association (QSMA) on the upper size limits for certain fish species to minimize ciguatera risks; and
- Area where catch is restricted (Platypus Bay), including species prohibited and further recommendations from QSMA of species that they believe should be prohibited.

In addition to the guidelines from the Queensland Seafood Marketers Association, outlined above, the Sydney Fish Markets Pty Ltd issues a schedule of ciguatera high-risk areas and species size limits to define which consignments will be rejected on the basis of ciguatera risk, which includes:

- Prohibited species;
- Prohibited supply regions and associated species; and
- Maximum size limits for high-risk species from various Australian states and Pacific countries

http://www.sydneyfishmarket.com.au/

### 7.2.2 United States

While the US FDA is the national regulatory body for public protection and seafood regulation, FDA "operates an oversight compliance program for fishery products under which responsibility for the product's safety, wholesomeness, identity and economic integrity rests with the processor or importer, who must comply with regulations promulgated under the Federal Food, Drug and Cosmetic (FD&C) Act, as amended, and the Fair Packaging and Labeling Act (FPLA)" (http://www.cfsan.fda.gov/~lrd/sea-ovr.html).

The US FDA publish a document entitled "Fish and Fishery Products Hazards and Controls Guidance" (http://www.cfsan.fda.gov/~comm/haccp4.html). This Guide relates to FDA's final regulations (21 CFR 123) that require processors of fish and fishery products to develop and implement Hazard Analysis Critical Control Point (HACCP) systems for their operations.

With respect to ciguatera fish poisoning, the guide comments that:
"An established water classification system similar to the molluscan shellfish system is not in place for controlling CFP in fin fish. However, some states issue advisories regarding reefs that are known to be toxic. In areas where there is no such advisory system, fishermen and processors must depend on first-hand knowledge about the safety of the reefs from which they obtain fish".

The FDA Food Compliance Programme for Domestic Fish and Fishery Products (http://www.cfsan.fda.gov/~comm/cp03842.html) includes procedures for monitoring HACCP programmes and carrying out confirmatory testing. Although this document contains reference to 'biotoxins', it appears to be limited to shellfish biotoxins.

Regional measures are in place to reduce the risk of ciguatera fish poisoning. In Florida restrictions on the sale of certain fish species are in place, while a limited fish monitoring programme is reported to be in place in Hawaii (van Egmond *et al.*, 1992).

### 7.2.3 Europe

Council Directive 91/493/EEC that lays down the health conditions for the production and the placing on the market of fishery products, states in Article 5 that:
"The placing on the market of the following products shall be forbidden:

- poisonous fish of the following families: Tetraodontidae, Molidae, Diodontidae, Canthigasteridae,
- **fishery products containing biotoxins such as ciguatera toxins or muscle-paralysing toxins**."

Species to be covered and analytical methodology are not further specified in the Directive.

A review of worldwide marine toxin regulations reported that France monitors the algal species *Gambierdiscus toxicus* and *Ostreopsis lenticularis* (van Egmond *et al.*, 1992). However, no details of this monitoring were provided or could be subsequently located.

## 7.3 Economic Costs

No assessment of economic costs associated with ciguatera fish poisoning has been carried out for New Zealand.

In ciguateric regions, ciguatera fish poisoning has the potential to result in public health costs, due to disease occurrence, and economic costs, due to lost trade and lost productivity. However, no systematic analysis of costs associated with ciguatera was found.

It has been reported that French Polynesia suffers annual losses in the region of $US 1 million due to a ban on the sale of reef fish (Bagnis, 1992). Economic costs in the Caribbean are estimated to be higher, with costs of approximately $US10 million associated with loss of toxic fish and adverse publicity (De Sylva, 1994).

Public health costs associated with ciguatera fish poisoning in French Polynesia were estimated to be in excess of $US1 million per annum (Bagnis, 1992). Todd (1997) estimated the costs associated with ciguatera fish poisoning in Canada to be of the order of $CND 1.2 million, based on a per case cost of $CND 4,000.

