FISH POISONING

1960s - 1990s

GEORGE BALAZS FILE

Cleaned fish not as likely

from the sea

mike markrich

Most people clean fish before eating them, probably not thinking much about it. Removing and discarding fish viscera in most cultures is something almost everybody takes for granted.

If you want a good reason for doing it this way, however, doctors and scientists can give you an answer.

They say that the possibility of getting serious fish poisoning may be 50 to 100 times greater for people eating the liver and intestines of fish bearing ciguatera toxins. They also say the only people who have ever died from ciguatera poisoning in the United States became ill after eating three different reef fish at Haleiwa Beach Park in 1964 without cleaning them.

There are, of course, people in Hawaii who do eat fish without cleaning them. Some think the limu inside the fish intestines is a delicacy.

Doctors and scientists who worked on a new state Health Department study on ciguatera that this does not necessarily mean that people are in great danger of getting ciguatera now. Most commercial fishing is done in deeper waters; the ciguatera-prone reef areas are largely fished by shore casters and others fishing for recreation.

Scientists say chances are slim that you could become ill by eating reef fish from most areas.

For more than 25 years, Professor Hank Banner of the Hawaii Institute of Marine Biology has studied ciguatera and other kinds of fish poisoning. He said that while certain reef fish such as ulua can be very toxic, "there are people who have been eating it for a long time that nothing has ever hap-

who was one of the authors of the study. Mild cases may not be treated at all or may be misdiagnosed.

Ciguatera is caused by small organisms called dynoflagellates that attach themselves to marine algae which are eaten by fish living on the reef. (Deep sea fish such as ahi, mahi-mahi and ono don't get it, but any reef fish can be toxic.)

Small fish get eaten by larger ones and the toxins in the dynoflagellates go with them. Scientists believe the toxin accumulates as it moves up the food chain with larger fish being more toxic than others. They also have concluded that it is more prevalent in some months than others. The greatest number of cases have been reported in July; the least, in February. Researchers don't yet know why.

But, as Banner explained, there is no guarantee that a large fish will be toxic and a small one will not. There is also no way to know if an ulua will

say that eating fish this way is not a good idea. Their report in this month's Hawaii Medical Journal says that ciguatera cases are on the rise, perhaps because of marine construction projects under way at places such as Pokai Bay and the Reef Runway.

Ciguatera was once almost unknown in Hawaii. There were only 59 incidents (affecting 450 people) from 1900 to 1974. But from 1975 to 1981, state doctors found 81 outbreaks affecting 203 individuals.

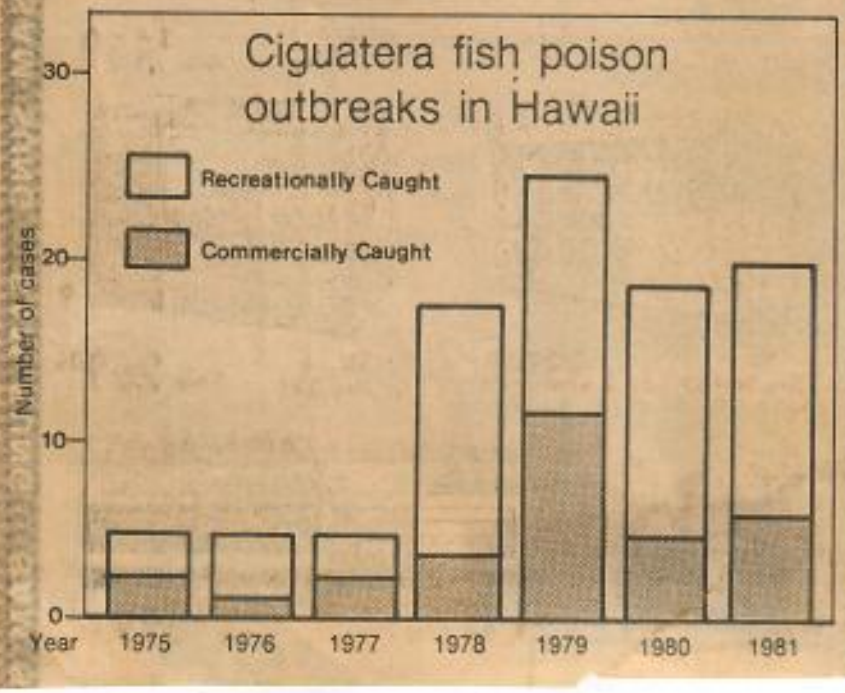
However, scientists caution

pened to. What they are doing is rolling dice and the dice are stacked in their favor."

According to the new state study, there are 2.5 cases of ciguatera or fish poisoning reported per 100,000 people in Hawaii each year.

These numbers may be of little comfort for the 25 or so people a year who have reported getting ciguatera since 1975 from at least than 18 different kinds of reef fish.

There may be many more cases of unreported ciguatera, according to Bruce Anderson



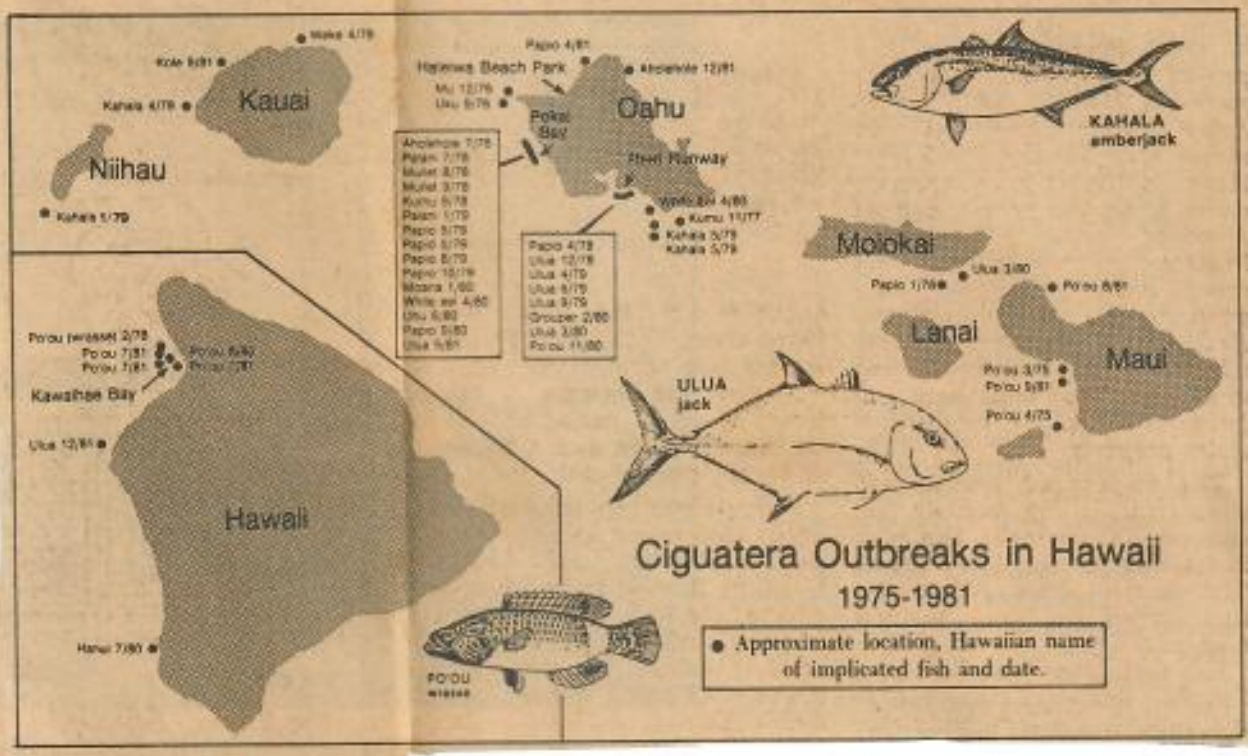
be toxic while another fish may not. It all depends on whether the fish came from an area known to have dynoflagellates.

Dr. Jack Randall of Bishop Museum explained that dynoflagellate-infested algae are usually found on new surfaces rather than old ones. When established reef areas are broken up by something — dredging, anchoring, even hurricanes — the dynoflagellate-laden algae are one of the first things to grow on the newly exposed surface.

Brooks Takenaka of the United Fishing Agency, the state's largest fish wholesaler, says he and people in the commercial business have been working with state agencies and the University of Hawaii to develop a fast and inexpensive way of checking to see whether fish is toxic. He said that he and other fish dealers have voluntarily taken fish they considered questionable off the market. But he also said the auction handles "thousands and thousands of pounds" without problems.

Dr. J.K. Sims, a coauthor of the Health Department study, says there is no way for individual consumers to test whether a fish is toxic before it is eaten. (UH has a laboratory test, but it is expensive and complicated.)

to have ciguatera poisons

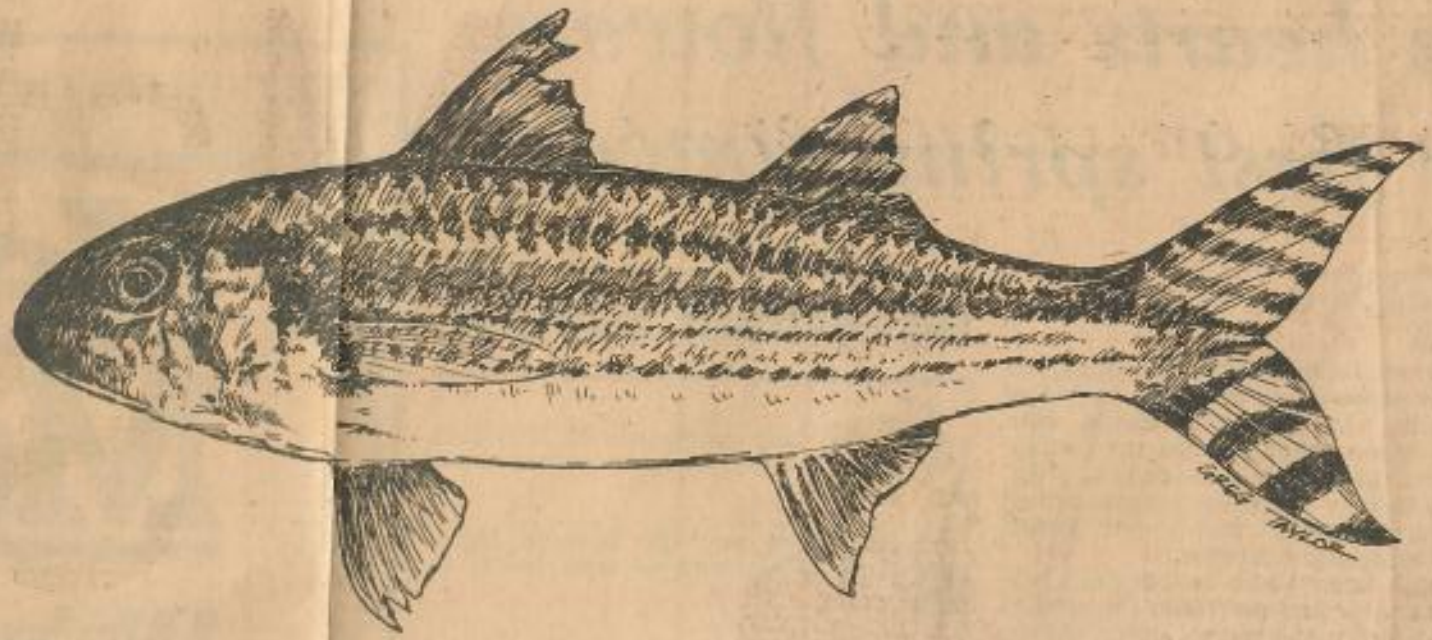


What happens if you get ciguatera? Sims says you usually become ill three to five hours after eating the toxic fish. The symptoms include a general feeling of weakness, painful tingling and numbness around the mouth, muscle pain and temperature reversal (where hot feels cold and vice versa).

Sims recommends not eating the head or tail of any fish caught in the tropics. But there are a lot of tropical fish and many people here eat the heads of fish. Besides, while fish experts agree the viscera or innards are more toxic in a fish with ciguatera, there isn't agreement on whether one part of the fish is more toxic than another.

Doctors, fish experts and others do agree on one thing, however: With a tiny fraction of 1 percent of the population affected by the toxin each year, there is no reason to give up the fish you like to eat.

"I don't what all the fuss about ulua is," said one woman on a Neighbor Island. "We've been eating it for years."



Raw-Fish Eaters Face Risk

Boston Globe

The growing popularity of sushi, sashimi, ceviche and other raw-fish dishes has raised the risk of tapeworm infection, according to researchers at the Mayo Clinic.

Dr. Joseph M. Kiley says in the current issue of the Mayo Clinic Health Letter that fish tapeworm has been found in freshwater fish in American lakes and in salmon from Alaska.

If infected fish are eaten raw, the worms can hatch and attach themselves to the intestinal wall, sometimes growing several feet long.

Alaskan processors occasionally ship fresh salmon to the markets.

Freezing or canning fish eliminates any chance that humans will contract the infection.

HSB 2/14/83 F12

'Weke pahulu' suspected cause of hallucinations

from the sea

mike markrich

The scientific name for the small striped goatfish known locally as weke pueo *s Upeneus arge*. Although it is considered by many to have a good taste, few Hawaiians in the old days ever ate it. They had another name for the fish: weke pahulu or nightmare weke.

People who have eaten the head of this fish have reported feelings of terror, imminent death and delusion. One man said he experienced feelings of being tortured by electric shocks. Another said he dreamed he was being engulfed and drowned by a large ocean wave.

Scientists have been interested in this phenomenon since 1927, when more than 30 Japanese laborers became paralyzed and delirious for one night after eating a large catch of these weke. The laborers recovered the next day, but a curious Queen's Hospital researcher took a fish head from the Molokai catch and fed it to a cat, "which at once went crazy."

organisms, they are affected by the minerals on the bottom. He said he was not surprised that the Lanai fish were affected because of the strange quality of the "red dirt" on the bottom there.

There are other theories that suggest the fish become contaminated only by eating certain kinds of seaweed that are known to be toxic, such as certain blue-green algae. But Banner says this is unlikely because fish that have caused severe reactions have come from areas where such algae is scarce, while safe weke come regularly from areas like Laie, where the water contains large amounts of the toxic seaweed.

Albert Banner of the Hawaii Institute of Marine Biology, who has published several articles on the subject, said it has been difficult to study the weke pueo because there were never enough fish with toxin in them to do adequate research.

The toxin is said by many to be concentrated in the brain, the head muscles and the spine of the weke. But people have said that they have suffered from hallucinations after eating only the flesh of the fish and it is widely believed that people respond to eating it in different ways.

Some people such as Aloha Kaeo, who is one of Hawaii's most experienced divers, have eaten the head of the weke pueo for years without ill effects. But Abraham Piianaia, also an experienced fisherman, said he had unusual dreams after he ate it. He said that once after he had eaten it, he dreamed he was being "chased down by a couple of dogs and mauled to pieces."

Banner discounts any suggestion that people's reaction to the weke toxin is psychosomatic and refers to an incident on Molokai, where young children were fed the fish "and woke screaming and trying to crawl out of their cribs."

After doing extensive research on the subject, Banner concluded that it was not necessarily the fish that was poisonous, but something the fish ate at a particular place and time.

Banner said, "We became convinced it was all right (to eat) most of the time but not at certain times or from certain locations."

The fish most likely to contain toxin are said to be from windward Oahu, Lanai, the southwest part of Molokai, and the Hanalei side of Kauai. Kaeo said he thought that, since the fish feed by foraging on the bottom for small crustaceans and other bottom dwelling

It is known that the substance that causes the hallucination can survive large amounts of heat because, in many of the reported cases, the fish heads were cooked before eating. Dr. J.K. Sims, who has studied this phenomenon, says such hallucinatory substances can be different from the ones that cause ciguatera fish poisoning. Victims of fish poisoning may take months to recover and even die, but there are no known fatalities from the hallucinagenic toxin in the weke.

Sims said he is particularly interested in the psychological reactions of people who have eaten weke pueo. There are few diseases that cause such depressed reaction and feelings of imminent death.

"This is a type of poison," he said, "that causes transient changes in behavior. If we study this, it may lead to explanations of alterations of behavior that last longer."

Sims said the symptoms include numbness, pressure on the chest, a loss of feeling in the fingertips, partial paralysis and even out-of-body experiences.

One of the explanations for the reaction may be that the fish contain something that breaks down substances in the brain and causes hallucinations similar to that experienced by people suffering from DTs or alcoholic withdrawal.

No one yet knows for certain, but people such as Piianaia who have experienced the reaction have said that "it is no nightmare, it is just bad news."

Epidemiology and Impact of Ciguatera in the Pacific: A Review

NANCY DAVIS LEWIS

Introduction

Ciguatera, first described in the Caribbean (Martyr, 1555), has a long recorded history in the Pacific as well. Sailors with the Spanish explorer, de Quiros, suffered from ciguatera in Vanuatu in 1606 (Dalrymple, 1770), and Anderson (1776) described an intoxication aboard one of Cook's ships, the *Resolution*, in the same island group. The pantropical distribution of ciguatera and the fact that the fish are unaffected by the toxin(s) suggest that it has existed far longer than the historical record indicates.

Ciguatera is a significant health and resource problem in tropical areas, largely because of its erratic and often unpredictable spatial and temporal distribution. The eating of toxic fish remains a risk for both seafarers and tourists, but it is a much more pronounced problem for the inhabitants of tropical islands, who depend on the resources of the seas for food and livelihood. The epidemiology of ciguatera in the Pacific has received considerable attention (Banner and Helfrich, 1964; Halstead, 1978; Bagnis, 1976, 1977; Lewis, 1981, 1984a, b). In this paper, the epidemiology is updated through 1983-84, and some considerations for health, nutrition, and specifically marine resource development are presented. Banner (1976), Withers (1982), and Baden (1983) have reviewed the etiology, chemistry, pharmacology, and ecology.

ABSTRACT—For inhabitants of the Pacific Islands, the mean reported incidence of ciguatera from 1979 to 1983 was 97/100,000. As a health problem, ciguatera also appears to be increasing on some Pacific islands that had formerly experienced lower levels of endemicity. Ciguatera also has important implications for nutrition and the development of inshore fishery resources. The epidemiology of ciguatera in the Pacific is presented, and viewing ciguatera as a phenomenon to which islanders have had to adapt for centuries, the implications for health, nutrition, and resource development are discussed.

Epidemiology

The most consistent, albeit incomplete, information on the existence of ciguatoxic fish in the Pacific is from morbidity reports. Intensive investigations of the distribution of both *Gambierdiscus toxicus* and toxic fish have been made at specific sites, but these are limited in both time and extent (Bagnis, 1969, 1973a, 1977; Yasumoto et al., 1979, 1984). Morbidity for selected diseases for the Pacific Island Region (not including Hawaii and Australia) is recorded by the South Pacific Commission's South Pacific Epidemiological and Health Information Service (SPEHIS) (South Pacific Commission, 1973-84). Ciguatera incidence for 1973-83 is presented in Table 1. The region is composed of 21 island states and territories (Fig. 1), and there are a host of cultural, economic, and practical factors that influence reporting. For example, sample surveys suggest a wide range (9-75 percent) in the number of individuals who go to a western medical facility when experiencing ciguatera (Bagnis, 1973a; Dawson, 1977; Lewis, 1981). There are often considerable differences between a country's central register of disease and the statistics reported to SPEHIS. In Fiji, Naryan (1980) indicated that there were 796 cases of ciguatera reported to the Ministry of Health between 1975-78. SPEHIS, based on reports submitted to it, listed 449.

The reported annual incidence of ciguatera for the 11 years from 1973 to 1983 was 97/100,000 for the region as a whole. Using the conservative estimate that this represents 20 percent of actual incidence, then actual incidence would be 500/100,000. The regional rate has been quite constant over the 11-year period, with 1973, 1979, and 1983 being years with marginally fewer cases reported. Lawrence et al. (1980) estimated that in Miami the actual incidence of ciguatera was 5/10,000 (50/100,000), making the estimated incidence for The Pacific ten times that of Miami.

Looking at the number of cases and mean rates for individual countries for the last 5 years of the period (1979-83) (Fig. 2), we see that some have reported annual morbidity rates several times that of the region. French Polynesia (585/100,000) had an average annual rate six times that of the region as a whole, and Tuvalu (484/100,000), five times. Kiribati (462/100,000) also had a mean incidence rate almost five times as high as the region as a whole. The reported rate for Tokelau, 1,338/100,000, was exceptionally high.

Nancy Davis Lewis is with the Department of Geography and the Social Science Research Institute, University of Hawaii, Honolulu, HI 96822. Views or opinions expressed or implied are those of the author and do not necessarily reflect the position of the National Marine Fisheries Service, NOAA.

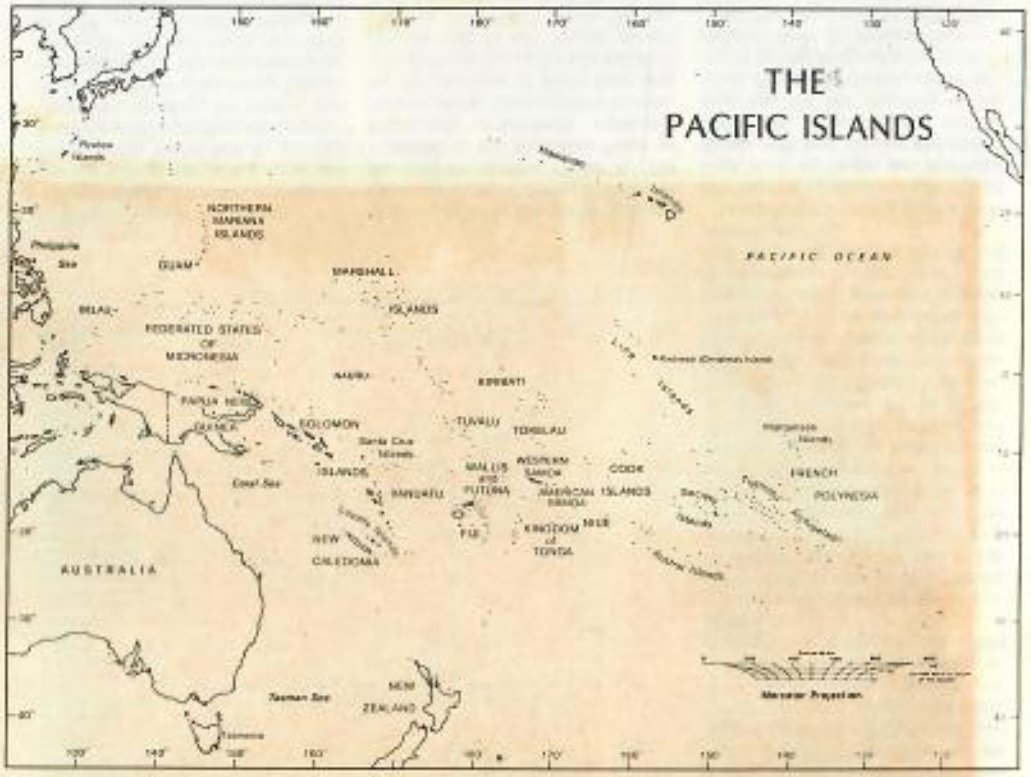


Figure 1.—The Pacific Islands.

Table 1.—Ciguatera morbidity, South Pacific Region, 1973-83, as reported to South Pacific Epidemiological and Health Information Service.

Country	Number of cases													Rate per 100,000 1973-83	Rate per 100,000 1979-83
	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	Total	Mean		
American Samoa	4	0	0	0	0	0	70	36	31	97	69	307	27.90	87	179
Cook Islands	0	0	0	0	0	0	0	1	2	0	0	3	0.27	1	3
Fiji	8	28	190	29	60	231	131	205	123	71	0	1,071	97.30	16	18
French Polynesia	607	667	625	680	532	621	677	837	1,149	831	789	8,081	798.18	545	585
Guam	0	0	21	16	6	6	9	6	4	3	21	86	7.82	8	7
Kiribati	101	176	187	77	41	36	78	167	396	418	414	2,063	102.00	324	462
Nauru	0	0	0	0	0	0	1	5	0	0	0	6	0.55	7	15
New Caledonia	0	260	618	847	457	458	168	147	107	150	112	3,024	274.91	200	95
Niue	7	1	35	4	0	0	0	3	3	0	0	53	4.82	136	38
Papua New Guinea*	0	0	16	0	0	0	0	0	0	0	0	16	1.46	<1	<1
Solomon Islands	1	7	0	7	6	6	0	4	4	0	2	37	3.36	2	1
Tokelau	0	0	0	8	0	0	14	0	3	17	73	115	10.45	663	1,338
Tonga	11	38	12	17	43	13	6	7	2	20	14	214	19.45	21	12
Tuvalu	240	264	208	313	325	295	191	317	183	118	105	2,459	223.18	170	114
Tuvalu	6	6	3	49	44	71	31	27	73	47	16	348	31.64	439	484
Vanuatu	6	6	35	28	53	53	67	0	32	12	0	277	25.19	25	19
Wallis and Futuna	6	3	3	7	0	0	0	0	0	0	0	19	0.91	9	0
Western Samoa	65	69	15	17	81	179	62	115	127	98	59	907	82.45	94	68
Total	1,042	1,557	1,925	1,879	1,655	2,172	1,617	1,948	2,308	1,875	1,689	19,368	1,760.36	97.1	96.1

*Papua New Guinea has been excluded from the regional analysis.

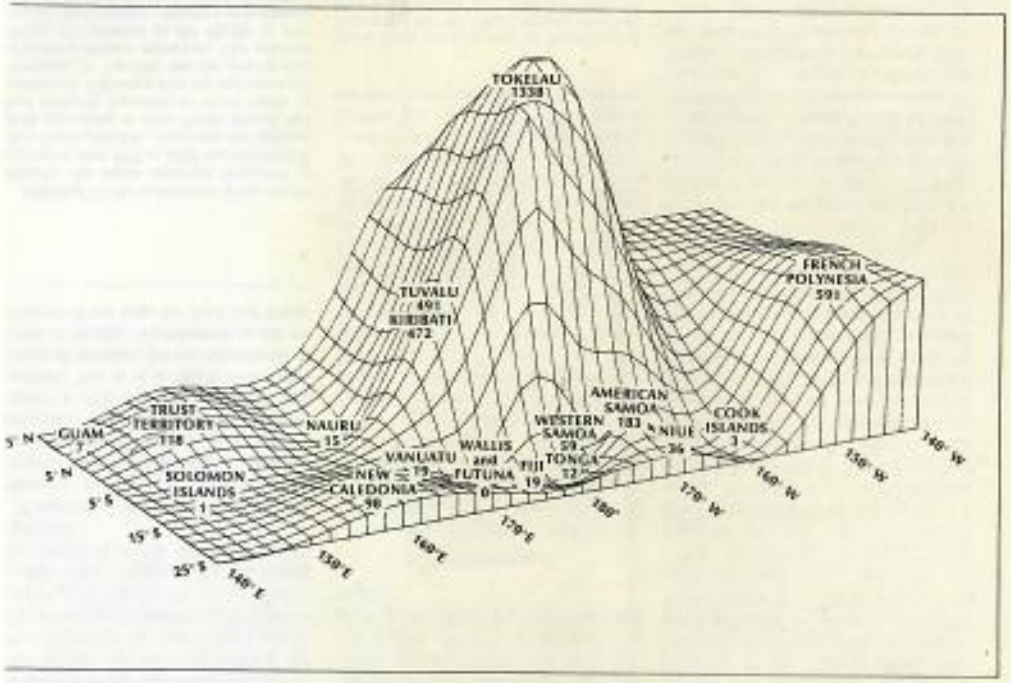


Figure 2.—Mean annual incidence of ciguatera (cases per 100,000), 1979-83.

Various patterns emerge in the three subregions of Oceania, Polynesia, Micronesia, and Melanesia. Incidence in French Polynesia has been consistently high throughout the period with a peak in 1981. While the longstanding research efforts of Raymond Bagnis and his colleagues at the Institut Recherches Medicales Louis Malardé in Papeete may have led to increased reporting, evidence suggests that ciguatera is a more serious problem in French Polynesia than in some other parts of the Pacific. The island groups within French Polynesia exhibit different levels of toxicity. It has been highest in the Gambiers, but this is not reflected in the morbidity statistics because of avoidance of locally caught fish. Species incriminated in the other groups are listed in Table 2. Disastrous hurricanes hit French Polynesia in 1982-83 and because of the long acknowledged association between disruption of marine substrate and increases in ciguatera, researchers are monitoring for increases in the causative organism. Islands to the west in Polynesia exhibit a less clear picture; the Cook Islands are in many respects a ciguatera anomaly with very low rates of ciguatera. Tonga also has relatively low rates of ciguatera. In Samoa, toxic fish were reported soon after the turn of the 20th century (Jordan, 1902; Spear, 1904) and the toxicity has persisted, but not at high levels. The species responsible (Table 2) are primarily carnivores. It is in the Tokelau that ciguatera seems to be most serious. Tokelau is a tiny territory of New Zealand made up of three coral atolls with a population of 1,600 and land area of only 10 km². The incidence rate was 1,338/100,000 for the period 1979-83. For 1983 alone the incidence was 4,867/100,000 and partial data from 1984 indicate an even higher rate. The seriousness of this, particularly in the last 2 years, which showed a precipitous increase, is obvious for this small population. They are isolated and highly dependent on the resources of the sea, and they are also distant from hospital facilities that can treat severe cases. Tuvalu is another small Pacific country with elevated rates of ciguatera, 484/100,000 annually from 1979 to 1983, based on a population of 7,600. On the single island nation of Niue, ciguatera was a more serious problem following World War II (Bagnis, 1973b), but ciguatera has been declining in recent years as it has in the French Territory of Wallis and Futuna (Table 1).

In Micronesia, ciguatera was a problem for both Japanese and American forces during World War II, and this focused scientific attention on ciguatera. Until 1982, statistics, for what is now the Trust Territory of the Pacific Islands, were aggregated. The annual incidence rate for the unit as a whole from 1979 to 1983 was 114/100,000. This is a decline from the period from 1973 to 1978 (232/100,000), which may in fact reflect a general decline in morbidity reporting that came with decentralization in mid-1978. The individual units show significant differences based on the 1982-83 statistics (Table 3). The 2/100,000 in the Federated States of Micronesia undoubtedly represents underreporting. Palau reported no cases, but the residents of Palau have long prided themselves on the fact that their fish are safe to eat. Earlier reports (Hiyama, 1943; Pacific Islands Territory High Commissioner, 1961; Banner and Helfrich, 1964) indicated that in the Caroline Island chain (with Palau to the west and the Federated States of Micronesia arching to the east), toxic fish were found in the eastern islands but absent in the west. The incidence for the Marshalls, 282/100,000 annually, was the highest reported from the former Trust Territory and may still represent significant underreporting. Ciguatera incidence in the Marianas archipelago, including Guam, is apparently increasing (Tables 1, 3).

Incidence is also low on the independent phosphate rich island of Nauru, 15/100,000. The low incidence may reflect poor reporting or reduced fishing effort. The Nauruans have one of the highest per capita incomes in the world and are highly dependent on imports. In Kiribati, ciguatera is a significant and an apparently increasing problem, with an annual incidence of 462/100,000, from 1979 to 1983. Kiribati is also one of the newly independent states of the Pacific facing the greatest development challenges. It is an atoll nation with limited land area, rapidly growing population (densities in urban Tarawa reach 1,137 km²), and there are very limited natural resources.

Less information on ciguatera is available from the Melanesian realm. By and large, these are larger, less developed islands with both interior and coastal populations. In general, they are less dependent on the resources of the ocean. Papua New Guinea, with 61 percent of the region's population, has been excluded from the regional computations. The population of Papua New Guinea is widely distributed in the Highlands and other interior locations. Many are not dependent on marine resources and morbidity reporting is very

Table 2.—Species perceived as commonly toxic in selected Pacific Islands (Lewis, 1984b).

Species	Marquesas: Hiva Oa	Marquesas: Tahuata	Marquesas: Fatu Hiva	Marquesas: Nuku Hiva	Marquesas: Ua Pou	Marquesas: Ua Huka	Tuamotus: Takaroa	Tuamotus: Manihi	Tuamotus: Rangiroa	Tuamotus: Other	Societies: Tahiti	Societies: Moorea	Societies: Huahine	Societies: Raiatea	Societies: Bora Bora	Samoa: Tutuila	Samoa: Upolu	Samoa: Savaii	Other: Tonga	Other: Fiji	Other: Vanuatu	Other: New Caledonia	Other: Hawaii
Gasterteidae																✱	✱	✱	✱				
Clupeidae																✱	✱	✱	✱				
Lycodontis sp.	●	●	●	●	●	●	□	□			■			■		✱	✱	✱	✱	✱	✱	✱	✱
Sphyraena barracuda	●		●		●	●	□	□	□	□	■	■		■		✱	✱	✱	✱	✱	✱	✱	✱
S. forsteri							□									○	○		✱				
Gymnosarda unicolor		●		●																✱	✱	✱	
Mugilidae											■									✱			✱
Acanthocybium solandri																					✱		
Scomberomorus commerson																					✱	✱	
Caranx sp.		●	●				□		□		■	■	■	■		○	○		✱	✱	✱	✱	✱
Seriola dumerili																							✱
Plectropoma maculatum																							✱
P. leopardus							□	□	□	□	■	■		■	■								✱
P. laevigatus																							✱
Variola louti			●											■							✱	✱	✱
Serranidae																○	○	○			✱	✱	✱
Cephalopholis argus	●		●	●			□	□	□		■	■	■	■							✱	✱	✱
Epinephelus cauvina	●		●				□	□		□	■	■		■									
E. coeruleopunctatus	●	●												■							✱		
E. fasciatus	●																				✱	✱	
Epinephelus sp.																							
Lutjanus monostigma																✱	✱	✱			✱	✱	✱
L. bohar	●	●	●	●	●	●	□	□	□	□	■	■	■	■	■	○	✱		✱	✱			
L. rivulatus											■	■	■	■	■	✱	✱	✱			✱	✱	✱
L. argentimaculatus																					✱		
L. gibbus	●	●	●	●	●	●	□	□			■	■		■		○	○		✱				
L. fulviflamma																					✱	✱	
Lutjanus sp.																							✱
Aphareus furcatus																							✱
Aprion virescens			●						□														✱
Monotaxis grandoculis				●	●	●	□	□			■												✱
Gnathodentex aureolineatus	●	●	●	●			□	□															
Diagramma picture																					✱	✱	
Lethrinus miniatus	●	●	●								■	■		■							✱	✱	
Lethrinus sp.								□															
Mullidae																○	○		✱	✱	✱		
Cheilinus undulatus							□	□	□	□	■			■	■								✱
Cheilinus sp.																							✱
Scaridae	●	●	●	●	●	●	□	□	□	□	■			■	■								✱
Chlorochaetus cyanognathus	●	●	●	●	●	●																	
C. strigatus							□	□	□	□	■	■	■	■	■								
Acanthurus olivaceus			●																				
A. achilles			●																				
A. lineatus																							✱
Acanthurus sp.																							
Naso brevirostris	●																						
N. lituri	●		●																				
Balistidae	●	●	●	●	●	●	□	□	□	□	■	■		■					✱		✱		
Balistoides viridescens							□	□	□														
Arothron sp.																○	○						

Table 3.—Ciguatera breakdown for Trust Territory of the Pacific Islands, 1982-83.

Islands	No. of cases 1982	No. of cases 1983	1982-83 rate per 100,000
Federated States of Micronesia	6	4	2
Marshalls Islands	100	88	282
Commonwealth of the Northern Marianas	17	31	130
Palau	0	0	0
Total	117	120	91

-oor. Inclusion of Papua New Guinea would only magnify underreporting in he region as a whole. Reports from the Solomon Islands are almost as poor. The reported incidence of 2/100,000 undoubtedly reflects underreporting, but t is difficult to get an accurate picture of the situation there. Somewhat better nformation exists for both Vanuatu and New Caledonia. In Vanuatu where morbidity reporting is poor and the annual reported incidence was only 19/100,000, Ilagnis (1977) estimated an incidence ate of 130/100,000. More recently, Bailllo (1984) estimated that there were 500 cases a year (512/100,000), thus Vanuatu would be experiencing the mean for the Pacific. The Melanesian island of New Caledonia is one of the places in the Pacific where there has been an apparent decline in ciguatera incidence over the last several years. Known locally as "la gratte" for the intense itching that is often one of its symptoms, ciguatera incidence was high from 1975 to 1978 (Table 1) and has declined since then. While reported ciguatera incidence is low in Fiji, researchers at the University of the South Pacific Institute of Marine Resources, led by U. Raj, have been monitoring the situation in the Fiji group. More than 17 species were confirmed to be toxic either experimentally or from medical records (Raj et al., 1982). Discussions with both researchers at the Institute of Marine Resources and medical staff indicate that ciguatera is indeed a problem and may be on the increase. Researchers Yasumoto et al., 1984) found two different toxins in the roe and another in the viscera of *Etelis carbunculus*, a deepwater snapper from Lauthala Island. The chromatographic properties of the toxins were different than those of ciguatoxin.

Hawaii continues to experience ciguatera outbreaks. Between 1975 and 1981 there were 81 outbreaks involving 203 individuals, giving an annual rate of 3/100,000 (Anderson et al., 1983). The authors estimated that this represented about 10 percent of the actual intoxications. In 1982, incidence was low with only 18 cases reported; 51 were reported in 1983 and 80 in 1984. It is tempting to speculate that the increase in 1984 was due to damage caused by hurricane Ewa which hit Hawaiian shores in November 1983. While the evidence is not conclusive, at least 50 intoxications have been reported from Kauai during the first 4 months of 1985. Kauai was the hardest hit of all the islands in the Hawaiian chain. In Australia, too, the ciguatera problem is apparently escalating. For decades, episodes of ciguatera poisoning have been reported sporadically from the great Barrier Reef of Northern Queensland. Reef species have usually been responsible, including the commercially important *Plectropoma maculatum*. However, since 1976, fish from Southern Queensland, in the vicinity of Hervey Bay, have been incriminated. It is the commercially important pelagic *Scomberomorus commersoni* or Spanish mackerel that is the main species responsible. Lewis and Endean (1983) have isolated the ciguatoxin-like substance from Southern Queensland *S. commersoni*.

The information that we have on the distribution of ciguatera in the Pacific basin comes from incomplete morbidity reporting and a few circumscribed, intensive investigations of the distribution of both *G. toxicus* and toxic fish. The information is far from complete and the fact that few cases are reported from the large less developed Melanesian island groups in the western Pacific, Papua New Guinea, the Solomons, and Vanuatu must be viewed with the realization that morbidity reporting for all causes from these groups is very poor. Despite this, it does seem that ciguatera is a more serious and escalating health problem in the eastern Pacific, notably French Polynesia, and in several of the more isolated island groups of the north central Pacific, Tuvalu, Tokelau, Kiribati, and the Marshalls. The anomalies in the Pacific distribution of ciguatera also deserve attention, e.g., the Cook Islands. One fruitful research frontier that may provide clues to help to unravel the ciguatera mystery is comparative analysis in the Pacific and Caribbean. It is disturbing that, in the Pacific, some of the most vulnerable island groups are the ones experiencing an elevated incidence of ciguatera.

Current Impact in the Pacific Region

While ciguatera remains a problem for islanders living in subsistence communities in the Pacific, its impact is exacerbated by the changing nature of Pacific residence, life-style, fishing, and marketing. Excluding the large, less developed islands of Melanesia, where ciguatera may generally be less of a problem, many Pacific nations have from 40 to 100 percent of their population living in the main urban center. Wage labor, often for the government, is common and individuals are moving farther and farther away from subsistence life-styles and modern aspirations are replacing traditional ones. At the same time, Pacific populations are growing, migration to the urban center continues, reef and shore modification projects are undertaken, particularly near urban centers, and there is an increased likelihood for the creation of ciguatoxic biotopes in the vicinity of the highest population densities. Populations are becoming increasingly dependent on the purchase of fresh fish in urban markets and fish export is being encouraged. I have written at length about how island dwellers have adapted to the existence of ciguatera, beliefs about etiology and strategies for detection, prophylaxis, and cure (Lewis, 1981, 1983, 1984b). Traditional strategies, relatively effective in subsistence situations, become less so in urban areas, and when export from the point of capture is commonplace.

Morbidity statistics underline the importance of ciguatera as a public health problem in the Pacific. Except in the most highly toxic locations, it is exceedingly difficult to assess the impact of ciguatera on community nutritional status. Disease patterns are now emerging in the Pacific that mirror those of the developed world with elevated rates of diabetes, hypertension, and cardiovascular disease. Furthermore, there may be a genetic predisposition to these conditions, at least in Polynesian populations (Prior, 1981). Dietary patterns that rely on western processed foods exacerbate the health risks in these vulnerable populations, and ciguatera may increase the tendency to rely more heavily on imported foods, both canned fish and meat and other refined, high-fat and carbohydrate foods. While it is still difficult to unravel the many factors involved, it may be easier to determine the impact of ciguatera on small-scale fisheries development.

