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Ciguatera fish poisoning

- Increasing due to reef destruction?

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Abstract

Ciguatera fish poisoning (CFP) can occur after ingestion of different reef fish that contain a toxin or several toxins known as ciguatoxins. The focus of this essay is to describe what CFP is and if there is a connection between environmental problems and the occurrence of CFP. Focus will be on the following questions: What is CFP? Is there any risk of being poisoned from CFP in Sweden? What environmental factors can increase the occurrence of ciguatera outbreaks? Are human activities responsible for ciguatera outbreaks?

The ciguatoxin origins from a benthic dinoflagellate called *Gambierdiscus toxicus*. CFP is a problem in the tropical regions of the world, and particularly in the small island countries around the pacific basin and the West Indies. Fish that cause the poisoning usually live in shallow waters close to coral reefs. The toxicity of the fish largely depends on where it lived and fed. Several species of fish have been associated with CFP. The moray eel is often regarded as the most poisonous species. Other species that often seem to cause the poisoning are species of snappers, groupers, barracudas and the narrow-barred Spanish mackerel.

The symptoms of CFP usually are combination of gastrointestinal symptoms, and neurological symptoms. There is no specific treatment so focus is on reducing the symptoms. CFP is rarely fatal but sometimes there are complications associated to cardiac problems. The symptoms can persist for a long time and can return after eating fish, chicken or drinking alcohol, several years after the poisoning. The severity of the symptoms seem to be dose dependent and the lowest toxic dose to humans is 0.05 µg of the toxin or 0.01 µg /kg bodyweight. The toxins remain stable after heating. Several different ciguatoxins (CTX) have been identified that are responsible for causing CFP. The most potent ciguatoxin is P-CTX-1, where P stands for Pacific.

CFP is not often reported to authorities and in Australia it is estimated that only 20% of the actual cases are reported. In 2001 the Swedish Poisons Information Centre reported that so far, to their knowledge, there had only been one single incident of CFP in Sweden where three people were poisoned during a vacation abroad.

It is not known what initiates blooms of *Gambierdiscus toxicus* that is responsible for the ciguatera outbreaks. Reef destruction seems to cause outbreaks of CFP. One of the reasons for this is that there are more substrates (consisting of dead corals) for the hosts (macroalgae) of the dinoflagellate to grow on. Many different types of reef destruction can increase the frequency of the ciguatera outbreaks. Some of these are land erosion and coral bleaching, a phenomenon strongly associated to El Niño and an increase in water temperature. In order to prevent CFP it is important to bear in mind the relationship between coral destruction and outbreaks of CFP. It is also important to refrain from eating large servings of the same fish.

Contents

1 Introduction

Ciguatera fish poisoning, or CFP, is a type of poisoning that can occur in humans after the ingestion of various types of reef fish that contains a toxin or several related toxins called ciguatoxins. It is a major health problem in the tropical regions of the world and can be mistaken with several other forms of marine poisoning. The major toxin is lipid-soluble but there may also be other, water-soluble toxins involved in this type of poisoning. The initial signs of poisoning are gastrointestinal symptoms such as diarrhoea and abdominal pain that usually are followed by neurological symptoms (Lewis and King, 1996).

Worldwide CFP is probably the most reported illness when it comes to seafood-poisonings. Still it is very under-reported, probably because the symptoms are not necessary severe and are easily confused with other seafood poisonings or diseases (Lehane and Lewis, 2000). The new technology and transportation techniques make it possible to transport marine organisms all over the world. But the knowledge about these exotic products is scarce in our parts of the world and it is therefore important to spread information to prevent possible food poisonings. In the Nordic countries there are only a few reports of CFP but there are people from Sweden who have been poisoned during their vacation to tropical parts of the world.

There are results indicating a relationship between the destruction of coral reefs and an increase in the frequency of CFP. This relation is not yet clearly understood, and the scientists are looking at several different factors that can affect the frequency of the outbreaks, such as water temperature, salinity and amounts of nutrients that are available. The purpose of this essay is to describe what CFP is and if there is a connection between environmental problems and the occurrence of CFP. Focus will be on the following questions: What is CFP? Is there any risk of being poisoned from ciguatoxins in Sweden? What environmental factors can increase the occurrence of ciguatera outbreaks? Are human activities responsible for ciguatera outbreaks?

2 The Project

This examination essay will be a part of a larger project initiated by Christer Andersson at the National Food Administration in Sweden. The project is funded by the Nordic council of ministers (Nordiska ministerrådet) and concerns natural toxins in seafood. Representatives from all of the Nordic countries are participating and contributing with their field of expertise. The completed project will be in the form of a dictionary concerning toxicological aspects of the consumption of marine organisms with regards to naturally occurring toxins. In this case the naturally occurring toxins mean those that the organism produces itself or substances produced naturally by another organism and then are accumulated into the organism via the intake of food. When completed the dictionary will be available in all the Nordic countries (Denmark, Finland, Iceland, Norway and Sweden).

3 Literature

The material used in this essay have been obtained via PubMed (http://www.ncbi.nlm.nih.gov/pubmed), Toxline (http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?TOXLINE), and FishBase (http://www.fishbase.org/home.htm). PubMed is a database service by the National Library of Medicine, USA. Toxline is the U.S. National Library of Medicine's collection of bibliographic information that concerns the biochemical, pharmacological, physiological and toxicological effects of drugs and other chemicals. FishBase is a large global online database of fish species that provides extensive species data, mostly on finfish.

4 Ciguatera Fish Poisoning

CFP is a form of food poisoning that can occur in humans after ingestion of fish that contains one or several poisons known as ciguatoxins.

4.1 History

CFP has been known to man for a long time, references of CFP go back as far as the T'ang Dynasty in China (618-907 AD) (Watters, 1995). Later, in 1606, sailors with the Spanish explorer Fernando de Quiros were reported to be affected by a fish called Pargos. Subsequently, many other reports of poisonings followed from the Caribbean region (Lewis and King, 1996). Captain Cook and four other crew-members got ill after eating a similar fish as the Pargos, caught in the waters of the New Hebrides. This was in July 1774 and was recorded in journals kept by two of the ship botanists. These two carefully took notes of the symptoms and at what time they started and ended. The only death that occurred on this occasion was a parakeet bird that had been fed the fish (Watters, 1995). The term ciguatera is believed to come from a marine turban shell called *Turbo Pica*, or cigua in Spanish (Lewis and King, 1996). This can be traced back to a Portuguese biologist, Don Antonio Parra, who noticed similar symptoms as from CFP, after ingestion of meat of cigua in 1771-1787 (Watters, 1995).

For several years the origin of the ciguatoxins was unknown. In 1958 it was suggested that the toxins are introduced into the food chain through herbivorous fish that feed on toxic microalgae. Five years later it was demonstrated that the toxins might pass through the food chain without apparent harm to the carrier. The actual compound responsible for the toxicity was isolated in 1967 and given the name ciguatoxin. Years later it was proposed that a precursor to the toxin come from a benthic dinoflagellate, initially identified as belonging to the genus *Diplopsalis*. The species was renamed in 1979 to *Gambierdiscus toxicus*, because it was first found on the surface of dead corals in the Gambier Islands. The molecular structures of the main Pacific Ocean ciguatoxin and the precursor toxin in *Gambierdiscus toxicus* were identified by Murata in 1989. (Lehane and Lewis, 2000)

The ciguatoxins are lipid-soluble polyether compounds consisting of 13-14 rings fused into a ladder-like structure. The toxins arise from the gambiertoxins and they increase in polarity and toxicity as they undergo oxidative metabolism and pass up the food chain of herbivorous and carnivorous fishes. The toxins are stable and remain toxic after cooking.

4.2 Occurrence of ciguatera fish poisoning

CFP is a problem originally occurring in tropical and subtropical Pacific and Indian Ocean regions, and in the tropical Caribbean (Lehane and Lewis, 2000). Fishes that cause CFP are those that inhabit warm seas around the world, particularly in the vicinity of coral reefs. (Lewis and King, 1996; Lehane and Lewis, 2000)

As reef fishes are becoming a food product widely exported all over the world, CFP is no longer a problem only regarding the tropical regions, but a potential problem all over the world. The toxicity of ciguateric fish depends to a large extent on the area where the fish lived and fed. Fishes that become toxic are usually bottom dwellers, but on occasion also pelagic fishes may be toxic. The toxicity is derived from compounds in the ingested algae. Most reef fishes are stationary. Therefore one reef fish can be toxic, whereas another one living nearby is not, due to the presence or absence of toxic microorganisms. Most reef-fishes in French Polynesia contain low concentrations of the toxins in their meat, but it is possible that the levels can increase to high-risk concentrations, within a short period of time. (Lehane and Lewis, 2000)

The geographic areas where CFP is a particularly serious problem are the small island nations of the pacific basin and the West Indies illustrated in Figure 1 (Lewis and King, 1996). But CFP is a global problem when consuming fish caught between latitudes 35° North and 35° South. This region includes the areas around the Caribbean, and the Indian and Pacific oceans. However, CFP is not common all over these areas. It is almost unknown e.g. in the Maldives, the Seychelles, Guam, the Solomon Islands, Wallis and Futuna (html 1). The distribution may be very local as illustrated by the example from the republic of Kiribati, an island nation located in the central Pacific Ocean, where two reefs have a high risk of CFP but the remaining reefs in this atoll are low risk areas (Lewis, 2000). In Australia the occurrence of CFP is predominant in the Northern Territory and Queensland (Lewis and King, 1996).

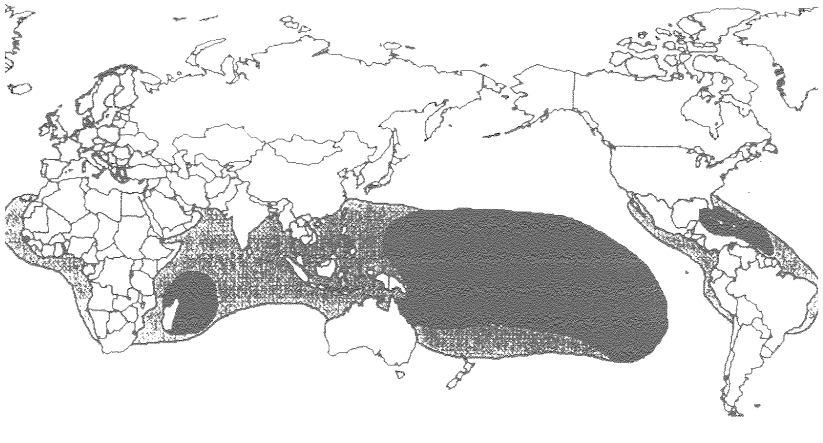

Figure 1 Map of the world where the high-to moderate-risk areas for CFP are heavily shaded and those of low or uncertain risk are shaded (From Lehane and Lewis 2000).

4.3 Species associated with ciguatera fish poisoning

It is not known how many species that can be ciguatoxic, the numbers given in the literature varies from 10 to over 400 (Lehane and Lewis, 2000). The moray eel (*Gymnothorax javanicus)* is generally regarded as the most poisonous fish when it comes to CFP.

In Australia, several different species of fishes have been known to cause CFP. These include the narrow-barred Spanish mackerel (*Scomberomorus commersoni*), reef cod (*Epinephelus tauvina*), barracuda (*Sphyraena barracuda*), grouper (*Epinephelus lanciolatus*), queenfish (*Scomberoidescommersonnianus*), snappers (*Lutjanus synagris*, illustrated in Figure 2) and kingfish (*Seriola hippos*) (Lewis and King, 1996). The moray eel (*Gymnothorax javanicus*), red bass (*Lutjanus bohar*), chinamanfish (*Symphorusnematophorus*), and paddeltail (*Lutjanus gibbus*) are reported to carry ciguatoxin, and are therefore not sold in Queensland, Australia (Lewis and King, 1996). In the Platypus-bay region, Australia, there is a ban on the capture of narrow-barred Spanish mackerel (*Scomberomorus commersoni*) and barracuda (*Sphyraena barracuda*) (Lewis and King, 1996; Lehane and Lewis, 2000). These species are frequently toxic and this area is the only true "hot spot" for ciguatera in Queensland. In this bay a small fish called blotched javelin fish (*Pomadasys maculatus*) has been known to cause the poisoning and should also be avoided (Lewis and King, 1996).

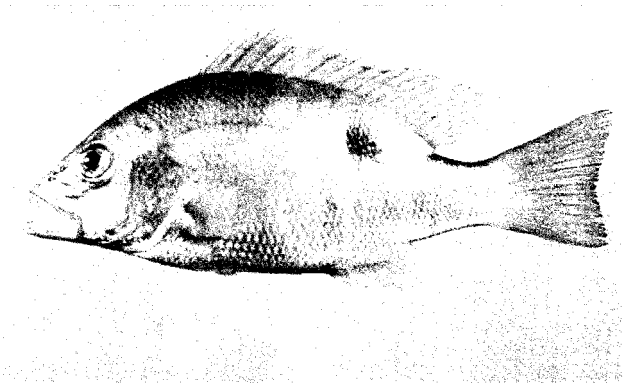

Figure 2 Lane snapper, *Lutjanus synagris*, which is known to cause CFP. (Photo by Flesher, D, Html 2)

Table 1 shows a list of species that are known to be ciguatoxic on occasion. This list is not complete. Many of these fishes are known under different names. The Swedish names are those that should be used according to the Swedish National food administration (SLVFS 2001:37). For more detailed information on the species, such as size, habitat and alternative names, see Appendix.

Table 1 List of fish species that have been reported to cause CFP.