# 8 CONCLUSIONS

## 8.1 Description of Risks to New Zealand Consumers

### 8.1.1 Risks associated with seafood products

Ciguatera fish poisoning is solely caused by the consumption of seafood contaminated with toxins produced by dinoflagellate microalgae, particularly *Gambierdiscus toxicus*. While there are isolated reports of ciguatera fish poisoning resulting from consumption of non-finfish species, the disease is usually caused by the consumption of large finfish species from circumtropical regions.

While there have been reports of potentially ciguatoxic dinoflagellate species in northern New Zealand waters, no cases of ciguatera fish poisoning have been associated with fish from these waters and, at least under New Zealand conditions, *Ostreopsis* and *Coolia* dinoflagellates appear to produce palytoxins, rather than ciguatoxins. All reported cases of ciguatera fish poisoning in New Zealand have been due to consumption of risk fish species imported from the Pacific Islands, principally Fiji, or consumed in the Pacific Islands. In all but one documented case of fish importation, the fish were imported by individuals for their own consumption.

In New Zealand approximately six cases per year come to the attention of the public health system. Internationally, it has been suggested that notified cases may only represent 2-20% of total cases. This would equate to an average number of annual cases in New Zealand of 30-300. As no diagnostic test for ciguatera is currently available and New Zealand physicians would generally be unaccustomed to diagnosing the disease, a considerable degree of under-reporting would be expected.

The risk of ciguatera fish poisoning amongst the general New Zealand population is likely to be very low and will mainly be associated with travel-acquired disease. The risk for the Pacific Island population will be considerably higher due to the practice of private importation of potentially ciguatoxic fish for personal consumption. There is currently little control on the private importation of seafood into New Zealand. With the information currently available, it is not possible to further categorise the level of risk within the Pacific Island community.

### 8.1.2 Risks associated with other foods

Ciguatera fish poisoning is not associated with any other foods.

### 8.1.3 Risk assessment options

Quantitative risk assessment would be hampered by a lack of information on the prevalence of ciguatoxicity in reef fish and scanty information on the frequency of importation and consumption of risk fish species.

## 8.2 Commentary on Risk Management Options

Currently risk management in New Zealand is by increasing awareness of the disease, to improve reporting and diagnosis rates.

Available information suggests that ciguatera fish poisoning in New Zealand could be substantially controlled by prohibition or stricter controls on personal importation of large fish from the Pacific Islands.

## 8.3 Data Gaps

The major data gaps identified in this document are:

- Frequency of importation and consumption of risk fish species; and
- Frequency of ciguatoxin contamination in risk fish species.

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## APPENDIX 1: CATEGORIES FOR RISK PROFILES

The assignment of a category for a food/hazard combination uses two criteria: incidence and severity.

*1. Incidence*

The incidence is an estimate of the proportion of the foodborne disease rate due to an individual hazard, that is transmitted by a single food or food group. The overall rate of foodborne disease caused by individual hazards can be derived from information in the published estimate of foodborne disease (Lake *et al.*, 2000). This estimate has been updated to reflect more recent notifications rates for the 12 months to June 2001, but still using 1996 census figures (3,681,546 population). Rates include estimates for unreported cases who do not present to a GP.

| Disease/organism | Food rate (/100,000 population) Calculated for 12 months to June 2001 | Food rate (/100,000 population) Calculated for 12 months to December 1998 |
|---|---|---|
| Campylobacteriosis | 1320 | 2047 |
| Listeriosis | 0.4 | 0.4 |
| VTEC/STEC | 1.9 | 1.4 |
| Salmonellosis | 176 | 230 |
| Yersiniosis | 38 | 62 |
| Shigellosis | 7 | 7 |
| NLV* | 478 | 478 |
| Toxins* | 414 | 414 |
| Typhoid* | 0.3 | 0.3 |
| Hepatitis A* | 0.4 | 0.4 |

* not recalculated.
VTEC = verocytotoxic or verotoxigenic *Escherichia coli* STEC = shiga toxin-producing *Escherichia coli*
NLV = Norwalk-like virus