By and large, local small-scale commercial fisheries are very poorly developed in the Pacific. In the region as a whole, the pelagic catch by Asian fleets is many times greater than the local fishery. With the establishment of the 200-mile Exclusive Economic Zones, island nations have begun, not without problems, to participate in this fishery, primarily through licensing agreements and joint venture projects. This participation is obviously important for economic development, but is does not obviate the need for the development of local small-scale commercial fisheries. There are many reasons to encourage subsistence and artisanal commercial fisheries development and the consumption of fresh fish, from benefits to the local economy, to a reduced dependence on imported food.

Accurate, comparable statistics on local catch, both subsistence and small-scale commercial fisheries, are very difficult to obtain. In the late 1970's, for example, in American Samoa, based on fish catch assessment surveys, local production was estimated to be 316,338 kg/year. Some other annual catches include: Cook Islands 606,654 kg; Fiji 5,030,362 kg; French Polynesia 2,885,667 kg; Tonga 1,039,089 kg, and Western Samoa 1,307,955 kg (this last includes subsistence catch only) (Lewis, 1981). Kent (1980), citing 1977 FAO data, gives local catch from the region as a whole as 82,696 t. This is approximately one-tenth of 1 percent of the total global catch and, while proportional to the region's population, is very low considering access to fish stocks. This figure includes local pelagic catch but not subsistence catch. Salvat (1980) estimated that the reef and lagoon catch for the region as a whole to be as high as 100,000 t. Nonetheless, in many Pacific locations as much as 90 percent of the fish eaten comes out of a can. It is difficult to generalize, but while overfishing and reef depletion have occurred where population densities are high, in the region as a whole, more fish could be taken on a sustained yield basis.

Ciguatera has been only one factor, and probably a relatively minor one, in fisheries development in the Pacific. With the changing nature of fish marketing and export, however, ciguatera or the specter of ciguatuera may have increasing impact. Many factors have contributed to change in traditional dietary patterns, to different agricultural and fishing practices, and to a dependence on imported food. One result has been that canned mackerel and sardines are often considerably less expensive than fresh fish. In 1977 the price of the least expensive fresh fish in the Cook Islands, French Polynesia, and Fiji ranged from (Australian) $1.34 to 1.50/kg. The price of canned mackerel (424 g), all of which is edible, was 43-48 Australian cents (A$ 1.00 = US$ 1.14). It is also widely distributed, relatively nutritious, and keeps without refrigeration. Furthermore, though the majority of Pacific islanders would respond that they prefer fresh fish, canned fish is a popular, easy, and relatively inexpensive protein source.

What evidence is there from the Pacific to suggest that ciguatera has affected or may affect the market for fresh fish? Urban consumers have a more generalized fish avoidance, e.g. "all the red fish" or "all the very big fish," and highly suspect species, *Lutjanus bohar*, *Lycodontis* (*Gymnothorax*) *javanicus*, or new or unfamiliar species, than individuals in subsistence situations (Lewis, 1981). This makes sense, as the urban consumer is commonly further removed from knowledge of the marine realm, may have no knowledge of who caught the fish, most likely will not know where it was caught, and, if it is filleted, may or may not know what species it is. Despite this, the ultimate responsibility for choosing a "safe" fish usually rests with the purchaser. A publicized outbreak of ciguatera associated with fish purchased from the urban market will have an adverse effect on the future marketability of that species, as it did with a catch of toxic *L. bohar* sold in the municipal market in Apia, Western Samoa. A gift to a church group of a large barracuda from an Asian longliner offloading at one of the canneries in American Samoa had a similar effect. In a follow-up study (Lewis, 1981), all those who had been poisoned said that they would avoid barracuda, and the church group voted not to serve it again.

Of all the intoxications reported in Tahiti in 1976, 52 percent were from fish caught outside of Tahiti and 18 percent were of unknown origin. Of those caught outside of Tahiti, 73 percent were caught in the Tuamotus. The population of French Polynesia is concentrated in the Papeete urban zone and the local reef resource is depleted. There is a ready market for reef fish in Papeete. Fish from several of the atolls in the Tuamotus to the north find their way to the urban market. Inhabitants on one atoll, no longer exporting fish, blamed an outbreak of ciguatera in the early 1970's for the cessation of their export. A change in interisland schooner schedules may have been equally responsible. As the statistics indicate, the fish are sometimes toxic.

Ciguatoxicity is not the only potential health problem associated with the consumption of these fish. They may be 6 days old and are often poorly iced. While the fish are not in prime condition, given the shortage of local reef fish and food preferences, they sell. This is probably the same reason that potentially ciguatoxic fish still have a market. Although one assumes that at some point if intoxications become more common or more severe, it will affect the marketability of these fish.

The Northern Line Islands of Kiribati, three equatorial islands 1,800 km south of Hawaii, loom large in that government's development plans. When there is regular airline service from Christmas Island to Honolulu, reef fish as well as spiny lobster are exported to Hawaii. Ciguatera has been a problem in the past and some species, primarily high-level carnivores, are toxic at the present time.

The potential for ciguatoxic biotopes may increase with proposed resettlement schemes. Ciguatera could also adversely effect the government's attempts to establish a sport-fishing-

oriented tourist trade. Until recently, the offshore bottom fishery (100-300 m) was poorly developed in the Pacific. It is receiving increasing attention, but some of the species caught can be ciguatoxic (Crossland, 1980).

Individuals in institutions may be at greater risk of intoxication. Large fish may be relatively inexpensive, perhaps because they are suspect, and hence find their way to institutional kitchens. In both Vanuatu and Fiji, inmates in the local jail were poisoned in the late 1970's. In 1980 in Kiribati, 54 students at the government boarding school were poisoned after a meal of barracuda (Marriott and Dalley, 1980). Large feasts are an important component of social interaction in the Pacific. The American Samoan example cited previously is only one where a single large fish prepared for a feast resulted in a common source outbreak.

Isolated populations, not only indigenous populations on outer atolls, but the crew aboard vessels or military personnel at isolated outposts can also be at risk. In 1982, 13 U.S. Navy personnel on Midway were evacuated to Hawaii after consuming a toxic *Seriola dumerili*.

Ciguatera can be a threat to the hotel and restaurant business in particular and tourism in general. The results of intoxication may include loss of business for the individual establishment and potentially for a circumscribed locale if the problem is severe enough. In the United States there is an added risk, given the litigiousness of our population.

The difficulty of assessing, except in the most highly toxic locations, the impact of ciguatera on fisheries and fisheries development has been stressed. In some parts of the Pacific, ciguatera may have been used as a convenient excuse for the lack of development. The threat of ciguatera may be as damaging as the actual incidence of intoxications. Scientific interest in the phenomenon has increased in recent years. In addition to articles in research publications, it has increasingly been the subject of letters to the editor, editorials, and articles in clinical journals in the United States, Canada, the United Kingdom, Australia, France, and the Pacific islands region. Many of these articles have alerted physicians in the United States or Europe to the fact that a patient returning from a tropical vacation with a perplexing set of symptoms may in fact be suffering from ciguatera.

Ciguatera has also become a subject of interest in the popular press. In an article in the *Australian Financial Review* (1984), the author commented that ciguatera was worrying both medical authorities and tourist promoters throughout the South Pacific. Even a few cases of ciguatera can have a drastic effect on the use of reef resources and fish avoidance can have an adverse economic impact on small-scale fisheries. In Hawaii, there is a generally increasing awareness of the problem, and interest in the origins of the red snapper that is commonly sold in large supermarket chains. It is carefully identified as New Zealand red snapper, undoubtedly because of this.

The research community, clinicians, fisheries officers, and the general public are becoming more aware of the existence of ciguatera. This can obviously have many positive effects from increased research funding, to correct diagnosis and appropriate detection and control strategies. However, care must be taken that the magnitude of the problem not be exaggerated. Tatnall et al. (1980) present a case where a West Indian was poisoned after consuming dried fish brought back from Antigua. They suggest that control in Britain rests on strong discouragement by such bodies as the Fishmongers' Company of commercial importation of moray eels and potentially ciguatoxic fish, such as amberjack and barracuda. Wholesalers do have a responsibility that should be self-evident. In the United States the potential for legal action elevates the stakes. The radioimmunoassay testing of amberjack (kahala) in Hawaii was successful; there were no intoxications from commercial purchased amberjack during the test period, but it was prohibitively expensive and also resulted in false positives. What is needed, of course, is a more complete understanding of the etiology, ecology, and epidemiology of ciguatera and a simple, inexpensive test to determine toxicity. Until then, we will have to rely on correct species identification, care to avoid both internal sale and export of toxic species, communication, and cooperation among fishermen, wholesalers, retailers, and the scientific and medical community.

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L.A. TIMES late January 1982

The dangers of eating raw fish

Physicians warn of parasites harbored by uncooked seafood, meat

By Mimi Sheraton

NEW YORK — The reported dangers of illness brought on by eating raw fish and meat often cause fear and confusion in the minds of those who enjoy such foods, either for their aesthetic or nutritional benefits.

Among foods posing such questions are clams and oysters, which can cause hepatitis if taken from polluted waters, and rare or raw meat in the form of steak tartare or beef *carpaccio*, which can transmit beef tapeworms to humans. Recognizing the possibility of infection, but uncertain of the likelihood, some people forgo the suspected food entirely, some continue eating it, ignoring the dangers, and many eat it and worry.

Most recently, such concern has been expressed over the dangers of eating raw saltwater fish in the forms of Japanese *sushi* and *sashimi*, Scandinavian *gravlax*, South American *ceviche* and raw or green herring, which is popular in the Netherlands.

When raw, most saltwater fish that are eaten can be carriers of anisakis simplex, a parasitic roundworm that may invade the gastrointestinal tract of humans, causing mild to serious complications. But again, the warnings of parasitologists and physicians are difficult to reconcile with the absence of known infection.

"So far we have not found any anisakis," said Dr. Edith Newman medical director of the Jetti-Katz Clinical Laboratory, the leading facility in New York City specializing in testing for gastrointestinal parasites. "But then we have never specifically looked for it."

Because raw fish in its various forms is increasing in popularity, the New York City Health Department will begin a full-scale investigation late next month to determine the incidence of parasite infection in the local fish supply. Jean Cropper, deputy commissioner for environmental services, said last week that fish in the city's wholesale and retail markets and restaurants would be examined for all possible parasite infection.

Mimi Sheraton writes for the New York Times.

Attention was focused on the dangers of such parasites last fall, when the Centers for Disease Control in Atlanta reported four cases of infection by the fish tapeworm diphyllobothrium, traced to the eating of raw salmon.

In investigating 10 samples of salmon in Seattle last July, inspectors for the Food and Drug Administration found that although those particular samples contained no tapeworm larvae, they did contain the anisakis larvae. Salmon, an anadromous fish, lives in both fresh and salt water, and so can contain both types of parasites. The dangers of tapeworm from freshwater fish have long been known and recognized and almost no freshwater fish is used for sushi and sashimi, except on some rare occasions in Japan.

Gravlax, which is only lightly salted, can be a cause of both types of parasite infection. In this country, the incidence of tapeworm infection is restricted primarily to those of Scandinavian descent and to Jewish women who taste raw freshwater fish such as carp, pike and whitefish in preparing gefilte fish. Freshwater fish is generally considered safe if it has been properly salted and smoked, or cured in a heavy salt brine, as for lox.

Anisakis simplex, however, is virtually unknown in this country, and because it is hard to identify, or not even examined for, it may go undiagnosed. Even if it is diagnosed, it is not a disease that must be reported, and, unlike tapeworm, cannot be cured by medication, another reason it is hard to document.

Dr. George J. Jackson, a parasitologist who is the acting branch chief for food and cosmetic microbiology with the FDA, said, "We have seen only a slight increase in the number of anisakis cases here, but I think they are just the tip of the iceberg. Symptoms may be too mild to notice or may be misdiagnosed, most often as appendicitis or stomach cancer."

The infection can take several forms, as described by Dr. Ronald R. Roberto, deputy chief of the infectious disease section of Department of Health Services of California.

"Man is not a definitive host, so the larvae will not grow or increase in number," Roberto said. "In the mildest and most frequent cases, the worm lodges in the throat and is coughed up. If it invades the intestinal wall it can cause inflammation, nausea, cramps and diarrhea and will last anywhere from a day to a week, after which it will be over.

"In the most serious cases," he continued, "it penetrates the intestinal wall and enters the peritoneal cavity, where it may cause chronic lesions or prompt white blood cells to gather around to fight it off, thus forming a granuloma, which is often mistaken for a tumor. Then if surgery is performed, the worm can be found within the granuloma. Nevertheless, we do not consider it a major problem, in spite of the potential. But we will probably see more of it as the eating of raw fish increases."

Symptoms of gastric anisakiasis are said to develop within four to 12 hours of eating contaminated fish (intestinal anisakiasis will develop within seven days).

There is no cumulative effect from eating raw fish. It is possible to eat 100 pieces and not get anisakiasis if it is not present in those pieces, and it is possible to become infected from eating only a single piece that contains larvae, usually about 1 to 1½ inches long.

In a report on anisakiasis written in 1976 for the Armed Forces Institute of Pathology, Ronald C. Neafie and John R. Dooley, both parasitologists, said, "Infected fish are commonly marketed in this country and elsewhere; for example about 90 percent of cod fillet sold in and around Washington, D.C., contain anisakine larvae." They recommended that "public health education should discourage the eating of raw or inadequately prepared fish or squid."

At various times, contaminated fish have been found off the waters of Massachusetts, and in 1970, the Beltsville Parasitological Laboratory in Maryland reported that infected fish were found in markets in Connecticut, Maryland and New York, as well as in fish from Canada. Fish from northern waters are generally considered more likely to have anisakis simplex larvae than those from tropical waters, and the proximity to mammals such as whales, seals and certain strains of porpoises also increase possibilities because those animals are important to the life cycle development of the parasitic nematodes.

Fish that have the anisakis larvae do not necessarily transmit it to humans. While the fish are alive, the worms live in their stomachs. Once the fish are dead, the larvae penetrate the tissue that is eaten.

It is safest, therefore, to gut fish as soon as they are caught to minimize chances of the larvae's spreading. It is also possible to remove anisakis larvae from fish as it is being cut, because such larvae are visible to the trained eye, especially those of experienced sushi cutters.

To be safe, however, responsible sushi shop owners buy only the

Raw food/D-17, Col. 1

Raw food

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freshest fish and never serve fish in which larvae were discovered. The most susceptible fish, such as mackerel or squid, are not served completely raw.

According to Ryudaburo Kawada, the owner of the Takezushi and Kurumazushi restaurants, all mackerel is salted and then marinated in strong vinegar and squid is purchased frozen, a process that kills the parasite. Kawada also said that all bonito is infected and so is rarely ordered by non-Japanese and the susceptible yellowtail is also purchased frozen.

Shrimp, eel and octopus are always cooked for sushi. Nevertheless, fluke, porgy and sea trout are traditionally served raw and all have been found to have anisakis at one time or another. There are no reports citing tuna as a carrier.

Because of an outbreak of the parasite a few years ago in the raw herring favored in the Netherlands, the law there now requires that all herring must be gutted immediately, then salted and frozen at 4 degrees below zero within 12 hours of being caught and for 24 hours thereafter. Such freezing, or heating to 140 degrees, kills the larvae.

But though anisakis simplex is known in Japan, it is not considered dangerous by most experts there and no regulations have been established. Dr. Noboru Kagei, chief of the second division of parasitology at the National Health Institute of Japan in Tokyo, said that the number of cases is considered negligible.

Nevertheless, there has been some increase of infection reported, most notably in the Ohita prefecture, where 42 cases were reported in 1980 as against 12 in 1977, an increase possibly a result of the eating of raw mackerel by a group of men at a party.

Acknowledging that he eats raw fish often and that doing so is a custom in Japan, Kagei said it is difficult to require all fish to be frozen because so many varieties are eaten raw. But he also stated that he believes that some regulations are needed.

Putting the matter in perspective, Roberto said, "There's no point in doing fish market surveys. The parasites are there all right and they can't be cleared up. You can't treat fish. People who like to eat raw fish because they enjoy it or feel it is healthful should be aware of the risk, and go only to good clean places and if they feel any gastric symptoms should see a physician."

George cy FYI

Wilmot B. Boone MD
Midway Island Dispensary
Midway Is. FPO 96614

2 August 1982

William G. Gilmartin, Leader
Marine Mammal and Endangered Species Investigation
National Marine Fisheries Service
Southwest Fisheries Center
P. O. Box 3830 Honolulu, Hi. 96812

Dear Sir:

Your letter and accompanying documents, encompassing current investigations & planning of studies in the Ciguatera area, and particularly the 3rd & 4th paragraph on page 8 of your article on phocid seal feeding experiments, are much appreciated.

I have been seeing patients with Ciguatera in many parts of the Pacific where I have worked during the past 25 years, and the vagaries of this toxin intrigue me tremendously. Here, in five months, I have seen at least 20 cases of very mild intoxication among our foreign employees who refused to believe the reef fish could be toxic---until empirically they learned their bitter lessons.

Since you are coming next month I have been particularly interested in viewing the local situation from the standpoint of the dinoflagellate population; of the many lagoons and reefs I have lived about, I have never seen the utter devastation of coral life as seen here. I suspect that 95% or more of the lagoon corals are dead, and covered with algae; I have swum as far and over as much area as I can, and find the water heavily clouded with algaform or other marine life; now everywhere the remaining detritus from dead gooneybird young, failing in their adaptation to their life on water, litters the surface; and the sewer outfall, with its heavy phosphorous load, all must add up to a nourishing media for dinoflagellate growth. I am wondering if the cyclic contribution of the dead birds, coincident with the warmer season, could locally trigger specific toxic forms of marine growth.

Also, as I had asked Ed Bowlby, if it may be of any value, I shall be happy to preserve (in our morgue) or autopsy any seals found dead & in condition for studies; but before I attempt any such tasks proper authorization would be needed, I presume.

Anticipating your arrival here, I am

Sincerely yours

Wilmot B. Boone MD

Ciguatera Fish Poisoning in Hawaii, 1981

Bruce Anderson taking samples from a black ulua caught during the Harrison Dudoit Annual Fishing Tournament on Molokai following an outbreak of ciguatera poisoning.

by Bruce S. Anderson, Epidemiology Consultant

Several other articles on ciguatera fish poisoning have appeared in *Hawaii Fishing News*. The following is not intended to be a comprehensive review of the problem (see also the September and October 1981 issues) but rather to bring the sports fisherman up-to-date on ciguatera poisoning in Hawaii and ways of minimizing the chance of illness.

Fish poisoning can result from eating spoiled fish or from toxic substances in fresh fish. Various different types of fish poisoning are recognized; some types are associated with specific fish, for example puffer fish poisoning and hallucinatory fish poisoning from mullet or weke; others occur when certain fish (mahimahi, ahi or aku) are eaten after they have spoiled (scromboid or histamine poisoning) usually because of improper refrigeration or storage. Ciguatera fish poisoning, perhaps the most serious of all, is a natural occurrence still not well understood.

Ciguatera poisoning is thought to have been unknown to early Hawaiian fishermen. Until recently most cases of illness were from fish brought in to Hawaii from other areas of the Pacific where it was common, from Johnston Island for example. Today in Hawaii ciguatera poisoning is a serious problem for both recreational fishermen and the already struggling fishing industry.

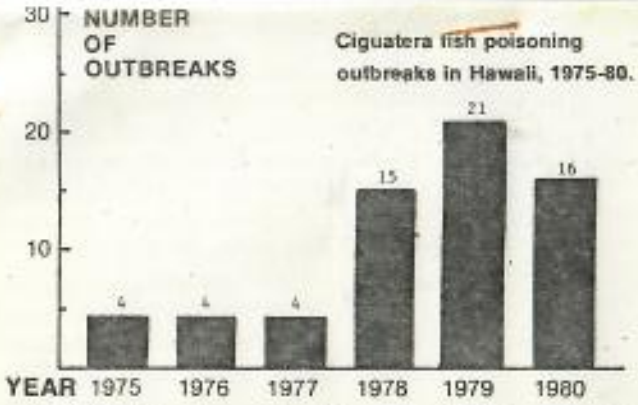
government funding. In fact, now there is no fish testing program whatsoever in Hawaii. Undoubtedly, these numbers grossly understate the actual number of fish poisoning incidences as they represent only those cases which were reported to the Department of Health.

Many species of fish which are highly esteemed as food have been implicated as causing serious illness. Ulua (jacks) and kahala (amberjacks) are at the top of the list as they have been most often responsible for ciguatera poisoning in Hawaii. Many other reef fish have been found to be toxic and are illustrated here.

The toxin (ciguatoxin) is produced by a microscopic marine organism, a dinoflagellate called *Gambierdiscus toxicus*, which grows on the surface of marine algae. Since the toxin accumulates in fish, beginning in those that eat the algae, any fish down the food chain can be toxic. Those fish that feed directly on the algae or coral reefs upon which the algae grows, the palani (surgeon fish), uhu (parrot fish), and others, may therefore be toxic, and the larger carnivorous fish that prey on them

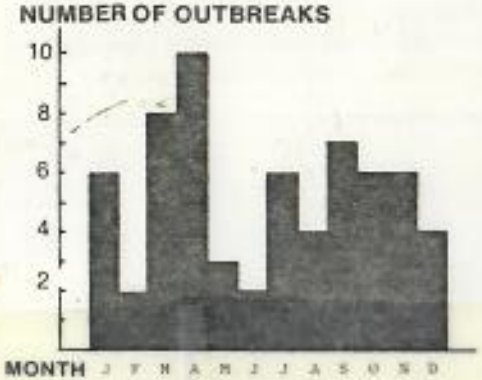
As a reminder, there are a few simple guidelines that, if followed, minimize the risk of being poisoned:

1. There are no reports in Hawaii of ciguatera associated with deep-sea fish such as ahi and aku (tuna), marlin, mahimahi or ono (wahoo). These fish should be safe to eat if they are fresh. Almost any reef fish though can be a potential carrier of ciguatoxin.
2. Because the toxin accumulates, larger predatory fish are more often toxic than smaller fish of the same species. Therefore, avoid eating large portions (of ulua or kahala for example) during the first meal.
3. DO NOT EAT THE ROE (EGGS), LIVER OR HEAD OF ANY REEF FISH. The toxin is concentrated up to 100 times more in these organs than in other parts of the fish. In fact, the only known deaths from ciguatera in Hawaii were caused by eating the viscera of toxic fish



Ciguatera fish poisoning outbreaks in Hawaii, 1975-80.

A total of 64 outbreaks involving at least 142 people were reported to the Department of Health during this 6-year period.



Combined number of ciguatera outbreaks by month in Hawaii, 1975-80.

Ciguatera outbreaks are difficult to predict or control. From 1900 to 1974 (a period of 75 years) a total of approximately 59 outbreaks of ciguatera were reported with at least 450 people becoming ill. Since 1974, 73 outbreaks have been reported (including 9 so far in 1981) involving at least 190 people (including 48 in 1981); thus, there have been more outbreaks (but fewer people involved) in a 5-year period than had ever been previously reported in Hawaii. The frequency of outbreaks has generally been increasing in recent years, although the numbers may be leveling off. A slight decrease in the number of reported outbreaks in 1980 (16) may be in part due to the effectiveness of a fish testing survey conducted by the National Marine Fisheries Service Program. This program, however, was recently discontinued because of a lack of support and herbivores, ulua (jacks), kahala (amberjacks), uku (snappers) and eels, for example, may be even more dangerously toxic because of this cummulative effect. None of the offshore palagic fish, ahi or aku (tuna), marlin, mahimahi or ono (wahoo), have been found to be toxic because the dinoflagellate grows only in shallow water.

An interesting phenomenon has been observed which may be significant in predicting outbreaks of ciguatera in Hawaii and elsewhere. Sometimes outbreaks follow the destruction of coral reefs as in dredging, blasting, and the construction of breakwaters. For example, on Bora Bora an outbreak followed the underwater blasting of a channel through the reef into a lagoon. In Hawaii there were apparent outbreaks following the construction of the "reef runway" and improvements on the breakwater in Pokai Bay. Ciguatera outbreaks have also followed natural catastrophes such as severe storms or tidal waves. It is thought that the destruction of the coral ecosystem as a consequence of such events leaves a denuded surface upon which the dinoflagellate producing the toxin is the first to grow back. Eventually other marine organisms displace the toxin-producing dinoflagellate and the problem seems to disappear. There is no indication that ciguatera poisoning in Hawaii is related to the weather or time of year.

Unfortunately, there is no way of detecting a toxic fish from its appearance, smell or taste. The fish seem to be unaffected by the toxin they carry. The freshness of a fish does not relate to its toxicity. Furthermore, it is not possible to destroy the toxin by freezing, cooking in any fashion, drying or salting the fish.

One way to determine if a fish is toxic is to feed a small portion to the family feline.

In areas of the Pacific where ciguatera poisoning is more prevalent, the usual way of determining if a fish is toxic is to feed a small portion to a cat. Cats are particularly sensitive to ciguatoxin, but other animals are susceptible too. If the animal shows no sign of illness after a few hours, the fish is eaten. This method is reported to be very effective. If the amount of fish fed to a cat is less than approximately 1/10 of the cat's body weight, the cat will survive and recover from a toxic fish (it usually regurgitates most of the fish). Other animals, mongooses for example, have also been reliably used to test for toxic fish. Currently, animal tests such as these are the only means available to determine if a fish is toxic.

4. Clean all reef fish promptly and thoroughly.

The symptoms of ciguatera vary greatly from one individual to another, even when they have eaten the same amounts of the same fish. Illness usually occurs within three to five hours after eating a toxic fish.

Briefly, common symptoms includes nausea, vomiting, abdominal pain and diarrhea in most cases. A numbness or tingling sensation around the mouth which later extends to the limbs is also common. The victim usually feels weak with muscular aches and pain. A headache, dizziness and nightmares may or may not be present. A distinctive reverse temperature sensation, a painful tingling of the palms of the hands and soles of the feet upon contact with cold water is also often present. In severe cases the neurological symptoms are very pronounced and may lead to muscular paralysis, coma and death from respiratory paralysis.

If illness does occur after eating reef fish:

- CALL YOUR PHYSICIAN IMMEDIATELY FOR TREATMENT. If your physician is unavailable, call the Hawaii Poison Center, 941-4411. A physician's care is needed to remove the unabsorbed toxin from the gastrointestinal tract and to treat the symptoms. The disorder may last days, weeks or months. Extreme sensitivity to the toxin sometimes develops after being poisoned once.
- DO NOT TAKE aspirin or any drugs without your physician's advice.
- Save the remainder of the fish (including the head and guts) in your refrigerator or freezer to be given later to the Department of Health. These toxic fish are being used to develop reliable tests to detect ciguatoxin.

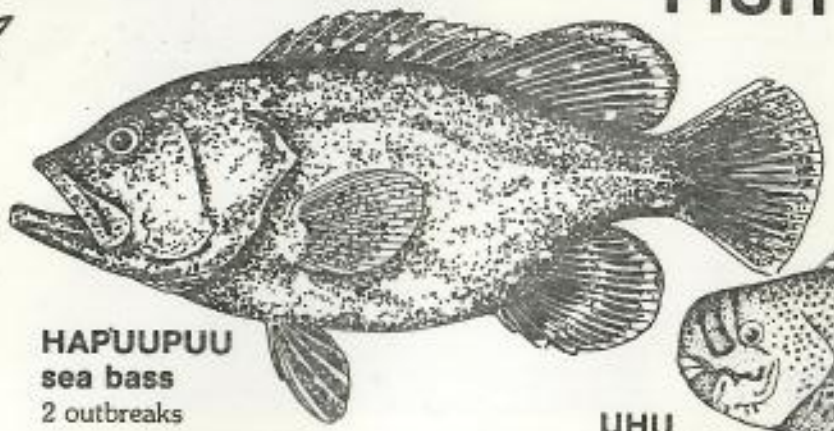
The present situation in Hawaii is frightening, especially because of the present lack of a good laboratory test to detect toxic fish. An advisory committee has been formed to investigate the problem and to recommend ways of dealing with it. Research work is continuing but drastic outbacks in funding have slowed progress considerably. Your support and a showing of concern to legislators and others responsible for the dispersement of funds is urgently needed if the problem is to be controlled before it gets worse. Please do not hesitate to call the State of Hawaii, Department of Health Epidemiology Branch (548-5986), or the Food and Drug Branch (548-3280) if you have further questions.

. . . Bruce

FISH IMPLICATED

in ciguatera fish poisoning in Hawaii 1975-1980

OPAKAPAKA
pink snapper
1 outbreak



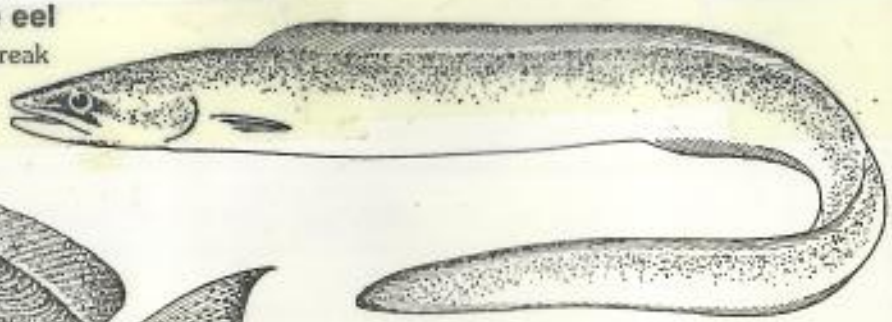
HAPUUPUU
sea bass
2 outbreaks

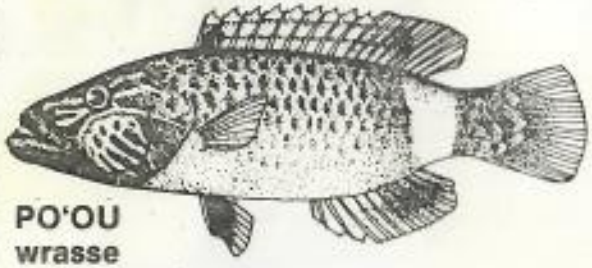
UHU
parrot fish
4 outbreaks

KUMU
goat fish
4 outbreaks

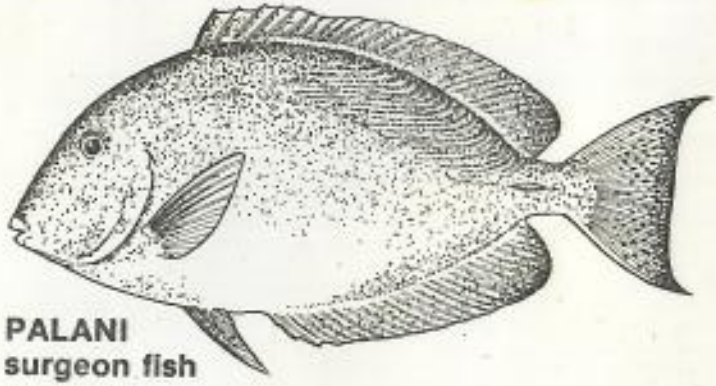
WEKE
goat fish
3 outbreaks

TOHE
white eel
1 outbreak

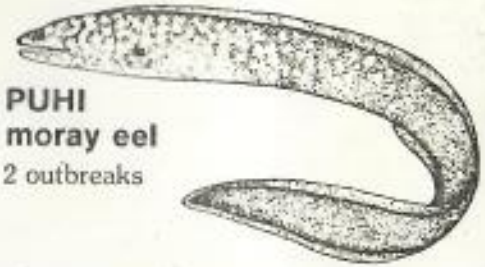




PO'OU
wrasse
5 outbreaks

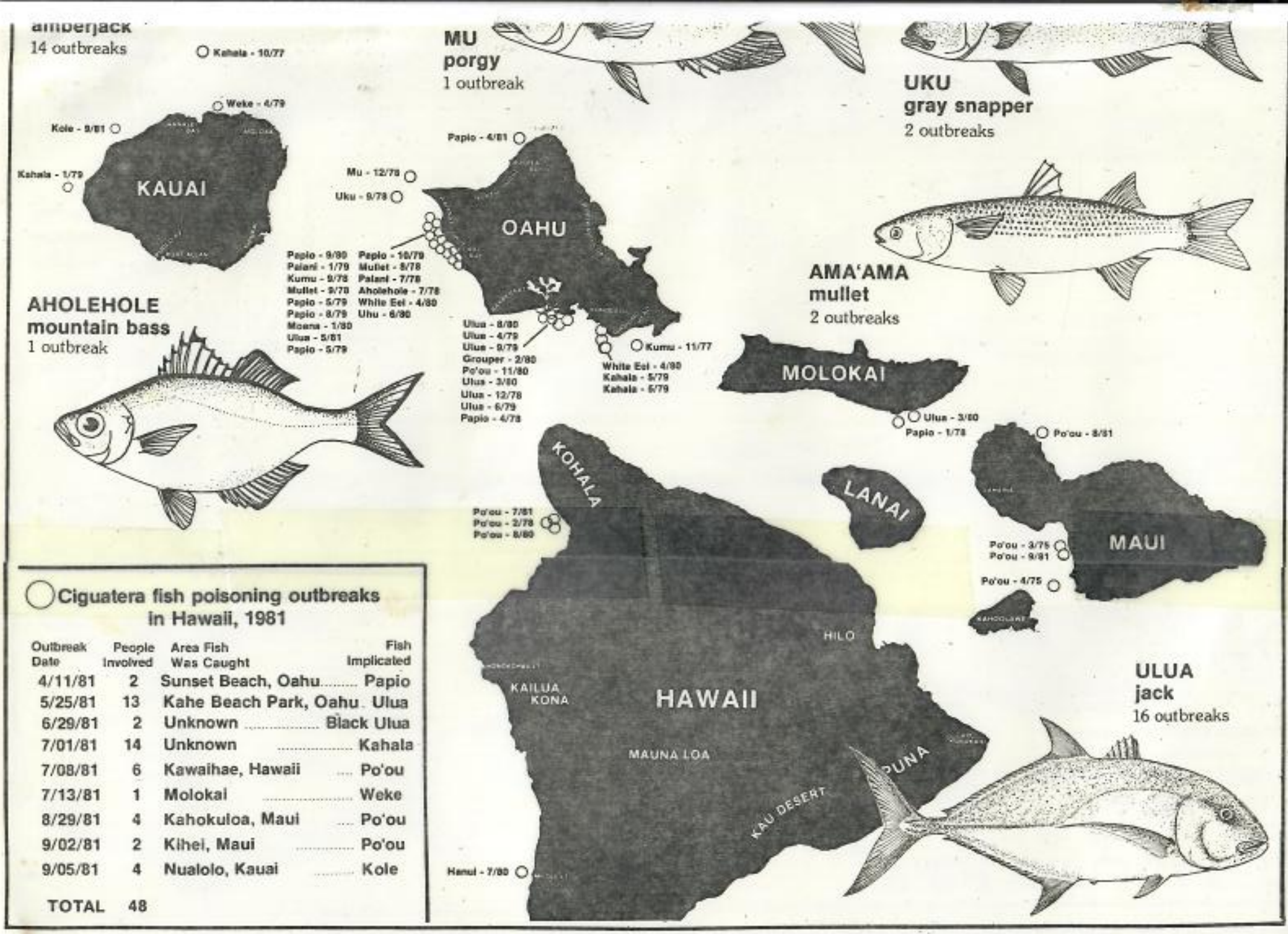


PALANI
surgeon fish
2 outbreaks



PUHI
moray eel
2 outbreaks

KAHALA



Ciguatera fish poisoning outbreaks in Hawaii, 1981

Outbreak Date	People Involved	Area Fish Was Caught	Fish Implicated
4/11/81	2	Sunset Beach, Oahu	Papio
5/25/81	13	Kahe Beach Park, Oahu	Ulua
6/29/81	2	Unknown	Black Ulua
7/01/81	14	Unknown	Kahala
7/08/81	6	Kawaihae, Hawaii	Po'ou
7/13/81	1	Molokai	Weke
8/29/81	4	Kahokuloa, Maui	Po'ou
9/02/81	2	Kihei, Maui	Po'ou
9/05/81	4	Nualolo, Kauai	Kole
TOTAL	**48**		

A PLAN FOR CIGUATERA RESEARCH IN THE PACIFIC

PREPARED BY THE

HONOLULU LABORATORY
SOUTHWEST FISHERIES CENTER
NATIONAL MARINE FISHERIES SERVICE, NOAA
HONOLULU, HAWAII 96812

ADMINISTRATIVE REPORT H-81-11
DECEMBER 1981

EXECUTIVE SUMMARY

The Problem

Ciguatera, a disease characterized by gastrointestinal and neurological symptoms caused by the ingestion of a variety of tropical marine reef fishes, is a long-standing problem in the many tropical and subtropical islands in the Pacific. Ciguatera spread to Hawaii and other central Pacific islands in the 1940s and the public health records show periodic outbreaks of the disease in Hawaii ever since.

The Program

What: The Honolulu Laboratory, Southwest Fisheries Center, National Marine Fisheries Service, NOAA proposes a multidisciplinary study of the ciguatera problem to solve the various (ecological, public health, pharmacological, etc.) problems associated with ciguatera.

Where: Field research will be carried out in various geographic locations in the Pacific, including the Hawaiian Archipelago, where ciguatera outbreaks may occur.

Who: Over the years many researchers in various disciplines have been working on the ciguatera problem in the Pacific. The program will continue to be a multidisciplinary effort and will involve scientists at the Honolulu Laboratory, various departments of the University of Hawaii, the Tohoku University of Japan, the Hawaii Department of Land and Natural Resources, and other research agencies.

How: Three overall objectives for ciguatera research were identified. A total of 22 research activities and subprojects were developed to achieve the objectives.

Goals and Objectives

The three overall objectives for ciguatera research are (1) to be able to "manage" the ecosystem, i.e., to predict the potential outbreak of ciguatera and to possibly interrupt the chain of events leading to potential outbreaks, (2) to develop a treatment for human ciguatera intoxication, and (3) to develop and implement a simple "dipstick" test for ciguatoxin and other toxins that can be used by laymen.

A PLAN FOR CIGUATERA RESEARCH IN THE PACIFIC

Ciguatera intoxication is a long-standing problem in many tropical and subtropical islands of the Pacific. Ciguatera is a disease characterized by gastrointestinal and neurological symptoms which are caused by the ingestion of any one of a variety of tropical marine reef fishes. It is evident that ciguatera outbreaks have occurred in oceanic islands of many areas in the Pacific Ocean at least as long as records exist from these areas and furthermore, it is likely that the Pacific islanders knew of the toxicity of fishes long before the beginning of European exploration in the Pacific. In more recent years, various researchers have pointed out the significance of ciguatera on the health of island populations in the Pacific. For example, one of the highest incidence of ciguatera intoxication on record occurred in the island of Hao in the Tuamotu Archipelago, where out of 514 individuals interviewed 224 (43%) reported that they had been intoxicated at least once.

The islands in the central Pacific including the Line Islands, Johnston Island, and the Hawaiian Archipelago apparently were free of ciguatera until about the 1940s. In response to the spread of ciguatera to these areas, beginning in the late 1950s to early 1960s a broad research program was undertaken by the Hawaii Institute of Marine Biology, University of Hawaii, in the chemical isolation, identification, pharmacology, and biological origin of the toxin or toxins causing ciguatera, as well as an epidemiological study of ciguatera intoxication in the Pacific. Much progress has been made to resolve the various facets of the ciguatoxin problem by the researchers at the Hawaii Institute of Marine Biology and other research groups at the University of Hawaii, as well as researchers at other laboratories throughout the Pacific.

The earliest work on the chemistry of the toxin began in the early 1940s and involved studies on its solubilities and other characteristics. It was noted that the toxin could be removed from fish flesh with ethanol and that the toxin was heat stable. Work on the chemistry of ciguatoxin--the name used for the agent responsible for ciguatera beginning in 1967--advanced to a point where the basic empirical formula $(C_{35}H_{65}NO_8)_n$ is now known. It was also determined that ciguatoxin "is apparently a lipid containing quaternary nitrogen, hydroxyl, and carbonyl functions."

Regarding the origin and transmission of toxin, the idea that ciguatoxin is endogenous in the fish, similar to tetrodotoxin in puffers, was early rejected. Researchers quickly recognized that ciguatera is of biological origin and is caused by a toxin that arises in the environment. The theory of the biological origin of the toxin or the food chain theory, was best advanced as early as 1958 that the original elaborator of the toxin was likely to be an "alga, a fungus, a protozoa, or a bacterium." In 1971, conclusive support for this theory became available with the discovery of ciguatoxin, or a compound which was indistinguishable from it at that time, in the gut viscera and flesh of a surgeonfish, Ctenochaetus striatus, which is a detrital feeder that cannot bite off pieces of attached algae. A breakthrough in this aspect of ciguatera research occurred in 1977-78 by the discovery of a new benthic dinoflagellate, Gambierdiscus toxicus, which was associated with the occurrence of ciguatoxin (and mitotoxin) in algal/detrital mixtures believed to be the causative agent in ciguatera outbreaks.