Scientific name	English name	Swedish name
Achantocybium solandri	Wahoo	Wahoo
Apsilus Dentatus	Black Snapper	Svart Snapper
Caranx latus	Horse-eye Jack	
Caranx sexfasciatus	Jack, Bigeye trevally	
Chelinus trilobatus	Maori wrasse	
Ctenochaetus striatus	Surgeonfish,	
Ephinephelus guttatus	Red hind	Röd prickig grouper
Ephinephelus striatus	Nassau grouper	Nassaugrouper
Epinephelus fuscoguttatus	Brown-marbled grouper	
Epinephelus lanciolatus	Giant Grouper	
Euthynnus affinis	Kawakawa	Kawakawa
Euthynnus alletteratus	Little tunny	Tunnina
Gymnothorax javanicus	Giant Moray	
Lutjanus analis	Mutton snapper	Fläcksnapper
Lutjanus bohar	Two spot red snapper	
Lutjanus buccanella	Blackfin snapper	Svartfenad snapper
Lutjanus campechanus	Northern red snapper	Röd snapper
Lutjanus gibbus	Humpback red Snapper	
Lutjanus griseus	Grey snapper	Grå snapper
Lutjanus monostigma	Onespot Snapper	
Lutjanus sebae	Emperor red Snapper	Kejsarsnapper
Lutjanus synagris	Lane snapper	Randig snapper
Lutjanus vivanus	Silk snapper	Sidensnapper
Mycteroperca bonaci	Black grouper	Svart grouper
Ocyurus chrysurus	Yellowtail snapper	Gulstjärtsnapper
Pomdasys maculatus	Saddle Grunt	Fläckgrymta
Scarus gibbus	Parrotfish	
Scomberoides commersonnianus	Queenfish	
Scomberomorus commerson	Narrow-barred Spanish mackerel	
Seriola aureovittata	Amberjack	
Seriola lalandi	Yellowtail amberjack	
Sphyraena barracuda	Great Barracuda	
Sphyraena jello	Pickhandle Barracuda	
Symphorus nematophorus	Chinamanfish	

4.4 Toxins found in ciguateric fish

The major toxins that are found in the ciguateric fish are a group of toxins called ciguatoxins, and they are the primary reason behind CFP. The involvement of other toxins has been suggested but is not yet fully understood (Lehane and Lewis, 2000). The ciguatoxins are a family of lipid-soluble, highly oxygenated cyclic polyether molecules consisting of 13-14 rings fused by ether links into a ladder-like structure.(Lehane and Lewis, 2000) They are stable molecules and remain toxic after heating to 100° C (Guzmán-Pérez and Park, 2000).

Recent studies have shown that the different variants of the toxin differ in polarity. They all seem to origin from a toxin known as gambiertoxin that is produced by the dinoflagellate *Gambierdiscus toxicus* (Lehane and Lewis, 2000). These toxins have been isolated from several sources, biodetritus containing wild *G. toxicus*, from toxic strains of cultured dinoflagellate isolated from different parts of the world, and from ciguateric fish (Lewis *et.al* 2000). The molecular structure of the toxins responsible for CFP has recently been established. They seem to arise from the metabolism of gambiertoxin, CTX-4A (GTX), in fish. The bioconversion of gambiertoxin to ciguatoxin in fish, results in an increase in toxicity up to ten times (Lewis and King, 1996).

The main ciguatoxins are called CTX- 1, -2 and -3. They have different effects in vivo and are present in fish in different relative amounts. Lewis and Sellin (1992) came to the conclusion that CTX-1, -2 and -3 are the major ciguatoxins present in the meat of fish. It has also been suggested that there are three chemically distinct classes of ciguatoxins relating to where the fish has been captured. It would therefore be a difference between the toxins found in the Caribbean Sea, Pacific Ocean ant the Indian Ocean (Lewis *et al.,* 2000; Vernoux and Lewis 1997). The main Pacific ciguatoxin is called P-CTX-1 where the P stands for Pacific. Pacific ciguatoxin is probably the most potent of the ciguatoxins (Lehane and Lewis, 2000). By using nuclear magnetic resonance (NMR) techniques the chemical structures of several Pacific ciguatoxins have been established (Lewis *et al.,* 2000). Also the structures of (Caribbean) C-CTX-1 and C-CTX-2 have been elucidated (Vernoux and Lewis 1997). In some articles the origin (Caribbean Sea or Pacific Ocean) of the toxins are not specified. In those cases I will only present the toxin as CTX-1, -2 or -3.

P-CTX-1 arises from acid-catalysed oxidative modifications of CTX-4A (Lewis *et al.,* 2000). Chromatographic properties of CTX-4A, the stereoisomer of CTX-4B (gambiertoxin), agree well with those of a toxin previously detected in Parrotfish, called scaritoxin. Therefore the name CTX-4A should be used instead of scaritoxin (Lehane and Lewis 2000).

P-CTX-1 is the major toxin present in carnivorous ciguateric fish, on the basis of both quantity and total toxicity. It is one of the most potent sodium channel toxins known. (Lewis *et al.,* 2000) In 1991 three major toxins from moray eel viscera were isolated and characterised. The structures for these toxins are shown in Figure 3.

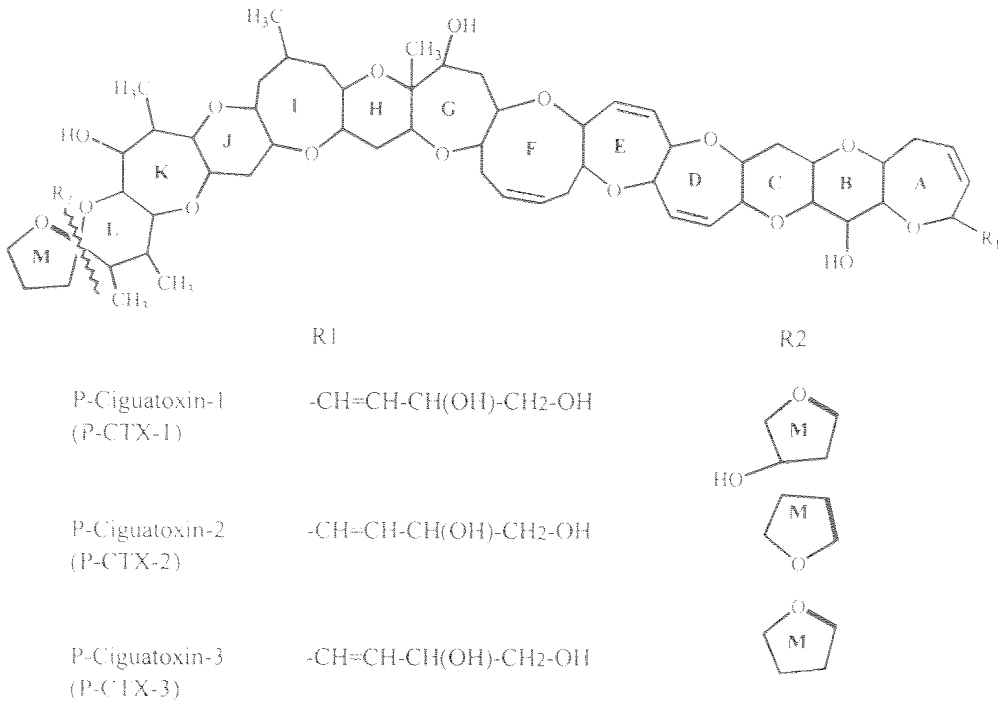

Figure 3 Structures for Pacific ciguatoxin-1,-2, and –3 (From Guzmán-Pérez and Park 2000).

The mechanism for the oxidation of gambiertoxin to ciguatoxins is not known in detail, but might be catalysed by cytochrome enzymes in the liver of fish. The biotransformation is shown in Figure 4. CTX-3 is apparently an intermediate in the biotransformation of CTX-4B to CTX-1. CTX-2 seems to originate from another toxin than CTX-4B, possibly CTX-4A (Lehane and Lewis, 2000). CTX-4A and -4B are sometimes referred to as GTX-4A and -4B as they are gambiertoxins.

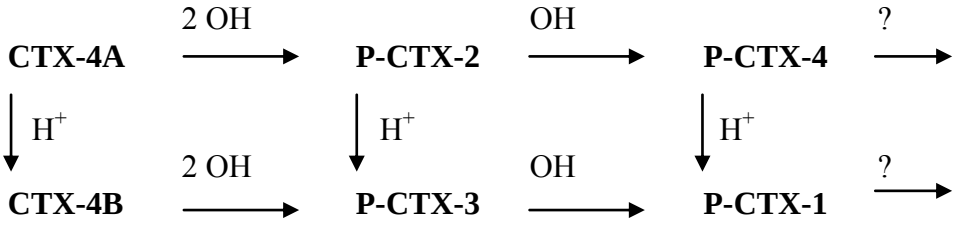

Figure 4 The biotransformation from gambiertoxins (CTX-4A and CTX 4-B) to different ciguatoxins through oxidation (After Guzmán-Pérez and Park, 2000).

It seems that the polarity of the toxins increases as they undergo oxidative metabolism. That means that the toxins become more potent as they pass up the food chain, from the dinoflagellate, through herbivorous and carnivorous fish and finally reaching humans as they are the top predators of this food chain So far, several different types of gambier- and ciguatoxins have been identified, nine from *G. toxicus* and the others from fishes (Lehane and Lewis, 2000). Some of them and their origin are presented in Table 2.

Table 2 Names, origin and potency of different ciguatoxins (both Pacific and Caribbean) involved in CFP. Potency of extracts of toxins after intraperitoneal injection in mice (μg/kg) (From Lewis *et al.,* 2000).

Name	**Origin**	**Potency* (μg/kg)**
P-CTX-1	Carnivore	0.25
P-CTX-2	Carnivore	2.3
P-CTX-3	Carnivore	0.9
P-CTX-3C	*G. toxicus*	2.0
2,3-dihydroxy P-CTX-3C	Carnivore	1.8
51-hydroxy P-CTX-3C	Carnivore	0.27
P-CTX-4A	*G. toxicus* / Herbivore	2.0
P-CTX-4B	*G. toxicus* / Herbivore	4.0
C-CTX-1	Carnivore	3.6
C-CTX-2	Carnivore	1.0

* Intraperitoneal LD_{50} dose in mice.

Regarding the difference between the toxins found in the Caribbean Sea, Pacific Ocean and the Indian Ocean, epidemiological studies indicate that gastrointestinal symptoms are more predominant and neurological not so dominant in the Caribbean cases as compared to Pacific cases of CFP. Fatalities seem to be more common after consumption of fish from the Indian Ocean than from the Pacific Ocean. Vernoux and Lewis (1997) have confirmed that Caribbean ciguatoxins indeed differ from Pacific ciguatoxins and that the Caribbean version, C-CTX-1, is less polar than the Pacific version P-CTX-1.

The main ciguatoxins CTX-1, CTX-2 and CTX-3 have different effects *in vivo* and are present in the fish in different amounts. This is one of the reasons for the variations in symptoms between different cases of CFP. The difference in ciguatoxin profile is generated as the toxin passes through the food chain. It is not yet determined how much the ciguatoxins vary between and within fish species. These variations may depend on the ratios of the different ciguatoxins and their precursors in the diet of the fish, and differences in the ability of fish to absorb, metabolise and excrete the toxins. CFP may also result after ingestion of less potent gambiertoxins and less oxidised ciguatoxins in herbivorous fish. Factors determining the concentration of toxins present in a fish are the rate of dietary intake, efficiency of absorption, the ability to metabolise the toxin, the rate of excretion and the growth of the fish. (Lehane and Lewis, 2000)

Fish convert the ingested gambiertoxin into ciguatoxin that accumulate in their meat. Large carnivorous fish prey on the smaller ones and the toxins are concentrated. Humans are the final link in this bio-chain. (Lewis and King, 1996)

Toxic fish have normal smell, taste and appearance but it is believed that higher levels of toxins may cause morphological and behavioural changes in the fish. One single fish can contain sufficient ciguatoxin in the meat to poison several humans, without showing any pathological signs. If the dose is too high it can even be lethal to the fish. This indicates that there is an upper level of how much toxin a fish can carry, which could be a reason why there are so few reported deaths among humans from CFP. (Lehane and Lewis, 2000)

4.5 Symptoms of ciguatera fish poisoning

CFP is likely to be underreported since it is easily confused with other types of poisoning. The diagnosis for ciguatera poisoning is clinical and it is important to exclude other possible reasons for being poisoned, such as exposure to Botulinus toxins, or toxins in shellfish or scombroid fishes.

The symptoms of CFP may begin rapidly, within less than one hour after ingestion of ciguatoxic fish, or may occur as late as 24 hours after consumption of the fish. All patients exhibit symptoms within 36 hours. The time to onset varies with the toxicity of the fish, the amounts that are consumed and the susceptibility of the consumer. CFP can be classified clinically into four different groups of symptoms:

- combined gastrointestinal/neurological (with the former symptoms predominating)
- combined gastrointestinal/neurological (with the latter symptoms predominating)
- purely neurological symptoms
- purely gastrointestinal symptoms

It is most common to observe a combination of gastrointestinal and neurological symptoms in a ciguatera patient. (Lewis and King, 1996)

In Australia the symptoms of CFP have been well studieda. Table 3 shows the most common gastrointestinal, neurological and non-specific symptoms observed in 360 cases. Cardiovascular complications are not so common in Australia but are documented from other parts of the world. In severe cases there is usually a combination of several of the symptoms named above. Gastrointestinal symptoms last for a few days, but the neurological symptoms may persist for several weeks or in some cases for several years. (Lewis and King, 1996)

Table 3 Symptoms of CFP and their frequency among patients (After Lewis and King, 1996).

Gastrointestinal symptoms	**Frequency (%)**
Diarrhoea	60
Abdominal pain, nausea	50
Vomiting	30
Neurological symptoms	**Frequency (%)**
Myalgia (muscle pain)	80
Burning sensation on skin upon contact with cold water, puritis (itching), arthralgia (joint aching)	70
Paraestesia (tingling and numbness) of hands, feet, mouth and lips, headache	60
Mood disorders (depression, irritability, anxiety)	50
Ataxia (unsteadiness) or vertigo (sensation of spinning), sweating, eye pain	40
Dental pain, tremor	30
Neck stiffness, demonstrable paresis (decreased power in specific muscle groups)	20
Salivation	10
Non-specific symptoms	**Frequency (%)**
Fatigue and lassitude	90
Chills	40
Skin rash, shortness of breath, pain on urination	20

4.5.1 Duration of symptoms and excretion rate of ciguatoxin

The excretion of ciguatoxins from the fish is slow. A study on moray eel indicated a half-life of 264 days (Lehane and Lewis, 2000). Maybe the excretion of the toxin is slow also in humans? A 40-year male experienced symptoms of CFP after eating the heads of several reef fishes. The symptoms persisted for about one month. Two years after he had suffered from CFP he drank half a pint of beer for the first time since the attack. The following evening he experienced a relapse of neurological symptoms and arthralgia. These symptoms disappeared after a week. According to Chan (1998) this relapse could indicate that he still had traces of the toxins in his body. Since the first attack he had also noticed an easily induced paraesthesia, for example when he sat with his arms resting on the arm support of a chair or sitting with his legs crossed. This observation fits well with other reports that symptoms of CFP can reappear as a kind of hypersensitivity when the patient ingests non-toxic fish, such as cold-water species. Even pork or chicken may on occasion bring back the symptoms of CFP. Therefore it is recommended that people refrain from eating fish (including shellfish) and drinking alcohol, for three to six months after CFP, in order to prevent the symptoms from reappearing (Lewis and King, 1996). Patients with pervious episodes of poisoning may experience increasingly severe and prolonged symptoms after further exposure to the toxins involved in CFP (Chan, 1998).