These are **total** foodborne rates, so it is probably safe to assume that in most cases the rates associated with a particular food are likely to be an order of magnitude lower. For instance, a category of ">1000" would only be assigned if it was decided that all campylobacteriosis was due to a single food/food type. The following categories are proposed for the rates attributable to a single hazard/food (or food group) combination:

| Category | Rate range | Comments/examples |
|---|---|---|
| 1 | >100 | Significant contributor to foodborne campylobacteriosis<br>Major contributor to foodborne NLV |
| 2 | 10-100 | Major contributor to foodborne salmonellosis<br>Significant contributor to foodborne NLV |
| 3 | 1-10 | Major contributor to foodborne yersiniosis, shigellosis |
| 4 | <1 | Major contributor to foodborne listeriosis |

NLV = Norwalk-like virus

A further category, of "no evidence for foodborne disease in New Zealand" is desirable, but it was considered more appropriate to make this separate from the others. Also separate is another category, of "no information to determine level of foodborne disease in New Zealand".

The estimation of the proportion of the total foodborne disease rate contributed by a single food or food group will require information from a variety of sources including:

- Exposure estimates
- Results from epidemiological studies (case control risk factors)
- Overseas estimates

For illnesses where the rate is <1 per 100,000 the ability to assign a proportion is unlikely to be sensible. For such illnesses it may be more useful to consider a Risk Profile across the range of all high risk foods, rather than individual foods or food groups.

*2. Severity*

Severity is related to the probability of severe outcomes from infection with the hazard.

The outcomes of infectious intestinal disease are defined in the estimate of the incidence (Lake *et al.*, 2000) as:

- Death
- Hospitalised and long term illness (Guillain-Barré syndrome (GBS), reactive arthritis, haemolytic uraemic syndrome (HUS))
- Hospitalised and recover
- Visit a GP but not hospitalised
- Do not visit a GP

The first three categories of cases were classed as severe outcomes. Some hospitalisations will result from dehydration etc. caused by gastrointestinal disease. However, for infections with *Listeria* and STEC hospitalisation will result from more severe illness, even if recovery is achieved. The proportion of severe outcomes resulting from infection with the hazards can be estimated from the proportion of cases hospitalised and recover, hospitalised and long term illness, and deaths (Lake *et al.*, 2000).

| **Disease/organism** | **Percentage of outcomes involving death or long term illness from foodborne cases** |
|---|---|
| Campylobacteriosis | 0.3 |
| Listeriosis | 60.0 |
| VTEC/STEC | 10.4 |
| Salmonellosis | 1.0 |
| Yersiniosis | 0.4 |
| Shigellosis | 2.7 |
| NLV | Assumed to be <0.5% |
| Hepatitis A | 15.4 |
| Typhoid | 83.3 |
| Toxins | Assumed to be <0.5% |

VTEC = verocytotoxic or verotoxigenic *Escherichia coli* STEC = shiga toxin-producing *Escherichia coli*
NLV = Norwalk-like virus

Categories for the probability of severe outcomes are suggested as follows:

| Severity Category | Percentage of cases that experience severe outcomes | Examples |
|---|---|---|
| 1 | >5% | listeriosis, STEC, hepatitis A, typhoid |
| 2 | 0.5 – 5% | salmonellosis, shigellosis |
| 3 | <0.5% | campylobacteriosis, yersiniosis, NLV, toxins |

STEC = shiga toxin-producing *Escherichia coli* NLV = Norwalk-like virus

There are a number of hazards for which the incidence of foodborne disease is uncertain. These have been assigned to the above severity categories as follows:

**Severity category 1:**

Bacteria

*Clostridium botulinum*

Protozoa

*Toxoplasma*

**Severity category 3:**

Bacteria

*Aeromonas/Plesiomonas*
*Arcobacter*
*E. coli* (pathogenic, other than STEC)
*Pseudomonas*
*Streptococcus*
*Vibrio parahaemolyticus*

Viruses

Others (e.g. rotavirus)

Protozoa

*Giardia*
*Cryptosporidium*
*Cyclospora*
Others (e.g. *Entamoeba*)

**Proposed Category Matrix**

| Incidence | >100 | 10-100 | 1-10 | <1 |
|---|---|---|---|---|
| **Severity 1** | | | | |
| **Severity 2** | | | | |
| **Severity 3** | | | | |

Alternatives:

No evidence for foodborne disease in New Zealand

No information to determine level of foodborne disease in New Zealand