Subjective assays or tests for toxins by the use of laboratory animals began in the early 1940s. Toxicity tests by injections of aqueous extracts from potentially toxic fish were also initiated in the early 1940s. The standard technique of using mongooses in feeding tests was developed at the Hawaii Institute of Marine Biology in 1960. In 1977, in another breakthrough, "a practical, sensitive, simple, and relatively specific radioimmunoassay test for the assay of ciguatoxin directly from natural sources" was developed.

It is evident that much has been done in solving the long-standing ciguatera problem. It is also evident that much still remains to be done. Presently a number of research organizations in Hawaii are actively working on various aspects of the ciguatera problem. While there has been an exchange of results and views among some of the ciguatera researchers, the need for a workshop to bring together these active investigtors for an in-depth discussion of the problems appeared warranted. In the spring of 1981 (March 18-20) the "Hawaii Ciguatera Workshop," sponsored by the Honolulu Laboratory and University of Hawaii Sea Grant, was held at the Honolulu Laboratory (Appendix A). The objective of the workshop was to develop working hypotheses to guide future research on the ciguatera problem. Some of the goals of ciguatera research are to fully understand the mechanism underlying the origin and transfer of the toxin(s) in the ecosystem and to determine if corrective measures can be developed to safely provide fish for human consumption. Considering earlier research and the status of ongoing research on the ciguatera problem, the workshop participants identified three basic goals and objectives for future ciguatera research. Also a total of 22 research projects/activities relating to the three goals/objectives were identified. The relationships of the research activities/subprojects and objectives are shown in Figure 1. The objectives and research activities/subprojects are described below.

OBJECTIVES

I. To obtain an understanding of the factors or mechanisms triggering the chain of events in the reef ecosystem leading ultimately to outbreaks of ciguatera intoxication in the human population so that the "management" of the ecosystem will be possible.

II. To elucidate the pharmacological aspects of ciguatera intoxication and to develop a viable treatment for human ciguatera intoxication.

III. To develop and disseminate for general use a quick and reliable field test to screen marine organisms for ciguatoxin and related toxins.

DESCRIPTION OF RESEARCH ACTIVITIES AND SUBPROJECTS

1. Monitor public health records

The Hawaii Department of Health maintains records on local outbreaks of ciguatera cases including the date and site of the outbreak, the species of fish implicated, and where it was caught. The data from these documented cases of ciguatera intoxications will be used to plan biological sampling of the reef habitat where the implicated fish was caught.

2. Sample substrate for dinoflagellates

The benthic dinoflagellate, G. toxicus, has been determined to be the source of ciguatoxin in the coral reef ecosystem. Based on current records of ciguatera outbreaks maintained at the Hawaii Department of Health, the coral reef habitat where fishes implicated in ciguatera outbreaks were caught will be sampled for the presence of G. toxicus.

3. Determine environmental conditions in dinoflagellate "bloom" and "no-bloom" areas

Data on environmental variables will be collected in dinoflagellate "bloom" and "no-bloom" areas to obtain baseline information for comparisons of environmental conditions in these areas.

4. Field and laboratory studies on transfer of toxin in food chain

Detailed field and laboratory studies to determine the transfer of ciguatoxin and other toxins from dinoflagellates to the fish carnivores in the reef ecosystem will be carried out.

5. Determine factors producing toxic dinoflagellate blooms

Analyze data collected in subproject 3 to determine environmental conditions producing toxic dinoflagellate blooms. A comparison of environmental conditions between areas with and without toxic dinoflagellate blooms should provide clues as to causative factors.

6. Determine best procedure to test for toxins in the marine environment

Instead of testing fish for the presence of toxins, it may be preferable to determine the presence of toxins at an earlier stage in the transfer of toxins in the marine environment. Studies should be conducted to determine the feasibility of testing for toxins at various stages of toxin transfer in the ecosystem.

7. "Manage" ecosystem (predict and interrupt chain of events leading to possible ciguatera outbreaks)

Analyze data to allow the prediction of potential ciguatera outbreaks and possibly to disrupt the chain of events to prevent ciguatera outbreaks in the human population.

8. Determine source of toxins

Ciguatoxin and other toxins are found in minute quantities in the flesh and viscera of fishes. Toxicity of fishes may vary with season and with species. Consequently large amounts of toxic fish are needed to extract only a small amount of toxin. Effort will be expended to locate highly toxic fish for the extraction of ciguatoxin and other toxins.

9. Obtain source of toxins

Once sources of toxic fish are located, attempts will be made to collect enough of them to extract an adequate amount of toxins required for various studies.

10. Laboratory study on toxicity to fish

On the surface it appears that ciguatoxin has no effect on the well-being of fishes. Laboratory experiments will be conducted to determine the effects of ciguatoxin on the growth and physiology of fishes. Also, the rate of toxin uptake and retention will be investigated.

11. Extract pure toxins

Using methods developed at the University of Hawaii, extract and purify ciguatoxin, maitotoxin, and other toxins.

12. Determine the molecular structure of ciguatoxin and other toxins

The empirical formula for ciguatoxin is $(C_{35}H_{65}NO_8)_n$, and has been known for some time. Much is also known about the molecular structure of ciguatoxin. It is anticipated that the complete molecular structure of ciguatoxin will become known when an adequate supply of toxin is available. The molecular structure of other related toxins should be revealed when an adequate amount of toxins becomes available.

13. Synthesize ciguatoxin and other toxins

The determination of the molecular structure of ciguatoxin and related toxins will allow the synthesis of these toxins.

14. Select target tissues/organs/test animals

Determine appropriate and suitable tissues/organs/test animals for pharmacological studies.

15. Pharmacology

Determine the effects of ciguatoxin and other related toxins on selected target tissues/organs/test animals. Test off-the-shelf drugs to counteract the effect of ciguatoxin and other toxins.

16. Test treatment on animals for ciguatoxin and other toxins

Using the results obtained in subproject 15, conduct experiments with off-the-shelf drugs on test animals in test treatment to counter ciguatoxin and related toxins.

17. Develop treatment for human ciguatera intoxication

Conduct clinical trials and develop suitable treatment for human ciguatera intoxication using off-the-shelf drugs.

18. Develop usable enzyme-linked immunosorbent assay test for ciguatoxin

Studies are underway to develop an enzyme-linked immunosorbent assay (ELISA) to test for ciguatoxin. Work is continuing on this project and it is believed that a usable ELISA test will soon be available.

19. Conduct field trials

Conduct field trials of ELISA on marine organisms.

20. Develop "dipstick" test for ciguatoxin

Following the development of a usable ELISA test for ciguatoxin, experiments will be conducted to adapt ELISA for a "dipstick" test, which will be a simple test, applicable for field use by laymen.

21. Implement "dipstick" test and educate populace

Following perfection of the "dipstick" test for ciguatoxin, the test material will be made available for use by the general populace. An education program for the public will be carried out.

22. Undertake research on Hawaii and Federal rules and regulations

Hawaii and Federal rules and regulations concerning public agencies' responsibilities on testing wholesomeness of fish relative to ciguatoxin and other toxins will be examined.

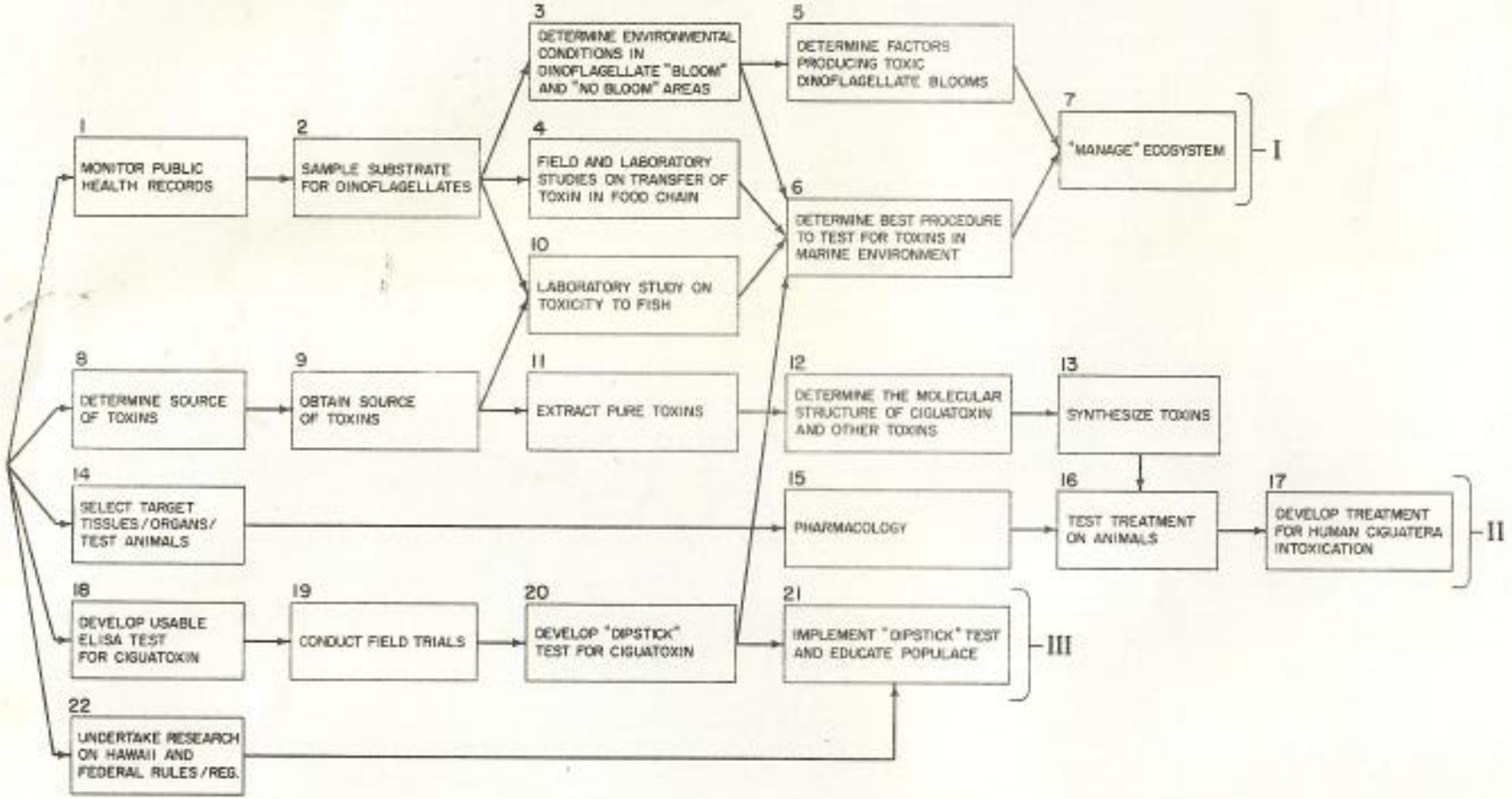


Figure 1.--Relationships of research activities/subprojects and objectives for ciguatera research in the Pacific.

APPENDIX A

SUMMARY REPORT OF THE PACIFIC CIGUATERA WORKSHOP
HONOLULU, HAWAII, 18-20 MARCH 1981

Richard S. Shomura, Chairman
Southwest Fisheries Center Honolulu Laboratory
National Marine Fisheries Service
National Oceanic and Atmospheric Administration
Honolulu, Hawaii 96812

Cosponsored by the Honolulu Laboratory and the
University of Hawaii Sea Grant

May 1981

The Hawaii Ciguatera Workshop, cosponsored by the Honolulu Laboratory and the University of Hawaii Sea Grant (UHSG), was held at the Honolulu Laboratory, Southwest Fisheries Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Honolulu, Hawaii from March 18 to 20, 1981. Mr. Richard S. Shomura, Honolulu Laboratory Director and Chairman of the workshop, opened the workshop by welcoming all the participants and observers and made a few introductory remarks including the rationale for holding the workshop. The objective of the workshop was to bring together some of the principal, active ciguatera researchers in the Pacific to help develop working hypotheses to guide future research on the ciguatera problem. It is recognized that the ultimate goal of ciguatera research is to fully understand the mechanism underlying the origin and transfer of the toxin(s) in the ecosystem and to develop corrective measures to provide nontoxic fish for human consumption.

The workshop agenda (Appendix 1) included the consideration of four broad areas or aspects of ciguatera research: toxins, the detection of toxins, the medical/pharmacological aspects of ciguatera, and the ecological aspects. Seven background papers were assembled for the workshop (Appendix 2) and nine working papers which described ongoing ciguatera research in the Pacific were presented (Appendix 3). In addition to the oral presentations on ciguatera research in the Pacific, presentations were made on the results of the recent World Health Organization meeting on ciguatera held in Noumea, New Caledonia, and on the status of ongoing ciguatera research in the southeastern United States. The workshop participants and observers are listed in Appendix 4.

TOXINS (WP/2, WP/6)

The discussion on toxins covered the topics of the chemical and molecular structure of ciguatoxin and other toxins and the origin of the toxins. The problem of solving the molecular structure of ciguatoxin has been hampered by the lack of an adequate supply of pure toxin for analysis. The toxin is diffusely distributed in minute amounts in the flesh and viscera of fishes so large amounts of toxic fish are needed to extract the toxin in a complex purification procedure. Dr. Paul Scheuer of the University of Hawaii (UH) now has 0.9 mg of ciguatoxin and Dr. Takeshi Yasumoto of Tohoku University, Sendai, Japan, has 0.4 mg. Although the analytic methods used to determine the molecular structure of the toxin is basically non-destructive, minute amounts of the toxin are lost in the analyses. Dr. Scheuer's 0.9 mg of ciguatoxin is presently at the California Institute of Technology for analysis. The basic, empirical formula of ciguatoxin, $(C_{35}\ H_{65}\ NO_8)_n$, has been known for some time. Although the molecular structure of ciguatoxin is not completely known, it is believed that the problem will be solved when more pure toxin becomes available. In this regard, it was announced that the UH will soon be acquiring a 300 MHz Nuclear Magnetic Resonance Spectrometer for the analysis of the molecular structure of complex compounds. This will greatly increase the instrument time available for the analysis of the molecular structure of ciguatoxin.

A breakthrough in ciguatera research occurred within the past 5 years when the source of ciguatoxin in the coral reef ecosystem was determined to be a benthic dinoflagellate, Gambierdiscus toxicus, a microscopic unicellular alga that grows primarily as an epiphyte on certain seaweeds on reef flats. This same species has been found near the Hawaii Institute of Marine Biology (HIMB)

in Kaneohe Bay and at other scattered locations around the island of Oahu. The results of experiments conducted at HIMB to determine the factors controlling toxin production, growth rate, and yields of laboratory cultures of the HIMB strain of G. toxicus were discussed. A problem in the research on culturing G. toxicus is the tremendous variability in the levels and kinds of toxins present in the cultures.

Elsewhere in the Pacific, the possible sources of secondary toxins have also been investigated. In addition to maitotoxin, the presence of secondary toxins have often been detected in the viscera of herbivorous fishes and it can be hypothesized that these secondary toxins, like ciguatoxin, originate from other benthic dinoflagellates. It was found that several species of dinoflagellates found growing with G. toxicus also produced toxins that were lethal to mice. Of interest are two fat soluble toxins of Prorocentrum lima, PL toxins I and II, which are practically indistinguishable in chromatographic properties from scaritoxin and ciguatoxin, respectively. A chemical and spectral analysis of PL toxin II showed that it is identical with okadaic acid, the cytotoxic component of a sponge.

DETECTION (WP/4, BP/2, BP/3, BP/6)

The early tests for ciguatoxin have been bioassays in animals such as mouse, cat, and mongoose. Among the shortcomings of these bioassays are that they are subjective and, in the case of the mongoose, the test animals are obtained from the wild and, therefore, are not homogeneous. A breakthrough in testing for ciguatoxin occurred with the development of a radioimmunoassay (RIA) for the direct examination of fish tissues for toxin. The advantages of the RIA are that it is simple and practical, relatively specific, and sensitive. One of the objectives of current research is to evaluate the RIA procedure for ciguatoxin based on two approaches: 1) identifying the reactive factor in the sheep anti-ciguatoxin serum used in the RIA, and 2) assessing the RIA procedure on fishes from clinically defined and documented cases of ciguatera intoxication and comparing results with those from nontoxic fish of the same species. The RIA results are also being evaluated by supportive bioassays, the mouse bioassay, and the guinea pig atrium assay.

There was much discussion on the technical aspects of the immunological reactions of the RIA procedure including questions on dose responses, extracts, and homogenates versus solids. It was pointed out that the whole basis of the RIA procedure is the assumption that ciguatoxin, being a lipid, is bound on the membrane surface or cells so that it can be detected by an antibody. Some of the difficulties associated with the RIA procedure is 1) sampling, i.e., there is uncertainty in the distribution of ciguatoxin in the fish flesh, 2) the possibility of false positives occurring in the RIA test and, 3) reproducibility owing to the fact that tissues are used and even within close proximity "hot spots" of ciguatoxin may vary.

Beginning in April 1979, at the request of the Hawaiian fishing industry and in cooperation with the Honolulu Laboratory, the RIA test was used to screen commercially landed kahala, Seriola dumerili, for ciguatoxin. The testing program made possible the marketing of large numbers of kahala larger than 20 pounds which, before the testing started, were ordinarily not marketed in the belief that large fish were more likely to be toxic. The most

significant result of the testing program has been the absence of any reported ciguatera cases from marketed RIA-negative kahala.

Mouse bioassays and guinea pig atrium assays have been used to evaluate the RIA procedure. Although the number of test samples is small, the data suggest direct correlations among the three assays. It was noted that ciguatoxin causes an initial massive release of neuroepinephrine in guinea pig atrium. However, one of the clinical symptoms of ciguatera intoxication in humans is reduced cardiac activity. In light of the effect of ciguatoxin on guinea pig atria, the question was posed regarding the effect of ciguatoxin on humans in the early stage of ciguatera intoxication.

Research is also underway to develop an enzyme-linked immunosorbent assay (ELISA) for ciguatoxin. ELISA is simpler and more rapid and unlike the RIA procedure, is not dependent on the use of radioisotopes. Current research on ELISA includes the establishment of toxicity ranges based on absorbence values of samples from clinically documented tissues from ciguatera intoxications and nontoxic tissues. A comparison of RIA and ELISA tests on kahala indicated that ELISA gave a lower rejection rate, which may be more realistic in light of the incidence of reported ciguatera intoxications by kahala in the past.

MEDICAL/PHARMACOLOGY (WP/7, WP/8, WP/9)

This section of the workshop dealt primarily with the public health aspect of ciguatera intoxication. The results of a study to determine the effect of ciguatera on the overall health of Pacific islands populations and how it has affected the utilization of marine resources were discussed. The study was an extensive comparative investigation of the perceived distribution and occurrence of ciguatera in the late 1970s, folk beliefs as to the etiology of the disease, and other aspects of the health and resource problems stemming from ciguatera. Statistics on ciguatera morbidity rates from the South Pacific Commission Health and Epidemiological Information Service (SEPHIS) area were presented. In 1978 the mean reported ciguatera morbidity rate for the area (excluding Papua New Guinea) was 12.01/10,000. The actual morbidity rate is undoubtedly considerably higher.

A summary of ciguatera intoxication in Hawaii covering a period from 1900-80 was presented. It was noted that cases of ciguatera intoxication were higher in the spring and autumn and that more patients rather than physicians are apt to report ciguatera poisonings. It was further noted that a 25% reporting rate is considered good.

A detailed account of the fatality caused by Marquesan sardine, Sardinella marquesensis, on Kauai in 1978 was also presented. Clinical data from this case were remarkably similar to what would be expected from a severe form of ciguatera intoxication, and it was suggested that clupeoid poisoning should not be excluded from the concern of those doing research on ciguatera poisoning. However, other workshop participants noted that clupeoid poisoning may be more closely related to paralytic shellfish poisoning (PSP) in ecological terms.

ECOLOGY (WP/1, WP/3, WP/5, WP/6, BP/1, BP/4, BP/5, BP/7)

Based on the correlation of the population density of G. toxicus with epidemiological information, the endemicity of ciguatera in various locations

in the Pacific was discussed. The results of surveys showed that the density of the G. toxicus population was correlated with fish toxicity. These results suggest that toxicity in any area can be predicted by the density of G. toxicus. Another possibility of a monitoring system for ciguatera is the assessment of the toxicity of fish livers. It has been shown that fish livers show far less variability in toxicity than the flesh of fishes.

The results of a study on the distribution of G. toxicus in French Polynesia (Tahiti and Gambier Island), New Caledonia, Guam, and Okinawa were also presented, including the effect of certain environmental factors on the density of G. toxicus. Temperature is not an important factor in the density of G. toxicus. However, salinity and light intensity appear to be important. Iron was not important in the growth of G. toxicus but phosphates significantly increased the growth.

Development of the RIA procedure made possible large-scale testing of fishes for ciguatoxin. As part of the cooperative survey of the marine resources in the Northwestern Hawaiian Islands (NWHI), the Honolulu Laboratory and the Hawaii Division of Fish and Game (HDFG) have been sampling inshore and nearshore fishes to determine the distribution of ciguatoxic fishes.

The Honolulu Laboratory's results of tests for ciguatoxin on fish and shellfish collected in the NWHI, Midway Islands, American Samoa and Western Samoa, and Guam and the Northern Mariana Islands were presented. The overall rejection (positive and borderline toxicity levels) rate was 15% for NWHI, 39% for Midway, 19% for American Samoa and Western Samoa, and 4% for Guam and the Northern Mariana Islands.

Flesh samples from fishes are obtained from the dorsal anterior region (A), ventral abdominal region (B), and the anal region (E). It was pointed out that there was no clear trend as to where (sites A, B, or E) the toxin is concentrated in the fish. The results were not only variable among species, but also between sexes. The results to date showed also that toxicity was not related to size or sex.

The results obtained by the HDFG indicate that fishes in the NWHI were relatively more toxic (in terms of numbers rejected in the RIA test) in the summers of 1977 and 1978 than in 1979 and 1980. In 1980 the nearshore fishes were more toxic in the fall than in the summer. Based on these results, it was suggested that the occurrence of ciguatoxic fishes appear to be cyclical. The feeding habits, distribution, and abundance of the fishes sampled were also described.

Also, some anomalous results in the HDFG data and the Honolulu Laboratory data were evident. In sampling around Midway in 1980, the Honolulu Laboratory samples indicated an overall high rejection rate for fishes caught from September 30 to October 4, whereas the HDFG samples collected around the same period showed a relatively low rejection rate.

The results of an analysis of data from the kahala sampling program between April 1979 and December 1980 were also discussed. The mean quarterly RIA scores showed a general trend of a decline in RIA scores from the second quarter of 1979 to a low at the end of 1979/beginning of 1980 and a subsequent increase in RIA scores through 1980. The trend was consistent for sampling

sites A, B, and E, for geographical origin of the fish (NWHI, Oahu, Penguin Bank, and Hawaii), and for both sexes. The correlation of RIA between sampling sites was high: (AB) = 0.74, (AE) = 0.69, and (BE) = 0.70. Also, the variation in RIA scores from two replicates from each sampling site (A, B, and E) was no greater than the variation among sampling sites A, B, and E. Other results indicated that: there was no relationship between kahala weight and RIA scores, and that sampling site B or E would be the best single site to sample for maximum RIA values.

Color slides of the various fish species implicated in ciguatera intoxication were shown and the distribution, food habits, and habitat of these fishes were described. The species that have caused ciguatera are shore fishes associated with reefs, are bottom-dwelling, but they may also be semipelagic open-water forms that range into the reef habitat to feed. They may be carnivorous or they may be benthic algal or detrital feeders. The carnivores that prey heavily on reef fishes are the most prone to be toxic whereas those that feed primarily on benthic crustaceans tend to be least toxic. The moray eel, Lycodontis (= Gymnothorax) javanicus, is highly toxic.

Other ecological aspects of ciguatera were also discussed. Young and old Hawaiian monk seals, Monachus schauinslandi, in the NWHI suffered high mortality in 1978. It was speculated that ciguatera intoxication could have been the cause of the mortality. In connection with this, the results of experiments where elephant seals, Mirounga angustirostris, were fed moray eels caught in the NWHI were described. Elephant seals were found to be highly susceptible to ciguatera intoxication.

RECOMMENDATIONS

The workshop identified three general objectives or goals for future ciguatera research. The first goal is to obtain an understanding of the factors or mechanisms triggering the chain of events leading ultimately to potential outbreaks of ciguatera intoxication so that these events could be predicted. The second goal is to elucidate the pharmacological aspects of ciguatera and to develop a viable treatment for ciguatera intoxication. The third goal is to develop and disseminate a quick and reliable field test to screen marine organisms for ciguatoxin and related toxins.

Several general areas of research were recommended to achieve the first objective:

1. Determine baseline conditions in (a) areas with no previous history of ciguatera outbreaks and (b) areas that could potentially produce ciguatera outbreaks. Related to this is the determination and verification of factors causing blooms of Gambierdiscus toxicus and the comparison of environmental conditions in areas containing and devoid of dinoflagellate blooms.

2. Verify the source of ciguatoxin in the reef ecosystem; determine the rate of toxin production from culture experiments and determine factors that influence the growth of toxin-producing dinoflagellates. Determine the source (cysts?) of dinoflagellate blooms.

3. Conduct field and laboratory studies to determine the transfer and metabolism of ciguatoxin in the food chain.

4. Determine the effects of ciguatoxin on fish including its toxicity to fish and its influence on growth rates; conduct experiments to determine if ciguatoxin is accumulated in fish or excreted and whether it would be possible to predict time/area fish toxicity based on dates/areas of dinoflagellate blooms; determine why some species are more toxic than others including a quantitative analysis of toxicity levels by species, size, and area, and compare findings with the food chain understanding; and determine whether crustaceans and molluscs in areas of dinoflagellate blooms are toxic.

5. Evaluate the ELISA technique for field work.

Research activities recommended to achieve the second goal were:

1. Determine source of toxin by locating toxic areas, e.g., by public health follow-ups, search for alternate source of ciguatoxin by bio-testing for possible new sources. A related activity is to determine factors that make dinoflagellates toxic.

2. Collect toxic fishes including moray eels, RIA-rejected kahala, and other species and develop cultures of Gambierdiscus toxicus to extract the crude toxins.

3. Assemble hardware and manpower to extract and purify toxins for experiments to a) compare toxin from moray eel with toxins from other species, b) determine the molecular structure of ciguatoxin and other toxins, and c) determine the metabolic pathways and target tissues/organs in tests on mice and other animals and muscle tissues, c) investigate the pharmacology of ciguatoxin to determine metabolic pathways and target tissues/organs, in tests on mice and other animals, and d) determine the efficacy of off-the-shelf drugs for the treatment of ciguatera in test animals and in clinical trials.

Research activities recommended for the third goal were:

Develop usable ELISA test for ciguatoxin and 1) improve specificity of ELISA test to sort out other toxins (scaritoxin, maitotoxin, okadaic acid) and 2) adapt ELISA procedure for a "dip stick" test.

APPENDIX 1

AGENDA

1. Introduction (R. Shomura)

   1.1 Participants
   1.2 Objectives of workshop

2. Report of SPC ciguatera meeting (A. Banner)

3. Toxins

   3.1. Chemical composition (P. Scheuer)
   3.2. Dinoflagellates (T. Yasumoto, N. Withers)

4. Detection

   4.1. RIA (Y. Hokama, L. Kimura)
   4.2 ELISA (Y. Hokama, L. Kimura)
   4.3 Muscle tests (J. Miyahara)

5. Medical/Pharmacology

   5.1. Case histories--Hawaii (M. Sugi)
   5.2 Case histories--Other Pacific areas (N. Lewis)
   5.3 Pharmacological (J. Miyahara)

6. Ecology

   6.1. Elaborators (T. Yasumoto, N. Withers)
   6.2. Fishes

      6.2.1. NWHI fishes (Honolulu Laboratory, Hawaii Division of Fish and Game)
      6.2.2. Kahala (Honolulu Laboratory)
      6.2.3. Other areas--American Samoa, western Pacific (Honolulu Laboratory)

   6.3. Other animals

      6.3.1. Hawaiian monk seal (Honolulu Laboratory)
      6.3.2. Others--birds, turtles (open)

APPENDIX 2

LIST OF BACKGROUND PAPERS

BP/1 Adachi, Rokuro, and Yasuwo Fukuyo.
1979. The thecal structure of a marine toxic dinoflagellate, Gambierdiscus toxicus gen. et sp. nov. collected in a ciguatera-endemic area. Bull. Jpn. Soc. Sci. Fish. 45:67-71.

BP/2 Hokama, Y., A. H. Banner, and D. B. Boyland.
1977. A radioimmunoassay for the detection of ciguatoxin. Toxicon 15:317-325.

BP/3 Hokama, Y., L. H. Kimura, and J. T. Miyahara.
In press. Immunological approaches to understanding marine toxins. Proceedings of the Sixth Food and Drug Administration Science Symposium on "Aquaculture - Public Health, Regulatory and Management Aspects," February 12-14, 1980, New Orleans, Louisiana (Office of Health Affairs, FDA).

BP/4 Humphreys, Robert L., Jr.
1980. Feeding habits of the kahala, Seriola dumerili, in the Hawaiian Archipelago. In Richard W. Grigg and Rose T. Pfund (editors), Proceedings of the Symposium on Status of Resource Investigations in the Northwestern Hawaiian Islands, April 24-25, 1980, University of Hawaii, Honolulu, Hawaii. Sea Grant Misc. Rep. UNIHI-SEAGRANT-MR-80-04, p. 233-240.

BP/5 Ito, Bernard M., and Richard N. Uchida.
1980. Results of ciguatera analysis of fishes in the Northwestern Hawaiian Islands. In Richard W. Grigg and Rose T. Pfund (editors), Proceedings of the Symposium on Status of Resource Investigations in the Northwestern Hawaiian Islands, April 24-25, 1980, University of Hawaii, Honolulu, Hawaii. Sea Grant Misc. Rep. UNIHI-SEAGRANT-MR-80-04, p. 81-89.

BP/6 Miyahara, J. T., C. K. Akau, and T. Yasumoto.
1979. Effects of ciguatoxin and maitotoxin on the isolated guinea pig atria. Res. Commun. Chem. Pathol. Pharmacol. 25:177-180.

BP/7 Randall, John E.
1980. A survey of ciguatera at Enewetak and Bikini, Marshall Islands, with notes on the systematics and food habits of ciguatoxic fishes. Fish. Bull., U.S. 78:201-249

APPENDIX 3

LIST OF WORKING PAPERS

WP/1 Polovina, Jeffrey J., and Bernard M. Ito.
An analysis of data from the kahala ciguatera sampling program, April 1979-December 1980.

WP/2 Withers, Nancy.
Pacific ciguatera.

WP/3 Uchida, Richard N., Bernard M. Ito, Paul M. Shiota, Darryl T. Tagami, Karen P. Wendel, Victor A. Honda, and Michael P. Seki.
Status of the Honolulu Laboratory ciguatera research on fishes of the Northwestern Hawaiian Islands, American Samoa, Western Samoa, Guam, and the Northern Mariana Islands.

WP/4 Hokama, Y., L. Kimura, and J. Miyahara.
Status report on detection methods for ciguatoxin.

WP/5 Okamoto, Henry.
Status report of ciguatera research in the Northwestern Hawaiian Islands by DLNR, Division of Fish and Game.

WP/6 Yasumoto, Takeshi, Yasukatsu Oshima, Akio Inoue, Yasuwo Fukuyo, and Takako Harada.
Studies on ciguatera.

WP/7 Kubota, Wilbert.
Ciguatera fish poisoning cases: A summary from 1900 - December 1980.

WP/8 Lewis, Nancy Davis.
Ciguatera morbidity in the island Pacific.

WP/9 Melton, Robert J.
Summary of data on fatal case of fish poisoning associated with Marquesan sardine, Kauai, 1978.

APPENDIX 4

LIST OF PARTICIPANTS

Bruce Anderson
Hawaii State Department of Health
1250 Punchbowl Street
Honolulu, HI 96813

Dr. A. H. Banner
Hawaii Institute of Marine Biology
University of Hawaii
P. O. Box 1346
Kaneohe, HI 96744

Deborah J. Burns
Department of Oceanography
University of Hawaii - CR 309
Honolulu, HI 96822

Dr. Jack R. Davidson
Director, Sea Grant
University of Hawaii - SPAL 255
Honolulu, HI 96822

Michael Gawel
East West Center, EAPL
P. O. Box 1806
Honolulu, HI 96848

William G. Gilmartin
Southwest Fisheries Center
National Marine Fisheries Service
P. O. Box 3830
Honolulu, HI 96812

Dr. John M. Gooch
Hawaii State Department of Health
1250 Punchbowl Street
Honolulu, HI 96813

Dr. Richard W. Grigg
Hawaii Institute of Marine Biology
University of Hawaii
P. O. Box 1346
Kaneohe, HI 96744

Sitiveni Halapua
IMR-USP, Fiji
Nukualofa, Tonga

Craig S. Harrison
U.S. Fish and Wildlife Service
P. O. Box 50167
Honolulu, HI 96850

Dr. Philip Helfrich
Hawaii Institute of Marine Biology
University of Hawaii
P. O. Box 1346
Kaneohe, HI 96744

Dr. Thomas B. Higerd
Medical University of South Carolina
171 Ashley Avenue
Charleston, SC 29425

Dr. Yoshitsugi Hokama
Cancer Research Center of Hawaii
1236 Lauhala St.
Honolulu, HI 96813

Bernard M. Ito
Southwest Fisheries Center
National Marine Fisheries Service
P. O. Box 3830
Honolulu, HI 96812

Dr. David Jollow
Department of Pharmacology
Medical University of South Carolina
171 Ashley Avenue
Charleston, SC 29425

Paul Kawamoto
Hawaii Division of Fish and Game
Department of Land and Natural Resources
1151 Punchbowl Street
Honolulu, HI 96813

Dr. Lucille Kimura
Cancer Research Center of Hawaii
1236 Lauhala St.
Honolulu, HI 96813

Nancy D. Lewis
Department of Geography
University of California
Berkeley, CA 94720

David J. Mackett
Southwest Fisheries Center
National Marine Fisheries Service
P. O. Box 271
La Jolla, CA 92038

Dr. Robert Melton
Department of Health
P. O. Box 671
Lihue, Kauai, HI 96766

Dr. James T. Miyahara
Department of Pharmacology
University of Hawaii - BIO-T 412A
Honolulu, HI 96822

Henry Okamoto
Hawaii Division of Fish and Game
Department of Land and Natural Resources
1151 Punchbowl Street
Honolulu, HI 96813

Dr. James D. Parrish
Hawaii Cooperative Fishery Unit
c/o University of Hawaii - EDM 165A
Honolulu, HI 96822

Dr. Jeffrey J. Polovina
Southwest Fisheries Center
National Marine Fisheries Service
P. O. Box 3830
Honolulu, HI 96812

Dr. Alf Pratte
Sea Grant Marine Advisory Program
University of Hawaii - SPAL 252B
Honolulu, HI 96822

Dr. John E. Randall
Division of Ichthyology
Bishop Museum
1355 Kalihi Street, Box 19000-A
Honolulu, HI 96819

Henry M. Sakuda
Hawaii Division of Fish and Game
Department of Land and Natural Resources
1151 Punchbowl Street
Honolulu, HI 96813

Holmes Saeve
IMR-USP
Fisheries Division
Ministry of Natural Resources
Honiara, Solomon Islands

Dr. Paul J. Scheuer
Professor, Department of Chemistry
University of Hawaii at Manoa - BIL 325
Honolulu, HI 96822

Robert E. Schroeder
Hawaii Cooperative Fishery Unit
c/o University of Hawaii - EDM 165A
Honolulu, HI 96822

Dr. Yuzuru Shimizu
Department of Chemistry
University of Hawaii - BIL 325
Honolulu, HI 96822
and
University of Rhode Island
Kingston, RI

Richard S. Shomura
Southwest Fisheries Center
National Marine Fisheries Service
P. O. Box 3830
Honolulu, HI 96812

Anthony Sudehum
Hawaii Cooperative Fishery Unit
c/o University of Hawaii - EDM 165A
Honolulu, HI 96822

Mitsugi Sugi
Epidemiology Branch
Hawaii State Department of
Health
P. O. Box 3378
Honolulu, HI 96801

Brooks Takenaka
United Fishing Agency, Ltd.
117 Ahui Street
Honolulu, HI 96813

Richard N. Uchida
Southwest Fisheries Center
National Marine Fisheries
Service
P. O. Box 3830
Honolulu, HI 96812

Dr. Nancy Withers
Hawaii Institute of Marine
Biology
University of Hawaii
P. O. Box 1346
Kaneohe, HI 96744

Dr. Takeshi Yasumoto
Faculty of Agriculture
Tohoku University
1-1, Amamiyamachi, Tsutsumidori
Sendai, Japan

Richard York
Hawaii Institute of Marine
Biology
University of Hawaii
P. O. Box 1346
Kaneohe, HI 96744

Howard O. Yoshida
Southwest Fisheries Center
National Marine Fisheries
Service
P. O. Box 3830
Honolulu, HI 96812

Parasite Lectures

By Jeanne Ambrose
Star-Bulletin Writer

His first love, he says, is sleeping sickness, but lately parasites have wormed their way into his heart.

Dr. Robert Desowitz, professor of tropical medicine and medical microbiology at the University of Hawaii's John A. Burns School of Medicine, says that during the course of his class on parasitology, many of his students develop anorexia nervosa, an ailment characterized by complete loss of interest in food.

When he lectures about the tapeworms, burrowing larvae and an assortment of other creepy crawlies that may be transported into the human body through rare hamburger and pork, uncooked freshwater crab and other food, most students lose their appetites.

As Desowitz says in his latest book, "On New Guinea Tapeworms and Jewish Grandmothers," to be published within the next couple of months: "Medical parasitology texts are like contra-cookbooks describing the ethnic and cosmopolitan gastronomic delights that serve as conveyances of infection .... For our students at the University of Hawaii school of medicine, particularly those of Japanese ancestry, only sashimi, raw marine fish, seems safe and it is a sad day when I have to give the lecture on anisakiasis that will dislodge them from their last refuge of culinary comfort."

ANISAKIS ADULTS, Desowitz goes on to say, "are parasites within the intestinal tract of marine mammals (porpoises, whales and seals), and their larvae, during the course of the obligatory life cycle, are present in marine fish."

Although hundreds of people each year in Japan were plagued by a mysterious, painful gastrointestinal ailment that produced a small ulcer, it wasn't until 1965 that the first diagnosis of human anisakiasis was made in that country. While examining an ulcer that had been surgically removed from a patient, the pathologist discovered a worm in the center of the inflamed tissue.

Studies done in the Netherlands a few years earlier turned up similar diagnoses on Dutch patients who suffered from sudden abdominal pain and stomach lesions. A Dutch parasitologist had identified the worms as anisakis, the same parasites found in the intestinal tracts of some fish.

Herring, salted or pickled and eaten uncooked in Holland, was implicated as the parasites' mode of transportation into the human body. When a human ate the infected herring, the anisakis parasite adapted to its new location by burrowing into the herring eater's tissues.

IN JAPAN, the ulcer-producing worms are linked to sashimi. Desowitz said about 1,000 new cases of anisakiasis occur each year in that country.

Patients who have ingested infected fish usually experience sudden painful attacks in their stomachs within hours, or at most a few days, after eating the fish. But, Desowitz said, "there is very little danger" of the ailment being transported to the Islands.

"As far as we know it has never been picked up here," he said.

Bite Into Appetites

"Unlike Japan's fish, our worms don't seem to be getting into the meat of the fish. They're mostly found in the stomach."

Normally the parasites remain in the intes.inal tracts of fish, but some of these worms may bore their way through the stomach walls into the fish flesh, Desowitz said.

One reason for the prevalence of the parasites in some fish may be the way the fish are caught and cleaned, he said. Instead of cleaning and "eviscerating" the fish at sea, many fisherman now put their catch on ice until the boat returns to port where the fish are cleaned.

IN AN EFFORT to keep warm, the larvae burrow out of the cooler stomach area and worm their way into the "meatier" areas of the fish.

Cooking or freezing the fish before eating usually kills the parasites. Neither of those options are available to sashimi lovers. As Desowitz says in his book: "... the Japanese would not forgo their deeply entrenched taste for raw fish in return for freedom from a disease that affects 'only' several hundred people each year."

There have been five documented cases of anisakiasis in the United States, he said. Three of those patients made the discovery themselves when they felt tingling sensations in their throats after eating fish and then coughed up the wiggling worms.