4.6 Pathological findings in experimental animals and man

Since CFP rarely is fatal to human, there is only limited information on post-mortem examinations from victims of the poisoning. In the period of 1964-1977 a large number of patients (3009) suffering from CFP were studied. In this study three cases were fatal, a case fatality ratio of 0.1 % (Bagnis *et al.,* 1979). However in severe outbreaks the mortality rate has been as high as 20%. This was the case on the east coast of Madagascar where 98 out of 500 persons died after eating a shark (Lehane and Lewis 2000). These examinations can give no clue to the full picture of the toxicity.

Terao (2000) refers to studies that have been made and concludes that available information shows evidence of acute visceral congestion (stocking of blood in intestines) but no discernible histological changes. However, there have been reports of histological changes in the liver, as well as swelling of the Schwann cell cytoplasm and changes in the sural nerve (backside of leg below knee, calf).

To improve the pathological picture derived from human post-mortem autopsies, studies have been performed both on mice exposed to a single high dose of ciguatoxin, and on mice repeatedly exposed to low doses of the toxin.

4.6.1 Single dose administration of ciguatoxin

Terao (2000) refers to a study performed by Terao *et al* (1991). In a single dose experiment, mice were given 700 ng of ciguatoxin per kilogram bodyweight, corresponding to 5 mouse units (MU). A watery stool occurred 15 minutes after the administration, probably as a result of accelerated mucus secretion in the colon. The diarrhoea was followed by loss of activity, lacrimation, and excessive salivation that continued for about 90 minutes. After several hours, during which the mice seemed to recover from the illness, suddenly severe dyspnea (breathing difficulties) and cyanosis appeared and about 70% of the mice died within 24 hours. Those mice that survived showed paralysis of the paws, spasms and penis erection accompanied by a markedly dilated and filled urinary bladder. (Terao, 2000)

On macroscopic examination, intoxicated mice at the state of dyspnea or death showed signs of a dilated heart and oedema of the lungs. Marked congestion of all other organs was also noted. Light microscopy showed that there were many single cell necroses in the cardiac muscle tissue, both in left and right ventricles. There was also a marked congestion of right ventricle and coronary veins. Electron microscopy revealed many swollen myocardial cells. Mitochondria became rounded and most organelles were separated from each other. Endothelial cells of the blood capillaries seemed to be more sensitive to the toxin than other cells in the heart. Occasionally an aggregation of platelets was seen in the lumen of many blood capillaries. Swollen erythrocytes were seen in the cardiac tissue. Mice with severe dyspnea showed marked oedema of the lungs. Histologically, alveolar spaces and bronchioles were filled with plasma-like effusion and prominent congestion. Marked swelling of nerve fibres and disappearance of synaptic vesicles was prominent. Ciguatoxin accelerated mucus secretion in the colon, resulting in prominent diarrhoea. There were many thrombus formations in the veins of various organs, such as the heart, liver and penis. (Terao, 2000)

4.6.2 Repeated administration of ciguatoxin

CFP produces no immunity. On the contrary the second attack often seems to be more severe than the first (Bagnis *et al,* 1979). Recurring or multiple attacks of CFP results in a clinically more severe illness compared to those patience that are being exposed to the ciguatoxins for the first time (Terao, 2000).

Repeated intraperitoneal injections of low doses of ciguatoxin (100 ng/kg body weight) to ICR mice for fifteen days support this theory and the effects of the ciguatoxin were manifested during later administrations. One single dose did not cause any discernible changes in the hearts of the mice. Dissection directly after the administration of 15 doses revealed marked dilatation of both ventricles of the heart. The changes were similar to those found in mice who had received a single dose of 700 ng/kg. In the case of the repeated administration the endothelial cells of blood capillaries in the heart were attacked more severely than other components of the heart. The myocardial cells were swollen and mitochondria were rounded.

Some negative effects such as hypertrophy of cardiac muscles still persisted one month after the end of the exposure. This experiment indicates that ciguatoxin has a cumulative effect on the cardiac tissue and although some changes were reversible, some were not. (Terao, 2000)

4.7 Treatment of ciguatera fish poisoning

Most attempts to find a specific therapy for CFP that is efficient in all cases have failed or the therapies are not effective enough (Terao, 2000). Therefore, the treatment of CFP is mostly symptomatic, especially to prevent dehydration that might occur after excessive vomiting and diarrhoea (Lewis and King, 1996). Other methods that are used include local anaesthetics and antidepressants (Lewis, 2000). It is known that intravenous infusion of D-mannitol can reduce the severity and duration of the symptoms, especially in patients suffering from acute poisoning after eating Pacific or Caribbean fish (Lewis and King, 1996; Lewis *et.al*. 2000). Mannitol is a crystalline alcohol obtained from glucose. The structure of mannitol is shown in Figure 6.

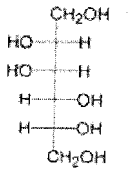

Figure 6 The chemical structure of D-Mannitol, a substance that is used for treating CFP (From: Html 3).

The treatment involves a 1g/kg intravenous infusion of 10 % or 20% mannitol solution over 30 minutes, given as soon as possible after CFP has been diagnosed. Some patients report a dramatic improvement of the neurological symptoms but some do not respond at all to the treatment. The reasons for these differences are unknown and require further studying but can be related to how long after the meal the mannitol was administered or to the size of the given dose. In non-responding patients a second infusion is recommended (Lewis and King, 1996). Mannitol does not remove the ciguatoxin molecule from its site of binding, but it reduces swelling of the Schwann-cells, which is induced by the ciguatoxin (Lewis *et.al.* 2000). Australian studies show that the diuresis that normally follows an infusion of mannitol, does not appear when it is used to treat ciguatera. The mannitol infusion should be administered as soon as possible but not until the patient is adequately hydrated (Lewis and King, 1996).

4.8 Prevention

When eating tropical fish from the affected regions, there is no certain way to avoid CFP since there is no simple way to detect the toxin. The toxic fish is normal in taste and appearance and is therefore impossible to detect (Lehane and Lewis, 2000). Still there are a few precautions that can be taken to reduce the risk. It is usually safer to eat many small servings (< 50 g) from several different fishes than to eat one large portion (>200g) from a single fish. The concentration of ciguatoxin in liver, viscera and roe is usually high and consumption of these parts should therefore be avoided (Lewis and King, 1996). Since the toxin accumulates along the food chain, large fishes and carnivorous species are usually more toxic than small fishes and herbivorous species (Ng and Gregory, 2000).

4.9 Methods to identify and quantify ciguatoxins

As mentioned before there is no easy way to test if a fish contains ciguatoxins. Over the years several methods have been developed but none have resulted in an easy user-friendly test that is also high in accuracy. People of the South Pacific have developed “traditional” ways of testing fish such as discolouration of silver coins that have been in contact with toxic fish, or if the fish repulses flies or ants. However all of these tests have been found to be completely invalid (Hashimoto, 1979).

The so called mouse bio assay is still the most commonly used assay to detect levels of ciguatoxins in extracts of fish. Mice are given an intraperitoneal injection of a lipid fraction of fish extract. The mice are observed for signs of intoxication such as hypothermia, hypersalivation, hind-limp paralysis and respiratory difficulties. This method has the disadvantage of not being sufficiently sensitive to detect small amounts of toxins. There is also an ethical concern when using mammalian assays in simple screening tests. (Lewis *et al.,* 2000)

The simplest assay that has been developed for detecting ciguatoxins is the fly assay. The test uses the larva of the meat-eating fly, *Parasarcophaga agryrostoma* which was chosen for its

ability to consume large quantities of proteins. Ten larvae are placed on 5 g of meat and the larvae are observed for 3-24 h. Samples that contain > 1 ng of CTX/ g of meat, kill the larvae in about 3 h. Samples containing lower concentrations, as low as 0,2 ng CTX/g, inhibit larval growth which can be noted with the naked eye. When the larval growth is determined by weighing concentrations as low as 0,1– 0,15 ng CTX/g can be detected. (Labrousse and Matile, 1996)

Attempts have been made to develop quick and easy methods for detecting ciguatoxins. Many have tried to use the possibility to bind specific antibodies to the toxin. Often the antibody is labelled so that it will be possible to detect it. The labels can for example be a radioisotope or an enzyme. This requires a high affinity antibody that is specific for the targeted compound. Some of the first immunoassays that were developed used a polyclonal antibody raised against ciguatoxin in sheep. The sheep antibody was purified and coupled to ^{125}I. Although the developed radioimmunoassay (RIA) was effective its complexity, cost and the antibody's tendency to cross react with other toxins, made it unsuitable for ciguatoxin screening. In 1984 the RIA was replaced by an enzyme linked immunosorbent assay (ELISA). This method was cheaper than RIA but still cross reactions occurred with other toxins resulting in inaccuracies, so the method was abandoned. (Lehane and Lewis, 2000)

Several attempts have been made to develop simple "stick tests" to detect ciguatera in fish. In the middle of the 1980's enzyme-labelled polyclonal antibody was used for this purpose. Unfortunately this test showed false positives but there were never any reports of the test showing false negatives. Still it was an inconvenient method since it took six tests per fish to determine the toxicity of those fish that contained low levels of toxin. Since 1990 monoclonal antibody to ciguatoxin (MAb-CTX), have been used. They have the advantage of being more specific, and more readily available than polyclonal antibody. (Lehane and Lewis 2000)

In 1998 a newly refined membrane immunobead assay, MIA, was developed (Hokama *et al.* 1998). It uses a MAb against purified moray eel ciguatoxin attached to coloured beads and a hydrophobic membrane laminated onto a plastic support. The toxin binds to the hydrophobic membrane and the specific monoclonal antibody to ciguatoxin (MAb-CTX) that is coated onto the beads will bind to the toxin, as shown in figure 7. If the test is positive there will be a visible change in colour and if it is negative the colour will not change. The intensity of the colour is proportional to the concentration of CTX attached to the membrane

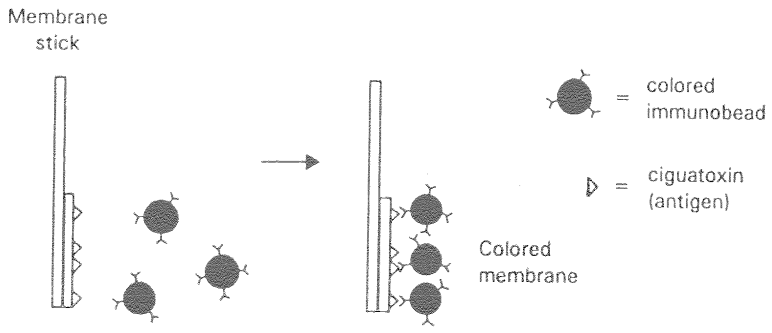

Figure 7 The principles for the membrane immunobead assay, MIA, where blue latex immunobeads bind to the membrane. If the test is positive the stick will turn blue (From Hokama *et al.,* 1998).

The test procedure is simple. A fish tissue sample (ca 5 mg) is placed in a test tube along with the membrane stick and 0.5 mL methanol. The stick is left in the test tube for 20 minutes. The

stick is removed and thoroughly air-dried for no less than 20 minutes. The dry stick is placed in 0.5 mL latex immunobead suspension for ten minutes. After that the stick is rinsed in saline water and once again left to air dry. Now the test is completed and if the test is positive a visible colour change will appear as shown in Figure 8. It is important never to touch the membrane part of the stick as this can cause false positive reactions (Hokama *et al*., 1998).

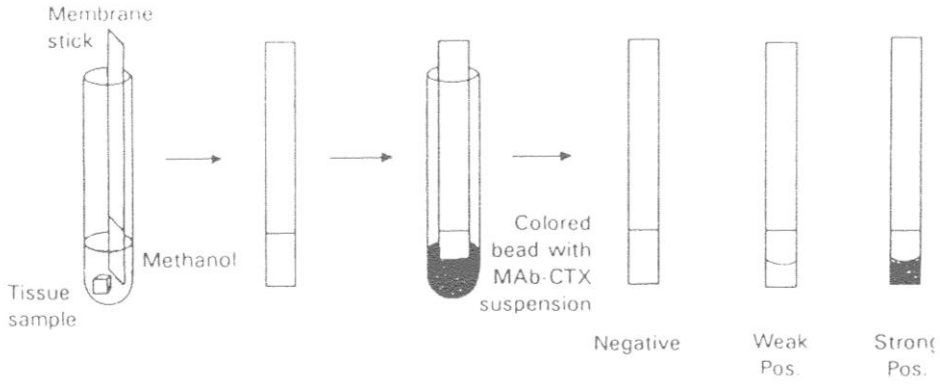

Figure 8 The membrane stick test for testing fish for ciguatoxin. A tissue sample is put in methanol along with the membrane stick. The stick is then air -dried and put into a suspension that contains coloured beads that bind to ciguatoxin. When the stick has been rinsed and air-dried the results will be shown as a visible change in colour on the stick (From Hokama *et al.,* 1998).

Another test, the Cigua-Check, is now available and claims to be more sensitive then any other test on the market, being able to detect levels of ciguatoxin at concentrations that cause clinical symptoms in human (≥0.08 μg /kg fish) (Html 4). Still fish that is considered non-toxic by the Cigua-Check may contain small amounts of ciguatoxins that could contribute to cumulative effects. A general problem with immunoassays is that they detect ciguatoxins based on structure. Therefore non-toxic substances may cross-react with antibodies against P-CTX-1 and therefore give rise to false positive results. (Lehane and Lewis, 2000)

Cell-based assays detect the ciguatoxins based on toxicity rather than on structure. Therefore these assays are more likely than the immunoassays to detect mixtures of different toxins that are associated with CFP (Lehane and Lewis, 2000). A cell-based assay for ciguatoxins based on cultured mouse neuroblastoma cells has been proposed. It reduces a substrate in the culture medium to a blue-coloured product. Samples of fish containing ciguatoxins affect the cells in such a way that the product does not change colour. The test is able to detect concentrations far lower than the mouse bioassay. (Lehane and Lewis, 2000)

A drawback when it comes to the development of chemical analytical techniques of ciguatoxins is that the compounds have no useful chromophore for selective spectroscopic detection. On the other hand, they contain a rather reactive primary hydroxyl group to which a label may be attached. Detectors such as fluorescence coupled to optimised HPLC have been used for chemical analysis of ciguatoxins in crude extracts from different fish (Lewis and Sellin, 1992). The analysis is time consuming but very valuable when confirming results obtained in rapid screening assays (Lehane and Lewis, 2000).