Desowitz, who said he'll continue to eat sashimi "worm or no worm," also is conducting research on another parasite that may be linked to asthma. Studies done at Kapiolani-Children's Hospital on young patients with asthma have produced promising results, he said.

A DRUG USED to treat a parasitic infection called filariasis has been shown to "act as an anti-asthmatic agent," Desowitz said. Results of the latest study are being analyzed now, he said.

His soon-to-be-published book is a combination of research, anecdotes and experiences he compiled while traveling throughout the world studying parasitic diseases. The 55-year-old researcher earned his doctorate in parasitology and a doctorate in science at the University of London.

He has conducted research or taught in Bangkok, Singapore and Nigeria. He has been involved in World Health Organization projects in Fiji, Tonga, Western Samoa and Indonesia. He has served as consultant to the Navy, the National Academy of Science Committee on the effects of herbicides in Vietnam, and the United Nations Educational Scientific Cultural Organization's tropical forests project, among others.

Another book written by Desowitz recently won a first-place award from the American Medical Writers Associatio "Hawaii doesn't have as many parasites as I'd like, but we make do," Desowitz said with a grin. His research is conducted in laboratories at Leahi Hospital.

Robert Desowitz

9/7/77 S-B

Needlefish Leaps, Stabs Kauai Boy in the Eye

LIHUE, Kauai—A 10-year-old Kauai boy suffered a critical injury Monday when he was stabbed in the eye by a needlefish as it leaped from the water into a small boat.

Aaron Carvalho was fishing with his father, John S. Carvalho, a Lihue Plantation engineer, and his younger brother when the freak accident occurred.

The father was setting a net in Hanamaulu Bay when Aaron suddenly fell into the bottom of the boat unconscious. The four-foot-long fish was thrashing about.

The injured boy was taken to Wilcox Hospital by his father for emergency help and was later taken to Straub Hospital in Honolulu.

A neurosurgeon at the hospital said the eye was seriously cut but may be saved.

The needlefish, known as aha aha or au au to the Hawaiians, feeds on the surface in a leaping pattern.

Boy Hurt by Fish Has Brain Injury

9/8/77 S-B

The 10-year-old Kauai boy who was stabbed in the left eye by a leaping needlefish in a freak fishing accident Monday is still in "critical condition" today with a brain injury, according to his neurosurgeon at Straub Hospital.

The physician said Aaron Carvalho remains unconscious and the extent and cause of an apparent bruise of his brain are not yet known.

He said earlier that the boy's eye was seriously cut but may be saved.

According to a Star-Bulletin report from Kauai, the youngster and his younger brother were fishing with their father, John S. Carvalho, a Lihue Plantation engineer, when a four-foot-long needlefish apparently leaped out of the water into their small boat.

The father, who was setting a net in Hanamaulu Bay, said Aaron suddenly fell to the bottom of the boat unconscious and the fish was thrashing about nearby. The boy was taken to Wilcox Hospital on Kauai and later transferred to the Straub Hospital in Honolulu.

10/2/80

George Balazs [redacted]

On 9/16/80:

I examined microscopically Turbinaria (relatively intact) taken from the stomach of a turtle from French Frigate Shoals. The algae did not harbor any apparent numbers of the epiphytic dinoflagellate, Gambierdiscus toxicus (~~6-10~~ 80 μ diameter), which (a) frequently attaches to Turbinaria and (b) is implicated as the primary source for ciguatera poisoning in the tropical Pacific + Atlantic.

Nancy Withers

(Nancy Withers)

22 April 79

Feeling confused? There may be some

By PAT HUNTER
Advertiser Medical Writer

There's no way to tell by looking at it if a fish you've caught over an island reef is going to give you ciguatera fish poisoning, according to a Honolulu physician who is doing research on fish poisoning.

"But you can give a little bit of the raw fish to your cat (or a mongoose or a dog) and see what happens," said Dr. J.K. Sims, whose interest in dangerous marine organisms in general and fish poisoning in particular grew out of his hobby of skin diving.

"If the cat throws up or becomes unsteady on its feet, don't eat the fish," he said.

Sims, who is training coordinator for the Hawaii Medical Association's Emergency Medical Services program, has mounted an exhibit at the Hawaii Medical Library on dangerous marine organisms and how to treat injuries or illnesses from them. It will be on view during library hours weekdays and Saturdays through May.

Most widely known of the sea-going, reef-dwelling creatures that sting is the Portuguese Man-o-War, a purplish-blue jelly fish that floats into shallow shoreline waters with the tide. If encountered in the water, or even after it has washed up on the beach, its dangling tentacles cause painful welts on the skin, where it has shot its tiny arrows.

Sims said the first thing to do to treat a Man-o-War sting is to remove the tentacles clinging to the skin, if any. "But don't do it with your bare hand. Use a knife blade or wear thick gloves to avoid getting stung yourself."

He said that while urine applied to the site of the sting is very effective, this treatment can be hazardous if the urine is infected. "I generally use a paste made from unseasoned Adolf's meat tenderizer and isopropyl (rubbing) alcohol to rub on the site of the stings. This reduces the pain and itching by breaking down the protein in the poison."

However, he cautioned, if the victim is allergic to rubbing alcohol, vodka or another high-proof liquor can be used instead. "If the person is allergic to papaya, use alcohol alone," he said. "And if the itching persists after this treatment, a saturated solution of sodium bicarbonate can be applied to the site of the sting."

Other reef-dwellers that can inflict painful stings are wana (sea urchins), cone shells, stingrays and scorpion fish.

"Most of these carry a toxin that is broken down by heat, so the site of the sting should be soaked in hot water," Sims said. "It will take only about 10 minutes to get rid of wana poisoning with the hot water treatment, but larger spines that may have lodged in tissue of a hand or foot should be removed by a physician."

He said that the hot water treatment takes longer for stings by other creatures — about an hour to remove stingray poison and a half-hour to an hour and a half for scorpion fish stings.

Sims said, in addition, that sometimes people who eat wana as a delicacy get sick from bacterial food poisoning, and that during certain seasons the creature's ovaries are extremely toxic.

"Red-orange sponges can cause sever dermatitis and peeling of the skin," he said. "Kaneohe Bay is loaded with them. The treatment for this ailment is vinegar soaks."

Sims said that a number of fish are dangerous to eat because they carry a variety of toxins within their brains, spinal cords, intestines or flesh.

"One should never eat balloon fish — the 'fugu' that men in Japan eat as a sign of male prowess — because the liver, flesh,

Having nightmares? thing fishy about it

skin and gonads of this fish are often loaded with a substance called tetrodotoxin which can kill you.

"Fresh-water eels, known to the Japanese as unagi, must be very well cooked before eating, because they have a toxin in their blood that can make the consumer very ill."

Other fish that have been implicated in ciguatera fish poisoning outbreaks are members of the mullet family, sardines and other reef fish, he said.

"Sharks and moray eels, too, can carry ciguatera poisoning because they are scavengers.

"But I want to stress that the fish that are usually used for sashimi are rarely implicated in ciguaters fish poisoning because they are deep-water fish."

Sims said the current theory of what causes fish to become contaminated with ciguatoxin involves what they eat. "Small reef fish live on tiny dinaflagellates that float in the sea," he said. "These cause the small fish to accumulate the toxin in their tissues. It's not easily eliminated.

"Then middle-sized fish eat the smaller ones, and still larger fish eat them. Each time the toxin accumulates in the tissues, and when the fish are caught and eaten by people, they contract ciguatera poisoning."

Sims said the illness has about 150 signs and symptoms, the most prominent of which are tingling and burning around the mouth and throat and in the hands and feet, which can progress to total body itching, and temperature reversal, in which cold is perceived as hot and vice versa.

"There are also vomiting and diarrhea, disorientation, weakness and paralysis, among other things. We think there may be a cluster of toxins involved, which would explain why the victims' symptoms can be so different," he said.

Sims said emergency treatment for ciguatera poisoning is to clean out the patient thoroughly by inducing vomiting, if necessary, and giving a cathartic. Then other symptoms are treated according to what has been manifested, he said. "If the patient's calcium level is low normal or below normal, intravenous calcium can be helpful."

Sims said that cooking by any method does not deactivate ciguatoxin, and cautioned persons who become ill after eating freshly caught reef fish or predators of reef fish to save some of it for testing for the toxin.

Other fish that can cause poisoning are some parrot fish which sometimes harbor toxins, one of which is similar to ciguatoxin, he said.

"And any of the sea-going mullet family can carry a toxin that causes numbness, tingling, delirium, confusion and terrible nightmares that last anywhere from three days to a week," he said.

"This illness could be avoided, since the toxin is only in the brain and spinal cord of the fish. But mullet also can carry ciguatoxin in any of their tissues.

"People in the tropics could avoid a lot of fish poisoning by following a few simple rules. Never eat the head or intestines, brain or spinal cord of a fish. Clean it thoroughly as soon as possible after it is caught, and refrigerate it to avoid bacterial poisoning developing.

"Never eat shark liver, and don't eat shark or moray eel flesh if there's a ciguatera poisoning outbreak."

Sims said that last year there were a number of ciguatera poisoning cases, as well as some sardine poisonings reported from Kauai and Oahu. On Oahu, uhua and other fish from Pokai Bay and the reef runway were implicated; on Kauai, it was sardines caught off Port Allen.

Advertiser photo by Edwin Tani

Fresh mullet comes ashore on Maui: Will it cause "numbness, tingling, delirium, confusion and terrible nightmares that last anywhere from three days to a week?"

WORLD NEWS IN BRIEF

Ship Tragedy

MANILA, Philippines (AP)—At least 12 people were killed and an unknown number injured when an interisland ship with 400 passengers burned and exploded today shortly after it left here, survivors said.

The number of survivors was not immediately known. Officials of Aboitiz Shipping Co., owner of the M-V Juan, refused to give information about the fire. Employees who asked not to be identified said at least 266 were rescued.

"I saw flames and later I heard two explosions like thunder," said Eufroncina Amar, a 25-year-old survivor who leaped off the flaming ship. She said at least 120 others who jumped were saved by another vessel.

"I went up to the pilot and asked for a life jacket," she said. "He refused to give me one saying the fire would die down soon. He was drinking gin from a bottle."

The director of a Manila funeral home, Carlos Suicon, told Associated Press 12 victims, including five children, were brought to his establishment by relatives.

The Philippine Coast Guard said it fished eight bodies from the waters of Manila Bay and rescued five people.

A Coast Guard statement said the fire broke out early this afternoon about seven miles off the port of Manila. The statement gave no other details.

Many passengers smashed cabin windows to get out of the flaming boat, survivors said.

Saved from Sea

KARACHI, Pakistan (AP)—American planes and two warships rescued 39 people in the Arabian Sea, some huddled on the deck of a burning Greek tanker and others adrift in rafts for six hours, authorities said today.

All personnel were saved and more than half the survivors were plucked from the 25,973-ton Irene Sincerity yesterday as the tanker blazed 130 miles southwest of Karachi, said U.S. Embassy spokesman James Thurber in Islamabad, the Pakistani capital.

"Everybody is well," ship spokesman John Koukis said of the 37 crew members and two officers' wives taken off the cruiser California outside Karachi harbor and brought ashore by trawlers today.

Koukis said the Irene Sincerity, returning empty from India to Bahrain, was still burning today. He said the fire started in the engine

Criticism Capped

Washington Star Service

WASHINGTON—The State Department has classified a Defense Department report that criticizes several close allies, primarily Japan and Canada, for failing to bear their share of defense burdens.

The 200-page report, entitled "Allied Commitments to Defense Spending," was ordered by Congress last year as part of an effort to monitor promises by other nations to follow the U.S. lead in increasing defense spending.

The basic report, stamped "SECRET—Not Releasable to Foreign Nationals," was signed by Defense Secretary Caspar Weinberger and delivered to Congress on March 3. However, an unclassified version of the report, also prepared by the Defense Department, was later embargoed for further study by the State Department.

A few unclassified exerpts from the report were released today by one of its primary sponsors, Sen. Carl Levin, D-Mich., who accused the State Department of "stonewalling" because the report "would ruffle some European and Japanese feathers."

Tapeworm Risk

ATLANTA (AP)—The national Centers for Disease Control today warned that the growing popularity of raw fish dishes has increased the risk of fish tapeworm disease for Americans.

The infection is caused by eating the larvae of parasites in raw or incompletely cooked fish, the CDC said in its Morbidity and Mortality Weekly Report.

The report cited an outbreak in California last fall, when four people were infected with fish tapeworm after eating sushi, a Japanese dish made with raw fish. At least two of the patients suffered abdominal pains, diarrhea and nauses, the CDC said.

Fresh salmon from Alaska was identified as the probable cause of the outbreak, the report said.

Hospitals Plea

WASHINGTON (AP)—The Reagan administration agreed yesterday to ask Congress for an extra $17 million to temporarily keep operating eight hospitals and 27 clinics, including one in Honolulu, which the president wants to close, health officials said.

The request for supplemental funds for the Public Health Service hospital system was made necessary

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Thurber said the California and aircraft carrier America were on routine patrol when they received distress signals from the burning tanker. Besides 30 Greeks, the crew included Indian, Sri Lankan, Ghanian, Pakistani and Bangladeshi nationals, officials said.

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The request for supplemental funds for the Public Health Service hospital system was made necessary when the hospitals and clinics ran short of money earlier than had been expected.

The hospitals and clinics notified their employees Wednesday that unless money is forthcoming, they may be released Aug. 15, said Dr. Leonard Bachman, acting director of the Bureau of Medical Services.

Jury Clears Market in Fish Poisoning Suit

By Pat Guy
Star-Bulletin Writer

A Circuit Court jury has determined that a fish market was not liable for the ciguatera seafood poisoning that made a family ill last year.

The verdict "doesn't mean a person who ate fish and got sick (as a result of ciguatera poisoning) won't be able to recover (any damages)," said Robert Nip, who filed the suit on behalf of Norman and Betty Jong and their children Brenton and Nadine.

Nip said different cases may have different results, depending on the factual situation in each case.

Ciguatera is the most prevalent form of fish poisoning in Hawaii and the Pacific region.

Craig Furusho, representing Wing Sing Corp. which sold the fish to the Jongs, said he believes there are at least two or three other ciguatera cases pending here.

BOTH FURUSHO and Nip said they believed the verdict returned Tuesday night was the first here involving ciguatera poisoning.

Nip said he is "considering further steps." He said he may ask Circuit Judge Ronald Greig, who presided over the week-long trial, to render a judgment in favor of the Jongs in spite of the verdict.

Nip's case was presented under the legal doctrine of strict liability, which says that the seller must bear the loss when he sells something to a consumer that has a defect that is "unreasonably dangerous."

"It's not a question of fault but who should bear the loss," Nip explained.

Jong, who works in the city Finance Department, his wife, who is a school teacher, and their children aged 13 and 14, ate kumu fish Sept. 2, 1979, that they had bought from Wing Sing, a family-owned fish market on North King Street.

After eating the fish, they became ill with most of the usual symptoms associated with ciguatera poisoning.

Jong had to be hospitalized. He was unable to sleep or eat because of hiccups caused by the poisoning and lost more than 20 pounds. The family also suffered nausea, tingling sensations in their mouths and limbs and dizziness. Jong reported having double vision, very low pulse and low blood pressure.

THE JONGS STILL have occasional pain in their joints and muscles and they cannot eat reef fish, which is a big loss to a Chinese family, according to Nip.

Furusho argued on behalf of Wing Sing that the company did nothing wrong in selling the fish. He noted that there is nothing humans can do to detect the poison through visual inspection or to prevent it by cleaning, scaling or cooking.

Furusho argued that Wing Sing should not be held liable because it bought the fish from KK Fish Market. Wing Sing filed a suit against KK Fish Market, but the Jongs did not.

The jury found that there was a defect in the sale of the fish, but that it was not unreasonably dangerous.

A team of University of Hawaii researchers is conducting a three-year study of ciguatera with a $100,000 federal grant.

UH Researchers to Study

By Alan T. Matsuoka
Star-Bulletin Writers

A team of University of Hawaii researchers has been awarded a $109,000 grant from the federal government to begin a three-year study of ciguatera, the most prevalent form of seafood poisoning in Hawaii and the Pacific region.

The study will center on the factors which contribute to dramatic growths, or blooms, in the population of a dinoflagellate which has been implicated as the cause of ciguatera outbreaks, according to Arnold Banner, a university marine biologist at the Hawaii Institute for Marine Biology who will coordinate the research.

The dinoflagellate, a microscopic marine organism displaying both animal and plant characteristics, was first identified three years ago around the Gambier Islands in French Polynesia by a Japanese scientist who was able to establish that it produced the poisonous substance ciguatoxin.

LARGE CONCENTRATIONS also were detected two years ago in Pokai Bay during a time when Waianae residents were experiencing "not an inconsiderable number of cases of fish poisoning," said Banner, who has been studying the problem for 25 years.

"We want to study the factors that might cause the dinoflagellate to change from just scattered individuals into massive blooms on the coral reef," Banner said yesterday. "If we can find out what those factors are, we might be able to account for these outbreaks of ciguatera."

Researchers also will look into the chemistry of the toxic substance and the way it acts on the mammalian body. The toxin affects the central nervous system and early symptoms — nausea, diarrhea, abdominal pains, skin rashes and breathing difficulties — could be felt within a half hour of eating a toxic fish.

As ciguatera becomes more severe, a victim can experience more severe disorders of the central nervous system such as an exchange in the tactile senses — cold items may feel hot and vice versa. Tingling sensations also may occur in the lips, mouth and extremities, and victims might find it difficult to maintain their balance. In extreme cases, paralysis or death may result.

THERE WERE 15 incidents of poisoning involving 43 people in 1978, 23 incidents involving 81 in 1979 and seven incidents involving 16 so far this year, reported to Mits Sugi, an epidemiological specialist with the state Department of Health. He said fatalities are rare, but one occurred in Hawaii during the 1960s.

The federal grant will allow continuation of research which has been going on for the last 20 years, said Ned Wiebenga, chief of the Health Department's epidemiology branch. While the dinoflagellate could be the cause, he said evidence at this point is circumstantial and there is other research which shows there may be as many as three toxins at work.

"We don't really understand the whole chain of transmission of the disease from fish to man," Wiebenga said. "But the circumstantial evidence indicates that some-

Seafood Poisoning

where in the food chain of fish and other aquatic organisms a material is ingested which is not dangerous to fish but is very toxic for human beings. They don't excrete it, but accumulate it, so a larger fish would probably become more dangerous."

BOTH WIEBENGA AND Banner noted that the outbreak at Pokai Bay occurred during a time when construction of a small-boat harbor was taking place there. The link has been seen elsewhere, also. "The proof may not stand up in court yet, but it's fairly interesting that there is a linkage," Wiebanga said. "The trouble is we don't have the definitive proof that we need to see what exactly is going on."

The incidence of ciguatera-causing fish has not been exceptionally high, but enough to cause concern among health officials and the fishing industry, which wants to expand into the Northwest Hawaiian Island chain. During the past year catches of all kahala, a large fish which is a popular food item, have been tested by a university pathologist before being auctioned on the open market by the United Fishing Agency.

The tests of about 1,600 kahala by Yoshitsugi Hokama, who developed a method of detecting ciguatoxin, showed that about 10 percent were toxic enough to be potentially dangerous if eaten. A variety of fish species caught in the Leeward Island chain also was tested by National Marine Fisheries Service researchers Bernard M. Ito and Richard N. Uchida, who found that 16 percent had levels of ciguatoxin considered hazardous to humans.

"THE OCCURENCE of ciguatera in some valuable food species found in the central Pacific affects not only the population by causing illness, but also, as is often the case, deprives them of a major source of much needed protein," they wrote.

Other research has indicated that ciguatera might have been responsible for the deaths of several Hawaiian monk seals, an endangered species, in the Leeward Island chain during 1978.

Announcement of the National Marine Fisheries Service grant was made by U.S. Sen. Spark Matsunaga's office this week. The original grant request was for $140,000.

Star-Bulletin

Health Page

Advice for You and the Latest Medical News

Section D

Friday, June 13, 1980 Honolulu

Clean Fish Catch Quickly to Avoid Poisoning Peril

D-12 Honolulu Star-Bulletin Monday, March 19, 1979

Fish eaters: Clean that fish as soon as possible after catching it.

That's the advice from the state Department of Health and the University of Hawaii concerning reef and offshore fish because of fish-poisoning danger.

The department, reporting on a year-end analysis of ciguatera fish poisoning, says that any reef fish can be implicated.

There were five incidents reported that involved 11 people in the Pokai Bay area, with uku, kumu, mullet or weke as the fish involved. Three incidents involving 10 people were reported from kumu caught off Waialae-Kahala, ulua from the reef runway, and mu and uku from Kaena Point.

A MARQUESAN sardine eaten on Kauai was implicated last fall.

The Island practice of pulehu, throwing the freshly caught fish onto a charcoal fire without gutting, puts the consumer in jeopardy of poisoning. That's why the Health Department advises fish eaters to clean the fish as soon as possible, discarding the gut, body organs and head.

There is no way of looking at the fish or testing the fish before eating. From 1 to 5 percent of fish caught at Pokai Bay have been implicated, the department says.

Any poisoning should be immediately reported to the department's epidemiology branch. Samples of the fish eaten should be packed in ice and sent to the department for testing.

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6

CIGUATERA: A DISEASE FROM CORAL REEF FISH

Albert H. Banner

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I. Introduction

In association with coral reefs throughout the tropical world are fish which, if eaten, may cause illness or even death. The fish range from small herbivores to large reef carnivores and belong to many orders; even some invertebrates have been implicated. The disease, or diseases, for there may be more than one, is known as "ciguatera." One of the outstanding characteristics of the disease is its ecology, for the same group of species of fish varies in toxicity in both space and time. A fish that is favored from the waters of one island may be highly toxic in an adjacent island, or an island that has not harbored any toxic fish for as long as tradition remembers may within a year have "all of the fish" become toxic.

The toxicity of the fish must have been known to the island people long before the advent of western exploration, for soon after the explorers

ventured into the region of coral reefs they began to report serious, and even lethal, cases of ciguatera. In 1601 the Dutch at Mauritius reported the disease; in 1606 the men of the Spanish explorer de Quiros were afflicted in the New Hebrides. Probably the most accurate early account, related by several observers, was illness of the crew of Captain Cook's *Resolution* after eating some *pajaros* (probably the red snapper *Lutjanus*) in the New Hebrides in 1774; the entrails also killed one of the captive pigs aboard (Cook, 1777).

The name ciguatera reaches back at least to the nineteenth century, for Poey in 1866 defined the disease as one contracted from eating fish. The name had been applied earlier to an illness found in Cuba that resulted from eating the "cigua" or "sigua," a turban shell, *Livona* (*Turbo*) *pica* L., and then was extended to the disease produced by fish (see Halstead, 1965).

The historical records of the disease and, to a lesser extent, the modern records are confused by use of the single term ciguatera to apply to a series of diseases resulting from a multiplicity of toxins of varying action. Many earlier workers lumped under the term all intoxications that resulted from eating any marine fish or invertebrates; modern workers have differentiated clearly among many of these toxins (see Section II). Even more confusing is the fact that the fish that bear the principal toxin causing typical ciguatera may concurrently bear other toxins, as reported by Banner (1967), Hashimoto *et al.* (1969b), Hashimoto (1970), Li (1970), and Yasumoto *et al.* (1971). In the main discussion that follows reference will be largely confined to the principal toxin, named "ciguatoxin" by Scheuer *et al.* in 1967. A brief discussion of the other toxins associated with ciguatera will be presented in a separate section.

The symptoms of the disease are primarily neurological, although gastrointestinal symptoms, including nausea and diarrhea, may be the first to appear. The characteristic early symptoms include the tingling of the lips, mouth, and tongue, which may be extended to an intense itching of the skin. This last symptom is so pronounced in New Caledonia that the disease is known as "La Gratte." Exhaustion and muscular weakness, particularly of the legs, become progressively worse, and in more intense cases, partial to complete paralysis may result. Generalized aches and pains, visual disturbance, and an "inversion of the senses," in which cool objects are reported to feel hot, and hot objects cold, are common. Profuse sweating, a rapid but weak pulse, and a loss of reflexes are often noted. In acute cases, the loss of reflexes becomes more widespread, clonic and tonic convulsions occur, and muscular fasciculation may be

pronounced. In these cases the patient often becomes comatose. Death, when it occurs, is the result of respiratory failure. The initial symptoms usually occur within six hours after ingesting the fish. Recovery may take months, and permanent damage has been recorded (see Halstead, 1967).

The severity of the attack naturally depends upon the amount of toxin ingested relative to body size; as shown below, the viscera contain relatively more toxin than does the flesh, and severe attacks usually result from eating liver and gonads.

The symptoms are not consistent (Banner and Helfrich, 1964, from Wake; Bagnis, 1968, from Tahiti). Bagnis reported that the gastrointestinal symptoms are more common in cases caused by herbivorous fishes and cardiovascular disorders and other symptoms in cases caused by carnivores. He suggested that this variability may be due to the presence of two or more toxins in the fish.

A person, once having suffered an attack of ciguatera, becomes sensitized in some fashion, so that he may have a recurrence of symptoms if he eats fish that do not cause symptoms in others. (How much of this is psychosomatic is problematical; the author has interviewed a physician in the New Hebrides who stated a patient had a recurrence of symptoms after eating canned sardines from the Mediterranean, and Australians who insist their symptoms recur after drinking beer.)

II. Differentiation among Marine Toxins

A large number of species of both fish and invertebrates, especially in the tropics, can cause intoxication when eaten. Only a few of these toxic forms have been studied in any detail; many of the reports are based on local knowledge and have never been actually observed by professionally trained personnel.

Most of the invertebrates found to be toxic have toxins that do not resemble ciguatoxin. These include such animals as the toxic sea anemone *Rhodactis howesi* Kent from Samoa (Martin, 1960), the crabs *Zozimus aeneus* (L.), *Platypodia granulosa* (Ruppell), and *Atergatis floridus* (L.) (Hashimoto, 1970, and other publications), and various mollusks.

However, ciguatera has been attributed to at least three pelecypods and one gastropod. The giant clam, *Tridacna maxima* Roding, was originally associated with ciguatera at Bora Bora, but Bagnis (1967) decided on the basis of the symptoms in two lethal cases that the causative toxin was not similar to ciguatoxin. Banner (1967) reported at least two toxins in this clam, but his preliminary chemical tests were not

definitive. McFarren *et al.* (1965) reported that an oyster, *Crassostrea virginica* (Gmelin), and a clam, *Venus mercenaria campechiensis* [= *Mercenaria campechiensis* (Gmelin)], from coastal waters of Florida contained a toxin they described as "ciguatera-like" as it was lipoidal and caused similar symptoms in test animals; neither the chemistry nor the pharmacology was investigated further. They also reported a similar toxin from a concurrent bloom of the planktonic dinoflagellate *Gymnodinium breve* Davis. The third pelecypod reported to cause symptoms similar to ciguatera was a clam of the family Arcidae from Wake Island; it has never been studied and the report of its toxicity was merely from hospital records (Banner and Helfrich, 1964).

The gastropod that was thought possibly to harbor ciguatoxin or a related compound was one of the Pacific turban shells, *Turbo angyrostoma* L. This has been reported to be toxic, particularly from the Tuamotus and from Marcus as well as several other places in the Pacific by Hashimoto *et al.* (1970). They made initial studies on the nature of the toxins and found two, one water soluble and one lipoidal; the lipoidal toxin, although similar to ciguatoxin in its chemical solubilities, caused different reactions in the test animals. The authors concluded that it was not ciguatoxin.

The only other invertebrate suspected of bearing ciguatoxin is the short-spined sea urchin, *Tripneustes gratilla* (L.). Randall, working in the Societies in 1956, heard a report from the Tahitians that this urchin, esteemed for food, was toxic in areas of the reef where the fish were toxic. He returned several specimens in the frozen state to the University of Hawaii for testing; one of these caused death in a test mongoose (Randall, 1958). No further studies have been made.

Among the fish in the tropics, there are at least four major types of toxicity in addition to ciguatera. The best known group is that labeled by Halstead (1967) as "Tetrodotoxic" and includes many species of puffers of the family Tetraodontidae as well as species in related families. The toxin appears to be endogenous in the fishes, and Tsuda *et al.* (1964) have elucidated the full chemical structure of the toxic molecule. The basic pharmacological action of the toxin appears to be an antagonism of the increased Na^+ ion permeability associated with electrical excitability in cellular membranes (Kao, 1966).

The second type of intoxication arises from a histaminelike response from eating tuna and related fish. Halstead (1967) called these fishes "Scombrotoxic." The causative toxin is produced in the early spoilage of the dark-fleshed tunalike fish by the action of the bacterium *Proteus morganii* that reduces histidine to toxic histamine and possibly related products. Patients have remarked that the toxic fish have a "sharp or peppery taste." The symptoms are typical of histaminic drugs. The disease was reviewed by Kimata (1961) and Halstead (1967).

The two other types of intoxication are less well known. The type of toxicity labeled by Halstead (1967) as "clupeotoxic" and confined to the suborder Clupeoidae, may be found in sardines, herring, shad, anchovies, and tarpon both in the Caribbean and the tropical Indo-Pacific. Because the disease may lead to death within 15 minutes to a few hours, and because the fish are plankton feeders, the toxin does not appear to be related to ciguatoxin, which has a slow onset of symptoms and is confined to fish associated by their food chain with the benthos. Nothing is known of the biological origin nor the chemical nature of the toxin; little has even been reported on the symptoms. The disease is discussed by Halstead (1967). Finally, a few species of mullet, surmullet, rudderfish, and possibly others from restricted localities are reported to cause short-lived hallucinations when eaten. Although hallucinations have been documented in a few cases (Helfrich and Banner, 1960), the disease has never been studied in the laboratory. For a review, see Halstead (1967) under "Ichthyoallyeinotoxic Fishes." (It is noteworthy that Halstead preferred his name, "ichthyoallyeinotoxism," to the term coined—and rejected—by Helfrich and Banner for the same syndrome, "ichthyosarcephialtilepsis," yet the earlier name has four more letters!)

In addition, Halstead (1967) listed other rare and unstudied types of toxicity in fish: sharks (some of which appear to carry toxins other than ciguatoxin); oil fish or escolars [whose oil, composed largely of wax esters according to Nevenzel *et al.* (1965) and Mori *et al.* (1966), is strongly purgative]; fish in several orders with toxic eggs; eels (some of which may have toxic blood); and a few perchlike fish that seem to have a toxin in their livers. On each category Halstead bestowed a characteristic name, some of which are shorter than those cited above.

III. Assays for Toxicity

In the South Pacific there are a number of popular "tests" for toxicity of fish; these include the discoloration of silver coins or copper wire and the repulsion of flies and ants. Banner *et al.* (1963) reported on controlled experiments on these popular tests and discarded them all as being completely invalid.

The basic test for any toxin or toxins that cause disease through ingestion must be a feeding test on a sensitive animal and be based on mea-

sured test meals given at a percentage of body weight. Many investigators from Hiyama (1943) on have used cats or kittens, feeding at 5–15% body weight. Although the cats are sensitive to the toxin, their absorption of the toxin cannot be quantified for they usually regurgitate part of the test meal. In a search for a better test animal, Banner *et al.* (1960) reported on attempts to use 37 species of animals, from protozoans to mammals, as test animals. Only five appeared to respond in a noticeable fashion to the oral administration of the toxic fish flesh. Two of these were mammals, the cat and the mongoose [*Herpestes auropunctatus* (Hodgson), an Indian mongoose introduced to Hawaii and elsewhere in the tropics]; the mongoose had the advantage that it seldom regurgitated the test meal. A third mammal, the mouse, did not respond reliably (it was afterward found that the laboratory mouse had such a high tolerance of the toxin that it could not eat enough of a highly toxic fish at one time to induce symptoms). The fourth animal, the painted turtle [*Pseudemys scripta* (Schoepff)] when sick or otherwise unhappy would merely withdraw into its shell, so there could be no quantification of degrees of reaction. The last animal, the stream crayfish introduced to Hawaii, *Procambarus clarki*, (Girard), could not be fed measured meals. The mongoose was chosen as the animal for standardized screening tests of raw flesh in Hawaii.

The standard technique evolved in Hawaii was to hold a trapped wild mongoose several days for observation. The animals to be tested were starved for 24 hours and then fed flesh at 10% body weight of the mongoose. The test animal was observed for 48 hours with its response rated from 0 (no response) to 5 (death within 48 hours). As the mongooses come from a wild population and are usually infected with a series of diseases (C. N. Stemmermann, personal communication), the response is only roughly quantitative, and critical tests must be run in duplicate or triplicate to avoid false positives.

Although feeding tests are necessary for screening fish for toxicity, they are not satisfactory for further studies of the toxin. The first injection tests of potentially ciguatoxic fish were initiated by Hiyama (1943), who used ethanol as a solvent. Hashimoto (1956) used acidulated methanol in his study on a toxic barracuda. Halstead, with numerous coauthors (see principally Halstead and Bunker, 1954), based a series of reports of faunal surveys upon aqueous extracts of flesh and visceral organs injected into mice by various routes. In a subsequent paper (Goe and Halstead, 1955) he cast some doubts on the reliability of his test when he reported the intoxication of a student by a fish pronounced nontoxic as a result of injection of an aqueous extract. The technique was subsequently questioned by Banner and Boroughs (1958) and most recently by Dammann (1969) and Brody (1970). A new type of aqueous injectant was reported by Keene *et al.* (1968) who used an emulsified fish tissue homogenate that subsequently was heat precipitated and centrifuged. This they injected intramuscularly into various crustaceans. The test was subsequently abandoned at the University of Hawaii because of false positives, especially with herbivorous fishes (A. H. Banner, unpublished observations). Dammann (1969) experimented with Halstead's aqueous extract on "fairy shrimp, cricket, hermit crab, fiddler crab, isopod, octopus, chicken, sand flea and fish louse" without finding a reliable test. Brody (1970) extended Dammann's list to two more species of crab and a mixed protozoan culture. Spandorf (1970) tried two species of bacteria, also with negative results.

In two papers Banner *et al.* (1960, 1961) reported on a more reliable mouse injection test. They found that the initial extraction by hot ethanol carried enough concentration of normally nontoxic compounds to prevent firm differentiation between nontoxic and moderately toxic fish. These normal components of nontoxic fish could be removed from the test extract by concentration of the ethanol solution, solvent–solvent partition of the residue between water and highly nonpolar solvents, such as petroleum ether, and the reextraction of the aqueous phase with a polar solvent such as diethyl ether. The concentrate of the diethyl ether solution was either suspended in a plant oil vehicle or emulsified with Tween 60 (polyoxyethylene sorbitan monostearate) or dried human serum (Kosaki *et al.*, 1968); it could be administered via the intravenous (IV) or intraperitoneal (IP) route. It gave a quantitatively reliable result, especially if an LD_{50} figure was obtained. Subsequent studies have indicated that any modification of the chemical extraction and purification that would give a comparable level of purification would give equally satisfactory results, and that the same syndrome in the test animals obtained from the initial ether extract was maintained through progressively higher levels of purity of the toxin. The symptoms of the test mice included diarrhea, inactivity, excessive salivation and lachrymation, pupillary miosis, and death after convulsive spasms. Aqueous extracts of other fish were found to cause death in animals, but with different symptoms (Banner, 1967; Yasumoto *et al.*, 1971). Kosaki *et al.* (1968) have indicated that the chick is as satisfactory as the mouse for this bioassay, and in an earlier paper (Kosaki and Anderson, 1968) reported that pupillary miosis in the rabbit could be used to judge the toxicity of extracts of the eel *Gymnothorax javanicus* (Bleeker).

IV. Species of Fishes Known to Be Ciguatoxic

Halstead (1967) listed well over 400 species of bony fish, exclusive of sharks, that have been reported in the literature to have produced ciguatera. However, Halstead pointed out in his footnotes that many of the older references did not differentiate between the now accepted types of toxicity such as that of the sardines and the histaminelike reaction to the pelagic tunas. Correspondingly, when the Pacific islanders report that all the fish on certain reef sections have become toxic, they are obviously referring to many or most of the fish that they commonly eat. In contrast, only a few species of fish have been tested by differential tests with appropriate solvents for lipoidal toxins; even fewer have been tested by exact pharmacological tests (see Banner, 1967; Baslow, 1969). The true figure must lie below that reported by Halstead and considerably above those that have been accurately tested.

In general, ciguatoxic species are limited to those fish that feed on algae or detritus of coral reefs, especially the surgeonfish (Acanthuridae), parrotfish (Scaridae), and the larger reef carnivores that prey largely upon these herbivores (some of the often indicted species are shown in Figs. 1–12). It is the larger carnivores that become the most

Fig. 1. Gymnothorax javanicus (Bleeker). Moray eel. Part of a day's catch at Johnston Island. These eels are the basis of the study at the University of Hawaii on the chemistry and pharmacology of ciguatoxin. (Photo by J. E. Randall.)

Fig. 2. Ctenochaetus striatus (Quoy and Gaimard). Surgeonfish. (Photo by J. E. Randall from Eniwetok, Marshall Islands.)

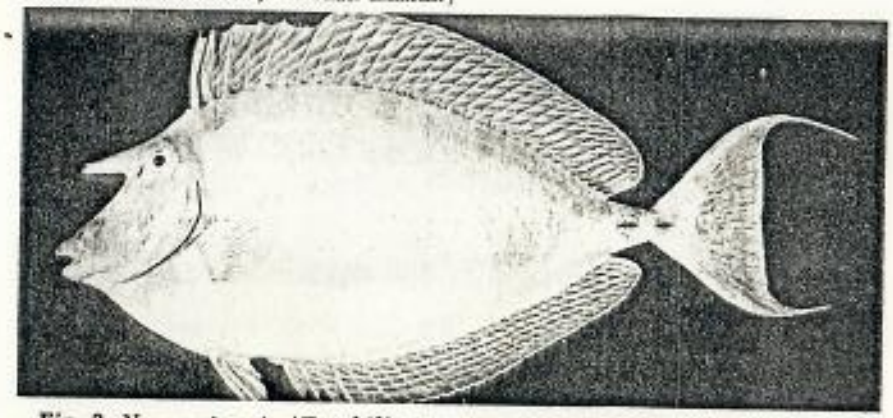

Fig. 3. Naso unicornis (Forsskål). Unicorn fish. (Photo by J. E. Randall from Hawaii.)

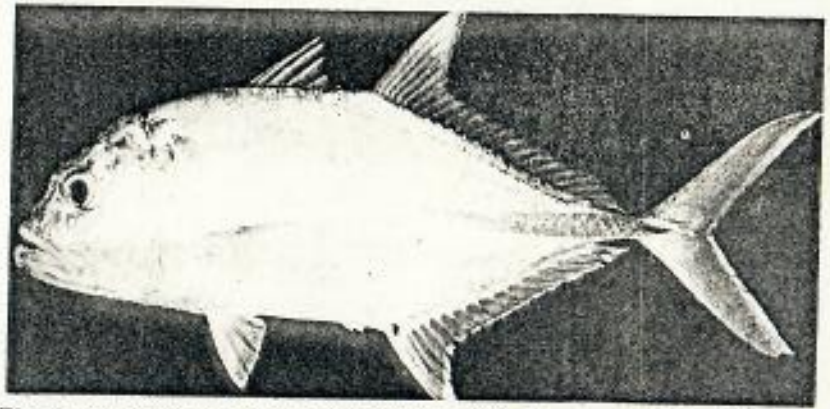

Fig. 4. Caranx sp. [probably *ignobilis* (Forsskål)]. Pompano, jack, or ulua. (Photo by J. E. Randall from Fanning Island, Line Islands.)

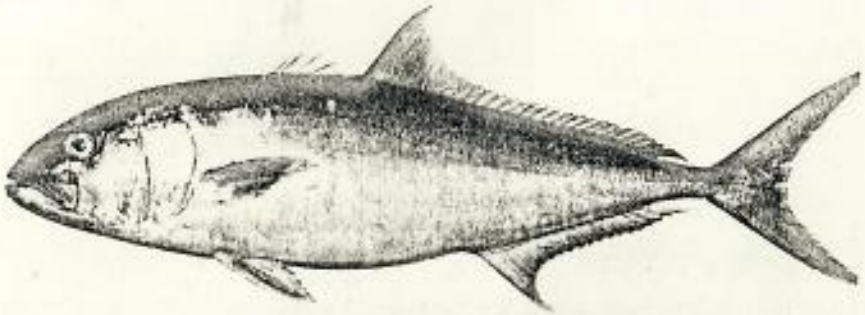

Fig. 5. Seriola dumerili (Risso). Amberjack. (Photo by J. E. Randall from St. John, Virgin Islands.)

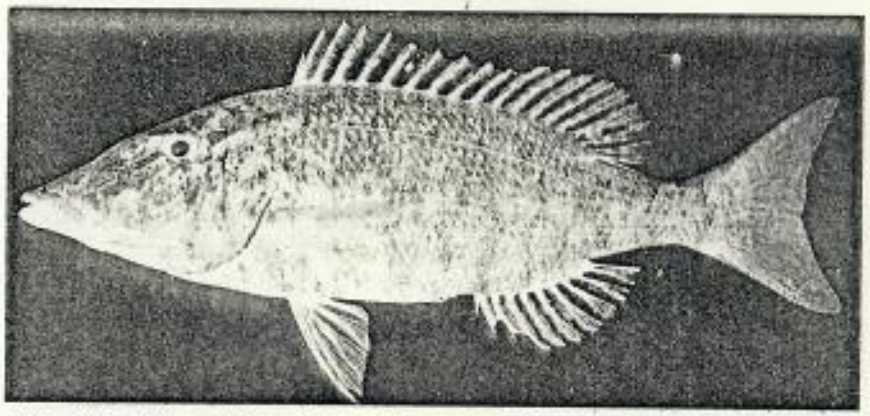

Fig. 6. Lethrinus miniatus (Forster). Scavenger fish. (Photo by J. E. Randall from Eniwetok, Marshall Islands.)

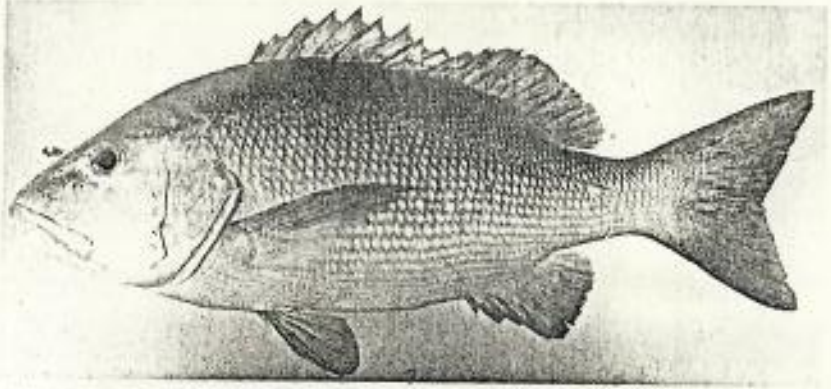

Fig. 7. Lutjanus bohar (Forsskål). Red snapper. (Photo by J. E. Randall from Ulithi, Caroline Islands.)

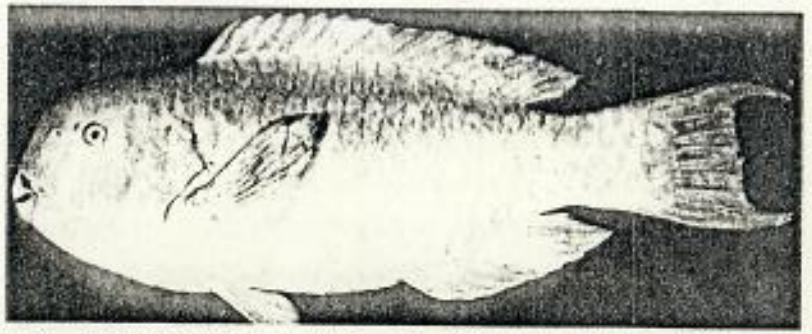

Fig. 8. Scarus jonesi (Streets). Parrotfish. (Photo by J. E. Randall from Tahiti, Society Islands.)

Fig. 9. Epinephelus fuscoguttatus (Forsskål). Grouper. (Photo by J. E. Randall from Eniwetok, Marshall Islands.)

Fig. 10. Cephalopholis argus Block and Schneider. Spotted Seabass or Spotted Grouper. (Photo by J. E. Randall from Tahiti, Society Islands.)

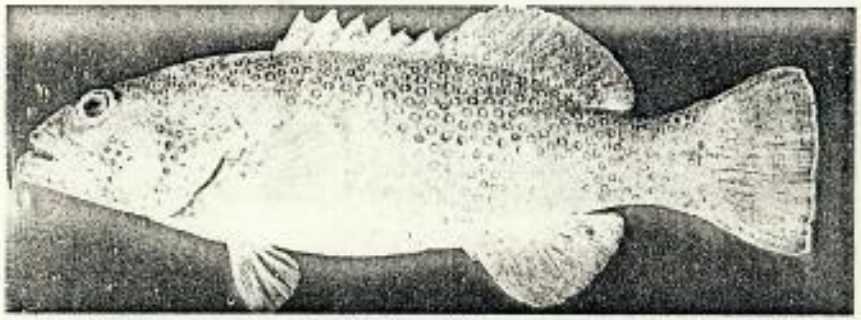

Fig. 11. Plectropomus truncatus Fowler and Bean. Grouper. (Photo by J. E. Randall from Eniwetok Atoll, Marshall Islands.)

Fig. 12. Sphyraena barracuda (Walbaum). Great Barracuda. (Photo by J. E. Randall from St. John, Virgin Islands.)

dangerously toxic: reef sharks (especially of the family Carcharhinidae), moray eels (Muraenidae), jacks or pompanos (Carangidae), wrasses (Labridae), snappers (Lutjanidae), scavengers (Lethrinidae), certain inshore tunas (Scombridae), groupers (Serranidae), and barracuda (Sphyraenidae). Many other reef fish not fitting exactly into these two groups may carry the toxin, as possibly do some of the triggerfish (Ballistidae). Likewise, in general, none of the pelagic fish and few of the reef plankton feeders, or those that feed over sand, mud, or turtle grass, have been blamed for ciguatera, and in those cases the identity of the disease in the strict sense may be questioned. It is difficult to state which species is the most highly toxic throughout the Pacific, but certainly the fish of the family Lutjanidae, especially the red snapper *Lutjanus bohar* Forsskål would be high, if not highest, on the list.

V. Distribution of Ciguatoxin in a Population

All of the fish in a single population are not equally toxic; Fig. 13 gives the cumulative data on moray eels (*G. javanicus*) weighing over 2.2 kg from Johnston Island, based on the mongoose test (Banner,

1974). It should be noted that even when the flesh of eels is rated as 0 in the mongoose test, the liver may have an appreciable amount of the toxin (Yasumoto and Scheuer, 1969).

With the selection from a fish population of only the large fish, the correlation of toxicity with size is not too apparent. In the 585 eels given in Fig. 13, those of 0 toxicity averaged 8.4 kg, whereas those rated as 5 averaged 9.5 kg (original data). In the samples of *L. bohar* from Palmyra Atoll where the size range was greater, the correlation is readily apparent (Fig. 14; the significance of the contrasting data from 1962 and 1968 will be discussed in the next section). As Helfrich *et al.* (1968) pointed out, not only are the larger fish more likely to be toxic, but they also are more likely to be highly toxic; the small fish, when toxic, usually rate 1–3 on the mongoose test, whereas the largest more often rate 4 and 5. This relationship between size and toxicity has been remarked upon before by many workers, but not objectively quantified with such large samples (see Halstead, 1967). Bagnis *et al.* (1970) have shown that fish from a toxic area that appear to be nontoxic or slightly toxic by a feeding test may contain enough extractable toxin to cause symptoms when injected in mice.

Previous writers have also discussed the distribution of the toxin within various parts of the fish. Studies (A. H. Banner, unpublished observa-

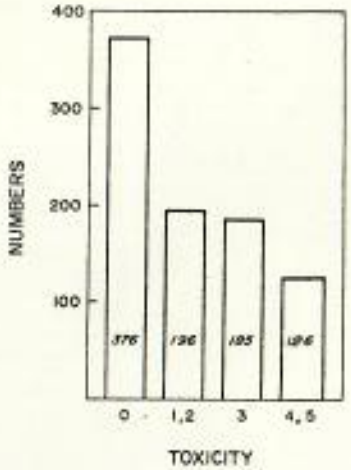

Fig. 13. Toxicity of *Gymnothorax javanicus* from Johnston Island, 883 eels all over 2.2 kg in weight (average 9.2 kg) caught from 1963 through 1969; toxicity ratings by the mongoose test. As toxicity ratings 1 and 4 appear to be transient, they have been combined with 2 and 5 in this graph. (From Banner, 1974 by the courtesy of Marcel Dekker, Inc.)

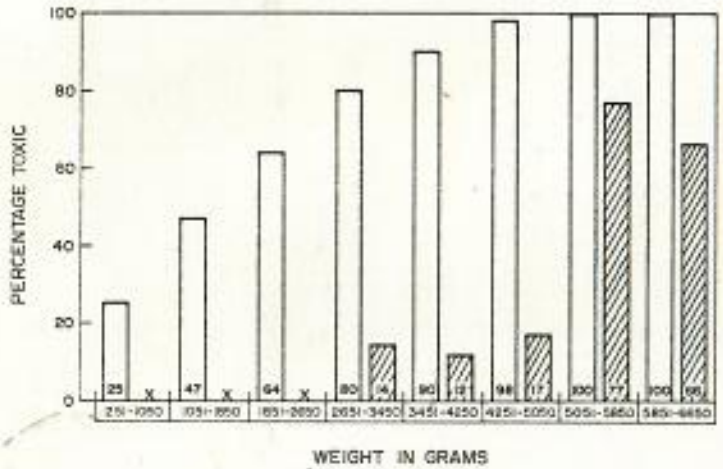

Fig. 14. Percentage of toxic *Lutjanus bohar* by weight categories, Palmyra, 1959 (solid) and 1968 (hatched) by the mongoose test; 1959 data based on 437 specimens; 1968 data based on 168 specimens. (Note: In 1968 only one specimen less than 2.6 kg was tested and only 25 in the two highest weight groups.) (From Banner, 1974 by courtesy of Marcel Dekker, Inc.)

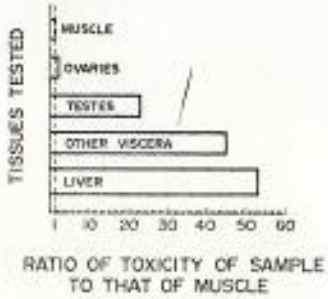

Fig. 15. Ratio of toxicity of pooled visceral samples to muscle in *Lutjanus bohar* from Palmyra; toxicity measured by the intraperitoneal injection of semipurified extract in mice and ratio based upon time to death. (Adapted from Helfrich *et al.*, 1968).

tions) at the University of Hawaii could show no statistically detectable difference between the flesh of the anterior and posterior halves of either *L. bohar* or *G. javanicus*. However, the viscera are definitely more toxic than the flesh. Helfrich *et al.* (1968) were able to show that the liver of *L. bohar* from Palmyra was about 50 times as toxic per unit weight as the flesh (Fig. 15). Yasumoto and Scheuer (1969) stated that the liver of *G. javanicus* is measurably toxic when no toxicity can be detected in the

flesh by feeding tests, but did not offer any comparative quantitative data (in personal communication Yasumoto estimated that the liver was about 100 times as toxic as the flesh per unit weight, which is roughly the ratio of the weight of the musculature to the liver—in other words, the small liver contained about the same total weight of toxin as did the body muscles).

VI. Distribution of Ciguateric Fishes in Space and Time

Halstead (1967) set the limits of ciguatera between 35°N and 34°S, but pointed out that the disease is largely confined to islands in the true tropics. Several authors besides Halstead have compiled lists of areas of known toxicity, either by broad areas (e.g., Whitley, 1943; Bouder *et al.*, 1962; Banner and Helfrich, 1964) or in particular archipelagoes (e.g., Jordan, 1929; Bartsch *et al.*, 1959; Cooper, 1964; Hashimoto *et al.*, 1969a). However, these compilations should not be relied upon as more than indications as to where ciguatera may occur for three reasons. First, almost all reports were compilations of second- or third-hand information, and almost none was based on laboratory testing. Second, often the informants did not distinguish between true ciguatera and other types of intoxications, even those as unrelated as puffer and sardine poisonings. Third, as the incidence of ciguatera varies in most areas from year to year, older surveys do not give the current picture (see below).

In both the Pacific and Caribbean, ciguatera seems largely confined to islands and is not found along continental margins. It is also apparently lacking in the waters of the great islands of the Western Pacific. When the author spent a year in Thailand and Malaya he found red snappers (*Lutjanus* spp.) to be a prized food fish and ciguatera was completely unknown; in the early 1960's he imported *L. bohar* from the Manila fish market to serve as nontoxic controls in contrast with the same species from Palmyra. Li, studying ciguatoxic eels in Hong Kong, stated that the fish were not caught in the vicinity of Hong Kong, but on offshore coral islands (K. M. Li, personal communication). Yip recently (1971) reported three toxic species of *Gymnothorax* from the Hong Kong fish market, but did not specify where they were caught; moreover, as he used an aqueous extraction for his tests, it is doubtful that he was reporting upon ciguatoxin. The rarity of ciguatera along the coasts of continents and large islands causes the odd reports of ciguatera from these waters to be viewed with suspicion. However, it has been reliably reported from the Ryukyu archipelago between Japan and Taiwan (Hashimoto

et al., 1969a) and may occur in *Seriola aureovittata* Schlegel (= *S. dumerili* Risso ?) from off central Honshu in Japan [Hashimoto and Fusetani (1968) had some doubts as to whether the toxin they extracted was identical with that reported by Scheuer *et al.* (1967) from the central Pacific].

Only two exceptions to the general rule have been reported, for de Sylva (1963) and Morton and Burklew (1970) have reported toxicity of barracuda from the coasts of Florida and Whitley (1943) for fishes along the Great Barrier Reef of Australia; these last may be lethally toxic (Tonge *et al.*, 1967). However, close examination shows that in Australia the toxic fish are not along the continental margin itself, but are found around the offshore reefs of the broad complex. In New Guinea there were a few rather vague reports on intoxications, but these again were from offshore islands (Banner and Helfrich, 1964); the last report on intoxications from the whole Indonesian archipelago was that of Stevenson (1914).

The situation in the Philippines seems to be somewhat controversial, for Banner and Helfrich (1964) reported "both medical and fisheries officers indicated that ciguatera is unknown to responsible authorities." Yet Halstead (1967) stated that "unofficial but reliable sources [report] that during 1957–1958 in fish poisoning outbreaks more than several thousand persons were involved." He stated the only known cause was the oceanic bonito—a fish that is more associated with scombroid intoxication than ciguatera. When the author revisited the Philippines in the spring of 1968 he inquired about ciguatera, and again all interviewed professed no knowledge of the disease. Even in the Sulu Archipelago, where coral reefs flourish and the water conditions are similar to those of isolated islands of the Central Pacific, not only the officials but also the fishermen knew nothing of the disease. This is not to say that some of the offshore islands, like the Cagayan Islands west of Negros, might not have unreported ciguatera.

In general, almost any of the islands of the Central Pacific and, to a lesser extent, those of the Indian Ocean and Caribbean Sea may have a few species of fish that may be sporadically toxic. An excellent example of this low level of toxicity is Hawaii where ciguatera-type of poisonings have been known intermittently since at least 1900 (Helfrich, 1963). The poisonings were of minor nature until 1964, when a sudden outbreak resulted in two deaths and one near death (Okihiro *et al.*, 1965); since then the cases have again been mild and sporadic. Yet in any of the tropical islands many of the safe fish may suddenly become highly toxic.

In the Central Pacific and in the Caribbean, peculiar discontinuous distributional patterns are found for toxic fish. One of the best documented areas is the sparsely inhabited Line Islands lying near the equator directly south of the Hawaiian Islands (for a review of the earlier work in this archipelago, see Helfrich *et al.*, 1968). Of the four major islands, Christmas Island, the southernmost, was only moderately toxic in the 1950's and 1960's; Fanning Island, next to the north, had an outbreak in 1946 but by 1960 even *L. bohar* was being eaten; on Washington Island, the following island, toxic fish were unknown except about a year after the breakup of a wrecked freighter in 1965; and Palmyra, about 250 miles northwest of Christmas, was perhaps the most toxic area in the Pacific in the late 1950's (see Fig. 14). In the Gilbert Islands, Cooper (1964) reported that of the 16 atolls, 10 harbored toxic fish in 1962–1963, all with decreasing toxicity since a peak in the decade around 1950. On the other six atolls, toxic fish were unknown in the postwar years. She also reported that the toxic areas around the atolls were limited to small patches, with the fish in the other waters considered safe; usually these toxic zones were associated with reef passages (Fig. 16).

The seasonal variability of the toxicity of fish has been reported by numerous authors; however, other authors reportedly could not detect any seasonality in their data (for summation, see Halstead, 1967). If other fish accumulate and store toxins as does *L. bohar* (Banner *et al.*, 1966), seasonal variation in the production of toxin would not be reflected in the toxicity of the fish. It may be, however, that the toxicity of some fish, such as acanthurids, does reflect the seasonally changing toxicity of the food making up their diets. A preliminary hint of this was given by Yasumoto *et al.* (1971), who found some specimens of *Ctenochaetus striatus* (Quoy and Gaimard) collected at a different season from their main collection, carrying what appeared to be different toxins. Two other factors may account for the reported seasonality. First, the nonseasonal nature of the fish bearing ciguatoxin may be confused with fish bearing other toxins of seasonal nature, as with sardines in Fiji. Second, the seasonal change in incidence of the disease may be a reflection of the intensity of the fishing effort. L. de Vambez, then the Fishery Officer for the South Pacific Commission, suggested that decrease in ciguatera in the winter in New Caledonia might be explained by the decrease in fishing effort during the period of rough seas (personal communication).

Ciguateric fish in any particular area may change in toxicity markedly over one to several years. Helfrich and Banner (1968) have pointed

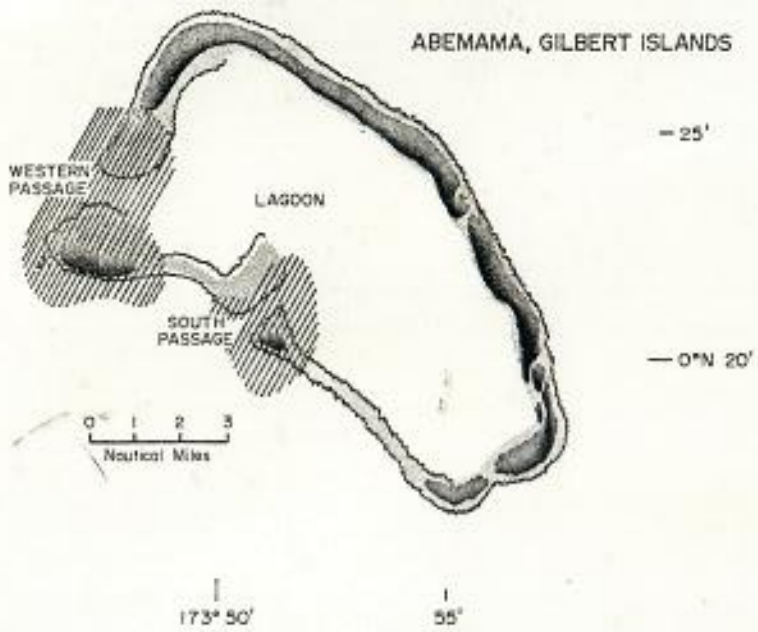

Fig. 16. Abemama Atoll, Gilbert Islands, showing areas (hatched) reported to be toxic by the inhabitants; they stated that in the stretch of reef between West and South Passages the fish were nontoxic. (Adapted from Cooper, 1964.)

out three general patterns of toxicity in the tropical Pacific: (1) where there was a sudden appearance, a rapid rise, and a slow decline in toxicity in areas where no previous toxicity was known—Palmyra is an example; (2) a similar rise and decline in toxicity in an area where ciguatera was previously known but of minor importance—the Marquesas are an example; and (3) where toxicity has apparently been continuous at the same level, high or low, over a long period of time—the New Hebrides and Jaluit Atoll in the Marshalls are excellent examples. The authors stated that when toxicity is rising, it is first noted in the reef herbivores, followed rapidly by the reef carnivores; in decline, the longer-lived carnivores such as snappers, sharks, and eels may remain toxic for years after the herbivores are reported to be nontoxic.

Several authors have recorded the rise of toxicity in areas where previously toxicity was unknown or very low (Bartsch and McFarren, 1962; Cooper, 1964). None has so fully documented the rise as did Bagnis (1969) for the atoll of Hao in the Tuamotus. His report has been reviewed by Banner (1974). Ciguatera had been unknown

to the atoll previous to its conversion by the French Atomic Energy Commission (CEA) to a staging base in January, 1965. The ciguatoxic fishes appeared in each area of the lagoon within 1.5–2.0 years after the changes in the marine environment. The geographic spread of ciguatoxic fishes is shown in Fig. 17; the rise in incidence of ciguatera is shown in Fig. 18. In 1971 the fishes of the western side of the atoll were still considered to be safe. As expected, the first fishes to cause ciguatera were herbivores, the acanthurids and scarids; much later did the carnivores, such as carangids and serranids, cause the disease. In all, Bagnis found 32 species in 15 families to have caused ciguatera.

The decline of toxicity is slow and less spectacular. The documentation of a decline on the basis of medical records is doubtful, for unless the drive for protein food impels the inhabitants to again and again sample potentially toxic fish as Cooper reported for the Gilbertese (1964), the falling off of case histories merely reflects that a person once or twice poisoned stops eating local fish. It is likely that the drop

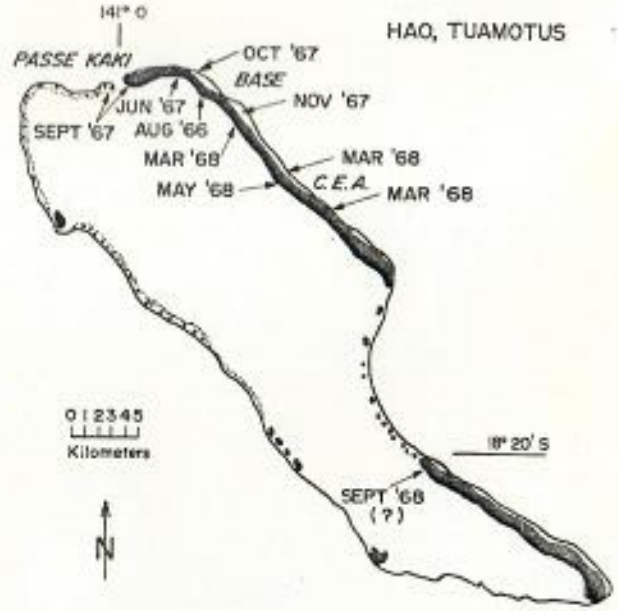

Fig. 17. Hao, Tuamotu Archipelago, showing spread of ciguatera from center of beaching area in August, 1966; data based on house-to-house survey in 1968. Even by 1971 no toxic fish were known from the southern and western portions of the atoll. (Adapted from Bagnis, 1969, in Banner, 1974 by courtesy of Marcel Dekker, Inc.)

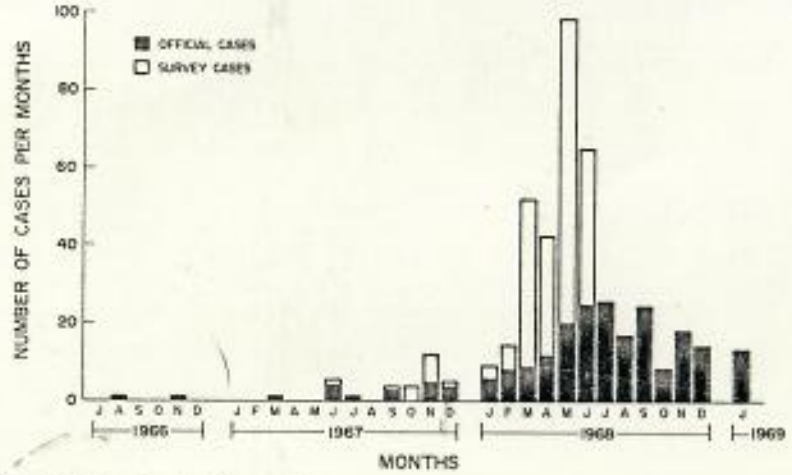

Fig. 18. Monthly incidence of ciguatera on Hao, Tuamotu Archipelago. Ciguatera was unknown to Hao previous to 1966. Solid bar represents the number of official cases reported to medical authorities; the open bar represents the number of additional cases that were not reported to medical authorities but that were discovered by a house-to-house survey. The epidemiological survey stopped in June, 1968; the official records were available through January, 1969. (Adapted from Bagnis, 1969, in Banner, 1974 by courtesy of Marcel Dekker, Inc.)

in cases shown by Bagnis from Hao thus does not represent a decrease in toxicity of the fish. However, the laboratory assays of toxicity of *L. bohar* from Palmyra for 1959 and 1968 given in Fig. 14 show a true loss of toxicity in the fish population; it is noteworthy that in 1968 it was the largest, therefore the oldest, fish that remained highly toxic.

VII. Origin and Transmission of the Toxin*

Many of the earlier suggestions as to the cause of ciguatoxicity in fishes have not survived modern scrutiny. The idea that the toxin is endogenous, similar to tetrodotoxin in puffers, has been rejected as the toxin is erratic in appearance in species of such broad phylogenetic span which are yet of such close ecological affinity. It cannot be a product

* This section is a summation of my review, "The Biological Origin and Transmission of Ciguatoxin," presented at the *Symposium on Physiologically Active Compounds from the Sea* at St. Petersburg, Florida in November, 1971 (Banner, 1974). This summation is included here as necessary for the development of this review; for fuller discussion and additional references, the reader should examine the parent article.

of bacterial decomposition of fish flesh, similar to scombrotoxicity, as the toxin is found in freshly frozen fish (Banner *et al.*, 1963). It cannot be from contamination with heavy metals, such as copper as previously suggested, or mercury as is found in the Minamata disease of Japan, for there are no heavy metals in the purified toxic extracts (see Section VIII). Instead, ciguatera is caused by a toxin that arises in the environment and is of biological origin.

Most workers in the field of marine toxins now accept the food-chain theory of transmission of ciguatoxin within the food web of coral reefs. The theory was best advanced by Randall (1958) who reviewed what was then known about the biology of ciguatoxic fishes. He rejected the idea that any of the larger reef forms, such as jellyfish, corals, balolo worms, pufferfish, and even the coarser attached algae, could be the elaborator of the toxin. He based this rejection on the knowledge of the food habits of toxic herbivores. He concluded (p. 257) that the original elaborator of the toxin was likely to be "an alga, a fungus, a protozoan or a bacterium," and he pointed out that if it were an alga, it would have to be filamentous and soft. This toxigenic form would then be eaten by the reef herbivores that in turn would be eaten by the larger reef piscivorous carnivores. He postulated that the toxin would accumulate in the carnivores; Helfrich and Banner (1968) suggested that the accumulation in the higher trophic levels could be comparable ecologically, but not physiologically, with the accumulation of modern insecticides in the trophic pyramid.

Since the publication of Randall's paper his theory has been confirmed both by field and experimental evidence. At Hao (see Section VI) the first fish to become toxic were the herbivores and detrital feeders, with the first carnivores causing the disease ten months later; presumably the lapse represents the time necessary for sufficient concentrations of the toxin to be passed through the food chain. In periods of declining toxicity, Cooper (1964) in the Gilberts, Banner (1974) at Palmyra (see Fig. 14), and Brock *et al.* (1965) at Johnston have recorded that only the larger carnivores remain toxic.

The experimental evidence also supports the food-chain hypothesis. Helfrich and Banner (1963) and Banner *et al.* (1966) reported on two experiments with captive fishes. In the first they fed a nontoxic omnivorous acanthurid, *Acanthurus xanthopterus* Cuvier and Valenciennes, with small portions of toxic fish flesh daily until the fish became lethally toxic to mongooses. In the second experiment they held a population of toxic *L. bohar* from Christmas Island in holding ponds on a nontoxic diet; at the end of 30 months of captivity, the fish showed

no detectable loss of toxicity. From these two experiments they concluded that the toxicity could be induced in fishes through diet, that the toxin in the diet caused no apparent harm to the fish, and that the toxin could be accumulated in the flesh and stored there for long periods of time.

The last and most conclusive support of the food-chain theory is the work of Yasumoto *et al.* (1971) on *C. striatus* from a toxic zone in Tahiti. The fish, a detrital feeder that cannot bite off pieces of attached algae, contained either ciguatoxin or a compound indistinguishable from it by present chemical and pharmacological tests. This toxin was found in the flesh, viscera, and gut contents. The presence of the toxin in the gut contents shows that the toxin originates at the base of the food chain in recognizable form and detectable quantities, and that there is no observed biochemical alteration of the toxic moiety by the metabolism of the fishes as it is passed through the food chain.

Although this evidence supports the theory of ecological transmission of the toxin, it leaves to conjecture both the identification of the elaborator and the ecological conditions that would cause the rapid rise and slow decline of ciguatoxin in a reef community. It may be possible that ciguatoxin originates in many species or even groups of the biota and that the appearance of ciguatoxin in a reef community may be caused by a variety of ecological conditions. However, in view of what is presently known about the toxin and its appearance, it would appear to be more logical to seek a single species or genus as the elaborator and to postulate that this form is stimulated into proliferation on separated reefs by similar ecological events.

If the reasoning of Randall and the evidence of Yasumoto *et al.* are accepted, the elaborator must be fine, but could be either a fine autotroph or heterotroph. The latter authors studied the food habits of *C. striatus* and reported that in the imperfect fractionation of the gut contents, the most toxic fraction was composed not of algae but of "unidentifiable particles"; they concluded that the elaborator(s) would be "both small in size and low in specific gravity."

The concurrent paper on the biology of ciguatoxin (Banner, 1974) reviews the unsuccessful search for the elaborator among the fine algae, mostly blue-green, and an inconclusive initial study by Gundersen of microbial heterotrophs and suggests that the originator may not be an alga but rather, as Randall had suggested, a bacterium, yeast, or mold. This microbial heterotroph could live in the bottom detritus—mostly broken fronds of seaweeds—where it would be picked up in quantity by the bottom-feeding *C. striatus*. However, the bacterium, yeast, or

mold might not occur in abundance in the coral reef environment but live as part of the nonpathogenic flora of the intestinal tract of the fish. There it would produce the toxin, harmless to the fish as has been shown, to be absorbed by the host and thus stored and transmitted to the rest of the ecosystem.

A number of hypotheses on the ecological causes of outbreaks must be discarded. Any postulated phenomenon that does not reach back into time would be improbable, for it could not account for the poisonings reported in the last several centuries. These postulations would include ecological disruption by the atomic testing program, modern insecticides, recent pollution, and similar changes. Similarly, ecological changes resulting from additional metal ions in the sea cannot be considered, as the atoll environments are notably deficient in metals.

Randall, in addition to supporting the food-chain hypothesis, also suggested that "new surfaces" on the coral reef might be the cause of the outbreaks. The new surfaces would be sections of the reef laid bare of its biota either by natural disasters such as hurricanes or by acts of man, such as anchoring of ships, shipwrecks, or the dumping of war surplus material. On these new surfaces would appear in early ecological succession the toxigenic form, which Randall suggests is most likely to be a blue-green alga. This influx of toxin would then spread through the ecosystem.

In the review, the author (Banner, 1974) carefully considers this hypothesis and cites more recent reef disruptions such as that on Hao, as supporting Randall's thesis. However, he also cites many cases—blasting of channels in the Gilberts, typhoon flooding in Fiji, reef disruption by the atomic testing program at Eniwetok, wholesale dredging at Johnston, even the denuding of reef surfaces by the starfish *Acanthaster*—that were followed by no reported increase in fish toxicity. He cites Randall's own report (1968) of failure to produce the toxigenic strain on artificial new surfaces (of plywood, asbestos board, etc.) in a toxic area in Tahiti.

Thus, no conclusions can be drawn about either the elaborator or the conditions that cause an outbreak. It is likely that the toxin itself is widespread and most ancient on the reefs, for all marine animals tested, in contrast with most freshwater and terrestrial animals, have developed immunity to the toxin (see Banner *et al.*, 1960). Some evidence pointed toward yeasts or molds, either in the reef environment or in the gut flora of fishes; these could be newly introduced into an ecosystem, as they could have on Hao by ship bottoms arriving from the then rampantly toxic waters of Tahiti, or they could exist on the

reef to be stimulated in growth by yet unknown factors. Any ecological speculation will be without foundation until the elaborator of the toxin is isolated and studied.

VIII. Chemistry of Ciguatoxin

The original work on the chemistry of the toxin was a mere excursion into its solubilities and other characteristics. Hiyama (1943) noted that the toxin could be removed from fish flesh with ethanol and that the toxin was heat stable so that cooking did not destroy the toxicity. Hashimoto (1956), working with only a 55 gm sample of barracuda, reported that the toxin was extracted from the fish by methanol and that it was soluble in organic solvents such as acetone and ether. Banner and Boroughs (1958) and later Banner *et al.* (1960) confirmed Hashimoto's results and found the toxin to be soluble in a series of polar organic solvents; they also noted that the toxin could not be extracted with water. They stated that there was no apparent loss of toxicity when the fish was stored below freezing for up to six months, when it was dried, or when the initial extraction was carried on with hot solvents. In the earlier paper they found that if secondary extractions were made under a normal atmosphere, toxicity was lost, but under a nitrogen atmosphere it was retained. To the contrary, Halstead and a series of associates (in papers from 1954 to 1958) presumed they were extracting the principal toxin with water, in some cases acidulated and in others in normal saline, for use in their bioassay. Similarly, Bartsch and McFarren (1962) and the group under Dammann (1969) also used aqueous extractions for their bioassay.

The use of column chromatography to purify the toxin was first hinted at, but not fully reported, by Banner *et al.* (1960). Hessel *et al.* in the same year reported on an unsuccessful technique of chromatography. In subsequent papers both Hessel and the group at the University of Hawaii reported successful chromatography (Hessel, 1961, 1963; Banner *et al.*, 1963; Banner, 1967; Scheuer *et al.*, 1967; Yasumoto and Scheuer, 1969).

The technique of extraction and purification at the University of Hawaii has gone through 14 modifications since 1962; the present standard technique, not previously reported in the literature, is given in Fig. 19. The yield is about 10 ppm of highly toxic raw fish. The only published reports on the structure of the molecule, named ciguatoxin, are by Scheuer *et al.* (1967). They reported (p. 1267) that they obtained a viscous oil, possibly pure (". . . the fact we have not obtained a

stable crystalline derivative keeps open the question whether ciguatoxin is a single substance with a tendency to decompose or whether it consists of several related compounds"). They obtained 0.75 mg of toxin per kg of highly toxic fish, and the final preparation had the toxicity of 0.5 mg/kg when injected intraperitoneally into mice. Combustion data gave the empirical formula of $(C_{53}H_{81}NO_9)_n$. Other experiments indicated that the molecule contains a quarternary nitrogen atom, one or more hydroxyl groups, and a carbonyl function. It should be noted that the statement that Halstead (1967, p. 304) attributed to "Yoshida *et al.* (1965)" that the toxin "in pure form is a phospholipid" was based on a manuscript reporting earlier studies of impure extracts and the statement was never actually published. The new procedure outlined in Fig. 19 gives a yield of 5–10 mg/kg and a toxicity of 0.025 mg/kg when injected intraperitoneally into mice (P. J. Scheuer and D. B. Boglan, personal commmunication), an increase over the 1967 report.

Baslow (1969) questioned the assumption made by the University of Hawaii group that the same toxin was found in the snapper *L. bohar*, the eel *G. javanicus*, and the shark *Carcharhinus menisorrah* (Müller and Henle) (= *C. amblyrhynchos* Bleeker). Scheuer *et al.* (1967) did not suggest that the whole lipoidal molecule was identical, but that the toxic moieties in the diverse fishes were of sufficient chemical similarity to give similar physiological activity. Rayner (see Section IX) has reported pharmacological characteristics for the toxin that appear to be unique, and has found these reactions caused by the toxin from the eel, the snapper, and the acanthurid *C. striatus* (Banner, 1974).

IX. Pharmacology of Ciguatoxin

Aside from the search for a suitable bioassay and the superficial observation of symptoms, the earliest work on pharmacology was that of Hessel *et al.* (1960), who reported that when the sciatic nerve of the frog was bathed in an emulsified acetone–ether extract of a toxic *L. bohar*, its action potential would drop by 25% or more in 70 minutes. Banner *et al.* (1963) reported that an extract made from a barracuda, originally extracted with ethanol and reextracted with diethyl ether, rapidly impaired the transmission of the nervous impulse to the muscle in a frog nerve–muscle preparation. As direct stimulation of the muscle caused contraction they concluded (p. 14) that "while the action potential of a nerve may be lost from long immersion in a solution of semipurified toxin, the immediate effect is on the nerve–muscle junction."

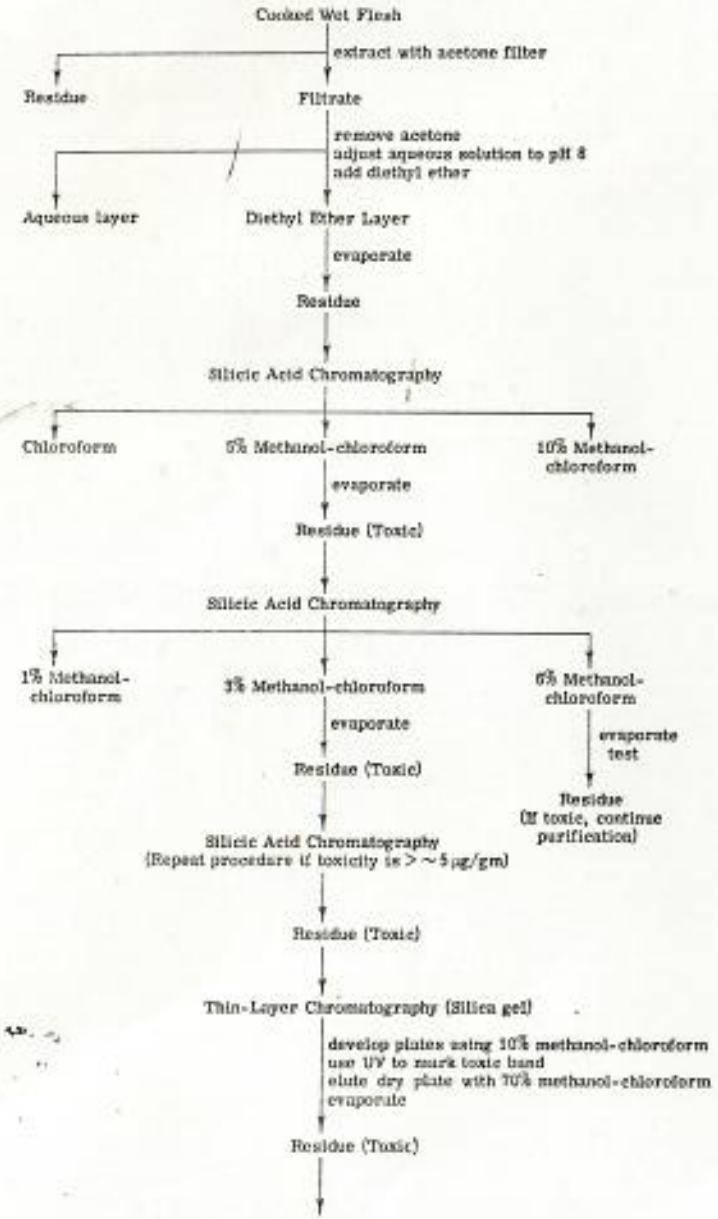

Fig. 19

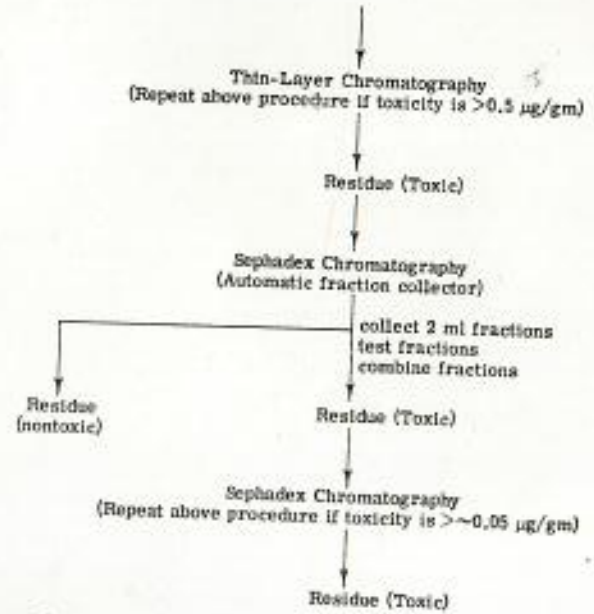

Fig. 19. Revised procedure for extraction and purification of ciguatoxin from flesh of *Gymnothorax javanicus* as developed at the University of Hawaii. (Note: All chromatography carried on in a cold room, near 4°C. Samples stored under inert atmospheres at about −20°C.)