4.10 Concentrations in fish and toxic doses

Most cases of CFP involve consumption of fish that contain 0.1-5 μg P-CTX-1/kg. The severity and duration of the intoxication appears to be dose-dependant and there are also indications that the toxins have a potential to accumulate in the human body. Another

complication is the possibility that several different toxins (not only different types of ciguatoxins) contribute to the symptoms. (Lehane and Lewis, 2000)

Mild cases of CFP have been associated with consumption of fish that contains no more than 0.1 μg/kg P-CTX-1. The lowest dose of P-CTX-1 that could be expected to be toxic in adults is about 0.05 μg of the toxin or 0.001 μg /kg bodyweight, corresponding to a consumption of 500 g of fish containing 0.1 μg/kg CTX-1 by a 50 kg individual. This dose will give symptoms of intoxication in two out of ten individuals. A ten times higher dose can be expected to be toxic to most humans and is easily ingested from a normal serving of 300 g of fish that contain ciguatoxins of levels that are commonly seen in fish. (Lehane, 2000; Lehane and Lewis, 2000) In one outbreak in Australia a family suffered from symptoms that seemed to be dose dependent both in severity and duration. The presence of ciguatoxin in the fish was confirmed by the mouse bioassay. The father who, reportedly, ate about one kg of the fish suffered from the most severe and long-lasting symptoms. The affected children consumed only small pieces of the fish and suffered only from minor gastrointestinal problems. The mother who consumed about 0.5 kg of the fish experienced severe gastrointestinal problems and some mild neurological problems. The concentration of P-CTX-1 in the fish was calculated to 1.3 μg/kg muscle or 0.25 MU/g muscle. MU is short for mouse units and is the LD_{50} dose for a 20-g mouse, which is equivalent to 5 ng P-CTX-1. This is a relatively high level and it is consistent with the rather severe poisoning seen in the adults. The total P-CTX-1 exposure of the man and the woman was 1.3 μg and 0.65 μg respectively. (Lehane and Lewis, 2000)

4.11 Mechanism of action

In the 1970 it was established that the mechanism of action of the ciguatoxins is related to its effect on voltage-sensitive sodium-ion channels (VSSCs). Such channels are present in most excitable cells and also in some non-excitable cells such as glial cells (Lewis *et al.*, 2000). The sodium channels are composed of large proteins and are important for propagation of action potentials. Therefore they are vital to the function of nerves and muscle (Lehane and Lewis 2000). The sodium channel consists of about 2000 amino acids, which form a pore in the plasma membrane that allows passive but selective movement of the Na^+ ion down the electrochemical gradient. These amino acids are organised in four repeated homologous domains (I-IV) each containing six α-helical segments, known as S1-S6, shown in Figure 9, which clump together and form the pore. Gating systems that control the closing and opening of the pores depend both on time and membrane potential. (Lewis *et al.*, 2000)

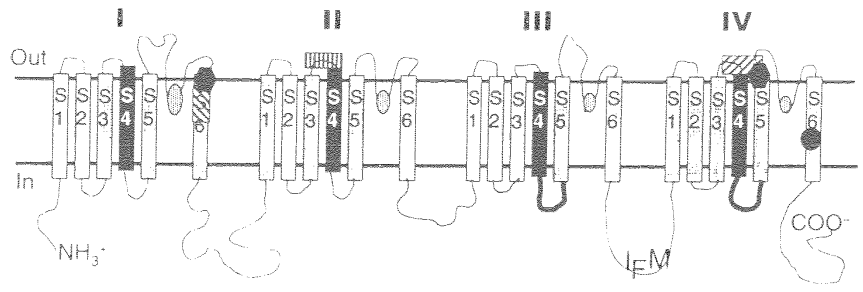

Figure 9 An illustration of a sodium channel, split open to reveal its structure. Domain IV is bound to domain I, and that is how the pore is formed. The ciguatoxin binds to S5 on domain IV (From Lewis *et al.*, 2000).

Ciguatoxin binds to the voltage-gated sodium channels in different types of cells causing the channel to open. The receptor used by ciguatoxin and also by brevetoxin (a toxin produced by another marine dinoflagellate *Ptychordiscus brevis*) is situated near the S5-S6 loop of domain IV on the sodium channel (Strachan *et al.*, 1999).

Ciguatoxin stimulates excessive Na^+ entry into cells by binding to the Na^+ channel. The entry of Na^+ depolarises the membrane that is, the cytosol becomes less negative. If this depolarisation reaches the critical level the membrane will generate an action potential (Lehane and Lewis 2000). CTX-1, -2 and-3 all inhibit the binding of brevetoxin to the sodium channel. The affinity of CTX-1 is about 30 times higher than that of brevetoxin. Ciguatoxin slows the nerve conduction and prolongs the refractory periods in human neurons. The sensory discomfort that often is experienced in relation to cold, a common symptom in CFP, is most likely a result of exaggerated and intense nerve depolarisation (Lehane and Lewis, 2000). Electrophysiological studies indicate that ciguatoxin induces a membrane depolarisation, which is prevented by tetrodotoxin (another potent marine toxin found in Puffer-fish) that also binds to the sodium channels (Lewis *et al.,* 2000).

4.12 Toxicokinetics

Not much is known about absorption, distribution, metabolism and excretion of ciguatoxin in humans. The toxin is fat-soluble, and absorption from the gut is rapid and almost complete. If the gastrointestinal problems (diarrhoea and vomiting) start immediately after ingestion of ciguatoxic fish, the absorption of the toxin will decrease. The finding that there is little or no difference in toxicity after intraperitoneal and oral administration might indicate that the absorption from the gut is rapid and almost complete. Manual cleaning of the fish has been reported to cause tingling of the hands, a sign that the toxin can penetrate the skin.

Ciguatoxins are bound to plasma proteins. In pregnant women, ciguatoxins can cross the placental barrier and reach the foetus. It is possible that the toxins can be excreted through vaginal secretions and sperm fluid since the partner of the affected person has experienced a local pain after intercourse. (Lehane and Lewis 2000)

There is evidence that ciguatoxins accumulate in the human body. The toxins are either bound to Na^+ channels, stored in fat tissue or bound to human serum albumin. If the ciguatoxin is stored in fat tissue it will probably not cause any problems. However if the fat tissue is broken down rapidly as in excessive weight loss, symptoms may reappear. Studies on brevetoxin indicate that the most important elimination route of this toxin is the bile. Since ciguatoxins and brevetoxin are very similar in structure it might be possible that their route for excretion is the same. (Lehane and Lewis 2000)

4.13 Ciguatera fish poisoning and cardiac toxicity in humans

CFP is rarely fatal but in some cases there are complications associated to cardiac problems. Ciguatoxins acts on the sodium-channels of the human cells including those of the heart. The usual findings in CFP associated to cardiac toxicity are hypotension and bradycardia. If these symptoms are severe the patient may need intensive care. When treating a patient with CFP it is important not to forget the negative effect that ciguatoxin has on the heart even if such symptoms have not yet occurred, since complications may arise (Miller *et al*., 1999).

4.14 Ciguatera fish poisoning and foetal development in humans

A question that has received special attention is whether adverse affects could occur in the foetus of a pregnant woman exposed to ciguatoxins. As already mentioned, ciguatoxins may cross the placental barrier (Lehane and Lewis, 2000). Another possibility for transport from

mother to offspring, although not well studied, is transmission of ciguatoxins via milk to breast-fed infants.

Pregnant women who have suffered from acute CFP have reported a pronounced increase in foetal movements shortly after having the meal containing the toxic fish (Senecal and Osterloh, 1991).

A 20 year old Californian woman was 16 weeks pregnant (second trimester) when she had a large fish meal containing ciguatoxin. Symptoms appeared around four hours after the meal. The early symptoms were abdominal pain, vomiting, diarrhoea, myalgia, fatigue, headache, blurred vision, vertigo, numbness of the extremities, hot-cold reversal, burning sensation, and increased foetal movements. Many of the neurological symptoms lasted for several weeks. The increased foetal movements lasted only for a few hours. Non-stress performed during week 40 and 41 of pregnancy were normal and the foetal sonograms showed nothing out of the ordinary throughout the pregnancy. 19 days past date she delivered a 3630 grams male infant by caesarean surgery. The child was born with a normal muscle tone. Follow-ups during the first ten months of life revealed no neurological problems and no signs of teratogenesis. (Senecal and Osterloh, 1991)

In another study six pregnant patients had symptoms of CFP. They all had neurological, neuromuscular and cardiovascular symptoms. All foetuses had a period of hyperactivity followed by a period of decreased activity. One of the women aborted during the acute phase of the poisoning. The other five women delivered apparently healthy children at or near term. (Rivera-Alsina *et al.,* 1991)

Lehane and Lewis (2000) have reported on two ciguatera poisonings in pregnant women. The first woman was poisoned during the first trimester of her pregnancy. She was treated with mannitol and gave 28 weeks later birth to a male infant. He suffered from mild respiratory distress and was very irritable soon after birth. The second woman was nine months pregnant when she consumed coral trout 2 days before the expected delivery. Within 4 hours after the meal she suffered from gastrointestinal and neurological symptoms of CFP and felt an increase of the foetal movements. These continued for 18 hours and then gradually decreased over the next 24 hours. Two days later she delivered, by caesarean section, a child that had left-sided facial palsy, possible myotonia (muscle cramp) of the small muscles of the hands and respiratory distress syndrome. All these symptoms disappeared within six weeks.

Taken together, these studies indicate that it is possible that ciguatoxin might have a negative effect on the foetus, although there seems to be little or no risk for any permanent defects of the child.

4.15 Cases

On June 17th 1998, a physician in Montreal, Canada reported several cases of CFP. A few days later, on the 26th, another physician reported other cases that believed to be part of the same event. The diagnosis of CFP was in this case based on symptoms: such as nausea, vomiting, diarrhoea, abdominal cramps, muscular and dental pain, weakness, dizziness, paraesthesia (tingling and numbness) of the hands and feet and dysesthesia (increased sensitivity to sensory stimuli). (Pilon *et al.,* 2000)

Symptoms that were compatible with CFP were found in seven people. These cases were found in three families, and they had all eaten barracuda. The cases showed the following

gastrointestinal symptoms: diarrhoea (7/7), abdominal pain (4/7), vomiting (2/7), and nausea (1/7). These neurological symptoms were present: dysenthesia (6/7), weakness (5/7), headaches (5/6), paraesthesia of the extremities (4/6), muscle pain (3/6), perioral paraesthesia (tingling and numbness around the mouth) (2/6), and dental pain (1/6). A number of other symptoms were also reported: Itchiness (4/7), skin rash (2/7), arthralgia (joint aching) (2/7), light-headedness (1/7), and profuse sweating (1/7). In two of the families, involving two and three cases respectively the symptoms began after 6 to 12 hours. In another family, involving two persons, the fish was eaten on three different occasions, 11th, 13th, and 14th of June, and the symptoms appeared on the 13th and 15th. Except for a 20-month infant, all those affected were adults, ages ranging between 32 and 42 years. (Pilon *et al.*, 2000)

On September 16, 1997 the Department of Human services, Victoria, Australia, began an investigation of what they suspected to be CFP. All patients that were studied had eaten at an Asian restaurant in Melbourne the day before. Initial information suggested that 18 of 36 people had been ill after eating fish as part of a banquet meal. The diagnosis of CFP in this case was based solely on neurological symptoms. It was concluded that 46 people (29 adults and 17 children) had eaten at the banquet. The children ate from a different menu and only one of the children ate any of the suspected fish. A questionnaire was used, and the interviews took place over the telephone. All banquet attendants or their parents were interviewed. All cases were advised to not eat any fish or drink alcohol for at least three months to prevent any recurrence of symptoms. Follow up interviews were conducted 3 and 10 weeks after the exposure in order to gather information on the duration of the symptoms and to see weather any new ones had appeared. (Ng and Gregory, 2000)

All 30 individuals who consumed the fish at the Asian banquet reported to have at least one symptom. The incubation period ranged from 2 to 27 hours. The severity of the symptoms varied. Out of the 30 that were infected 17 persons sought medical attention. Three persons were admitted overnight at a hospital and nine were treated with intravenous mannitol (Ng and Gregory, 2000). Three weeks after the exposure 73% still showed some symptoms. These symptoms were exclusively neurological and included, paraesthesia of the extremities, weakness, and myalgia (muscle pain). A few patients developed late symptoms of itch, dysuria (painful urination) and rash. Of the nine individuals given mannitol, one had recovered completely. The others felt better, but still had some symptoms such as paraesthesia and hot and cold temperature reversal. Ten weeks after the exposure nine patients were symptomatic. (Ng and Gregory, 2000)

These poisonings came from a maori wrasse (*Chelinus trilobatus*) that on 15 September 1997 was used to prepare four separate dishes at the Asian restaurant. All parts of the fish were used including the head and intestines for the banquet meal. The fish had been caught off Trunk Reef in Queensland, Australia. (Ng and Gregory, 2000)

4.16 Ciguatera fish poisoning in the world

In the United States 94 outbreaks (418 cases) have been reported to the Center for Disease Control and Prevention (CDC) through the national food borne surveillance system from 1970 to 1980. This is believed to be a much underreported disease and therefore it is probable that the number of cases should be much higher (Morris, 1980). From 1983 to 1992 there were 129 outbreaks that involved 508 persons that were reported to the CDC. Several of these reports came from Hawaii (111) and Florida (10), but there were also sporadic reports from California, Vermont, New York and Illinois. No deaths have been reported (Html 5).

In Australia 50-100 cases are reported each year and this is estimated to be about 20% of the actual cases. There are usually not any reports of deaths but it is likely to be one every ten years in Australia (Lehane and Lewis 2000). Data have been collected from the years 1965 to 1988 and showed 617 cases from 225 outbreaks. The risk of being poisoned by eating coral trout (member of the serranidae family), a fish often associated with ciguatera, is less than 1/5000. (Lehane, 2000)

4.17 Ciguatera fish poisoning in Sweden

The problem of CFP is not very well known in Sweden. The Swedish Poisons Information Centre (Giftinformationscentralen) only knows of one case where three persons had eaten fish on a vacation to the Dominican Republic. They sought medical attention when they arrived in Sweden, ten days after the poisoning, due to neurological symptoms. The symptoms had initially been gastro-intestinal and the neurological symptoms were paraesthesia and reversed cold-heat sensations in their hands, feet and throat. (Giftinformationscentralen, personal communication) The National food administration in Sweden has a ban on selling and distributing fish that contain measurable amounts of ciguatoxin (SLVFS 1994:2). There are fish distributors that sell species that are associated with CFP. One distributor sells barracuda and snapper and sells about 700 kg/year of barracuda and 500 kg/year of snapper. These species are only sold to restaurants (Nordhav, personal communication).

5 Environmental aspects on ciguatera fish poisoning

5.1 Biology of *Gambierdiscus toxicus*

G. toxicus is a species of dinoflagellate (figure 10) and is responsible for CFP. Most dinoflagellates are unicellular, but colony forming species exist. Each species has a distinct form made of internal plates of cellulose. They have two flagella and their beating makes them move in a spinning way. The growth rate of this particular species is relatively low. It divides only about once every third day. The normal habitat for this dinoflagellate is coral reefs. Normally it is found attached to certain macroalgae on the reefs. Some believe that there can be more than one species of dinoflagellate involved in CFP, but *G. toxicus* is still regarded as the major source for the toxins involved in CFP. Not every genetic strain of this species produces gambiertoxins.

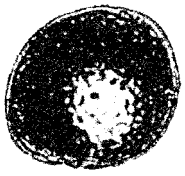

Figure 10 The dinoflagellate *Gambierdiscus toxicus* that is responsible for producing the toxin that causes CFP (From Harmful Algal Blooms, 2000).

G. toxicus is a photosynthetic species that normally grows as an epiphyte, which means that it nourishes itself but grow on other algae for support. The largest population of *G. toxicus* has been found in Hawaii and it was associated with the red alga *Spyridia filamentosa*. The main types of algae on which the *G. toxicus* lives are: *Turbinaria* sp, *Jania* sp, *Spyridia* sp, *Laurencia* sp, and any red, brown or green algae. Other dinoflagellates that possibly are

involved in CFP are *Ostreopsis lenticularis* and *Prorocentrum lima*. Their average abundance is much lower than the one of *G. toxicus.* (Chinain *et al*., 1999)

5.2 Maitotoxin

Maitotoxin is a toxin that is often discussed in relation to CFP. It is a very potent toxin and it is present in some of the ciguatoxic fish and can be produced by the dinoflagellate *G. toxicus*. The structure is shown in figure 11. In the surgeon fish (*Ctenonhaetus striatus*), that is often associated with CFP, maitotoxin is the dominating toxin in the alimentary tract. Both maitotoxin and ciguatoxins are present in the liver but only ciguatoxins are present in the meat. It is unlikely that maitotoxin accumulates in the meat of these fish or that this causes human poisonings because of this poor accumulation and it's rather low potency when administered orally (Lehane and Lewis, 2000).

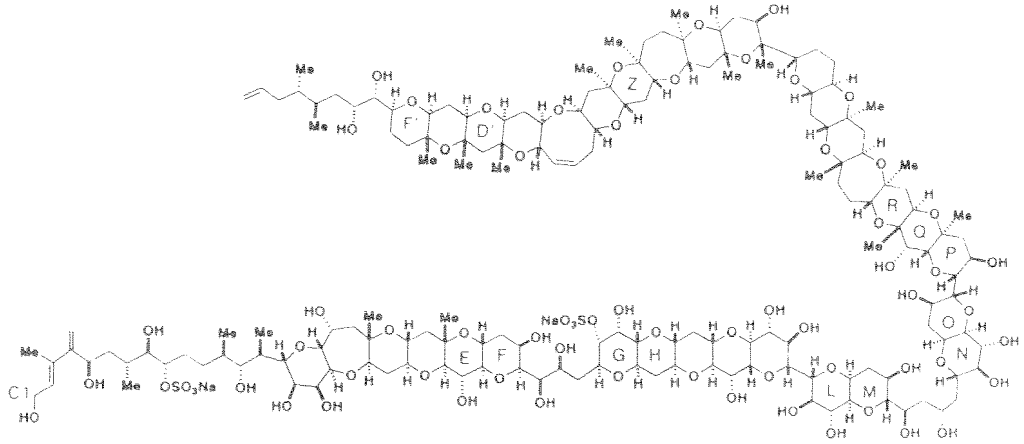

Figure 11 The structure of maitotoxin, a toxin that can be involved in CFP (From Estacion, 2000).

In culture *G. toxicus* may produce maitotoxin but little or no ciguatoxin (Lehane and Lewis 2000). Different cultured strains of *G. toxicus* produce different types of maitotoxin, with one strain only producing one single type. Maitotoxins have up to 32 ether rings and even though they are analogous in structure they have no partial structure corresponding to ciguatoxin. After intraperitoneal injection maitotoxin is much more toxic than ciguatoxin, but maitotoxin is about 100 times less toxic than ciguatoxin, when administered orally. (Lewis *et al*., 2000)

5.3 Environmental conditions causing ciguatera outbreaks

What is it that initiates blooms of *G. toxicus* and why is there often an increase of CFP after storms and hurricanes? Reef destruction and disturbance of the reef ecosystem and its relation to an increase in CFP is not yet clearly understood. Still there is evidence that it may play an important role on the frequency of outbreaks of CFP in a specific area. Additional research is required to explain the importance of different environmental factors, such as temperature and nutrients, on growth and toxin production of *G. toxicus* (Lewis and King, 1996). Dead coral surfaces covered with different micro-algae provide a favourable environment for this particular dinoflagellate. *G. toxicus* seem to become more common after both natural and man-made disturbances of the reefs, probably because there is an increase in the amounts of dead coral substrates, on which the hosts, different macroalgae, may grow. Coral bleaching is now well documented and seems to indicate extreme stress in coral colonies mostly due to increased water temperature. Bleaching will eventually kill the coral colony just like hurricanes or anthropomorphic activities, which creates new substrates for the algae to grow on (Lehane and Lewis, 2000). *G. toxicus* is endemic in most areas of the French Polynesia and

blooms of dinoflagellate are responsible for outbreaks of CFP. It is believed that the incidence of CFP outbreaks will increase due to the global warming and coral bleaching (Lehane, 2000).

5.3.1 Bacteria

Some studies indicate that certain bacteria live symbiotically with the dinoflagellate and play a role in the production of the toxins. When bacteria were added to cultures of dinoflagellate the growth rate increased. The bacteria might produce nutrients that are assimilated by the dinoflagellate, nutrients that are necessary for producing the toxins. Another possibility is that the bacteria synthesise the toxins, which is then phagocytosed by the dinoflagellate. The most common bacteria associated with ciguatera include different strains of *Pseudomonas* but also *Nocardia, Vibiro, Aeromonas, Flavobacterium* and *Moraxella*. (Lehane and Lewis, 2000)

5.3.2 Destruction of corals

There are many documented incidences where natural disturbances **of** the coral reefs have caused an increase in the incidences of CFP. Following a severe storm in the Bahamas in 1908, there were several cases of poisoning. In 1979 hurricane David crossed the West Indies and several cases of the poisoning followed and two of them were lethal. There are other examples where adverse changes in the environment of the coral reefs, such as mechanic destruction, have been followed by an increase in the amounts of *G. toxicus* in the affected area. On the other hand several hurricanes and other disturbances have struck on the coral population and not increased the occurrence of the algae.

Listed below are a number of examples of human activities that have a negative effect on the coral reefs.

- Tourism, such as recreational activities, collecting of marine animals and fishing
- Sewage and eutrophication
- Pollution from industries that contain petroleum hydrocarbons, heavy metals and different poisons.
- Degrading and filling for the construction of docks and piers
- Military activities, for example underwater or shoreline explosions
- Boats dumping ballast waters from different oceans or regions

(After Lehane and Lewis, 2000)

5.3.3 Coral bleaching

In the process of coral bleaching the coral polyps spontaneously force out the algae that live in them in symbiosis. The corals will then die and gradually lose their colour, leaving only the calcareous skeleton, hence the name coral bleaching. This is caused by stress provoked by pollution and changes in salinity, temperature and sedimentation. The corals are sensitive to changes in their environment and bleaching might occur if the average summer water temperature rises only one degree above normal. Since 1997 fifty countries have reported seeing the phenomenon of coral bleaching in their waters. There is also a strong correlation between coral bleaching and the changes of ocean currents that occur during the El Niño, which will be discussed in sector 5.4. (Lehane and Lewis, 2000)

5.3.4 Land erosion

The coral reefs of Mayotte, a volcanic island of the Comoro Archipelago between northern Mozambique and Madagascar, have been studied for several years. During this time a degrading process, due to land erosion, started in the beginning of the1980s. First there was a growth of soft corals and sponges that killed the hard corals. There was an increase in the population of the seastar *Acanthaster planci,* which feeds on coral polyps, and there was a development of algal communities. An increase in the amounts of the dinoflagellate *G. toxicus* was seen in this area between 1979 and 1988. These dinoflagellate blooms might have been associated with climatic parameters but also with seasonal development of macroalgae. However, it is possible that these blooms are a consequence of major changes in the erosion of the land, connected to the building of the main roads on the island. If that is so, this would mean that not only factors that directly affect the reefs such as building of harbours but also land erosion that origins from changes in the land environment may affect not only ecosystems on land, but also the coral reef ecosystem and therefore increase the occurrence of CFP. (Thomassin *et al.,* 1992)

5.3.5 Increase in nutrients due to coral destruction

Several studies have been made to determine if different concentrations of nutrients might have an effect on toxic blooms. Nitrates and phosphates are correlated to the density of the toxic algae (Chinain, *et al*., 1999). Areas that are of high risk for an increased population of *G. toxicus* are often those that are disturbed by human activities. In the normally functioning coral reef ecosystem there is a balance between nutrients and organisms that normally absorb them, zooxanthellae (algae that live symbiotically within the coral polyp) and benthic calcareous algae. In a disturbed ecosystem these species might not be as frequent as they would be in an undisturbed ecosystem, or perhaps they are not present at all. The nutrients will then be available for other species of algae or macroalgae, including *G. toxicus*. A bloom of *G. toxicus* may therefore follow local death of corals. When the corals start to recolonize the affected area the amount of nutrients that are available to the toxic algae will decrease and the bloom will collapse. This scenario is not only a possibility in reef destruction induced by human activities but also as a consequence of natural stress such as coral bleaching or other destruction from weather phenomenon such as cyclones and storms. During times of stress or disturbance a new environment is created and new substrates for *G. toxicus* are formed. (Rougerie and Bagnis, 1992)

5.3.6 An acidic environment

Some scientists have proposed that the increasing levels of carbon dioxide in the atmosphere will create a more acidic environment for the corals, since the carbon dioxide reacts with the surface water. Ocean acidification leads to an increased solubility for calcium carbonate which will weaken the skeletal structure of the corals. When the structure weakens the corals are more sensitive to national erosion, such as storms heavy rain or tourism and will crumble more easily. This disturbance of the coral reefs may increase outbreaks of ciguatoxic blooms. Toxic fish are often found on the windward side of a reef where the damage from storms is greater than on the other side of the reef. (Lehane and Lewis, 2000)

5.4 Correlation between ciguatera fish poisoning and El Niño

In order to investigate possible relationships between CFP and El Niño Southern Oscillation (ENSO), Hales *et al*., (1999) used a database of reported fish poisoning. ENSO is a reoccurring climate pattern that causes variations in the climate in the tropical regions of the

world. The changes are seen in rainfall, ocean currents and in sea surface temperature (SST). The ENSO is a semi regular cycle with two extremes, the El Niño and the La Niña. The phenomenon of El Niño normally makes the water temperature higher than normal in the eastern equatorial pacific as illustrated in Figure 12.

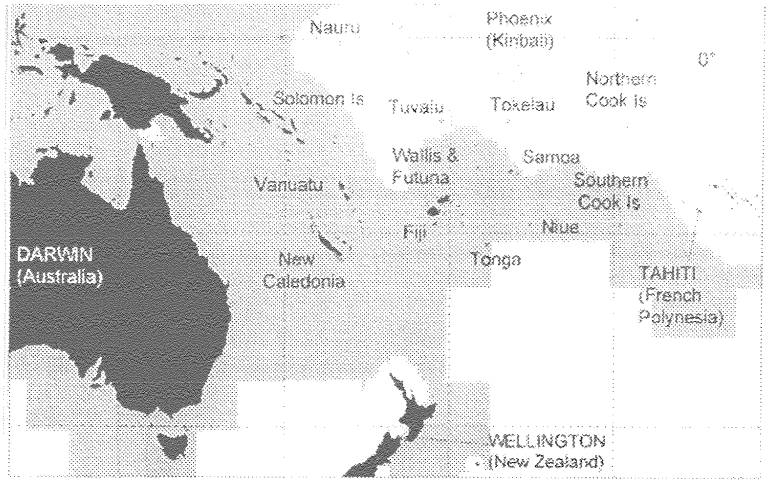

Figure 12 Map that shows the changes in sea temperature during the phenomenon of El Niño. Those areas that experience a lower temperature than normal are shaded in the figure and those areas that experience a higher temperature than normal are the light areas (From Hales *et al.,* 1999).

The results that were obtained by Hales *et al*. (1999) showed that in the eastern group of islands that were studied there was a positive correlation between fish poisoning and the local SST anomalies. CFP is known to appear after physical disturbance to the reefs and the coral ecosystem is definitely affected by such events as an increased water temperature, where one of the effects might be coral bleaching. The surfaces of dead corals are suitable for colonisation by macroalgae and therefore also *G. toxicus*. The environmental factors that affect CFP are complex, but it is highly possible that there will be an increase in CFP outbreaks if the climate continues to get warmer as a result of the greenhouse effect. Another aspect of this is that CFP or the abundance of *G. toxicus* might be a sensitive indicator of environmental stress in coral reef systems (Hales *et al.,* 1990).