Li (1965a,b, 1970) reported that the toxins from the red snapper, the moray eel, a grouper [*Epinephelus fuscoguttatus* (Forsskål)], and a shark (*C. menisorrah*) all behaved as anticholinesterases. These conclusions were based upon the following: (1) the similarity of symptoms in animals when injected with either crude extracts of ciguatoxin or known inhibitors of cholinesterase; (2) *in vitro* tests of cholinesterase activity in human red blood cells when bathed in a solution containing ciguatoxin; and (3) the recovery of intoxicated test animals when treated with Protopam Chloride (2-formyl-1-methylpyridinium chloride oxime), a drug known for its activity against anticholinesterases. Li (in Banner, 1967; Li, 1970) pointed out that all toxic fish do not have the same anticholinesterase activity.

The first doubt of the supposed anticholinesterase activity was cast when Okihiro *et al.* (1965) reported (p. 355) on attempting to treat a near fatal hospital case of ciguatera with 2-PAM, a cholinesterase regenerator: "The results were startling, unexpected and almost disas-

trous." Only heroic measures, including a bedside tracheotomy and massive dosages of atropine and magnesium sulfate saved the patient. Rayner and associates in two papers (1968, 1969) reexamined the contended inhibition of cholinesterase activity. In the first paper they studied the effects of other cholinesterase inhibitors and of ciguatoxin upon the reaction of the respiratory system; in the second they used a similar series of drugs and measured the reduction of the cholinesterase activity in the blood cells in living animals. From these two studies they concluded that while there may be some inhibition of cholinesterase in *in vitro* preparations, the action of the toxin is definitely not that of an anticholinesterase in living systems.

Reviewing the recent work carried out in the laboratory at the University of Hawaii, Rayner (1970b) concluded that ciguatoxin has a rather widespread action on excitable membranes involving an initial increase in excitability followed later by conduction block; the hypothesis was put forward that increased Na^+ permeability might be the causative mechanism for these changes. Subsequent studies (Rayner, 1970a; Rayner and Kosaki, 1970; Setliff *et al.*, 1971) have confirmed that ciguatoxin increases the passive permeability of frog skin preparations and frog muscle membranes to ^{22}Na, and that ciguatoxin extracted from both *Gymnothorax* and *Ctenochaetus* produces a depolarization in frog muscle cells that is specifically antagonized by the action of tetrodotoxin. This action appears to be associated with the competitive inhibition by ciguatoxin of the membrane stabilizing action of Ca^{2+} ions. The more common acute symptoms associated with ciguatera poisoning appear to be consistent with the proposed mechanism of action and a rational therapy would be to treat the membrane effects of ciguatoxin with magnesium sulfate and calcium gluconate (M. D. Rayner, personal communication). Those symptoms associated with the disfunction of the autonomic nervous system have been shown to be relieved by atropine (Okihiro *et al.*, 1965). On the other hand, it cannot yet be suggested that this is the only mechanism responsible for all of the symptoms which characterize the ciguatera syndrome.

Intoxications by ciguatoxic fish may cause pathological damage. Banner *et al.* (1960) quoted a neuropathologist who stated he could find no changes in the central nervous system of two mongooses maintained intoxicated on ciguateric fish for over a month. However, Li (1970) reported demyelination in both spinal cord and sciatic nerve in adult hens that had been given a large sublethal dose of a toxic extract intramuscularly two weeks before; he did not state the purity or the source of the extract.

X. Other Toxins

Ciguatoxin, or a compound of similar solubilities and similar pharmacology, appears to be the principal toxin in the carnivorous fish of the Pacific, being found in fish as diverse as sharks, moray eels, snappers, etc., from almost all parts of the Pacific (Banner *et al.*, 1960, 1963; Banner, 1967). However, the positive results obtained by Halstead in the 1950's with his bioassay utilizing aqueous extracts gave an indication that other toxins might be found in "ciguateric" fishes. This was also indicated by reports of seasonality in toxicity, as in Fiji where the fish are reported to be highly toxic during the spawning period of the balolo worm (Banner and Helfrich, 1964), for ciguatoxin appears to be stored in at least some species of fish (see Section VII). Differences in human symptoms were also remarked upon. Dr. E. Massal was quoted as stating that the symptoms of ciguatera in Tahiti were different from those in New Caledonia (Banner *et al.*, 1963). More recently, Bagnis (1968) reported (p. 28) from 350 cases in French Polynesia that he had attended (see also Section XII): "Gastrointestinal and neurologic symptoms are more common in cases caused by eating herbivorous surgeonfishes; cardiovascular disorders and more varied symptoms are common when piscivorous groupers or snappers have caused the poisoning."

The first actual laboratory report of a different toxin was by Banner *et al.* (1963) in which they reported that *C. striatus* had a toxin that was removed by alcohol, but not soluble in diethyl ether. Banner (1967) reported three different types of intoxications from fish based primarily upon pharmacology and supplemented by tests of *in vitro* anticholinesterase activity. The fishes of the first group, which included the carnivores *Gymnothorax, Lutjanus,* and *Epinephelus,* caused death by respiratory failure and gave high anticholinesterase activity. The fishes of the second group, with *Cheilinus* and *Acanthurus* being mentioned, caused death by cardiac failure and had lower *in vitro* anticholinesterase activity. The third group, which included *Caranx* and *Sphyraena,* was intermediary between the first two. He concluded (p. 161) that these differing reactions may "indicate the presence of other toxins." This differentiation was also discussed by Li (1970).

Hashimoto and Yasumoto (1965) and Hashimoto *et al.* (1969b) reported a water-soluble toxin that they named "ciguaterin" in the liver, and in some cases in the flesh, of five species of snappers, groupers, and eels from the Ryukyu and Amami Islands. In some cases they also found an oil-soluble ciguatoxin-like compound. Ciguaterin was found to be unstable in frozen raw fish, but stable when frozen after being cooked; it

caused vomiting in kittens when administered either by feeding or subcutaneous injection, but in all cases the test animal would recover in 24–48 hours.

The most definitive work on toxins other than ciguatoxin was that of Yasumoto *et al.* (1971) who worked with the acanthurids *C. striatus* and *Acanthurus lineatus* (L.) from Tahiti. Both fish contained an oil-soluble toxin either identical or very similar to ciguatoxin from Johnston Island eels, both in chemistry and pharmacology. In addition both carried a second toxin which was water-soluble, nondialyzable, and precipitated from concentrated solution by the addition of acetone; the last stage of purification reported upon was on a Sephadex G-100 column. When the toxin was injected intraperitoneally into mice, the mice displayed a "remarkable loss of activity, weakness in limbs, and convulsions before death that took place rather slowly: after several hours to three days, depending upon the dose" (Yasumoto *et al.* 1971, p. 730). The extract was also hemolytic and caused death in guppies when they were immersed in a 0.00044% solution. Like the ciguatoxin in the same fish, the toxin was found in the gut contents and in the liver; none, however, was found in the flesh.

The same authors said they had indications of possibly different toxins from the same species of fish in samples at the same locality but at a different season, but their sample was too small for further investigation.

From this evidence the authors concluded that a fish labeled as "ciguateric" may harbor a variety of toxins of different chemistry and pharmacology, and the variable symptoms reported probably reflect the variability in toxins. They suggested (p. 733) "As our knowledge of the differing toxins and the symptoms they produce increases, it would be well to apply more specific designations to the various illnesses."

XI. Dealing with Potentially Toxic Fish

As indicated in Section III, there is no simple test for toxicity in fish. If a fish is suspected of carrying toxin, the only way to ascertain its toxicity away from the laboratory is to feed its viscera to a cat or other animal and to watch it for symptoms for 24–48 hours. An early sign of intoxication in the cat is the loss of the ear-flick reflex when the inner ear hairs are touched.

Correspondingly, there is no way of preparing a fish carrying ciguatoxin that would render it safe for consumption. Washing does not remove the oil-soluble toxin, nor does freezing or cooking detoxify it. No studies have been made on the other toxins except that of Hashimoto *et al.* (1969b), who reported that ciguaterin is detoxified with freezing.

XII. Remedies for Intoxication

A number of remedies has been suggested for the treatment of ciguatera, including (1) native plants (Loison, 1955; also in Banner *et al.*, 1963); (2) vitamins of the B complex (Bouder and Cavallo, 1962); (3) Protopam chloride or the related PAM (2-pyridine aldoxime methochloride)* (Li, 1965b); and (4) neostigmine methylsulfate (trimethylammonium methylsulfate dimethylcarbamate) with other drugs (Banner *et al.*, 1963). In some cases the drugs appear to have contributed to recovery but laboratory studies with pure or semi-pure ciguatoxin do not confirm the efficacy of the drugs. Thus Shaw reported (in Banner *et al.*, 1963) what appeared to be a spectacular cure with the use of the anticholinesterase neostigmine in a case of barracuda poisoning, but Li (1965a, p. 1581) reported that physostigmine (a related compound) injected before the administration of a ciguatoxic extract in rats had "little protective action." Similarly Rayner *et al.* (1968) have reported that no aid was derived from PAM (similar to Protopam chloride) or from vitamin B_1 (M. D. Rayner, personal communication).

One of the best documented cases of an almost fatal case of ciguatera and its cure was that of Okihiro *et al.* (1965). They recorded a symptomatology in part parallel with that of a calcium deficiency. After an almost fatal attempt to use 2-PAM, they changed to massive doses of atropine for the muscarinic functions and magnesium sulfate, which they titrated against the muscular fasciculations. Later, calcium gluconate was given with methylphenidate for the coma. The use of magnesium and calcium ions thus preceded the recommendations of Rayner (see Section IX). Atropine, used in this and other cases (Shaw, in Banner *et al.*, 1963), also appears to be helpful but as yet is without a basic pharmacological rationale derived from a knowledge of specific actions of ciguatoxin.

Complicating any treatment is the possible presence of other toxins in the fish body which may demand drugs different from those used to treat ciguatoxin alone. Thus Bagnis (1968) reported that in 350 cases

* Li (1965b) and Okihiro *et al.* (1965) used a different chemical name for this drug than did Li (1965a, cf. p. 25).

he had treated in Tahiti the following types of symptoms as tabulated below were found to predominate.

Predominate symptoms	Number of cases
Neurologic	112
Digestive	58
Itching	29
Erythema	14
Cardiovascular	7
Neuromuscular	7
Sensory disturbances	4

From what is now known about the presence of other toxins in at least some fishes, this variety of symptoms could have been caused by a mixture of various toxins. Bagnis reported that he treated most cases symptomatically.

XIII. Public Health Significance

To one who has visited the Pacific Islands, the public health significance lies beyond the cold statistics of incidence. Statistics do, however, reflect the impact upon island cultures. Bartsch and McFarren (1962) reported that on the island of Majuro, Marshall Islands, during the year 1957, 9.3% of the island population was treated in the hospital, but that interviews of families living on various islets gave the figure of 14–15% incidence during the year. Malardé *et al.* (1967) carried out a house-to-house survey in rural Tahiti and interviewed 33,085 individuals. They reported that a total of 8.45% of those interviewed had fish poisoning during the year 1966, but that in some districts the incidence was as high as 22.84%. The highest incidence yet reported was on Hao, Tuamotus, where Bagnis (1969) found 224 of the 514 interviewed reported to have been intoxicated at least once, giving an incidence of 43%, and counting multiple attacks, there were actually 271 cases. The last six months of Bagnis' study saw a decrease in medically treated cases, either because of a decrease in toxicity in the fish or a decrease in use of local fishes.

It is rare for death to result from the intoxication (Bagnis, 1968), but the illness imposes an economic strain upon island economies, for those affected cannot be productive during their days or weeks of illness. Malardé *et al.* (1967) reported that 80% of the cases they investigated were of people of the "active age," in the 15–64 age group. In more

subsistence economies, such as in the Gilbert Islands, the father and husband is unable to gather coconuts and fish for his family; in more advanced economies, the poisoned individual is unable to work for wages. Malardé *et al.* estimated that during their study year, 6580 mandays of labor were lost. In addition to the loss of labor, the economies may be displaced by the loss of market to fishermen.

An indirect effect of the toxicity of the reef fishes is the loss to the island diet of the principal source of protein. Especially on the atolls where domestic animals cannot be raised easily as food—pigs and chickens, for example, are a "feast food"—the routine protein comes from the sea. Where all of the fish become toxic, this source is denied to the people and malnutrition results unless the individuals have sufficient cash income to buy imported tinned fish and meat. There have been no studies on the results of the dietary imbalance or economic loss.

Ciguatera has been found to interfere with the development of domestic and export fisheries in some tropical "underdeveloped" areas. A noteworthy example was the attempt of the United States and the Virgin Islands governments to increase income in the Virgin Islands by increasing the local fisheries. In 1967–1968 the local fisherman landed a catch worth $781,896, yet during the same period $1,416,726 worth of seafood was imported (Dammann, 1969). The project to improve the fisheries started in 1965; in 1970, Dammann (p. 3) concluded: "This problem [of ciguatera] was viewed as being so severe, in fact, that there was little possibility of improving the inshore fishery until the total problem could be solved." Similarly, Halstead (1970b, p. 1) reported that the FAO Caribbean Fisheries Development Project vessel landed a catch of *Caranx* in Kingston, Jamaica that "severely poisoned . . . about 60 persons at the Police Officer's Mess"; Halstead cited several other outbreaks resulting from the exploratory fishing effort. For the development of tropical reef fisheries there is the vital need of either an infallible predictability as to which fish will be toxic (as individuals, as species, or from certain areas), or a simple and reliable method of testing individual fish for toxicity.

XIV. Conclusions

The disease ciguatera results from the ingestion of fishes associated with coral reefs and is widespread in the tropical Pacific and Caribbean. Although research on the principal causative toxin, ciguatoxin, has been quite productive in spite of the complexity of the problem, all areas of research must advance before any definitive answers are achieved. Bio-

logical research has shown that the toxin originates environmentally on the coral reef and is transmitted through the food web, but both the elaborator of the toxin and the ecological factors causing the increase of the toxin in the environment are unknown. Ciguatoxin has been isolated, but is not structurally identified; other toxins in fishes labeled as "ciguateric" lack more than initial exploration of their chemical characteristics. One of the pharmacological activities of ciguatoxin, that of disruption of the ionic balance of excitable cell membranes, has been established but it is not known whether this is the sole action of ciguatoxin; the actions of other toxins have not been investigated. No treatment other than symptomatic for the disease has been developed that considers the action of other possible toxins in the fishes.

The disease is of considerable importance to the public health and to the present economy of oceanic islands. The development of new fisheries in much of the tropics, as is now being tried, demands extensive research on all aspects of the problem. This research will require laboratory work to confirm field observations, and will also require consideration of all other toxins that may be found in "ciguateric fishes."

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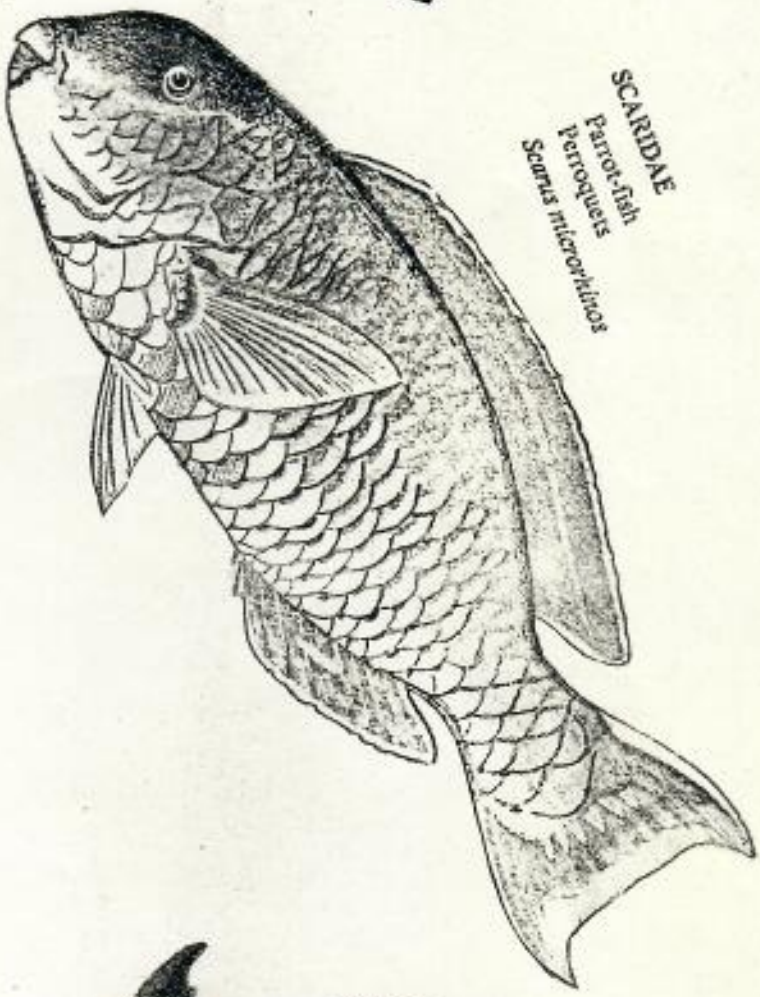
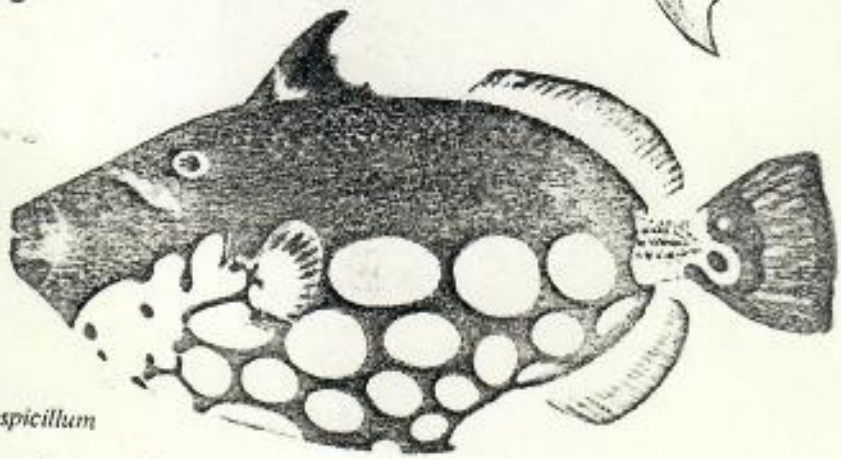


Recent advances in knowledge of ciguatera fish poisoning

By TIM KUBERSKI, SPC Epidemiologist



Ichthyosarcotoxism is the frighteningly long scientific term for fish poisoning. Literally, it means "poisoning by the flesh of fishes" and is a general term applied to the various different types of poisoning caused by the ingestion of toxic fish. In the South Pacific, where fishing and fish consumption have important nutritional and economic connotations fish poisoning has become a serious problem.

The important implications of fish poisoning to the health and economy of people in the South Pacific was recognized over ten years ago by the South Pacific Commission. In 1968 the Commission organized the First International Seminar on Ichthyosarcotoxism which took place in Tahiti. Since then, it has continued to be vitally concerned with investigations into the various causes of fish poisoning and has published a handbook on this topic.

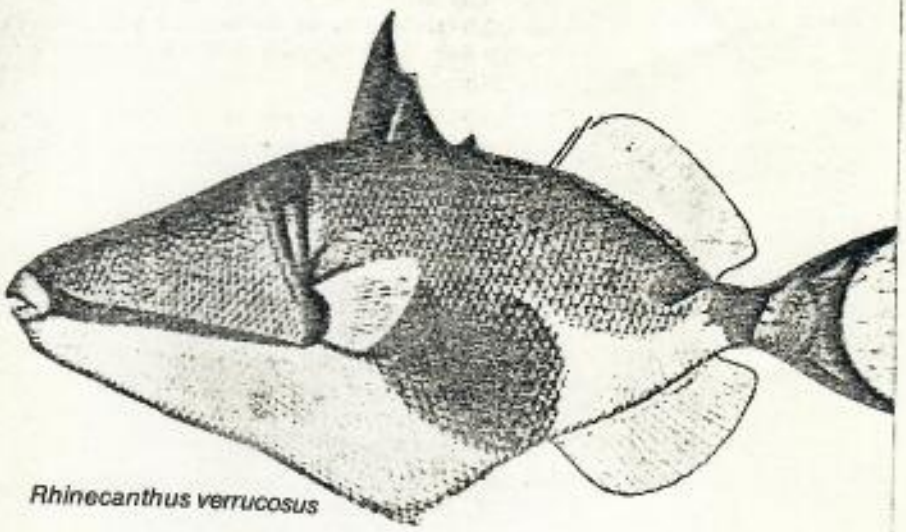
Results from the ongoing research and surveys have been slow in developing, but within the past few years great strides have been made in the knowledge of fish poisoning, particularly a type of poisoning referred to as *ciguatera*. The term ciguatera was coined many years ago by a Cuban fish expert after he recognized the similarity of symptoms between this form of fish poisoning and poisoning due to eating shellfish known in the Spanish Antilles as *cigua* (*Turbo* sp.).

The symptoms observed in a person who has ciguatera fish poisoning can be diverse, with intestinal complaints such as nausea, vomiting and diarrhoea being very common. The most distressing symptoms which occur, however, are the peculiar skin sensations such as itching, pain or numbness which usually involve the arms and legs or occur around the mouth. Weakness and lack of co-ordination can also occur. A few patients complain that touching cold objects results in a sensation of heat and that hot objects feel cold.

These symptoms usually last for only a few days, but they can linger on for several weeks, depending on the sensitivity of the particular individual and the amount of toxin ingested. Some persons appear to be more sensitive to the toxin than others and therefore tend to have a more severe reaction. The symptoms described above are not unique to fish poisoning and similar findings can be observed in other types of intoxications; however their appearance within less than 24 hours of eating types of fish should suggest very highly the possibility of ciguatera fish poisoning. Treatment with anti histamines, which are drugs frequently used for allergic conditions, appears to relieve some of the symptoms.

What causes ciguatera fish poisoning? Scientific evidence gathered in the past few years suggests that a microscopic organism known as a dinoflagellate (*Diplopsalis* sp.) may be the primary source of the ciguatera toxin. This micro-organism, which lives around coral and is frequently found associated with bottom fixed algae, is incorporated in the food of many of the relatively small and intermediate sized fish which feed on the ocean bottom and off coral reefs. Familiar examples of these fish which man consumes would be the parrot fish and the surgeon fish.

The dinoflagellate toxin, known as ciguatoxin, becomes incorporated into the flesh of these fish, making them dangerous for human consumption. However, small fish generally contain comparatively small amounts of toxin and as a general rule poisoning is less likely to occur with small fish than with certain types of larger, more predacious fish. The larger fish, such as groupers, snappers, jacks, barracudas and emperors (also the moray eel), all prey on the small bottom-feeding fish and thus indirectly become contaminated with ciguatoxin.



Rhinecanthus verrucosus

The large fish are amore serious threat since they have been found to contain larger amounts of the toxin, apparently because they tend to concentrate the ciguatoxin acquired from numerous smaller fish. Therefore, a toxin produced by a micro-organism gradually moves along the ocean food chain to reach eventually a concentration in certain fish which makes them toxic to man. The ciguatoxin can be found in both the flesh and entrails of fresh fish and cannot be removed by salting, washing or cooking the fish. It must be emphasised that not every fish of a certain species will be toxic; toxicity is dependent on the distribution in nature of relatively large quantities of the toxin-producing dinoflagellate, as well as the presence of the susceptible fish which feed indirectly on them.

It is difficult to assess accurately how great a problem fish poisoning is in the South Pacific, because many cases are never reported, but several thousand cases of fish poisoning in Pacific Islanders do get reported to the South Pacific Commission every year and the indications are that it is becoming even more commonplace. An explanation for this observation may relate to changes in the environment which are conductive to the growth of the micro-organisms which produce the ciguatoxin. These organisms appear

to increase rapidly in areas of the ocean that experience sudden and drastic natural or man-made changes.

Such things as an unusually heavy rainfall; building of a new channel through a reef or a wharf; wrecks; or dredging in areas where this form of micro-organism exists only in small quantities, frequently will be followed by a great upsurge in their numbers. This eventually results in an increase in the number of toxic fish. It is not impossible that the appearance of toxic fish and recognized human fish poisoning may not be evident for months to years after the increase in the toxin-producing dinoflagellate has occurred. However, further studies of this aspect of fish poisoning are needed before a definite conclusion can be reached. The complicated manner in which the different varieties of fish become toxic has made recognition of the association between human fish poisoning and this small organism very difficult.

Much research has yet to be done before ciguatera fish poisoning as it occurs in the Pacific can be completely understood. The current major efforts of scientists are to determine how to detect toxic fish and how best to deal with patients who become ill with ciguatera intoxication. In many areas of the Pacific, certain fish are not eaten or are completely banned from sale because of their potential toxicity to man. If a simple method could be devised to determine which members of a certain type of fish were toxic, a number of fish identified as safe from ciguatera fish poisoning could then be marketed.

How to deal with individuals who have been poisoned is the other priority research problem of scientists working on ciguatera. This form of fish poisoning is usually not a life-threatening disease; however, a peculiar thing happens to persons repeatedly exposed to the ciguatoxin. They become hypersensitive to fish in general and are unable to tolerate eating any fish, whether toxic or not. Simply eating fish which are apparently toxin-free causes a relapse in their symptoms.

This results in the individuals having to restrict severely the amount of fish in their diet. This has important implications, because some South Pacific Island populations rely heavily on the ocean for food and in some instances very few alternatives are available. Scientists are currently puzzled by this phenomenon, but its importance to human health has made it an area of intensive investigation.

Although there have been major breakthroughs in studies of ciguatera fish poisoning, a number of things need to be done before adequate control and preventive measures can be implemented. A better understanding of the environmental circumstances resulting in the increase of the toxin-producing micro-organism has to be a long-term goal of investigations into ciguatera poisoning. Through a better understanding of the ecological requirements of the toxin-producing dinoflagellate, it might be possible to predict when an increase in these micro-organisms will occur, outline toxic areas and possibly even control outbreaks. But developing a safe method to prevent fish from becoming toxic without upsetting the ecology will be a most difficult task.

Current efforts are now concentrating on effective methods of recognition and control before the problem becomes much larger or insurmountable. This research takes on some element of urgency as the world demand for fish increases and new fishing grounds are continually being sought.

Readers are encouraged to inform the Commission, or their local health authorities, about cases of suspected fish poisoning which undoubtedly occur more frequently than we know about. □

SPC EPIDEMIOLOGIST

The South Pacific Commission has appointed to the post of Epidemiologist Dr Timothy Kuberski, formerly on the staff of the Pacific Research Section, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Honolulu, Hawaii.

Dr Kuberski has been certified by the American Board of Internal Medicine as a specialist in the field of infectious diseases and has undertaken studies involving research and epidemiology of infectious diseases found in the South Pacific. He has held the post of Assistant Clinical Professor of Tropical Medicine and Medical Microbiology at the University of Hawaii. He is particularly interested in dengue, eosinophilic meningitis and venereally transmitted disease, and has extensive laboratory and field experience in work on dengue fever. Dr Kuberski's surveillance and control of dengue fever and other infectious diseases; the organisation of a programme for prevention and detection of diabetes; the formulation of a reporting system for cancer; and responsibility for the South Pacific Epidemiological Health Information Service. He took up his appointment with SPC at the beginning of March.

DR T. KUBERSKI

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Fish poisoning is occurring more often in Hawaii in recent years, and may present you with a puzzling clinical picture to untangle.

Fish Poisoning in Hawaii

PHILIP HELFRICH, Ph.D.,* *Honolulu*

• *Fish poisoning has affected more than 433 persons in over 54 recorded outbreaks in Hawaii since 1900. Of the four categories of poisoning reported in Hawaii (ciguatera, hallucinatory mullet poisoning, tetraodon or puffer fish poisoning, and scombroid or histamine poisoning), only poisoning by the puffer fish has caused deaths—seven of them. Gymnothorax (moray), elasmobranch (shark) and clupeid (herring) poisoning have not been recorded in Hawaii. Ciguatera, caused by a neurotoxic substance, is the most serious hazard of all: it is a recent affliction here, difficult to predict or control, and produced by many species of fish that are highly esteemed as food; and the toxin seems to have a cumulative effect.*

OUTBREAKS of fish poisoning, or ichthyotoxism, caused by the ingestion of the flesh or viscera of fish containing toxins of nonbacterial origin, have been relatively uncommon in Hawaii, and most local physicians have seldom been confronted with such cases. A study of this problem of fish poisoning, including a search of literature and records as well as numerous personal interviews, has revealed that at least 54 outbreaks involving more than 433 persons and resulting in seven deaths have occurred in Hawaii since 1900.

*Assistant Marine Biologist, Hawaii Marine Laboratory, University of Hawaii.

Most of these outbreaks were apparently caused by toxins endogenous to the fish or acquired from sources other than the micro-organisms normally encountered on "spoiled" fish. Because outbreaks have been infrequent and usually involve only a small number of persons, physicians encountering cases for the first time have been perplexed by some aspects of the unfamiliar syndrome, particularly by manifestations of neurological involvement that are frequently observed in certain types of nonbacterial fish poisoning.

RESEARCH FOCUSED

A recent spread of some types of fish poisoning in the central Pacific, as well as increased demands on the resources of the sea by Pacific peoples, has been instrumental in focusing the attention of researchers on this problem. This increased interest in fish poisoning in the Pacific has resulted in the initiation of a number of related research projects on various aspects of the problem at the Hawaii Marine Laboratory of the University of Hawaii, supported by national and local agencies. The Japanese have been actively engaged in research on marine fish toxins for a number of years, and recently a few workers in North America have been studying related problems.

It is the purpose of this discussion to review the presently defined categories of fish poisoning, their symptomatology, treatment, and other information

that would be of value to physicians and public health officials in recognizing and handling outbreaks. Results of past and current research will be discussed, as well as the significance of the spread of fish poisoning in the central Pacific, with emphasis on outbreaks in Hawaii. It is hoped that this discussion will be of sufficient interest to stimulate an exchange of information with those who have had first-hand experience with cases of fish poisoning, in order to expand our present knowledge of toxic fishes and to aid in our research upon them.

Fishes that are poisonous to eat are widely distributed throughout the warm seas of the world, being particularly prevalent around certain islands in the Pacific and Caribbean.[12, 15, 19, 39, 45, 47] Outbreaks of fish poisoning have occurred in almost all of the major oceanic island groups in the Pacific. In some islands, such as New Caledonia and the New Hebrides, fish poisoning is known to have been a problem for hundreds of years, while in other areas such as the Line and Hawaiian Islands it appears to be largely a problem of recent origin (Fig. 1). Inquiries made of medical officials throughout the tropical Pacific during the past four years revealed that fish poisoning of one variety or another is ubiquitous on islands within 30 degrees north and south of the equator.

EARLY REPORTS

The first report of illness from the ingestion of toxic fish in the Pacific is believed to be that of the Spanish navigator, Fernandez de Quiros, in 1606, when he and his crew were poisoned in the New Hebrides.[42] Kaempfer[32] reports deaths among the Japanese from eating improperly cleaned puffer fish as early as 1690, and his awareness of the highly toxic qualities of this fish is evident from his statement that it, ". . . if eat [*sic*] whole, is said unavoidably to occasion death. . . ."

The famed Pacific explorer, Captain James Cook, and members of his crew were poisoned twice in the New Hebrides and New Caledonia in 1774.[14] The journals of other explorers, missionaries, naturalists, and others, contain numerous accounts of episodes of fish poisoning on various Pacific islands, attesting to a widespread existence of this condition prior to the present century.

The occupation of many Pacific islands by military forces during World War II served to emphasize the gravity of the poison fish problem, for, lacking the native's knowledge of potentially toxic species, these sojourners to the islands were often seriously afflicted.[21]

CLASSIFICATION

Fish poisoning has been classified into a number of categories, primarily upon the syndrome manifested in cases of human intoxication and the taxonomic classification of the fish implicated. Only one type of fish poisoning (by the tetraodon or puffer fish) has been extensively investigated, and further research is required to determine whether all of the categories of fish poisoning discussed below validly reflect the action of a distinct toxin harbored by a defined taxonomic group of fishes.

Based on extensive published literature as well as numerous unpublished reports, the following categories of fish poisoning are presented with a condensation of the available pertinent information on them. This discussion will not include fishes with venomous spines or those with a reputation for aggressive attacks on humans.

CIGUATERA POISONING

Ciguatera is a term used to describe a disease characterized by neurological and gastrointestinal symptoms resulting from the ingestion of any of a number of tropical marine reef fishes, notably (but not exclusively) snappers, groupers, ulua, barracuda, and surgeonfish.

The term ciguatera, of Spanish origin, was first used in the Caribbean area to designate intoxication caused by the ingestion of the poisonous marine snail, *Turbo pica,* which the early Spanish settlers called "cigua."[5] Unfortunately, the term gradually came into common usage and is now widely accepted as descriptive of a particular type of poisoning, due to the ingestion of certain fishes encountered around islands both in the Caribbean and the Pacific, which produces the characteristic symptoms described below.

Ciguatera is a disease that is not well understood, and a broad program of research has been undertaken at the Hawaii Marine Laboratory on the chemical isolation, identification, pharmacology, and biological origin of the toxin or toxins involved, as well as an epidemiological study of this and other types of fish poisoning in the Pacific.

It has been suggested that fish causing ciguatera become toxic through factors existing in their environment. Numerous possible sources of the toxin in the environment have been proposed, including dumped war material,[48] poisonous plankton,[30] and "flowering" coral.[42] The most plausible theory in the light of existing evidence is that the toxin originates in a benthic organism (possibly an alga) and is transmitted to other species of fish through the food chain in the process of normal feeding.[47] It has also been suggested that nuclear testing programs in the Pacific may have been instrumental in causing an increase in outbreaks of ciguatera in the Marshall Islands. The results of a recent study show that no relationship exists between radioactivity and toxicity in snappers caught near the atomic test sites in the Marshall and Line Is-

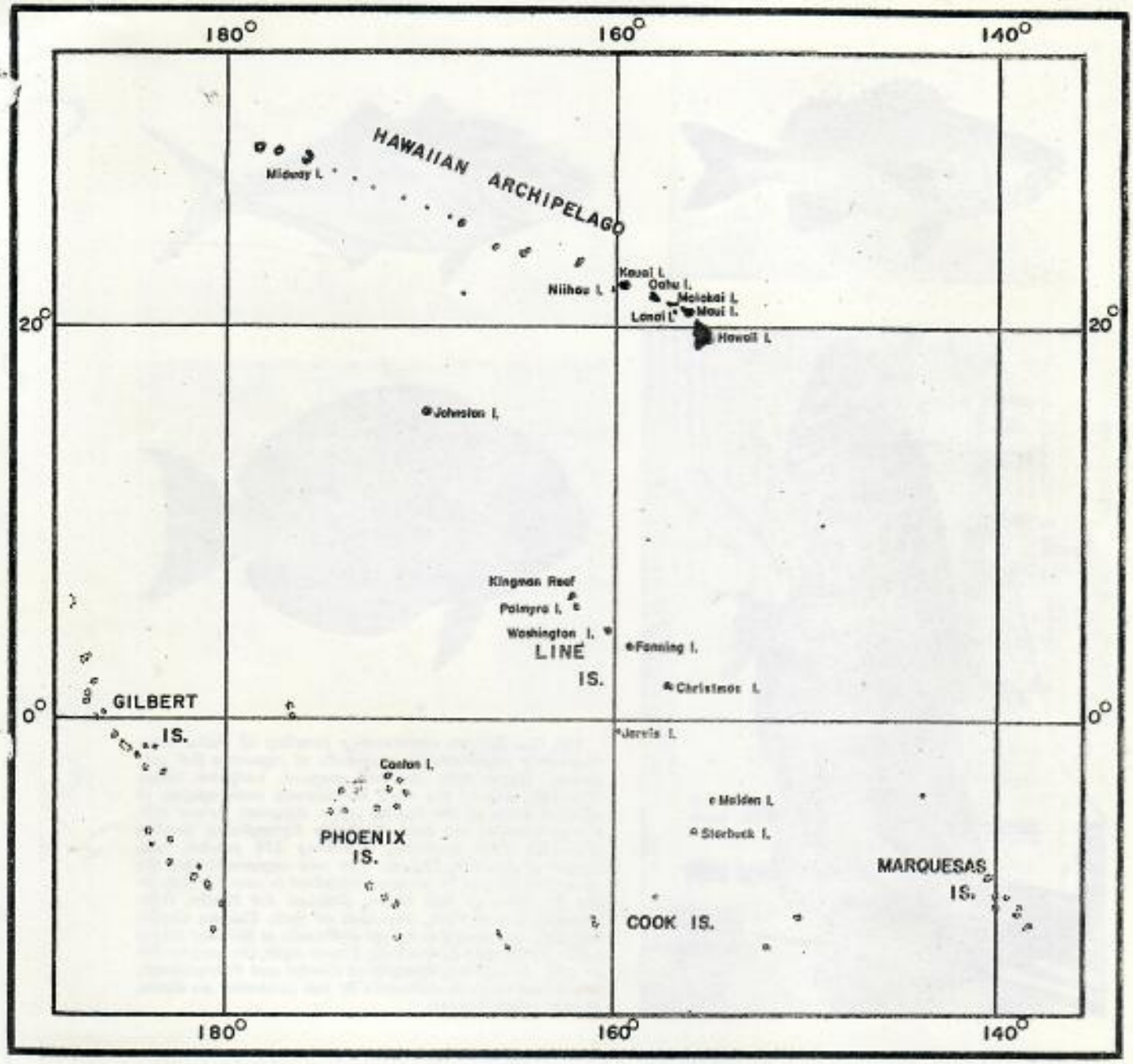
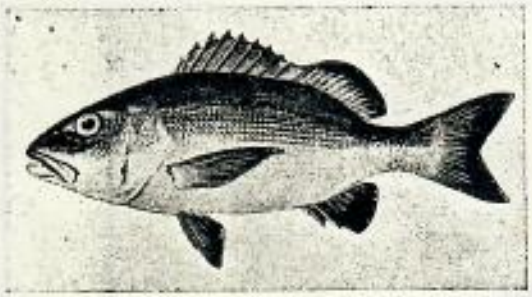


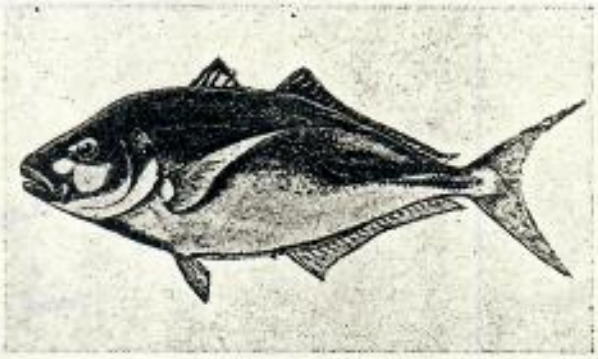
FIG. 1.—*Map of the central Pacific Ocean showing the Hawaiian Archipelago, Line Islands, and other areas pertinent to the distribution of poisonous fishes discussed in the text.*

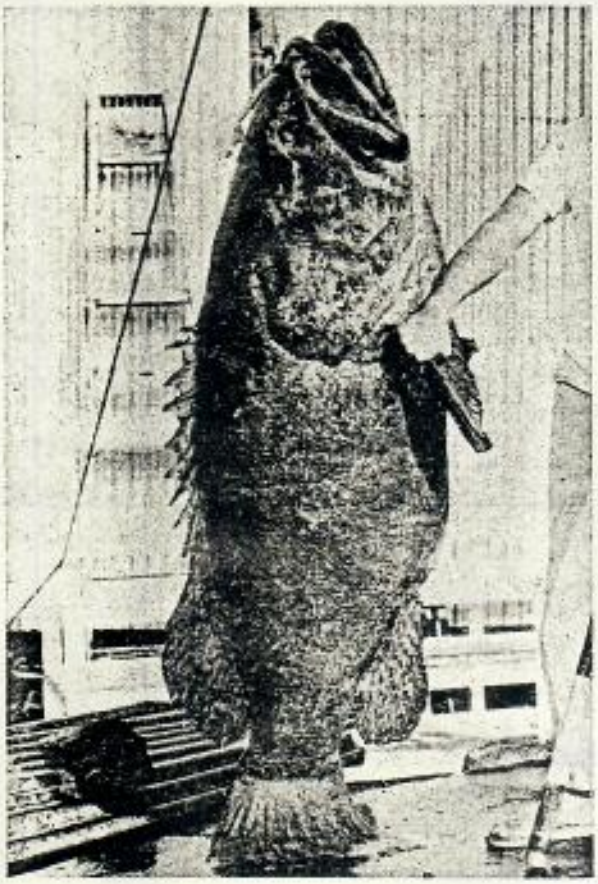
lands, which is to be expected in the light of the past history of the poisonous-fish problem in the Pacific.[24]

Reports indicate that outbreaks of ciguatera have occurred on oceanic islands of the western, southwestern, and southern parts of the Pacific at least as long as records exist from these areas. Islands of the central Pacific, including the Line Islands, Johnston Island, and the Hawaiian archipelago have apparently been free of ciguatera until recently.[22] A spread of ciguatera to this area in the past 20 years is a source of concern to public health officials as well as the inhabitants, and its significance is discussed further below.

Species most frequently implicated in outbreaks of ciguatera include certain snappers (Lutjanidae), groupers or sea bass (Serranidae), ulua, jacks or pompano (Carangidae), barracuda (Sphyraenidae), and the surgeonfishes or tangs (Acanthuridae). Other species less frequently consumed as food, including the parrot fishes (Scaridae), wrasses (Labridae), squirrelfishes (Holocentridae) and







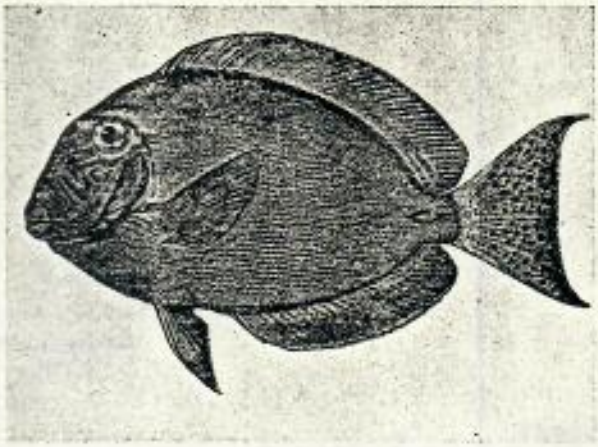


FIG. 2.—*Species representing families of fishes most frequently implicated in outbreaks of ciguatera fish poisoning. Upper left, the red snapper,* Lutjanus bohar *(Forskål), one of the most consistently toxic species in affected areas of the Pacific (after Hiyama). Lower left, the grouper or sea bass, probably* Epinephelus tauvina *(Forskål). This specimen, weighing 238 pounds, was caught at Kapoho, Hawaii, and was responsible for the poisoning of over 30 persons described in case 52, Appendix B (photo by Roy Ogata, Kilauea Art Studio, Hilo, Hawaii). Upper right, the ulua or jack,* Caranx cheilio *(Snyder), implicated in recent outbreaks at Midway Island (after Jordan and Evermann). Lower right, the surgeonfish or tang,* Acanthurus dussumieri *Cuvier and Valenciennes, implicated in three outbreaks of fish poisoning on Oahu, Hawaii (after Aoyagi).*

others may also carry ciguatera toxin.[12, 18, 19, 47] Some of the species implicated in outbreaks of ciguatera in Hawaii are illustrated in Fig. 2.

The clinical picture in ciguatera is often bizarre, and not all patients display the same symptoms, even, in some cases, when all of the individuals have eaten portions of the same toxic fish. Ciguatera can usually be recognized on the basis of a few initial characteristic symptoms that may appear from a few minutes to ten hours or more following the ingestion of a toxic fish. The first symptoms are usually experienced within about three hours after ingestion, and consist of nausea and vomiting, followed by tingling and numbness about the lips, tongue, and throat. These may be followed by any of a multiplicity of other symptoms, including abdominal pain and cramps, diarrhea, arthralgia, muscular weakness, incoordination, numbness and tingling of the extremities, malaise, chills, low-grade fever, and prostration. Hypotension, profuse sweating, dyspnea, restlessness, insomnia, headache, intermittent dizziness, dilatation of the pupils, ptosis, divergent strabismus with diplopia, reduced vision, dryness of the mouth, a metallic taste, and myalgia (particularly severe in the back and thighs) also occur. Hyperesthesia, urinary retention, and diminished-to-absent knee and ankle reflexes have been reported. Patients often experience dysesthesia that consists of a confusion of temperature sensation; when touching a cold object, the patient reports that it gives the sensation of burning, tingling, or "dry

ice," and hot objects feel cold. When tap water is swallowed, it often gives the sensation of being carbonated. In severe cases of ciguatera, shock, convulsions, muscular paralysis, and death may occur.

It appears as though the body does not rapidly neutralize or eliminate ciguatera toxin, for the recovery period is characteristically quite prolonged. In moderate cases of poisoning, most of the symptoms subside in 24 hours, with the exception of the muscular weakness, tingling, and numbness, which may last from four to seven days. During the recovery phase, some patients report vague neuralgic pain about the teeth, and intense itching,* especially on the palms and soles. In severe cases, symptoms may persist for several weeks or months, and complete recovery from sensory disturbances, weakness, and weight loss may require much longer.[5, 8, 9, 10, 12, 18, 21, 39, 43, 47]

An attack of ciguatera does not impart immunity; on the contrary, patients who have been poisoned previously report a mild recurrence of symptoms after eating a potentially toxic fish, while others who have never been poisoned experience no symptoms when eating the same fish.[47]

Usually treatment recommended for ciguatera is symptomatic, consisting initially of emptying the digestive tract by means of gastric lavage, emetics, and saline purges. A variety of other treatments have been suggested and tried with varying palliative effect, but none have been consistently successful; these include injections of calcium gluconate, injections of vitamin B complex, infusions of glucose in normal saline, phenobarbital, belladonna, paregoric, morphine, codeine, and aspirin.[3, 9, 11, 12, 18] In one outbreak of ciguatera involving five persons, procaine hydrochloride infusions were inadvertently substituted for the calcium gluconate recommended and the patients displayed a regression of symptoms and general improvement. Subsequent administration of calcium gluconate in place of the procaine hydochloride resulted in a recurrence of symptoms, followed by complete recovery of the patients within a week.[17]

A recent outbreak of ciguatera on Guam was caused by the ingestion of a portion of a 53-pound barracuda. One of the victims, a 19-year-old girl, experienced severe depression and paralysis, and treatment with hydrocortisone, Tensilon, and neostigmine seemed to have a beneficial effect.[8] Investigations in Japan, Ohio, at the Hawaii Marine Laboratory, and elsewhere indicate that the toxins found in barracuda, snappers, and ulua are similar and perhaps identical; all produced characteristic ciguatera symptoms in man and laboratory animals. The toxin from the red snapper (*Lutjanus bohar*) from the Line Islands has been the object of intensive chemical and pharmacologic studies in laboratories in Hawaii, Ohio, and California.[6, 8, 27, 28, 41] In the Hawaiian investigation it was found that the crude toxin from this fish is thermostable, initially soluble in 95 and 100 per cent ethanol, acetone, chloroform, or diethyl ether, and insoluble or only slightly soluble in water, butanol, benzene, or petroleum ether. It has been rendered in a very nearly pure state by initial extraction and washing with 95 per cent ethanol, petroleum ether, and diethyl ether, and by repeated chromatographic separations with solvents of varying polarity.[6]

No simple rapid test for identifying ciguatera toxin exists. Preliminary experiments with *Lutjanus bohar* indicate that the liver contains the highest concentration of toxin, followed by the viscera (less liver and gonads), the testes, ovaries, and muscle, in order of decreasing toxicity. Therefore, as an expedient, the liver or other portions of the viscera may be fed to susceptible animals such as cats, dogs, or mongooses at doses of 10 per cent of the weight of the test animal. If the flesh is sufficiently toxic to cause illness in man, the ingestion of such a sample will cause muscular weakness beginning with flexion of the wrists, ataxia, hypersalivation, prostration, and probably death in a test animal (mongoose or cat) within 24 hours. A roughly quantitative bioassay has been developed which entails the intraperitoneal injection of an alcohol-ether extract of the suspected sample into laboratory mice.[6, 7, 8, 41] A more refined bioassay utilizing the blocking action of the semipurified toxin at the synapse in nerve-muscle preparations is presently being perfected. Hessel and his coworkers have developed a test which records the loss of action potential in a frog sciatic nerve preparation which is bathed in a fine emulsion of the toxin.[27, 28]

TETRAODON POISONING

Fish causing tetraodon or puffer fish poisoning are primarily from the family Tetraodontidae and are known in Hawaii by a variety of names including puffers, blowfish, balloon fish, 'o'opu-hue, makimaki, keke, or *fugu*. Puffer fish have a peculiar appearance; they are without pelvic fins, are generally feeble swimmers, and have a habit of inflating themselves when disturbed to assume a spheroid form almost twice their original size.

Many species of puffer fish occur in the tropical Indo-Pacific area. Many species have been reported to contain a very virulent toxin, and reports of human intoxications from their ingestion are numerous and widespread geographically.[12, 15, 18, 19, 55] Five species of Tetraodontidae

* The French in New Caledonia commonly refer to ciguatera as "*la grante*" (= the itch).

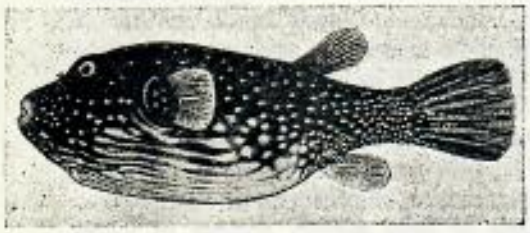


FIG. 3.—*The common puffer fish or fugu,* Arothron hispidus *(Linnaeus) implicated in four outbreaks of tetraodon poisoning in Hawaii which resulted in seven fatalities (after Hiyama).*

occur in Hawaii;[16] *Arothron hispidus* (Fig. 3) is the most common local species, and, according to available reports, the only species implicated in cases of fish poisoning in Hawaii.

The toxin in puffer fish is endogenous and appears to be concentrated in certain organs and tissues; the ovaries are said to always be toxic, while the liver, bile, skin, and flesh may occasionally be toxic. Special care in the cleaning and preparation of puffer fish can render them innocuous; normally the viscera are carefully removed under running water, and the flesh may be soaked in repeated changes of fresh water to leach out the water-soluble toxin. Ironically, the puffer fish is considered a delicacy by many Oriental people. Despite regulations in Japan requiring that persons who cook puffer fish or *fugu* commercially be specially trained and licensed, puffer fish poisoning is the greatest single cause of fatal food intoxication in Japan.

In Hawaii, seven deaths from the ingestion of puffer fish have occurred in the past 60 years, despite a general recognition by Hawaiians and Orientals alike of the potentially toxic qualities of this fish.[1, 18, 36] In Honolulu, where no special regulations control its preparation, puffer fish or *fugu* usually commands a premium price when it is available at the few restaurants and tea houses that feature this delicacy.

In addition to toxic Tetraodontidae, there are a number of other closely related families of the order Tetraodontiformes (Plectognathi) that have been reported toxic when eaten. However, in the present state of our knowledge it is not possible to say whether or not these toxins are identical or similar to that found in the puffer fish. These other closely related families include the filefishes or 'o'ilis (Monocanthidae), the boxfishes, trunkfishes, or cowfishes (Ostraciidae), the triggerfishes or humuhumus (Balistidae), the sharp-backed puffers (Canthigasteridae), and the spiny puffers (Diodontidae).

Only one case of poisoning attributed to plectognath fishes other than puffer fish is recorded from Hawaii. In 1956 an outbreak was reported from the Island of Hawaii, caused by the ingestion of the black triggerfish or humuhumu-'ele'ele, *Melichthys buniva* (see case 32, Appendix B). The nature and distribution of toxins in plectognath fishes in Hawaii are presently being studied at the Hawaii Marine Laboratory.

Puffer fish (Tetraodontidae) may be highly toxic, resulting in the onset of symptoms within ten minutes after ingestion. However, in some cases recorded, more than three hours passed before the onset of symptoms. Initial symptoms include tingling of the lips, tongue, and fingertips, followed by progression of the numbness which may involve the entire body, and extreme weakness, associated with nausea, vomiting, headache, profuse sweating, subnormal temperature, hypersalivation, dysphagia, dysphonia, dyspnea, and a constricting sensation in the chest. In severe, acute cases, the patient rapidly develops a weak, rapid pulse, hypotension, aphonia, marked dyspnea, cyanosis, and an ascending paralysis with death resulting from respiratory failure.[1, 12, 18, 36] The mortality rate of persons ingesting toxic puffers has been estimated to be greater than 60 per cent.[19] Deaths usually occur within 24 hours after ingestion; if the patient survives this time the prognosis is good.

The toxin from puffer fishes, called "tetrodotoxin" has been studied extensively and isolated by the Japanese, but the molecular structure has not been determined.[51, 52, 53, 54] A number of investigators have studied the pharmacology of tetrodotoxin, which has a curare-like action on the neuromuscular junction, depresses conductivity in peripheral nerves, and depresses the medullary center.[12, 43]

A specific antidote has not been developed for tetraodon poisoning and the treatment recommended is symptomatic. Japanese physicians, who have encountered numerous cases of this disease, recommend the following: Coramine (niketha-mide, Ciba), Metrazol (pentamethylentetrazol, Knoll) or other suitable respiratory stimulants, together with artificial respiration, in event of respiratory failure; administration of Vasopressin (Parke Davis) and rapid digitalization to correct hypotension and circulatory collapse; and physostigmin in an attempt to reverse the neuromuscular block.[12, 44]

Puffer fish or *fugu* is usually prepared as a soup consisting of pieces of the flesh and liver in a thin broth. It is generally reported by those who have consumed this dish in Hawaii that it produces more profuse perspiration than might be expected from the ingestion of an equal quantity of another hot soup. Many others have reported a feeling of "warmth and well-being" and a few have claimed it causes muscular weakness and localized numbness (around the mouth), and that it is a powerful aphrodisiac.

Some of these effects may be due to the inges-

tion of small doses of the toxin, while others may be psychosomatic. The author, from personal experience, can attest only to the profuse perspiration. No research on tetraodon poisoning in Hawaii has been reported since the preliminary work of Larsen,[36] and at least a comparison should be made of the structure and mode of action of the toxin of the common local puffer, *Arothron hispidus,* with those studied extensively by the Japanese.

The lack of reported fatalities from tetraodon intoxication in recent years is probably due to a more widespread understanding of the potential dangers adjunct to the ingestion of improperly prepared puffer fish. It is improbable that local specimens have become less toxic in recent years, as checks during 1961 on the ovaries of mature female specimens of *Arothron hispidus* from Kaneohe Bay, Oahu, produced rapid fatalities when fed to mongooses in the laboratory.

No simple, rapid test has been reported to assess the toxicity of puffer fish; oral feeding to carnivorous mammals such as cats, dogs, and mongooses will result in a marked response in a few hours if the sample fed is highly toxic. A bioassay standardized by the Japanese requires extraction and partial purification of the water-soluble toxin, and injection of the extract intraperitoneally into laboratory mice.[23]

HALLUCINATORY MULLET POISONING

Hallucinatory mullet poisoning is a seasonal condition occurring only during the months of June, July, and August in restricted areas on the islands of Kauai and Molokai. Old residents of the affected areas report that the condition has existed for as long as they can remember, and most of them have been stricken with this poisoning at least once. The areas affected are on the northeast coast of Kauai, principally in the vicinity of Anini, but extending from Pilaa to Haena, and in a region around Pilaau on Molokai.[1, 2, 26]

The species implicated include the mullets, *Mugil cephalus* Linnaeus ('ama'ama) and *Neomyxus chaptalii* (Eyodoux and Souleyet) (uouoa); the surmullet or goatfish, *Upeneus arge* (Jordan and Evermann) (weke pueo, weke pahula, nightmare weke, or crazy surmullet), and *Mulloidichthys samoensis* (Gunther) (weke, weke'a'a); occasionally the rudderfish, *Kyphosus cinerascens* Forskal (nenue, nenue parii, manaloa), and the surgeonfish, *Acanthurus sandvicensis* (Streets) (manini or convict tang). The first and third are pictured in Fig. 4.

The toxin appears to affect the central nervous system when ingested by humans, and produces symptoms of dizziness, loss of equilibrium, ataxia, hallucinations, and mental depression if the onset

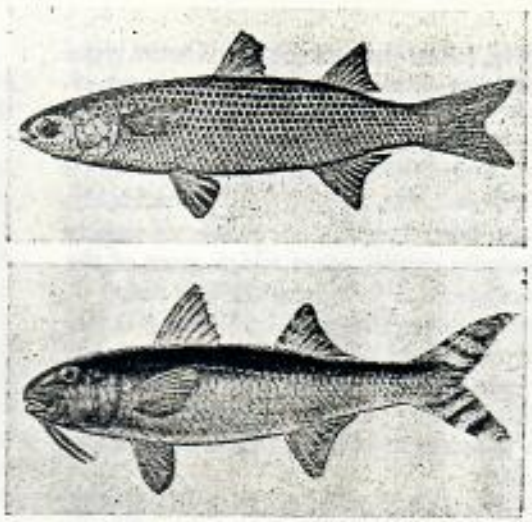


FIG. 4.—*Two species most commonly implicated in outbreaks of hallucinatory mullet poisoning in Hawaii; Upper, the common mullet,* Mugil cephalus *Linnaeus, and, lower, the goatfish or surmullet,* Upeneus arge *(Jordan and Evermann), which is also referred to locally as the "nightmare weke" (after Jordan and Evermann).*

of symptoms occurs when the patient is awake. If the onset of symptoms occurs while the patient is sleeping, he experiences terrifying nightmares. Other symptoms which may occur are malaise, itching or burning of the throat immediately after ingestion, muscular weakness, and partial paralysis. Gastrointestinal upset, nausea, or diarrhea are rarely reported in these cases. The onset of symptoms is from ten minutes to two hours after ingestion.

The evidence available indicates that this toxin is not a result of bacterial action, as persons have been afflicted after eating freshly-caught fish. Similar symptoms have resulted from fish that were boiled, fried, steamed, and eaten raw, and from the ingestion of various portions of the fish. Although the head alone is frequently implicated as containing the greatest concentration of the toxin, cases of poisoning were discovered in which the victim had eaten only the eviscerated body of the fish.[1, 2, 26]

Preliminary investigations of this hallucinatory mullet poisoning have stimulated interest among pharmacologists, who plan to further investigate the hallucinogenic properties of the toxin involved.

SCOMBROID POISONING

This report has been restricted largely to acquired or endogenous toxins in live fish, believed to be the result of other than bacterial action upon the fish after capture. A departure from this categorical restriction will be made to discuss scombroid poisoning, the result of the rapid action of a strain of bacterium, producing a toxin often unaccompanied by the usual signs of putrefaction,

and, therefore, sometimes mistaken for other types of fish poisoning. Scombroid poisoning is an allergic type of intoxication, resulting from ingestion of improperly stored tuna, mackerel-like fishes, swordfishes, and others, including those known locally as ono, aku, ahi, ahi-pahala, a'u, and mahimahi. These fish comprise a group of highly esteemed pelagic species upon which most of the Hawaiian commercial fishing industry is based.

The toxin causing scombroid poisoning is not produced by the usual enterotoxic pathogens, but by certain strains of the bacterium, *Proteus morganii*, which, under proper conditions, act on histidine, a naturally-occurring substance in scombroid fish flesh.

Free histidine is known to occur in the flesh of many fishes, and it is particularly abundant in dark-fleshed fish, sometimes reaching concentrations of more than 2,000 mg/100 g. The dark flesh of such pelagic species as the frigate mackerel, *Auxis thazard* (Lacépède); the Japanese mackerel, *Scomber japonicus* Houttouyn; the mahimahi or dolphin, *Coryphaena hippurus* Linnaeus, and the striped marlin, *Maxaira audax* Philippi, contain concentrations of histidine up to two orders of magnitude greater than that found in white meat fish.[29, 49] *P. morganii*, normally found on the surface of freshly-caught fish under certain conditions, causes a decarboxylation of free histidine to produce histamine and possibly a synergistic agent known as "saurine."[33, 34]

Proteus morganii is a motile, gram-negative rod, 0.5 x 1.0-1.4 microns. Optimum temperature for the production of histamine by this organism is 20-25°C. In this temperature range, the decarboxylation of histidine to form histamine may be very rapid, and considerable histamine may be formed before ammonia or other putrefactive by-products can be detected. The critical concentration for human poisoning by histamine in fish flesh seems to be about 1 mg/g.[35]

The histamine and saurine produced in improperly refrigerated fish cause a severe allergic type of reaction upon ingestion. The presence of these toxic substances, detectable by a sharp or peppery taste, may be masked by sauces or seasoning. Symptoms develop within a few minutes to three hours after ingestion and are often sudden in onset. They include erythema of the face and upper body; severe occipital headache; giant urticaria; conjunctivitis and periorbital edema; edema of the lips, tongue, and throat; respiratory distress; tachycardia; abdominal pain; malaise; or generalized weakness and giddiness. Fever and mild diarrhea may occur as well as nausea, though patients rarely vomit. The acute symptoms usually persist for eight to twelve hours, after which the patient experiences a rapid recovery; few fatalities have been reported.[12, 18]

The treatment recommended is immediate evacuation of the stomach contents, followed by the administration of antihistaminic drugs.[18]

At least two outbreaks of what appears to be scombroid poisoning, from ingestion of ahi, *Neothunnus macropterus* (Schlegel), and swordfish, have occurred in Hawaii[37, 40] (Cases 37 and 40, Appendix B), although neither was confirmed by culturing of *P. morganii* from the implicated sample. An additional outbreak of fish poisoning attributed to mahimahi, *Coryphaena hipurus*, which is known to have a high histidine content, may fall into this category, although scombroid poisoning has not been previously reported from this species, and the causative agent was not disclosed (Case 24, Appendix B).

Mahimahi, tuna, mackerel and other allied pelagic fishes are widely exploited by commercial fishermen throughout the Pacific. It should be emphasized that these fishes are not known to contain any endogenous or acquired toxin while in the sea. Strains of *P. morganii*, occurring in the normal bacterial flora, may produce the above described conditions only in improperly refrigerated specimens.

Other types of fish poisoning, unknown to the Hawaiian Islands, occur sporadically in islands to the south and east of Hawaii, and may extend their range to the Hawaiian Archipelago, just as ciguatera has. Such speculation is based on an ignorance rather than a knowledge of these diseases and their means of dissemination. Until we know more of the etiology of toxicity in the fishes themselves, we must be wary of the planned or accidental introduction of marine organisms from areas where these conditions are prevalent.

CLUPEID POISONING

Clupeid poisoning has occurred sporadically in the Marshall Islands, New Caledonia, Fiji, the Society Islands, Indonesia, and Ceylon. It may result from the ingestion of certain herring or sardine-like fishes. The symptoms include dyspnea, cyanosis, cold sweat, painful cramps, and dilated pupils; occasionally death results. It has been suggested by Randall[47] that since the clupeids are plankton feeders, the toxic condition might be related to blooms of tropical planktonic algae, similar to the dinoflagellates which cause paralytic shellfish poisoning in temperate regions. Species which cause poisoning elsewhere do not occur in Hawaii, and closely related species are not prominent in the local fish markets.

GYMNOTHORAX POISONING

Gymnothorax (moray eel) poisoning is a category of fish poisoning proposed by Halstead and

APPENDIX A.—*Compilation of fish poisoning outbreaks in Hawaii described in Appendix B.*

CLASSIFICATION OF FISH POISONING BASED ON AVAILABLE EVIDENCE	NO. OF OUTBREAKS RECORDED	NO. OF PERSONS AFFLICTED	FATALITIES	PERCENT DEATHS IN PERSONS AFFLICTED	PERCENT OF TOTAL OUTBREAKS ATTRIBUTED TO THIS CATEGORY OF FISH POISONING	CASE NO. (APPENDIX B)
Ciguatera						
a. Fish originating in main Hawaiian Is.*	4	38+	0	0	7.4%	33, 43, 51, 52
b. Fish originating in other than main Hawaiian Is.	19	183+	0	0	35.2%	11, 12, 15, 16, 17, 21, 22, 29, 35, 38, 39, 41, 42, 44, 47, 48, 49, 53, 54
Hallucinatory Mullet Poisoning	9	50+	0	0	16.7%	7, 9, 25, 26, 27, 28, 30, 31, 46
Tetraodon Poisoning	4	12	7	58.3%	7.4%	1, 3, 4, 6
Scombroid Poisoning	3	57+	0	0	5.5%	24, 37, 40
Undetermined Classification	15	93+	0	0	27.8%	2, 5, 8, 10, 13, 14, 18, 19, 20, 23, 32, 34, 36, 45, 50
TOTALS	54	433+	7		100.0%	

* The "main Hawaiian Islands" refers to the populated islands of Hawaii, Maui, Molokai, Lanai, Oahu, Kauai, and Niihau.

APPENDIX B.—*Reports of outbreaks of fish poisoning from Hawaii.*[1]

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
1.	April 1903 Kamalo, Molokai	'O'opu-hue or Makimaki." *Arothron hispidus.* Place caught unknown.	1 (1 death)	Tightness and obstruction in breathing; giddiness, tingling, burning and creeping sensations; nausea, vomiting, involuntary purging; rapid, irregular heart action; tendency to syncope; cold hands and feet; failing voice, vision, and hearing; body bathed in cold perspiration; pupils markedly dilated; face pale; great prostration; delirium; convulsive twitching of limbs and muscles of face and body.	Report of A. Mouritz, M.D., Mapulehu, Molokai, in Cobb, J. N., 1904. *The Commercial Fisheries of the Hawaiian Islands in 1903; Report U. S. Fish. Comm. for 1904*, p. 500.
2.	1909—Aboard Cruiser Tennessee at Honolulu	Not recorded.	Not recorded.	Not recorded.	Mann, W. L., 1938. *U. S. Navy Med. Bull.* 36:631-634.
3.	1910—Federal Immigration Sta.	"Puffer fish." Probably *Arothron* sp.; place caught unknown.	7 (3 deaths)	Symptoms not reported, only "Three died, four were very sick."	Anonymous 1925. Fish Poisoning, *Queen's Hospital Bulletin* 2(1). (Nils P. Larsen, M.D., of Honolulu claims authorship of this report.)
4.	1924—Waialua, Oahu	"Dried eggs of Puffer fish." Probably *Arothron* sp.; place caught unknown.	1 (1 death)	Symptoms not reported, only "She (the victim) died within four hours."	Same as above.
5.	Prior to 1925 Molokai	"Ulua" (Family Carangidae). Caught on the sheltered (south) side of Molokai.	6	General ill-feeling and apprehension, nausea, dizziness and tingling in the fingers and toes and all over the body, feeling as if they had been drugged. Two felt and acted definitely intoxicated as with alcohol. In 24 hours all symptoms disappeared.	Same as above.

[1] Only those outbreaks which occurred in Hawaii are recorded (i.e. the fish was ingested here although it may not have been caught in Hawaii). Cases in which the outbreak took place elsewhere and the victims were brought to Hawaii for treatment are not included in this compilation. Common names of fish as in report or scientific names after Gosline and Brock, 1960.

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
6.	1925—Honolulu	"Puffer fish." Probably *Arothron* sp.; place caught unknown.	3 (2 deaths)	Onset 3½ hrs. after ingestion; weakness, nausea, vomiting, severe headache, general numbness; two deaths in few minutes after onset of symptoms.	Larsen, N. P., M.D., *Proc. Sixth Pac. Sci. Congress* 1939. 5:417-421.
7.	1927 (?) Molokai	"Weke pueo." *Upeneus arge;* place caught unknown.	30 to 40	Delirium and "mental paralysis"	Jordan, D. S., B. W. Evermann, and S. Tanaka. 1927. *Proc. Calif. Acad. Sci.* 4(16):20.
8.	1936—Molokai	"White ulua." Probably *Carangoides ajax;* caught near the mouth of Waialua stream, Molokai, Hawaii	3	Nausea, vomiting, tingling of the skin. Medical treatment received.	Personal interview with Mr. Daniel Naki, Waialua, Molokai.
9.	1939 or 1940 Pilaa, Kauai	"Mullet." Probably *Mugil cephalus;* caught near Pilaa, Kauai.	About 12	Drowsiness, extreme weakness in legs, feeling of impending death; frightening dreams.	Report by Mr. George Akau, Bureau of Pure Food & Drugs, Dept. of Health, T. H.
10.	Mid-August, 1943 Honolulu	Flatfish, "Pakii" or "'Ui'ui." Probably *Bothus mancus;* about 100 yds. off Kuhio Beach (Waikiki, Honolulu).	2	Fish fried and eaten at evening meal, a few hours after capture. Onset of symptoms about 5 hrs. after ingestion; symptoms included nausea, vomiting, diarrhea, parched feeling in throat, nightmares and mental depression. Symptoms subsided about 4½ hours after onset.	Report by Dr. Yoshio Kondo, Bishop Museum, Honolulu.
11.	Nov.-Dec. 1944 Honolulu	*Variola louti;* caught at Midway Is., Hawaiian Arch.	24	Onset—4 to 6 hours; vomiting, diarrhea, severe aches and pains in extremities; feeling of numbness in hands and feet.	Lee, R. K. C., M.D., and H. Q. Pang, M.D. 1945. HAWAII MED. JOUR. 4(3):129-132. Also in *Amer. Jour. Trop. Med.* 1945. 25(3):281-285.
12.	Nov.-Dec. 1944	"Black Sea Bass," *Serranus* (=*Epinephelus*) *fuscoguttatus;* caught at Christmas Is.	14	Onset—2 to 6 hours; vomiting, diarrhea, severe aches and pains in extremities, feeling of numbness in hands and feet.	Lee, R. K. C., and H. Q. Pang. 1945. HAWAII MED. JOUR. 4(3):129-132. Also in *Amer. Jour. Trop. Med.* 1945. 25(3):281-285.
13.	Feb. 1946 Waipahu, Oahu	Unident. "red fish"; caught at Waipio Pt., Oahu, Hawaii.	12	Onset 1 hour after ingestion; complete collapse, prostration; shock; extremities cold and clammy; severe diarrhea and dehydration. Pulse 30-40; BP 80/60.[a]	M. M. Chandler, M.D., Waipahu Hospital, Waipahu, Oahu, in report to Dept. of Health, T. H.
14.	June 1947	"Snapper" (Lutjanidae), "Ulua" (Carangidae) and "Mullet"; place caught unknown.	12	Onset 4 to 5 hours after ingestion; stomach cramps, headache, burning and numbness of lips and cheeks, vomiting, diarrhea, and paralysis of arms and legs. Recovery in 24 hours.	Report of Mr. Jack P. Kapua, Pure Food & Drugs Bureau, Dept. of Health, T. H.
15.	Sept. 1947 Honolulu	"Red Snapper." Probably *Lutjanus* sp.; caught in vicinity of Christmas Island, Line Islands.	16	Not recorded.	*Honolulu Star-Bull.*, 3 Oct. 1947 and Report of R. K. C. Lee, M.D., Dept. of Health, T. H.
16.	28 Sept. 1947 Honolulu	"Red Snapper." Probably *Lutjanus* sp.; caught at Fanning Island, Line Islands.	4+	Onset within ½ hour after ingestion; nausea, vomiting, diarrhea, "numbness of body," and a stinging sensation.	Report of Mr. Andrew S. Orlando, Pure Food & Drugs Bureau, Dept. of Health, T. H.
17.	Dec. 1947 Honolulu	"Red Snapper." Probably *Lutjanus* sp.; caught in vicinity of Christmas Island, Line Islands.	17	Chills, diarrhea, tingling and numbness about mouth, pains and weakness in legs, headache, dizziness, and burning sensation of mouth when drinking cold water.	Report of R. K. C. Lee, M.D., Dept. of Health, T. H.

[a] Laboratory tests on implicated fish revealed that the causative agent was probably staphylococci.

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
33.	10 Oct. 1956 Honolulu	"Palani," *Acanthurus dussumieri;* speared on the Mokuleia side of Kaena Pt., Oahu	1	Onset of symptoms about 24 hours. Symptoms included vomiting, diarrhea, numbness about the mouth, hypersalivation, prickling sensation on palms of hands, tingling and itching over entire body, intermittent periods of aching joints during which time the patient could not stand (duration of about 30 min.), and insomnia. Tingling and numbness persisted 3 days. 2 cats ate the head and suffered ataxia, loss of appetite.	Reported by Mr. Michio Takata, Terr. Division of Fish & Game, and F. I. Gilbert, M.D., Honolulu.
34.	30 July 1957 Honolulu	Specimen not seen; from description thought to be "Weke-'ula," *Mulloidichthys auriflamma;* place of capture unknown.	3	Onset of symptoms 3½ hours after ingestion; duration 4 hours. Symptoms included intermittent numbness of fingertips, tongue and mouth, and chills.	Reported by Mr. Michio Takata, Terr. Division of Fish & Game, Honolulu, Hawaii.
35.	October 1957 Wahiawa, Oahu	Specimen not seen; from description it appears to be a Gray Snapper, probably *Lethrinus* sp.; caught at Canton Is., Phoenix Islands and brought to Honolulu frozen.	2	Onset of symptoms 2-3 hours after ingestion of the fish fried. Patients hospitalized with severe vomiting and diarrhea, hypersensitivity of extremities to cold, inability to drink cold water.	Reported by Mr. K. K. Tomomitsu, Food & Drug Inspector, Terr. Dept. of Health, Honolulu.
36.	1958 Kauai	"Manini," *Acanthurus sandvicensis;* caught in the Pilaa-Haena region, Kauai, Hawaii.	1	Patient reported "poisoned" from eating Manini; symptoms not recorded.	Report of Mr. George H. Akau, Bureau of Pure Food & Drugs, Terr. Dept. of Health, Honolulu.
37.	26 Aug. 1958 Honolulu	"A'u" or "Swordfish"; species unknown. Caught either off Hilo, Hawaii, or the Waianae coast of Oahu.	4+	Onset of symptoms 15 minutes after ingestion. Symptoms included diarrhea, nausea, vomiting, throbbing headache, and hot-flushed face. A four-year-old victim developed urticaria. Duration of symptoms was about two hours.	Reported by Mr. K. K. Tomomitsu and Mr. Y. S. Lee, Bureau of Pure Food & Drugs, Terr. Dept. of Health, Honolulu.
38.	May 1959 Honolulu	"Grouper," *Cephalopholis argus;* caught at Palmyra Island, Line Islands.	4	Onset of symptoms about 10 hours after ingestion, included tingling about mouth, numbness in extremities, nausea, and lethargy. Remains fed to mongooses produced kills at 10% and 20% body weight equivalent doses.	Report of Col. F. L. Boling and Maj. F. Clayton, U.S.A.F., Hickam Air Force Base, Hawaii.
39.	Oct.-Nov. 1959	"Thick-lipped Ulua, Pig Ulua, or Butaguchi," *Caranx chellio,* caught in lagoon at Midway Is.[5]	25+	Numbness and tingling about the mouth and extremities; weakness in legs; reversal of temperature sensation, insomnia, nausea, vomiting, diarrhea.	Report of W. M. M. Robinson, M.C., USN, Medical Officer, U. S. Naval Station, Midway Is., and Banner *et al., Ann. N. Y. Acad. Sci.* 90(3):770-787.
40.	1 Sept. 1959 Honolulu	"Ahi," *Neothunnus macropterus* bought from a Honolulu fish market, place caught unknown.	2	Severe cephalgia, pounding and frontal, (histamine type), circumoral tingling, diffuse irregular erythema, severe sensation of burning in skin but no itching, conjunctival congestion, BP normal, pulse 112. Nausea w/o vomiting. Symptoms abated after IV inj. antihistamine. Patient thought fish tasted "hot" or "wrong" at time of ingestion.	Report of R. Butler, M.D., Honolulu, Hawaii, to State Dept. of Health.

[5] Periodic sampling of this species from Midway Lagoon was carried on during 1960. Samples produced ciguatera syndrome in laboratory animals.

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
18.	Dec. 1947 Honolulu	"Mahimahi," *Coryphaena hippurus;* place caught unknown.	31	Employees of Libby, McNeill & Libby Co. became very ill after eating in company's cafeteria; detailed symptoms not reported.	Report of Mr. George Akau, Pure Food & Drugs Bureau, Dept. of Health, T. H.
19.	About 1948 Hilo	"White Eel." Probably *Conger marginatus;* caught in a trap on island of Hawaii, exact location unknown.	"Several"	Patients became violently ill; symptoms both gastrointestinal and neurological with recovery in 5 to 7 days. Remains of eel fed to cat which developed paralysis of limbs and died from respiratory arrest.	Personal correspondence from W. S. L. Loo, M.D., Hilo, Hawaii.
20.	Oct. 1949 Honolulu	"Surgeonfish and other unidentified varieties"; caught at Johnston Is.	10	Not recorded.	Report of Mr. George Akau, Pure Food & Drugs Bureau, Dept. of Health, T. H.
21.	Aug. 1950 Honolulu	"Black Bass" (Sea Bass). Probably a serranid; caught at Canton Is., Phoenix Islands.	5	Onset 12 to 36 hours after ingestion; abdominal pain, diarrhea, tingling and numbness of hands and feet; when drinking water it tasted hot and salty; showering caused itchiness over the entire body, fetal movements ceased during 3 days of sickness in pregnant women.	Lewis M. Nutting, Pure Food & Drugs Bureau, Dept. of Health, T. H.
22.	Feb. 1951 Maui	"Manini," *Acanthurus triostegus triostegus;* caught at Palmyra Is.	31	Vomiting, diarrhea, abdominal pain, nausea, tingling and numbness about the mouth, extreme weakness, pains in joints of arms and legs, reversal of temperature sensation; itchiness of soles and palms.	Reports of Mr. George Akau, Dept. of Health, T. H., and T. G. Lathrop, M.D., Dept. of Health, Wailuku, Maui, Hawaii.
23.	1953-1955 (?) Maalaea, Maui	"Akule," *Trachurops crumenophthalmus;* caught at Maalaea Bay, Maui, Hawaii.	5+	Gastrointestinal upset; numbness and tingling about the mouth.[3]	Report by W. B. Patterson, M.D., Puunene, Maui, and personal interview with one of the victims.
24.	17 Aug. 1954 Honolulu	"Mahimahi," *Coryphaena hippurus;* caught 30-40 miles SE of Diamond Head, Oahu, Hawaii.	51	Onset 15-30 minutes after ingestion; resembled histaminic reaction; diarrhea, nausea, vomiting, headache, flushing, some conjunctival congestion. No neurotoxic symptoms.	Report of James R. Enright, M.D., Chief, Bureau of Epidemiology, Dept. of Health, T. H.
25-28.	Between May and Aug. 1954-1959 (4 separate outbreaks) Molokai	"Mullet," *Mugil cephalus;* caught in Palaau region of Molokai, Hawaii.	4	Onset 2 hours after ingestion; dizziness, loss of balance, pounding heart. Symptoms of short duration (about 1 hour), and the same in all four outbreaks.	Personal interview with victim at Kaunakakai, Molokai.
29.	1955 Honolulu	"Red Snapper." Probably *Lutjanus* sp.; caught at Canton Is., Phoenix Islands.	4	Diarrhea, extreme weakness, difficulty in breathing, decreased heartbeat, numbness of lips, hands, and feet; warm water felt cold and penetrating.	Mr. Y. S. Lee and Mr. K. K. Tomomitsu, Food & Drug Inspectors, Dept. of Health, T. H.
30.	1955-56(?) Kaunakakai, Molokai	"Mullet," *Mugil cephalus;* caught at Palaau, Molokai, Hawaii.	1	Dizziness, loss of equilibrium, ringing in ears, tingling sensation over entire body; duration about 30 min.	Personal interview and correspondence with victim at Kaunakakai, Molokai.
31.	1956 Molokai	"Mullet," *Mugil cephalus;* caught at Palaau, Molokai, Hawaii.	1	Onset of symptoms 10 minutes after ingestion—included nausea, vomiting, tingling sensation of skin. (Fish prepared and eaten 20 minutes after being caught.)	Personal interview with victim at Pukoo, Molokai.
32.	Sept. 1956 Kohala	"Humuhumu-'ele'ele," *Melichthys buniva;* caught near Mahukona, Hawaii.	1	Onset 14 hours after ingestion; pain, muscle spasm in arms and legs, severe pain in wrists and ankles. No nausea or vomiting. More severe effects after ingestion of portion of the same fish 3 weeks later.[4]	Personal correspondence from F. L. Tabrah, M.D., Kohala, Hawaii.

[3] Fishermen report that Akule "spoil" rapidly after removal from water. Conflicting evidence exists as to the freshness of these fish when eaten, and therefore bacterial action cannot be ruled out as the causative agent in this case.

[4] Second ingestion caused more severe symptoms including an overpowering lethargy and a feeling of cold and painful feet. A burning pain in soles continued for almost 12 months requiring Demerol occasionally to enable the patient to work.