5.5 Toxicity of *G. toxicus*

There are numerous reports on the coexistence of toxic and non-toxic strains of *G. toxicus*. One example is the report on two cultures that grew under identical conditions and came from individual cells isolated from Papara, French Polynesia, and only one of them proved to produce toxin. Toxic and non-toxic clones have been known to coexist in the coastal waters of Puerto Rico. All of this suggests that the ability to produce toxin in this species is genetically determined, and therefore the severity of CFP incidents depends mainly on the type of strain than the number of cells present. (Chinain, *et al*., 1999)

5.6 Can outbreaks of ciguatera fish poisoning be predicted?

To be able to predict the outbreaks of CFP in an area it is necessary to monitor the area for a long period of time. Several speculations have been made about the role of environmental factors, such as salinity, temperature and the amounts of nutrients that are available. Chinain *et al (1999)* have studied the fluctuations in the population of *G. toxicus* in relation to temperature and salinity. Data were taken from February 1993 to December 1997 in Papara, outside Tahiti, French Polynesia. Macroalgae specimens, agglomerated *Jania* sp and

Amphiroa sp, (approximately 150 g per sampling site) were collected weekly, and water temperature and salinity were measured. Every time a bloom was detected efforts were made to collect larger quantities of the blooming cells. The cells of the harvested *G. toxicus* were stored at -20° C pending toxicological analysis. A total of 58 blooms were recorded during this period of which 48 were recovered and 43 screened for their toxicity. Eighty-five percent of the blooms that occurred in 1993 to 1994 took place in the hot season. In January to April in 1994 the water temperature was elevated to unusual levels for the season. Severe coral bleaching in large areas of the reef followed this increase in temperature and there was an increase in blooms, both in density and frequency from October 1994 to December 1996. During this period the blooms were evenly distributed throughout the year, regardless of season and temperature. These blooms also yielded an exceptionally high biomass. (Chinain *et al*., 1999)

The salinity was only monitored during 1994 and 1995. During this period there were little or no changes in the salinity of the water. Salinity ranged between 34.3 and 36.1 ‰ with the lowest levels during the summertime, which corresponds to the rain season in this area. There was no correlation between the salinity and the density of *Gambierdiscus* spp during this period. (Chinain *et al*., 1999)

The largest amounts of toxins were observed in 1995 when there were several toxin-producing blooms. In 1994 there was a peak in toxicity stemming from a single bloom. No correlation was found between the concentrations of toxin present in a bloom and its biomass. Also no relationship was found between water temperature and toxicity of the algae. (Chinain *et al*., 1999)

In this long-term study it was not possible to see an obvious correlation between season and the density of *G. toxicus*. On the other hand more refined analysis revealed a seasonal cycle in densities from February 1993 to May 1995. There are other studies that support this theory and there are those that show that there is no relationship between season and bloom of *G. toxicus*. In laboratories *G. toxicus* produces more toxins at higher temperatures than at lower and the lack of seasonality might be due to the relative constant water temperature in the regions where the dinoflagellate lives. (Chinain, *et al*., 1999)

Atimaono barrier reef outside Tahiti, French Polynesia, was exposed to elevated water temperature in the year of 1994 from January to April. This resulted in a massive coral bleaching, which was well documented. The increase in densities and frequency of blooms, in October 1994 through 1995 and 1996, correlates well with the maximum toxin production. These observations suggest that coral bleaching and other adverse effects on the coral reefs could be another factor important for naturally occurring blooms of *G. toxicus* by providing new surfaces for colonisation by the macroalgae that are hosts for the toxic dinoflagellate. The relation seen between coral bleaching and blooms suggests that outbreaks of CFP can occur some time after destruction of reefs. The study made by Chinain, *et al*. (1999) indicates that the time between the reef mortality and the increased risk of CFP may only be a few months, in this study it was seven months.

Other factors that might be relevant to the toxicity of *G. toxicus* could be both extrinsic (such as bacteria) and intrinsic (such as genetic) factors. The bacterial hypothesis should not be completely ruled out until the absence of endosymbiotic bacteria (= a prokaryote cell living inside a eukaryote cell) in this particular dinoflagellate is proven. There is also some evidence supporting the theory that the variability in toxin production in *G. toxicus* might be due to genetic differences within the species. For example two clonal cultures of *G. toxicus* grown

under identical conditions from individual cells resulted in one being toxic and the other one being non-toxic. There have also been reports of coexistence of toxic and non-toxic clones in the waters off Puerto Rico. (Chinain *et al*., 1999)

All of these studies indicate that there indeed is a correlation between the destruction of corals and an increase in the frequency of CFP.

6 Conclusions

CFP is a common type of food poisoning in humans in the tropical regions of the world. The toxin is lipid soluble and can be found in reef fish. Due to very effective transportation that allows us to ship fresh and frozen marine products all over the world CFP is now becoming a potential problem worldwide. Cases of CFP have begun to appear in countries where knowledge about this poisoning is limited. This might be a very serious problem since it is important to make the correct diagnosis in order to be able to give the right treatment. Today there is no evident risk of getting CFP from eating reef fish imported to Sweden, even though some of those species that can contain the toxin are sold in Sweden. Still it is not impossible that cases of CFP will appear in Sweden in the future. However, CFP is something that tourists should have in mind when travelling to the tropics, and they should refrain from eating large portions of a single fish.

Regarding the environmental aspects it seems highly possible that there is an increased risk for CFP after different kinds of reef destruction. Those environmental factors that may increase the occurrence of ciguatera outbreaks are coral bleaching, a lowered pH in the waters of the oceans, land erosion close to coral reefs or a disrupted balance of nutrients in the waters. Several of these factors will lead to the other, which makes this a complicated web of events that may result in an outbreak of ciguatera. Storms or hurricanes have been known to cause reef destruction that is followed by an outbreak. This is supported by several studies but is at the same there are several storms that have not been followed by an outbreak of ciguatera.

Not only natural factors are responsible, but also human activities such as building of harbours and environmental pollutants cause the ecosystems of the reefs to crumble. The indications that coral bleaching and other disrupted balances of the ecosystems might increase the frequency of ciguateric fish is alarming and gives us even more reasons to be aware of how fragile the environment is. This is important to have in mind when planning to build new harbours or roads that pass close to the water. It is necessary to conduct further research on this matter until it is fully understood why there are sudden outbursts of CFP and if they can be prevented.

The risk of being poisoned from ciguatoxins when eating tropical fish in Sweden is probably small. However since there are no easy ways to detect toxic fish the risk cannot be completely ruled out. And it cannot be guaranteed that every fish that is imported is free from ciguatoxins. Still it is more likely that Swedes will suffer from CFP when travelling to those countries that are affected. It is therefore important to inform tourists that they should bear in mind the risk of CFP when eating tropical fishes.

Acknowledgements
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Appendix

- **Family: Muraenidae Moray eels**

Marine fish, on occasion some species may enter freshwater. Live in tropical and temperate seas (Nelson, 1994). This group consists of moderate size carnivorous fish that live in the coral reefs in shallow waters and down to depths of hundred meters or more. They hide during the day under rocks or in holes and come out to feed at night. Moray eels have sharp long teeth and are often seen lying in a hole with their head sticking out, waiting for something edible to pass before them (Halstead, 1988).

Subfamily: Muraeninae

Gymnothorax javanicus Bleeker 1859, **Moray eel** (Hashimoto, 1979) **Giant Moray** (www.fishbase.org 2001-10-25) Shown in Figure A 1.
Lives in lagoon and seaward reef, depth ranging from 0-50 meters. Feeds primary small fish but on occasion it eats small crustaceans. This species is the largest in the Indo-Pacific and can reach up to 3 meters in length. Distribution: Indo-Pacific: Red Sea and East Africa to the Marquesas and Oeno Atoll, north to the Ryukyu and Hawaiian Islands, south to New Caledonia and the Austral Islands, throughout Micronesia. The maximum size is 300 cm and maximum weight 30 kg (www.fishbase.org 2001-10-25).

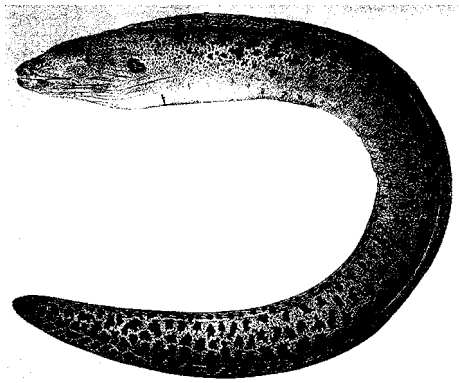

Figure A 1 Picture of *Gymnothorax javanicus,* or Giant moray, a species that is known to cause CFP (From www.fishbase.org 2002-01-1, Randall J.E photographer).

- **Family: Haemulidae Grunts**

Pomadasys maculatus Bloch, 1793, **Blotched javelin** (Lewis and Sellin, 1992) **Saddle Grunt** (www.fishbase.org 2001-10-25)
Found in coastal waters over sand near reefs. Feed on crustaceans and fish. The maximum size is 60 cm and it is found at depths from 20 to 110 meters. Distribution: Indo-West pacific: Throughout the Indian Ocean and the western Pacific, Northeast to China and Southeast to Australia.

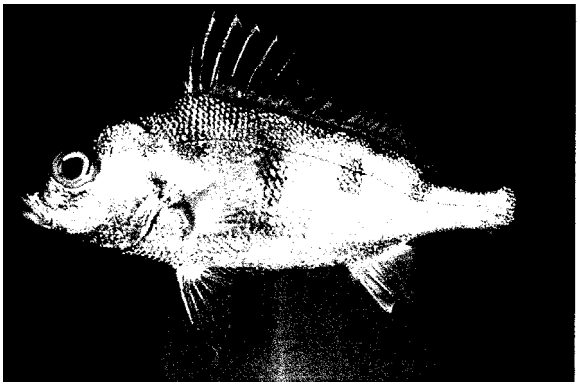

Figure A 2 *Pomadasys maculatus* Saddle grunt or Blotched javelin. This species is a member of the Haemulidae family, and is known to cause CFP (From www.fishbase.org 2002-01-11, Randall J.E. photographer).

- **Family: Serranidae, Groupers** (Crump *et al*., 1999)

Groupers or Seabass are sturdy predacious fish that inhabit a variety of biotope but are most frequently found in shores of tropical waters (Halstead, 1988). There are a few freshwater species (Nelson, 1994). They are considered good food fish and can attain great size (Halsted 1988).

Subfamily: Epinephelinae

Ephinephelus guttatus Linnéus, 1758 **Red hind, Röd prickig grouper** (www.fishbase.org 2001-12-21)
This species is found in shallow reefs and on rocky bottoms. It lives solitary and feeds mainly on crabs, other crustaceans and fish. Some fish undergo sexual inversion when they obtain a length of 28 cm and most fish over 40 cm are male. It is an excellent food fish and is easily approached by divers. The fish can obtain a size of 76 cm and a weight of 25 kg. Found in the Western Atlantic from North Carolina USA to Venezuela. It is the most common species of Ephinephelus in the West Indies. Also found in Bahamas, Antilles Central and South American coasts (www.fishbase.org 2001-12-21).

Ephinephelus striatus Bloch, 1792 **Nassau grouper, Nassaugrouper** (www.fishbase.org 2001-12-21)
Occurs from the shoreline to at least 90 m depths, usually close to caves. Feeds mainly on other fish but also on crabs and molluscs. Lives solitary but can on occasion form schools. Spawns at special spawning sites where as many as 30 000 individuals may be present. Heavily fished species and is particularly vulnerable when migrating or spawning. It is the most important commercial grouper in the West Indies. The maximum length is 122cm and it can weigh up to 25 kg. Distribution: Western Atlantic: Bermuda, Florida, Bahamas, Yucatan Peninsula and throughout the Caribbean to southern Brazil. It is not known to exist from the Gulf of Mexico except at the Campeche Bank off the coast of Yucatan, at Tourgas and off Key West (www.fishbase.org 2001-12-21).

Epinephelus fuscoguttatus Forsskål, 1775, **Flowery cod** (Lehane and Lewis, 2000) (Hashimoto, 1979). **Brown-marbled Grouper** (www.fishbase.org 2001-10-25)
This species lives in lagoon pinnacles, channels and outer reef slopes, in coral rich areas with clean water. They feed on other fish, crabs and cephalopods. They can attain a weight of 11 kg and a length of 120 cm. It is cultured under experimental conditions in the Philippines and is a candidate for aquaculture in Singapore. Distribution: Indo-Pacific: Red Sea and along the east coast of Africa to Mozambique; east to Samoa and the Phoenix Islands, north to Japan,

south to Australia. It is unknown in the Persian Gulf, Hawaii and French Polynesia (www.fishbase.org 2001-10-25).

Epinephelus lanciolatus **Grouper** (Lehane and Lewis, 2000) *Epinephelus lanceolatus* Bloch, 1790, **Giant Grouper** (www.fishbase.org 2001-10-25)
This is the largest bonefish found in reef waters. It can attain a size up to 270 cm and a weight of 400 kg. Despite its size it is common in shallow waters, and individuals of over one meter have been caught from shore and in harbours. It feeds on fish, but also small sharks, young see turtles and lobsters. There are some unconfirmed reports on human attacks. It is now becoming rare in heavily fished areas. Distribution: Indo –Pacific: Red Sea to Aloga Bay, South Africa and eastward to the Hawaiian and Pitcairn Islands, north to southern Japan, south to Australia. Absent in the Persian Gulf (www.fishbase.org 2001-10-25).

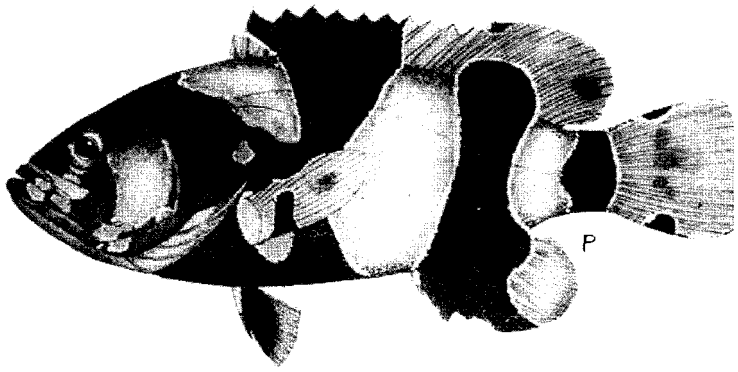

Figure A 3 *Ephinephelus lanceolatus,* Giant grouper. This fish is a member of the Serranidae family, where several species is known to cause ciguatera poisoning. (From www.fishbase.org 2002-01-11, Randall J.E. illustrator).

Mycteroperca bonaci Poey, 1860 **Black grouper, Svart grouper** (www.fishbase.org 2001-12-21)
This is a solitary species that inhabits rocky bottoms or coral reefs. Common but can be difficult to approach. The meat is said to be of excellent quality. Large species with a maximum size of 133 cm and a maximum weight of 100 kg. The adults feed primarily on fish and the juvenile mainly on crustaceans. Distribution: Western Atlantic: Bermuda and Massachusetts, USA to southern Brazil, including the southern gulf of Mexico, Florida Keys, Bahamas, Cuba and throughout the Caribbean. Adults are unknown from the north-eastern coast of the USA (www.fishbase.org 2001-12-21)

- **Family: Carangidae Jacks and Pompanos**

This is a large group that consists of Jacks, scads and pompanos. They are particularly common within the coral reefs. They are mostly carnivorous in their feeding habits and they are valued as good food (Halstead, 1988). Marine fish, only rarely brackish, exists throughout Atlantic, Indian and the Pacific Oceans (Nelson, 1994).

Subfamily: Scomberoidinae

Scomberoides commersonnianus Lacepède 1801, **Queenfish** (Lehane and Lewis, 2000) **Talang queenfish** (www.fishbase.org 2001-10-25)
This fish inhabits coastal waters frequently found near reefs and offshore islands, usually in small groups. Feeds on fish, cephalopods or other pelagic prey. The maximum size is 120 cm. Distribution: Indo-West pacific: in tropical waters (www.fishbase.org 2001-10-25).

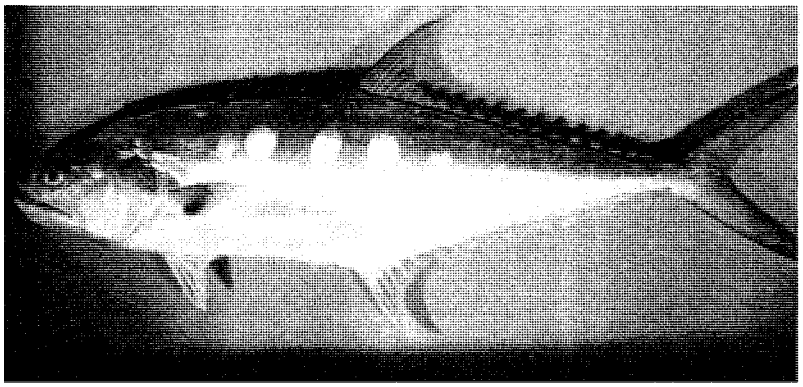

Figure A 4 *Scomberoides commersonnianus*, Talang queenfish. This species is a representative of the Carangidae family, and has been known to cause CFP (From www.fishbase.org 2002-01-11, Randall J.E. photographer).

Subfamily: Nauctratinae

Seriola aureovittata **Amberjack** (Hashimoto, 1979) *Seriola lalandi* Valenciennes, 1833, **Yellowtail amberjack** (www.fishbase.org 2001-10-25)
This is a pelagic species that is found in coastal and oceanic waters. They live solitary but may occur in small groups near rocky shore, reefs and islands. Feed on small fish and crustaceans. Distribution: Circumtropical, entering temperate waters in some areas. Indo-Pacific: Japan, Great Australian Bight and South-eastern Australia. Eastern Pacific: British Columbia, Canada to Chile. Eastern Atlantic: St. Helena, South Africa. The maximum size is 250 cm and the maximum weight is 70 kg (www.fishbase.org 2001-10-25).

Subfamily: Carangniae

Caranx latus Agassiz, 1831, **Horse-eye Jack** (Vernoux and Lewis, 1996)
Pelagic fish found in schools in offshore reefs. Sometimes it is found in brackish waters or rivers. It feeds on fish, shrimp, and other invertebrates. The maximum size is 80 cm and it can attain a weight of 5,4 kg. Distribution: Western Atlantic: New Jersey Bermuda and northern Gulf of Mexico to Rio de Janeiro, Brazil, also entire Caribbean. Eastern Atlantic: St. Paul's Rocks, Ascension Islands, and two confirmed records from the Gulf of Guinea (www.fishbase.org 2001-10-25).

Caranx sexfasciatus Quoy & Gaimard, 1825, **Jack** (Hashimoto, 1979) **Bigeye trevally** (www.fishbase.org 2001-10-25)
Reef fish that inhabits both coastal and oceanic waters. On occasion it may enter rivers. Lives in slow-moving schools outside the reef during the day at depths from 20-50 meters. It feeds at night and lives on fish and crustaceans. The length is up to 120 cm and the maximum weight is 18 kg. Distribution: Indo-Pacific: Red Sea and East Africa to Hawaii, north to southern Japan and the Ogasawara Island, south to Australia and New Caledonia. Eastern Pacific: south-western coast of Baja California Sur, Mexico and the Gulf of California to Ecuador and the Galapagos Islands (www.fishbase.org 2001-10-25).

Family: Lutjanidae Snapper (Crump *et al*., 1999)
Marine fish, only rarely in freshwater or estuaries. Lives in tropical and subtropical regions. They are important food fish that is found near the bottom in shallow waters to depth of 550 meters. Tree species are only found in freshwater or estuaries. Snappers are abundant in all

warm oceans. They are carnivorous and live in rocky, coral reef areas. They are popular as game fish and in some areas they have great commercial value (Halstead, 1988).

Subfamily: Apsilinae

Apsilus dentatus Guichenot, 1852 **Black snapper, Svart snapper** (www.fishbase.org 2001-12-21)
This species mainly inhabits rocky bottoms, but young fish can be found near the surface. Feeds on fish and other benthic organisms. Can obtain a size of 65 cm. Found in Western Central Atlantic: West Indies. Florida Keys, western Gulf of Mexico and western Caribbean (www.fishbase.org 2001-12-21).

Subfamily: Paradicichthyinae

Symphorus nematophorus Bleeker, 1860, **Chinamanfish** (www.fishbase.org 2001-01-25) (Lehane and Lewis, 2000) *Glabrilutjanus nematophorus* **Chinamanfish** (Hashimoto, 1979)
This species lives in coral reefs from shallow waters to depths of at least 50 meters. It usually swims solitarily and feeds mainly on other fish. It can obtain a size of 100 cm. Distribution: Western-Pacific from New Caledonia throughout the Indo-Australian archipelago (from New Guinea and northern Australia to the Malay Peninsula) and north to the Ryukyu Islands. Also known from the eastern Indian Ocean off north-western Australia. The maximum size is 100 cm and the maximum length is about 12 kg (www.fishbase.org 2001-01-25).

Subfamily: Lutjaninae

Lutjanus analis Cuvier, 1828 **Mutton snapper, Fläcksnapper** (www.fishbase.org 2001-12-21)
Found both in continental shelf areas and round islands. The adults are usually found in rocky or coral areas whereas the young are found over sandy or vegetated bottoms. Feed both day and night on fish, shrimp or crabs. The size is up to 85 cm and the maximum weight is 16 kg. Found in the Western Atlantic as far north as Massachusetts, USA, Bermuda, and southward to south-eastern Brazil, including the Caribbean Sea and the Gulf of Mexico. It is most abundant in the Antilles, Bahamas and off southern Florida (www.fishbase.org 2001-12-21).

Lutjanus bohar Forsskål, 1775, **Red bass**, (Lewis and King, 1996) (Lehane and Lewis, 2000) (Hashimoto, 1979) **Two spot red snapper** (www.fishbase.org 2001-10-25)
Inhabits both sheltered lagoons and outer reefs at depths from 1-180 meters. Often found single but sometimes in groups. Feeds mainly on fish but also takes shrimps, crabs. The maximum size is 90 cm and the maximum weight is 13 kg. Distribution: Indo-West Pacific: East Africa to the Marquesas and Line Islands, North to the Ryukyu Islands, south to Australia. This species is more common around oceanic islands than in continental areas (www.fishbase.org 2001-10-25).

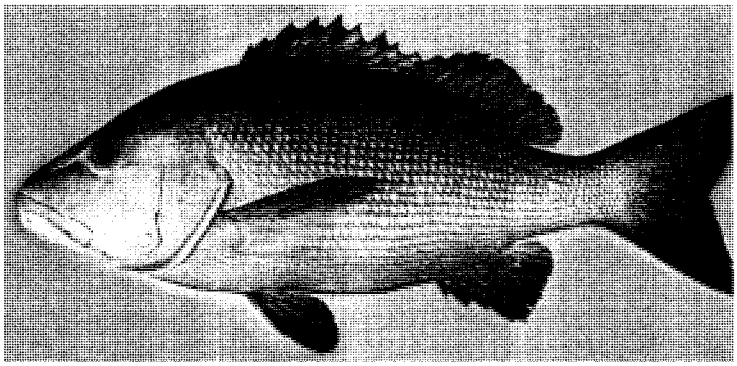

Figure A 5 *Lutjanus bohar,* Two spot red snapper. A member of the Lutjanidae family, where several species are known to cause CFP (From www.fishbase.org 2002-01-1, Randall J.E photographer).

Lutjanus buccanella Cuvier, 1828 **Blackfin snapper, Svartfenad snapper** (www.fishbase.org 2001-12-21)
Good food fish that is found in deeper waters over sandy or rocky bottoms near ledges. When young they live on sandy bottoms in shallow waters. Feed mainly on fish. The maximum size is 75 cm and the maximum weight is 14 kg. Distributed in the Western Atlantic as far north as North Carolina, USA and South to Bermuda, Trinidad and Northern Brazil including the Gulf of Mexico (www.fishbase.org 2001-12-21).

Lutjanus campechanus Poey, 1860 **Northern red snapper, Röd snapper** (www.fishbase.org 2001-12-21)
When young, this species live over sandy bottoms in shallow waters and when adult it is mostly found over rocky bottoms in deeper waters down to 190 meters. Feeds on fish, shrimp, crabs, worms and some plankton. It is a popular fish to eat and has been heavily exploited in America, where it is now protected. The maximum size is 100 cm and the reported maximum weight is 22 kg. Lives in the Gulf of Mexico and eastern coast of the USA, extending northward to Massachusetts. Also found in Florida and the Gulf of Mexico, but is rare in North Carolina (www.fishbase.org 2001-12-21).

Lutjanus gibbus Forsskål, 1775, **Paddeltail**, (Lewis and King, 1996; Lehane and Lewis, 2000) **Humpback red Snapper** (www.fishbase.org 2001-10-25)
This species mainly inhabits coral reefs, sometimes forming large aggregations that are mostly stationary during the day. Lives on fish, crabs, shrimp, and lobster. They can attain a size of 50 cm. Distribution: Indo-West Pacific: Red Sea and East Africa to the Society and Line Islands, north to Southern Japan, south to Australia (www.fishbase.org 25/10 2001).

Lutjanus griseus Linnaeus, 1758 **Grey snapper, Grå snapper** (www.fishbase.org 2001-12-21)
Lives in large aggregations and feed at night. This species is found in both coral reef areas as well as offshore waters. They have also been found over rocky bottoms and in mangrove areas or estuaries. Juveniles have even been found in freshwater in Florida. Good fish to eat and is easily approached by divers. The size is up to 90 cm and the weight is up to 20 kg. Distribution: Massachusetts, USA and Bermuda southward to Rio de Janeiro, Brazil, Including the West Indies, Gulf of Mexico and Caribbean sea (www.fishbase.org 2001-12-21).

Lutjanus monostigma Cuvier 1828, **Snapper** (Hashimoto, 1979) **Onespot Snapper** (www.fishbase.org 2001-10-25)
Lives in coral reef areas usually close to caves wreckage or large coral formations that it uses as shelter. It usually lives alone but sometimes form small groups. Feeds at night on small fish and benthic crustaceans. Distribution: Indo-Pacific: Marquesas ad Line Islands, north to the Ryukyu Islands, south to Australia. The maximum length is 60 cm (www.fishbase.org 25/10 2001).

Lutjanus sebae Cuvier, 1816, **Red emperor** (Lehane and Lewis, 2000) **Emperor red Snapper** (www.fishbase.org 2001-10-25) **Kejsarsnapper**
Lives close to coral or rocky reefs, often over adjacent sand flats and gravel patches. It is found at depths of 5-180 meters. Juveniles live in shallow waters or in mangrove areas. As they grow they move too deeper areas. This species lives solitary or from schools with similar sized individuals. They feed on fish, crabs or benthic crustaceans. Can reach a size of 116 cm and weigh up to 33 kg. Distribution: Indo-West Pacific southern Red Sea and East Africa to New Caledonia, north to southern Japan, south to Australia (www.fishbase.org 2001-10-25).

Lutjanus synagris Linnaeus, 1758 **Lane snapper, Randig snapper** (www.fishbase.org 2001-12-21)
Found over all types of bottoms but mainly around coral reefs at depths from 10 to 400m. Good food fish that often is found in large schools, especially during the breeding season. Feeds mainly on small fish and bottom-living crabs. The maximum size is 50 cm and max weight is 2.6 kg. Lives in Bermuda and North Carolina, USA, to south-eastern Brazil, including Gulf of Mexico and Caribbean Sea. Most abundant in Antilles and the northern coast of South America (www.fishbase.org 2001-12-21).

Lutjanus vivanus Cuvier, 1828 **Silk snapper, Sidensnapper** (www.fishbase.org 2001-12-21)
Good food fish that is common near the edges of continental and island selves. It has also been found in deeper waters, below 200 m, but is often enters shallow waters at night. Feeds mostly on fish, shrimp, crabs and gastropods. The size is up to 80 cm ant the weight is up to 8.5 kg. Found in the western Atlantic as far north and Bermuda and North Carolina, USA and south to central Brazil. It is most abundant in the Antilles and Bermuda (www.fishbase.org 2001-12-21).

Ocyurus chrysurus Bloch, 1791 **Yellowtail snapper, Gulstjärtsnapper** (www.fishbase.org 2001-12-21)
This species inhabits coastal waters, mostly around reef areas. Often seen in aggregations not to close to the bottom. Mainly feeds at night on a combination of plankton and a variety of marine animals, including fish. The size is up to 86.3 meters and a maximum weight of about 3.7 kg. Distribution: Northward to Massachusetts, USA and Bermuda, south to Brazil, Gulf of Mexico and Antilles. However they are most frequently found in the Bahamas, outside the coast of Florida and throughout the Caribbean (www.fishbase.org 2001-12-21).

- **Family: Labridae Wrasses**

This is the second largest family of marine fish. It is one of the most diversified families, where size ranges from 4.5 cm to 230 cm. They are usually very colourful. Most species bury themselves in the sand at night (Nelson, 1994). This species is known for their large separated conical teeth in the front of the jaw. They are widely distributed over the world but are most abundant in warmer waters. Most species are carnivorous, but some are herbivorous (Halstead, 1988).

Subfamily: Cheilininae

Chelinus trilobatus **Maori wrasse** (Lehane and Lewis, 2000) or *Cheilinus trilobatus* Lacepède, 1801 **Tripletail wrasse** (www.fishbase.org 2001-10-25)
This fish inhabits seaward reefs and lagoons at depths from 1 to 30 meters. It is usually solitary and feeds on molluscs and crustaceans, but occasionally takes fish. This is a shy fish that is difficult to approach. The maximum length is 45 cm. It exists in the Indo pacific: East Africa to Tuamotu and Aostral islands; north to the Ryukyu Islands, south to New Caledonia, throughout Micronesia (www.fishbase.org 2001-10-25).

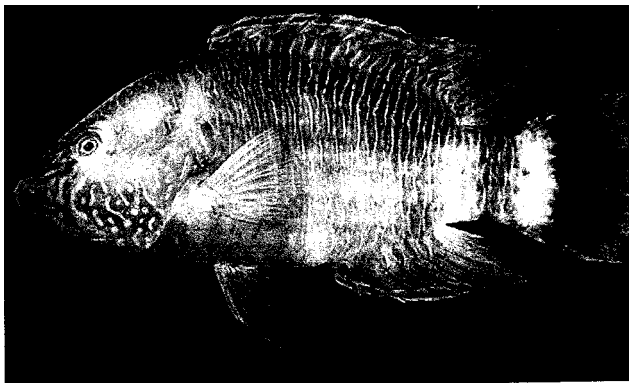

Figure A 6 Picture of a Maori wrasse, also known as Tripletail wrasse, *Cheilinos trilobatus.* Several species of the Labirdae family have been known o cause CFP (From www.fishbase.org 2002-01-11, Randall J.E photographer).

- **Family: Scaridae Parrotfish** (Crump, *et al*., 1999) (Lehane and Lewis, 2000)

Marine fishes that mainly lives in the tropical regions. This family consists of herbivorous fish that feed on dead coral substrate. They rarely feed on living corals. Sex change appears to be common in this family. They are very colourful and the colour-pattern is an important factor in identifying several species. However this can vary greatly with gender and growth (Nelson, 1994). Their appearance is similar to the Wrasses, but their teeth are fused into plates in an almost beak-like manner. They are shallow water shore fish and are common on lagoons and reef areas. They play an important role in the making of fine sands since they return the pulverised rock and skeletal material to the bottom as faecal composition (Halstead, 1988).

Subfamily: Scarinae

Scarus gibbus **Parrotfish** (Hashimoto, 1979) or *Chlorurus gibbus* Rüppel, 1829 **Heavybeak parrotfish**
This fish usually live in schools on the outer reef. Feeds on algae. Maximum length of this species is 70 cm and it can weigh up to 4 kg. Distribution: Indo-Pacific: Red Sea. The spices *C. Gibbus,* in the Red Sea, *C. Strongylochephalus* in the Indian Ocean a *C. microrhinos* in the west central Pacific needs comprehensive study to determine the taxonomic status of the various forms (www.fishbase.org 2001-10-25).

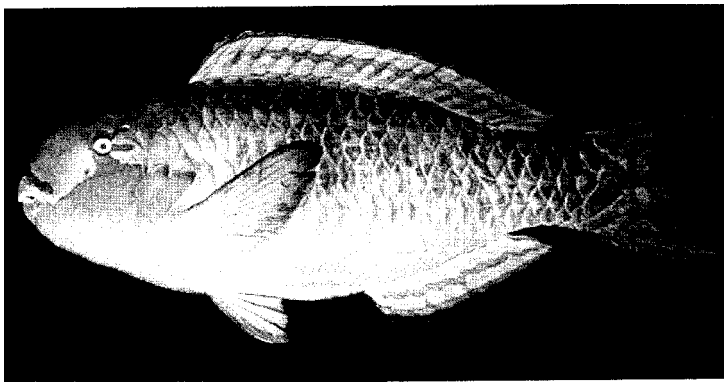

Figure A 7 A member of the Scaridae family, the *Scarus gibbus*, Heavy beak parrotfish that have been known to cause CFP in humans (From www.fishbase.org 2002-01-11, Randall J.E photographer).

- **Family: Acanthuridae Surgeonfishes, Tangs and Unicornfishes**

Marine species lives in all tropical and subtropical seas although not in the Mediterranean (Nelson, 1994). They are shore fish and are especially common in surge channels and shoal areas. Surgeonfish are mostly herbivorous fish and feeds on algae. Most surgeonfish are small to moderate in size (Halstead, 1988).

Subfamily: Acanthurinae

Ctenochaetus striatus Quoy & Gaimard 1825, **Surgeonfish** (Hashimoto, 1979) **Striated surgeonfish** (www.fishbase.org 2001-10-25)
Inhabits reefs and lagoons to a depth of over 30 meters. It is found over coral, rock, or rubble substrates. Is occurs single as well as in large mixed-species schools and can attain a length of 26 cm. Feeds on algae and diatoms that make this species especially important in the ciguatera food chain. Distribution: Indo -Pacific: Red Sea south to Natal, South Africa and east to Tuamotu Island, north to southern Great Barrier Reef and Rapa (Austral Is.), throughout Micronesia (absent from Malden and Jarvis Islands) (www.fishbase.org 2001-10-25).

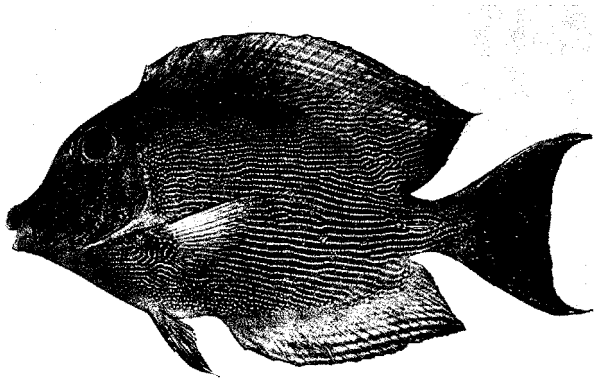

Figure A 8 *Ctenochaetus striatus,* Striated surgeonfish. A member of the Acanthuridae family. This species is herbivorous and is believed to be the link between the toxin producing algae and the carnivorous fish that contain ciguatoxin (From www.fishbase.org 2002-01-11, Randall J.E photographer).

- **Family: Sphyraenidae Barracuda** (Crump *et al.,* 1999)

This species is known to attack humans and in some regions they are feared more than sharks. The maximum length is about 1,8 meters (Nelson, 1994). Barracudas are long, slender, carnivorous and extremely voracious fish. They have long slender sharp teeth. Some species attain large size. They are common in lagoons, passageways and coral reefs. The meat is excellent but may be toxic especially during the reproductive season (Halstead, 1988).

Sphyraena barracuda Walbaum, 1792, **Barracud**a (Hashimoto, 1979) **Great Barracuda** (www.fishbase.org 2001-10-25) This species is mostly found at or near surface but it can be found at depths down to 100 meters. Adults are found in a wide range of habitats from harbours to open seas. This fish usually swims alone, but can also be found in small aggregations. It feeds mostly on fish but sometimes it eats shrimp. This is a large fish and can attain a size up to 200 cm. There have been reports of it attacking humans but these are extremely rare and are rarely fatal. Distribution: Indo-Pacific Red Sea and East Coast of Africa to Hawaii, Marquesas and Tuamotos Islands, throughout Micronesia. Western Atlantic: Massachusetts, Bermuda and throughout the Caribbean Sea to Brazil. Eastern Atlantic: Sierra Leone, Côte d'Ivoire, Togo Nigeria, Senegal, Mauritania, St. Paul's Rocks, São Tomé Island (www.fishbase.org 2001-10-25).

Figure A 9 *Sphyraena barracuda,* Great Barracuda. This species belong to the Sphyraenidae family and is frequently associated with CFP (From www.fishbase.org 2002-01-11, Trevor, M., photographer).

Sphyraena jello Cuvier, 1829 **Barracuda** (Lehane and Lewis, 2000) **Pickhandle Barracuda** (www.fishbase.org 2001-10-25)

Found near current swept lagoons or seaward reefs at depths from 20 to 200 meters. It has also been found in bays estuaries and inner lagoons. Lives alone but the juveniles form schools. Feeds mainly on fish but also take squid. The maximum size of this species is 150 cm and the maximum weight is around 7.5 kg (www.fishbase.org 2001-10-25).

- **Family**: **Scombridae Mackerel** (Crump *et al*., 1999) (Lewis and Sellin, 1992)

Marine fishes that are rare in freshwater. Lives in tropical and subtropical seas.
They are fast swimming and are valuable as game fish and commercial fishing (Nelson, 1994). Its streamlined bodies and smooth scales distinguish this species. The colour has a metallic shimmer and they have several small fins behind the dorsal and anal fin. This is a large family and only a few genera have been involved in ciguatera poisoning (Halstead, 1994).

Achanthocubium solandri Cuvier, 1832 **Wahoo** (www.fishbase.org 2001-12-21)

This is a pelagic fish that lives in the topical regions of the world at depths from 0-12 meters. Solitary species that on occasion forms small loose aggregations. Maximum size is 250 cm and maximum weight is 83 kg. Found in tropical and subtropical waters of the Atlantic, Indian and Pacific oceans. Also found in the Caribbean and Mediterranean seas (www.fishbase.org 2001-12-21).

Subfamily: Thunninae

Euthynnus affinis **Kawakawa** (www.fishbase.org 2001-12-21)
This species is found in open waters but always close to the shoreline. It is a highly migratory species. Forms large schools, depending on individual size with other species of the Scombroidae family, of 100 to 5000 individuals. Feeds on small fish, squids, and sometimes zooplankton. The size of this species is up to 100 cm and it can weigh 14 kg. Found in the warm waters of the Indo-West Pacific including oceanic islands in archipelagos. A few individuals have been found in the Eastern tropical Pacific (www.fishbase.org 2001-12-21).

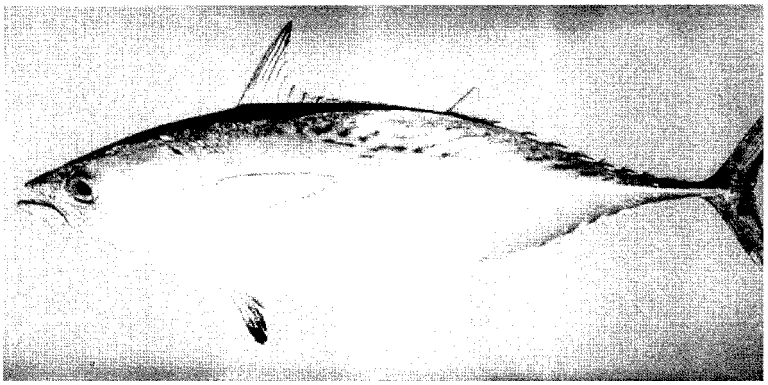

Figure A 10 *Euthynnus affinis* Kawakawa. This species is a member of the Thunninae family and have caused CFP in humans. (From www.fishbase.org 2002-01-11, Randall J.E photographer).

Euthynnus alletteratus Rafinesque, 1810 **Little tunny Tunnina** (www.fishbase.org 2001-12-21) This migratory species is found in schools in open waters not far from shore. Feeds on almost anything they can find such as fish, crustaceans and squids. Fished commercially and is also a popular game fish. Found in the tropical and subtropical waters of the Atlantic Ocean. Including the Mediterranean, Black Sea, Caribbean Sea and the Gulf of Mexico. The maximum weight is 16 kg and the maximum length is 122 cm (www.fishbase.org 2001-12-21).

Subfamily: Scombrinae

Scomberomorus commerson Lacepède 1800, **Narrow-barred Spanish mackerel** (Lehane and Lewis, 2000).
This species lives in several different types of surroundings, near the edges of continental shelves, in shallow coastal waters or in gently sloping reef or lagoon waters. They live in small school and are known to migrate, but permanent resident populations seem to exist. This fish primarily feed on smaller fish like anchovies but also eat squids and shrimp. A lipid soluble toxin, similar to ciguatoxin has been found in the meat of specimen caught on the East Coast of Queensland, Australia. This species can obtain a length of 240 cm and weigh as much as 70 kg. Distribution: Indo-West pacific: Red Sea and South Africa to Southeast Asia, north to China and Japan and south to Southeast Australia and to Fiji. Have migrated to eastern Mediterranean Sea through the Suez Canal. Also reported from St. Helena (www.fishbase.org 2001-10-25).

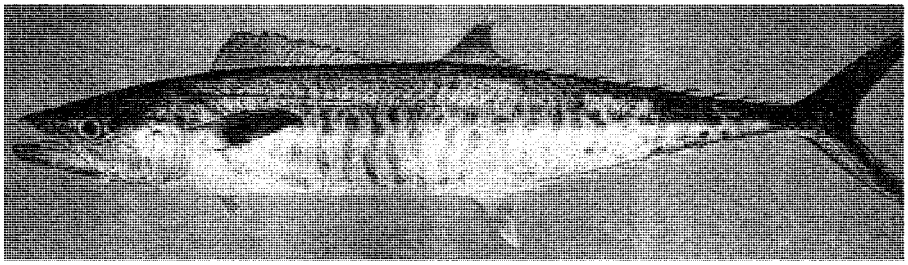

Figure A 11 *Scomberomorus commerson* Narrow-barred Spanish mackerel, a member of the Scombridae family. This species is very frequently associated with CFP, and in some parts of Australia there is even a ban on catching this species (From www.fishbase.org 11/1 2002, Randall J.E photographer).

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