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
41.	Nov. 1959 Kaneohe, Oahu	"Ulua," *Caranx ignobilis;* caught off channel at west end of Palmyra Is., Line Islands.	1	Onset of symptoms about 20 hrs. after ingestion; diarrhea, headache, dizziness, unsteady gait, flushed feeling, malaise around lips, tongue, and throat, aching joints (especially in hips), weakness in legs, lethargy, burning sensation when touching cold objects. Mild symptoms persisted for 5 days.	Personal interview with patient by author.
42.	11 Dec. 1959 Kailua, Oahu	"Ulua" (Carangidae), species unknown; fish caught off Johnston Is.	3	Onset of symptoms about 12 hrs. after ingestion; weakness in limbs, lethargy, dizziness, metallic and carbonated taste when food ingested, aching joints, burning sensation when touching cold objects, itchiness of skin over the entire body. No vomiting or diarrhea. Mild symptoms persisted about 7 days.	Personal interview with patient by author.
43.	2 Mar. 1960	Thought to be "Palani" (*Acanthurus dussumieri*) bought from Honolulu fish market; place caught unknown.*	2	Attending physician familiar with *ciguatera* from other areas and stated symptoms similar including nausea, vomiting, paresthesia, numbness in extremities, reversal of sensations of heat and cold.	Report of H. L. Arnold, Sr., M.D., Honolulu, Hawaii.
44.	18 June 1960	"Ulua" (Carangidae), species unknown; caught at Midway Is., Hawaiian Arch.	4	Onset of symptoms 5 hours after ingestion; included numbness of leg muscles and joints, reversal of temperature sensation, burning pains, sweating. Diarrhea, fever, headache not present. Examined by physician who diagnosed it as acute food poisoning. Lab tests on fish tend to confirm *ciguatera* fish poisoning.	Mr. Thomas M. Naito, Food Inspector, Pure Food & Drugs Div., State Dept. of Health.
45.	25 June 1960 Hauula, Oahu	"Black Hinalea," an unidentified labrid fish, and the ovaries of the sea urchin, *Podophora atrata* both caught in shallow water at Hauula, Oahu.	1	Onset of symptoms about 18 hours; weakness of legs, nausea, later swollen feeling in tongue and hand, numbness in cheeks and tingling in fingers (like pins and needles).	Report by victim's physician, Mary A. Glover, M.D., Kaneohe, Hawaii, and personal interview with the patient.
46.	25 Oct. 1960	Unidentified species of "Weke" (Mullidae). Place caught unknown. (Obtained from peddler.)	2	Dizziness; paresthesia; temporary paralysis; ineffectual attempts to urinate; malaise; feeling of floating in the air; mental anguish; nightmares.	Report of Teru Togasaki, M.D., Honolulu, Hawaii.
47.	29 Oct. 1960	Ulua, believed to be *Caranx cheilio,* caught in lagoon, Midway Island.	8	Burning and tingling about the mouth; tingling in extremities; weakness in legs; pain in joints; reversal of temperature sensation; malaise, nausea, vomiting, and diarrhea.	Report of M. A. De Harne, M.D., Wahiawa, Oahu, and James Enright, M.D., State Dept. of Health, Honolulu, Hawaii.
48.	1 June 1961	"Kahala, Amberjack, or Yellowtail," *Seriola dumerilii,* (Carangidae) caught at Midway Island.	12	Diarrhea; vomiting; tingling and numbness in the extremities; sensation of electric shock experienced when touching objects. Adult patients later developed hives.	Report of K. K. Tomomitsu, Food & Drug Inspector, Bureau of Pure Food & Drugs, State Dept. of Health, Honolulu, Hawaii.

* The remains of the implicated fish were discarded before they could be examined, but the physician asked the patient to purchase another fish just like the one that had poisoned him. The patient purchased a "palani" (*Acanthurus dussumieri*). It should be noted that this species is very similar to two other species commonly found in the local fish markets, *Acanthurus mata,* and *Acanthurus xanthopterus,* and the three species might be easily confused by those not familiar with the rather subtle distinguishing characteristics. The fish purchased by the patient was fed to test animals with negative results.

NO.	DATE AND SITE OF OUTBREAK IN HAWAII	SPECIES OF FISH AND PLACE CAUGHT	NO. OF PERSONS INVOLVED	SYMPTOMS	SOURCE OF INFORMATION
49.	20 July 1961 Naval Air Sta. Barber's Pt., Oahu	"Rainbow Runner, Kamanu, or Hawaiian Salmon" *Elagatis bipinnulatus* (Carangidae) caught in shallow water off Eastern Is., Midway.	6	Tingling and numbness about lips, tongue and throat; abdominal cramps; diarrhea; numbness and tingling of hands and feet; malaise, slow heart rate; restlessness; insomnia; peppery taste and thirst; dysesthesia; itching palms and soles; hypotension. Neurotoxic symptoms prolonged.	Report of CWO R. J. Mazza, Sanitation Officer, U. S. Naval Station, Barber's Point, Hawaii. Also *U. S. Navy Medical News Letter* 38(9):29, 1961.
50.	1 Apr. 1962 Kealia, Kauai	"Marlin" (species unknown) caught near Kauai. Fish weighed 520 lbs.; only the head and liver were known to be involved in intoxications.	3	Onset of symptoms about 12 hrs. Liver eaten on 2 occasions; after 1st meal patient experience frontal headache, after 2nd meal, patient and wife both experienced nausea, vomiting, diarrhea, aching joints, and weakness in legs. Weakness persisted for 8 days. Face began to peel 3rd day after ingestion. One patient who both handled and ingested the liver experienced exfoliating dermatitis of hands after 7 days. A 3rd person ate only the head and experienced drowsiness and "heaviness of legs."	Peter Kim, M.D., Samuel Mahelona Memorial Hospital, Kealia, Kauai.
51.	9 Apr. 1962 Honolulu	"Palani," *Acanthurus dussumieri* caught off the Waianae coast of Oahu and sold in a Honolulu fish market. Two fish involved—about 9" and 18" in length.	5	Fish appeared wholesome when prepared. Fish boiled and eaten; onset of symptoms about 10 hours after ingestion. Symptoms included headache, malaise, tingling of extremities, aching of joints and teeth, generalized weakness, abdominal cramps, sore throat, and diarrhea (black stools). One victim had been previously poisoned; his symptoms persisted for 3 weeks. In others, symptoms lasted 1½ weeks.	Reported by Mr. K. K. Tomomitsu, Food & Drug Inspector, State Dept. of Health, and R. C. Durant, M.D., Honolulu.
52.	9 July 1962 Island of Hawaii	"Black Sea Bass" possibly *Epinephelus tauvina* caught at Kapoho Beach. Fish weighed 238 lbs.	30+	Symptoms in order of frequency reported: "tired feeling" or weakness in arms and legs, diarrhea, vomiting, prickling and itchiness of palms and soles, itching of mouth, itching of fingers, "tender tongue," "stomach distress," hives, swollen lips, itchiness over the entire body, rash 2-3 days after ingestion. Stomach of fish appeared to be the most highly toxic.	Report of Mr. Horace Kawamura, Area Sanitarian, Hawaii State Dept. of Health.
53.	7 Sept. 1962 Waianae, Oahu	"Red Snapper" (species unknown) from lagoon, Canton Island, Phoenix Is.	2	Onset of symptoms about 4 hrs. Numbness of facial area and extremities, severe diarrhea, vomiting, prostration, and sensitivity to cold. One victim was hospitalized for 3 days.	Report of Mr. K. K. Tomomitsu, Food & Drug Inspector, Bureau of Pure Food & Drugs, State Dept. of Health.
54.	24 Oct. 1962 Midway Is.	"Ulua" (species unknown) caught near lagoon reef; total length—20 inches.	1	Onset of symptoms 90 minutes after ingestion with sudden extreme fatigue, nausea, feeling faint. Other symptoms—chills, sweating, stiff neck, cold liquids burned when swallowed, diarrhea, itching and burning of skin, abdominal cramps, aching muscle (especially thighs), aching joints, reversal of temperature sensation over entire body. Patient responded well to neostigmine therapy.	Robert L. Altman, M.D., Pearl Harbor Naval Shipyard Dispensary, Hawaii.

Lively,[21] with a syndrome similar to that of ciguatera. It differs from ciguatera in that the onset of symptoms is more rapid, and convulsions and paralysis are more prominent.

Randall[47] suggests that the difference may be only quantitative; he points out that the syndrome in moderately severe cases of ciguatera is almost identical to that encountered in cases in which the victim consumed small quantities of toxic moray eel. Ralls and Halstead[46] claim that a difference in the initial solubilities of the two toxins exists, although validity of this work has been questioned by other investigators.[6] Experimental feeding of large specimens of the moray eel, *Gymnothorax javanicus,* from Palmyra Island, indicated that these eels contained a much higher concentration of toxin per unit weight than did the most toxic red snapper, *Lutjanus bohar,* from the same area, and the symptoms produced in laboratory animals from the snappers and moray eels were identical. Moray eels of the genus *Gymnothorax* are all predacious on fishes, and they possess adaptations for capturing and ingesting relatively large prey. It is hypothesized that continued ingestion of large fishes such as snappers and groupers would allow the moray eels to acquire sizable quantities of the toxin over a period of time. The longevity of moray eels is favored by the fact that they have no known predators. If they excrete only a small percentage of the toxin ingested (as is the case with *L. bohar,* studied by Takata[50]), the high concentration of toxin in their flesh would be an indication of the quantity of toxin (in the form of toxic fish) that they had ingested.

Further elucidation of the relationships of ciguatera to gymnothorax poisoning requires the chemical isolation and identification of the toxins involved as well as ecological studies on the relationship of moray eels to species harboring ciguatera toxin. Based upon the above hypothesis, however, one might expect moray eels to become toxic sometime after ciguatera had extended into a previously unaffected area.

No cases of gymnothorax poisoning have been reported from Hawaii, although small quantities of these eels are regularly consumed locally by certain ethnic groups.

ELASMOBRANCH POISONING

Sporadic outbreaks of elasmobranch poisoning have occurred from the ingestion of sharks, particularly shark livers, in the Line Islands, Samoa, the Society Islands, the Marshall Islands, and perhaps elsewhere. Only mild symptoms have been attributed to eating of the flesh, but more severe poisonings and deaths have resulted from ingestion of shark livers. Symptoms may begin within 30 minutes after the ingestion of a toxic liver; they include headache, nausea, vomiting, diarrhea, aching joints, heaviness of limbs, prostration, delirium, feeble pulse, diaphoresis, respiratory distress, thoracic pain, burning sensations of the tongue, throat, and esophagus, and tingling of the lips and extremities. In cases of elasmobranch poisoning, coma and death may result.[12, 19]

Little is known of the nature or mode of action of the toxin involved. It has been postulated that the toxicity of sharks is the result of their feeding upon ciguateric fish. Others feel that the intoxication in humans is due to an overdose of vitamin A. There is probably no relation to scombroid poisoning, for shark flesh contains no free histidine.[35]

Elasmobranch poisoning has not been reported from Hawaii, and it would only become a potentially serious problem if Hawaiian sharks were to be exploited commercially for human consumption. Present laws which require the labeling of packaged food items have discouraged the former practice of using shark flesh in the preparation of fish cake for local consumption, and a fishery for sharks no longer exists in Hawaii. Therefore, at the present time, the primary danger presented is not in the ingestion of sharks by humans, but vice versa.

REVIEW OF OUTBREAKS

In the course of research on toxic marine organisms over the past four years, numerous case reports of fish poisoning were discovered. A search of the records of the State Department of Health, as well as personal interviews, resulted in the uncovering of 54 outbreaks since 1900; the pertinent data on these outbreaks are presented in Appendix B. Although undoubtedly incomplete, these data probably represent a fair picture of the relative frequency of various types of fish poisoning described. Therefore, the pattern of previous outbreaks is examined as indicative of possible trends.

Fish poisoning outbreaks of various types, occurring in Hawaii and listed in Appendix B, are sumarized in Appendix A. The data in Appendix A indicate that 42.6 per cent of all outbreaks of fish poisoning are attributable to ciguatera. Hallucinatory mullet, tetraodon, and scombroid poisoning outbreaks combined accounted for almost 30 per cent of the outbreaks, with about 28 per cent in the unidentified category. Tetraodon poisoning accounted for the only known fatalities, with a mortality rate of almost 60 percent in four outbreaks. This reported mortality rate is probably considerably higher than the actual figure, because of publicity surrounding fatalities of this type, and the lack of information on mild cases.

A compilation of outbreaks of fish poisoning from the standpoint of families of fish involved

TABLE 1.—*Compilation of families of fishes most frequently involved in outbreaks of fish poisoning in the Hawaiian Islands with the geographical area from which they originated. (Data from Appendix B.)*

FAMILY OF FISH		GEOGRAPHICAL ORIGIN						TOTAL	
		Hawaii (Main Is.)	*Midway*	*Line Is.*	*Johnston Is.*	*Phoenix Is.*	*Origin Not Determined*	*No.*	*%*
Carangidae (Ulua, Jacks, Pompano)		4	6	1	1		1	13	23.2
Mugilidae (Mullet)		6				1	1	8	14.3
Acanthuridae (Surgeonfish)		3		1	2			6	10.7
Lutjanidae (Snappers)				3		2	1	6	10.7
Serranidae (Sea bass)		1	2	1		1		5	8.9
Tetraodontidae (Puffers)		4						4	7.1
Mullidae (Goatfish)		3						3	5.4
Other		9						9	16.1
Family not determined		1					1	2	3.6
TOTAL	No.	31	8	6	3	4	4	56*	100.0
	%	55.4	14.3	10.7	5.4	7.1	7.1		100.0

* This total is greater than the number of outbreaks recorded in Appendix B because three species of fish were involved in a single outbreak.

and their geographical origin is presented in Table 1. About half of all outbreaks of fish poisoning in Hawaii were attributable to fish in three families: Carangidae (uluas), Mugillidae (mullets), and Acanthuridae (surgeonfishes). Over 37 per cent of all outbreaks were caused by toxic fish imported from Midway, Johnston, and the Phoenix and Line Islands, despite a regulation prohibiting such importations.* In view of the nuclear testing and missile programs on Johnston Island and the Line Islands, further outbreaks of poisoning from fish originating in these areas might be expected unless a more active program of education and control is pursued.

An examination of the number of cases of fish poisoning occurring in periods since 1900 is presented in Table 2. Although, admittedly, greater attention has been given to fish poisoning outbreaks in the past four years, the marked increase in the number of outbreaks (particularly of ciguatera) in the five-year period, 1956-1960, may be indicative of an increase in the absolute incidence of these occurrences, and it might well warrant the attention of public health authorities.

DISCUSSION

Of the various types of fish poisoning herein discussed, only four are likely to be encountered by the practicing physician in Hawaii: ciguatera, hallucinatory mullet, tetraodon, and scombroid. This is not to imply that other types may not become problems, or that yet undescribed ichthyotoxins may not be discovered in the Hawaiian Islands.

* Part c-1, Second Amendment to Chapter 4, Public Health Regulations of Hawaii, April, 1954.

In the author's opinion, of the types of fish poisoning likely to be encountered, ciguatera poses the greatest potential health problem in the Hawaiian Islands for the following reasons: (1) the spread of this disease to the Line Islands, Johnston Island, and Midway Island is well established,[6, 20, 22, 37, 38] and in at least four outbreaks reported from Hawaii, the characteristic ciguatera syndrome strongly suggests that the etiologic agent in the environment of the fish has invaded Hawaiian waters; (2) the condition is particularly insidious in that it defies ready detection: it is not possible to distinguish a toxic fish from a nontoxic fish without time-consuming test feeding to mammals or completion of a complex extraction and bioassay; (3) among the fishes normally affected are those highly esteemed as food, such as the carangids (ulua, papio), barracuda, and acanthurids (manini, palani, pualu), as well as a number of other reef fishes which are heavily fished by sportsmen and some commercial fishermen (practically all reef species are consumed as food by some segment of the population in Hawaii); (4) because of the vicissitudes of phenomena associated with ciguateric fishes, regulation of consumption in a general outbreak would be difficult and wasteful (for example, in the Society Islands, there is a species of fish which is toxic in one area of a bay, and nontoxic in another,[47] and there is evidence that the toxicity of fish waxes and wanes within a toxic area); and (6) the apparently cumulative effect of ciguatera toxin causes patients to display what appears to be an increased sensitivity upon repeated exposure.

Hawaii State Health Department and conservation authorities are aware of the potential problem

TABLE 2.—*Periods since 1900 during which outbreaks of fish poisoning of various categories have occurred in the Hawaiian Islands. (Based on data from Appendix B)*

CLASSIFICATION OF FISH POISONING	DATES						TOTAL	
	40 yrs. 1900-40 (Incl.)	*5 yrs. 1941-45 (Incl.)*	*5 yrs. 1946-50 (Incl.)*	*5 yrs. 1951-55 (Incl.)*	*5 yrs. 1956-60 (Incl.)*	*1 Jan. 1961-1 Jan. 1963*	*No.*	*%*
Ciguatera								
a. Hawaiian origin*	0	0	0	0	2	3	5	9.3
b. Non-Hawaiian origin	0	2	4	2	7	4	19	35.2
Hallucinatory Mullet Poisoning	2	0	0	2	5	0	9	16.7
Tetraodon Poisoning	4	0	0	0	0	0	4	7.4
Scombroid Poisoning	0	0	0	1	2	0	3	5.5
Undetermined Classification	3	1	5	1	4	0	14	25.9
TOTAL {No.	9	3	9	6	20	7	54	100.0
TOTAL {%	16.7	5.5	16.7	11.1	37.0	13.0		100.0

* Hawaiian origin refers to "main Hawaiian Islands" as defined in Appendix A.

posed by the increased incidence of ciguatera; it is hoped that this publication, as well as the results of research presently being pursued, will allow the practicing physician to better understand the problem of fish poisoning and to recognize and treat cases which he may encounter.

Among other categories of fish poisoning found in Hawaii, tetraodon poisoning is not viewed as a major health problem because the potential danger appears to be well understood by those persons who regularly consume puffer fish. Carelessness, misjudgment, and ignorance by those preparing local puffer fish will probably result in occasional outbreaks, as they have in the past.

Hallucinatory mullet poisoning is more of academic interest than a threat to public health, since it is a relatively mild condition which has been restricted to the same areas for many years. Existing sanitary regulations and normal precautions will probably prevent scombroid poisoning, except for an occasional case.

EPIDEMIOLOGICAL STUDY

Recognition of the value of more complete data on fish poisoning to aid in better understanding the problem has prompted the initiation of a broad epidemiological study of fish poisoning in the entire tropical Pacific. Cooperating in this study are the National Institutes of Health, the South Pacific Commission, and the University of Hawaii. French and English editions of an informational booklet on fish poisoning,[25] as well as questionnaires on (1) local knowledge, (2) biological aspects, and (3) medical aspects of outbreaks of fish poisoning are being distributed through the Executive Officer for Health of the South Pacific Commission to medical personnel throughout the tropical Pacific. Because of the apparently low incidence of fish poisoning in the Hawaiian Islands at the present time, the wholesale distribution of this literature in Hawaii is not planned. However, the cooperation of local physicians is solicited in reporting future outbreaks as well as information not previously reported on past outbreaks. Questionnaires are available for this purpose from the Epidemiology Branch, State Department of Health, and from the Hawaii Marine Laboratory, University of Hawaii.

SUMMARY

Fish poisoning from the ingestion of fish containing toxins largely of nonbacterial origin have been responsible for at least 54 outbreaks involving more than 433 persons and resulting in seven deaths in Hawaii since 1900. A recent spread of ciguatera fish poisoning to areas not previously affected, including the Hawaiian Islands, has resulted in increased research on various aspects of the problem. A description of the defined categories of fish poisoning found in the Pacific is presented, together with the species implicated in each, the syndromes of the diseases in humans, and other available information. The categories discussed are ciguatera, hallucinatory mullet, tetraodon, scombroid, gymnothorax, clasmobranch, and clupeid poisoning. Only the first four categories listed have been implicated in Hawaiian outbreaks. Of these, only scombroid poisoning is known to be caused by the action of a bacterium upon the fish after capture, but, because of its very rapid development under certain conditions, it may be confused with the other categories of poisoning discussed in which fish contain endogenous or acquired toxins when alive. An analysis of past outbreaks of fish poisoning in Hawaii is presented, revealing that ciguatera from imported fish was responsible for a majority of the cases. Ciguatera fish poisoning is considered to be the most serious potential health problem in Hawaii because it has recently spread from the south and west to the Hawaiian Islands, it is difficult to detect,

it normally occurs in a number of species that are highly esteemed as food in Hawaii, its vicissitudes make prediction and control difficult, and the toxin appears to have a cumulative effect on patients receiving repeated doses. An epidemiological study of fish poisoning in the entire tropical Pacific has been initiated in order to learn more about the various categories of fish poisoning and their distribution.

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ifornia Morbidity

ekly Report from the
Infectious Disease Section
State Department of Health

October 28, 1977
No. 42

CIGUATERA FISH POISONING

On June 21, 1977 a Bay Area hospital pharmacist called the Infectious Disease Section regarding the availability of botulinum antitoxin for a 33 year-old San Francisco woman. On 6/20, about 2 hours after eating a homemade fish chowder prepared from frozen fish caught at Midway Island, she had acute onset of "chilliness", nausea, abdominal cramps and watery diarrhea. The next morning she complained of circumoral and limb numbness, malaise, weakness and difficulty in swallowing and breathing. Symptoms worsened and she was admitted to hospital. Examination was unremarkable except for slight weakness of the upper extremities and diminished deep tendon reflexes. No cranial nerve or respiratory abnormalities were noted. There was no history of ingestion of mussels, clams or oysters. The clinical and epidemiologic data suggested ciguatera fish poisoning rather than botulism and an investigation was started to trace the distribution of any remaining fish.

During a stopover on 5/2/77 at Midway Island a merchant seaman caught six jackfish (Carangidae) weighing 15-20 pounds each. The whole ungutted fish remained in the ship's freezer until return to the Port of Oakland on 6/19 when they were distributed to 5 families in San Francisco, Oakland and Sacramento. All remaining fish were quickly recovered but in four of the five households some fish had already been eaten and cases had occurred in each family. Three persons were hospitalized.

In each household fish was prepared in traditional Philippine style as a chowder (by boiling heads, flesh and viscera with vegetables for 10 to 15 minutes) or by deep frying fish steaks served in vinegar/herb sauce (escabeche).

A total of 24 persons ate the fish but 8 had only 1 or 2 tablespoonfuls of chowder broth poured over boiled rice. None of the 8 developed symptoms. The attack rate was 100% in the remaining 16 individuals, all of whom consumed at least 1/2 cup of chowder. The mean incubation period was 4 hours (range 2-6 hours). Symptoms included malaise, chilly sensation, sweats, nausea, vomiting, diarrhea, abdominal pain, metallic taste, oral burning, numbness and tingling of the lips, mouth and extremities, vertigo, headache, arthralgias, myalgias, weakness, blurred vision, breathing discomfort and paradoxical temperature sensation. Severity of symptoms and incubation periods were dose-related. Acute symptoms generally subsided within 24 to 48 hours, however paresthesias, myalgias and weakness persisted in 4 persons for more than 2 weeks. Ciguatoxin was identified in fish samples by Professor Y. Hokama, University of Hawaii at Manoa, using an RIA method (Hokama, Y. et al, Toxicon, *15*:317, 1977).

COMMENT: Ciguatoxin is produced by certain dinoflagellates attached to algae on coral reefs in warm tropical ocean waters. Small fish feed on the algae and are in turn eaten by larger bottom dwelling shore fish and so on up the food chain. The larger the fish, the more potentially toxic. More than 400 fish species have been implicated between latitudes 35° N and 35° S. Ciguatoxic fish do not look or taste spoiled and ordinary cooking does not destroy the heat-stable toxin. Ciguatera poisoning has been reported frequently on Midway and other islands of the Hawaiian archipelago (MMWR, 25(27):219, 1976).

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TABLE 1. REPORTED CASES OF SELECTED NOTIFIABLE DISEASES
CALIFORNIA, 42nd WEEK ENDING October 22, 1977

DISEASE	Cases Reported This Week			Total Cases Reported to Date		
	1977	1976	1975	1977	1976	1975
Coccidioidomycosis	1	3	2	290	406	308
Encephalitis, Total	8	2	3	106	133	125
Primary	8	1	3	83	99	81
Post-infectious	-	1	-	23	34	44
Food Poisoning (excludes Botulism)	758	-	7	2,213	243	2,852
Gonorrhea	2,516	2,748	2,142	104,669	98,902	93,972
Hepatitis Type A (Infectious)	106	131	145	4,312	4,688	4,079
Hepatitis Type B (Serum)	57	94	42	2,734	2,532	2,034
Hepatitis Type Unspecified	44	41	25	1,732	1,006	761
Measles	29	48	28	9,312	2,071	5,073
Meningitis, Viral	25	29	21	667	538	421
Meningococcal Infections	3	3	5	100	116	91
Mumps	8	19	38	759	1,647	3,140
Pertussis	5	2	1	138	60	102
Rabies, Animal	5	8	4	366	282	229
Rubella (German Measles)	11	13	7	1,559	806	1,357
Salmonellosis	58	57	38	1,895	1,677	1,561
Shigellosis	108	106	112	2,925	3,492	3,097
Streptococcal Infections, Respiratory	298	255	179	16,929	11,941	11,939
Syphilis, Total	243	334	245	10,084	9,928	11,167
Primary and Secondary	91	137	110	3,161	3,810	4,053
Tuberculosis (Total Reports)	79	75	67	2,895	3,072	3,119
Typhoid Fever	1	3	4	62	88	77

Toxicon, 1968, Vol. 6, pp. 55–58. Pergamon Press. Printed in Great Britain

MARINE TOXINS FROM THE PACIFIC—IV PHARMACOLOGY OF CIGUATOXIN(S)*

THOMAS I. KOSAKI and HAMILTON H. ANDERSON
Hawaii Institute of Marine Biology, University of Hawaii, Honolulu, Hawaii, U.S.A.

(*Accepted for publication* 11 *December* 1967)

Abstract—Ciguatoxin(s) in the methanol extract stage from Central Pacific fishes were derived by previously described methods. LD_{50} Data in chickens and mice determined rat lethal doses for quantitative study of systems' responses. Impaired respiration occurred first, followed by cessation; hypotension occurred and later blood pressure returned to near normal; cardiac irregularities, viz. bradycardia and arrhythmias followed. Partial blockage of nerve-muscle response was observed. In animals given non-lethal extracts, slightly irregular respiration and transient hypotension were observed.

INTRODUCTION

HESSEL *et al.* [1] found ciguatera toxin to have an inhibitory effect on action potentials, and concluded that more than one poison was present. Their characteristics are similar [2, 3], and further studies provide indications that more than one toxin probably occurs in fish previously labelled as 'ciguateric' [4]. Quantitative pharmacologic analysis has been made of neuromuscular and other system responses of mammals.

Ciguatoxin(s) given intravenously within the lethal range provided somewhat similar responses to those noted previously [5]. KOSAKI *et al.* [6], have established the lethal range of ciguatoxin(s) in comparison with oral toxicity in the mongoose. Comparative bioassays (i.v./i.p.) in mice and chickens were made to permit studies of systems' responses in rats.

Pupillary meiosis in the rabbit following topical use of 1 per cent ciguatoxin(s)† correlated well with *in vitro* inhibition of cholinesterase, using the pH stat Titrimeter. This has been confirmed by other *in vitro* methods (G. Ellman, personal communication) [7].

Utilizing the Grass polygraph, systems studied were: respiration, blood pressure, electrocardiography, and sciatic nerve/gastrocnemius muscle *in situ*. In every instance, except when extracts of non-toxic *Naso unicornis* were studied, the i.v. dose in the rat was within the lethal range.

MATERIALS AND METHODS

Ciguatoxin(s) methanol extracts were prepared by SCHEUER *et al.* [3] Ciguatoxin was

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†Methanol extracts of four species of fish known to be 'ciguateric' [2].

TABLE 1. COMPARATIVE PHARMACOLOGIC EFFECTS ON VARIOUS SYSTEMS

Species of fish and source	Total dose-i.v. (in rat) (mg/kg)	Grass polygraph recordings: Effects on respiration (RR-resp. rate)	Blood pressure response (mm change)	Electro-cardiography (rate/min)	Neuro-muscular response (mm)	Miosis in rabbits (maximum change mm)
Gymnothorax javanicus R—157	11·8	RR-↓ amplitude-↑ Cheyne Stokes	Initial ↓ (20) long recovery period then returns (30)	Heart rate from 360–300 beats arrythmia	↓8/13	1·20
Gymnothorax javanicus R—171	26·0	RR-↓ amplitude-↑ Cheyne Stokes	Initial ↓ (50) long recovery period, returns to slightly subnormal level (40 below)	Bradycardia: 30 min from 360–240 beats arrythmia	↓4/13	1·50
Gymnothorax javanicus R—749	76·6	RR-↓ amplitude-↑ irregular respiration	Initial ↓ (10) remains low then (25)	Bradycardia: 360–120 beats arrythmia	↓4/8	2·00
Gymnothorax javanicus R—606	75·0	RR-↓ amplitude-↑ Cheyne Stokes	Initial ↓ (80) long recovery period, returning subnormal (20 below) going above normal (20)	Bradycardia: 360–240 beats arrythmia	↓6/13	1·70
Seriola dumerilii B—446	105·0	RR-↓ amplitude-↑ irregular respiration	Initial ↓ (50) long recovery period returning subnormal (20 below)	Heart rate 360–300 beats arrythmia	↓2/9	1·50
Gymnothorax javanicus R—570	168·0	RR-↓ amplitude-↑ irregular respiration	Initial ↓ (80) long recovery period returns subnormal (60 below)	Bradycardia: 360–180 beats	↓1/5	1·20
Epinephelus fuscoguttatus EPF—1	465·0	RR-↓ amplitude-↑ irregular respiration	Initial ↓ (55) short recovery period returns to normal	Heart rate 30 min from 360–300 beats arrythmia	↓3/9·5	1·34
Epinephelus hexagonatus EPH—1	438·0	RR-↓ amplitude-↑ irregular respiration	Initial ↓ (100) short recovery period remains subnormal (20)	Heart rate 30 min from 360–300 beats arrythmia	↓2/9	1·20
Gymnothorax javanicus R—745	1260·0	RR-slightly↓ amplitude-↑ irregular± respiration	Initial ↓ to (100) returns to slightly subnormal level	Heart rate 30 min from 360–300 beats	No change	0·05

↓ Indicates decrease ↑ increase in response from normal.

Topical application, 10 per cent dilution.

Source of Specimens: R—Johnston Island; B—Oahu (Hawaii); EPH and EPF—Palmyra Island.

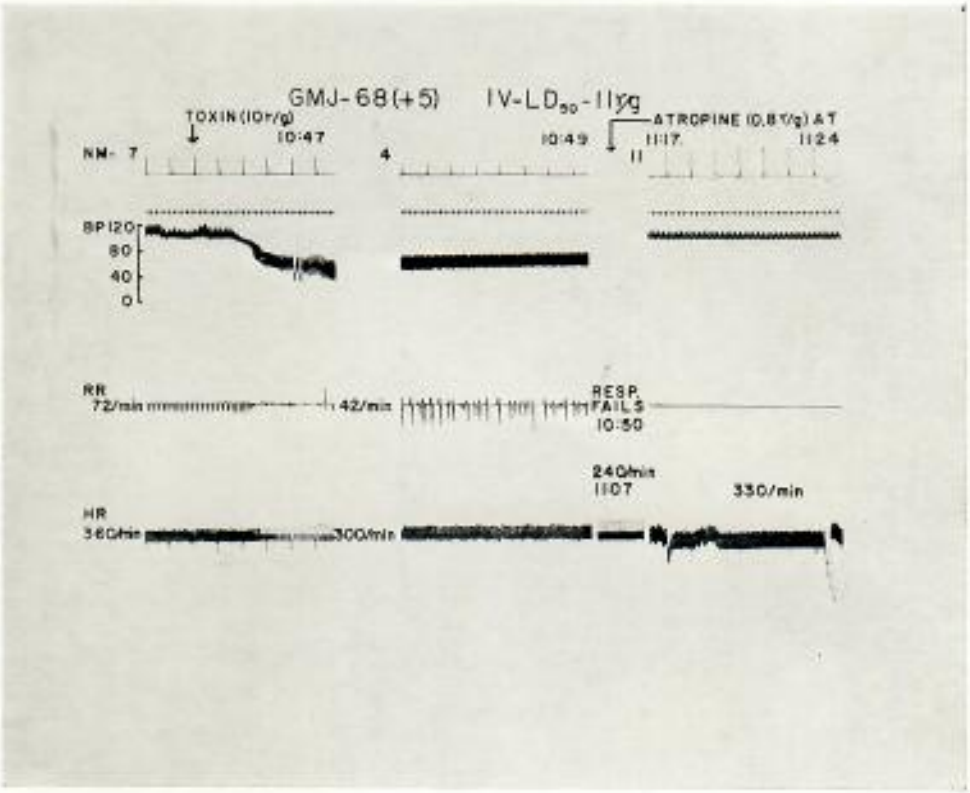

FIG. 1. PHYSIOLOGIC RESPONSES IN THE RAT TO *Gymnothorax javanicus* TOXIN. NM = nerve muscle: height of contraction (mm). Time (sec); BP = blood pressure (mm of mercury); RR = respiratory rate and amplitude; HR = heart rate: electrocardiographic recordings of Lead II.

considered to be a lipid containing a quaternary nitrogen atom, one or more hydroxyl groups, and a cyclopentanone moiety [3]. Extracts were prepared from toxic *Gymnothorax javanicus, Seriola dumerilii, Epinephelus fuscoguttatus,* and *Epinephelus hexagonatus.*

As suspending agents, ethanol, buffered saline 0·9 per cent, and Tween-60 (4·5 per cent) were used. With less soluble extracts serum and saline were satisfactory. Used alone, the diluents were not toxic.

Toxic extracts were injected in rats anesthetized with sodium pentobarbital, 40 mg per kg. Grass polygraph recordings were made *in situ.*

Respiratory rate was measured by conventional methods. When respiration was impaired or stopped, an animal respirator maintained respiration. Arterial blood pressure and electrocardiograms (Lead II) were recorded in the conventional manner. Neuromuscular response was measured by a modification of the method of BROWN *et al* [8] for recording results of indirect stimulation of the sciatic nerve-gastrocnemius preparation *in situ* (at 0·1 mV per 0·2 sec, every 4 sec).

RESULTS

The four most toxic extracts of *Gymnothorax javanicus* (see Table 1) were lethal at doses from 11·8 to 76·6 mg per kg. All depressed respiration, with decreased rate and irregular amplitude to cessation. Prompt fall in arterial pressure, with slow recovery, occurred in 2 of 4 animals (Fig. 1). Bradycardia and arrhythmias developed following administration of 3 of the 4 extracts studied. Neuromuscular response was reduced in all 4 experiments.

In 2 of the toxic extracts studied, from *Seriola dumerilii* and *Gymnothorax javanicus* respectively (lethal at 105 and 168 mg per kg), depressed respiration was followed by cessation; reduced arterial pressure followed by recovery occurred, with bradycardia. The neuromuscular response was reduced by one-fifth or more.

The toxic extracts from *Epinephelus fuscoguttatus* and *E. hexagonatus* (lethal at 438 and 465 mg per kg), caused irregular respiration followed by cessation. Arterial pressure dropped, cardiac rate decreased, and arrhythmias developed. Neuromuscular response was as above.

Drug antagonism studies were made, with special reference to neuromuscular response in 9 specimens tested. It was found that both eserine, 0·2 mg per kg, and atropine, 0·8 mg per kg, antagonized the ciguatoxin(s)-induced neuromuscular block. This would suggest that more than one site of action is involved. There is evidence that ciguatoxin(s) may have a central action and produce ventricular extrasystoles without definite heart block.

DISCUSSION

Toxins of ciguatera-like fish caused damage to the systems examined. There is apparent correlation between pupillary miosis and other pharmacologic activities, notably neuromuscular activity [9]. Preliminary studies of pathologic damage to nerve tissue (K. M. Li, personal communication) indicate that demyelination of peripheral and central nerve tissue may occur. Our findings are in agreement with the *in vitro* studies on toad sciatic nerve-gastrocnemius muscle previously reported [10].

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MARINE TOXINS OF THE PACIFIC—VIII CIGUATOXIN FROM MORAY EEL LIVERS*

TAKESHI YASUMOTO and PAUL J. SCHEUER
Department of Chemistry and Hawaii Institute of Marine Biology,
University of Hawaii, Honolulu, Hawaii, U.S.A.

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Abstract—Moray eels (*Gymnothorax spp.*) from Johnston Island, the Society Islands, Okinawa, and Hawaii were screened for ciguatera by mongoose feeding. The livers of all eels from a ciguateric area and the toxic extracts were assayed by i.p. injection in mice. Comparison of toxicity levels showed that livers of all eels from a ciguateric area are toxic, regardless of the results of the screening test on the eel flesh. It further showed that the toxicity level of the livers is approximately the same from 'nontoxic' (zero rating) and moderately toxic (three rating) eels. The results of the study on Okinawan eels present a special situation, which is discussed.

INTRODUCTION

CIGUATERA is a disease caused by the ingestion of a wide variety of tropical reef fishes. Many aspects of ciguatera fish poisoning have been reviewed by HALSTEAD (1967). The molecular structure of ciguatoxin—the active principle which causes ciguatera—is not known, but an isolation procedure and some chemical and physical characteristics of the toxin were reported by SCHEUER *et al.* (1967). Recent research on the pharmacological action of ciguatoxin was published by RAYNER *et al.* (1968).

Our source of ciguatoxin has been a number of carnivorous reef fishes from Pacific archipelagoes, chiefly the red snapper, *Lutjanus bohar* (Forskål), and the moray eel, *Gymnothorax javanicus* (Bleeker). Yields of homogeneous toxin from fish flesh have been in the range of 1–10 ppm. In a comparison of the toxity of various *L. bohar* tissues, HELFRICH *et al.* (1968) demonstrated that the toxin concentration in the liver is *ca.* 50-fold the toxin concentration in the flesh. In an attempt to utilize this finding we extracted (T. Yoshida, unpublished data) a supply of toxic shark livers [*Carcharhinus menisorrah* (Müller and Henle)], but were unable to isolate homogeneous ciguatoxin from this source, probably because of the presence of certain constituents which promoted toxin degradation during purification.

In spite of these discouraging results we once again extracted a stock of eel livers (*G. javanicus*) and much to our surprise succeeded in isolating homogeneous ciguatoxin. This report describes the results of the isolation of ciguatoxin from eel livers of varying degrees of toxicity from different areas in the Pacific.

MATERIALS AND METHODS

Isolation procedure

Chopped eel liver (150 g) was homogenized with three successive portions of acetone

*Contribution No. 344 from the Hawaii Institute of Marine Biology.

(450, 450, 300 ml) for 2 min each in a Waring Blendor. The combined extracts were cooled to −20° and filtered. The insoluble residue was discarded and the filtrate was concentrated to *ca.* 50 ml. The concentrate was diluted with 150 ml of 90 per cent methanol and extracted with two successive portions (200, 100 ml) of *n*-hexane. The hexane extract was only marginally toxic and was discarded. The methanol phase was concentrated to *ca.* 50 ml and extracted with five 100 ml portions of ether. The bulk of the toxin (*ca.* 175 mg) was in the ether extract. An additional amount of toxin (*ca.* 9 mg) was obtained by extraction of the methanol-water phase with l-butanol and by partitioning of the butanol residue between water and ether. All bioassays were carried out with extracts at this stage of purification. LD_{99} of these extracts was *ca.* 100 mg per kg.

Bioassay

Eels were screened for toxicity by feeding raw eel flesh to mongooses, as described by BANNER *et al.* (1960). Toxicity was rated on a scale of 0 (no symptoms) to 5 (death) based on the observation of the assay animal. The eels from Okinawan waters were similarly screened by Professor Y. Hashimoto and co-workers, but their assay animal was the cat.

Toxic liver extracts were assayed by i.p. injection in mice as described by BANNER *et al.* (1961). For ready comparison, toxicity is expressed in mouse units per g of raw liver (M.U.). A mouse unit is defined as the ratio of toxic extract in mg derived from 1 g of liver to the LD_{99} value of the extract in mg per kg as determined by i.p. injection in mice.

RESULTS

Table 1 summarizes the data obtained from a series of eels, all of the same species, collected in one area, Johnston Island.

TABLE 1. COMPARISON OF *G. javanicus* FLESH TOXICITY ESTABLISHED BY MONGOOSE SCREENING WITH EEL LIVER TOXICITY DETERMINED BY ASSAY OF THE EXTRACT*

Mongoose rating of eel flesh	Toxicity of the liver in M.U.
5	550
4	300
3	130
2	160
1	130
0	130

*All fish collected from Johnston Island.

Table 2 presents the details of a similar study in which the toxicity of eel flesh derived from different species from several areas is compared with the toxicity of their livers. Some of the areas are known to harbor ciguateric fish, while others are not.

DISCUSSION

Table 1 clearly demonstrates that livers of all eels from one area, Johnston Island, are ciguateric. The study further shows that the toxicity level is essentially the same for

TABLE 2. COMPARISON OF EEL FLESH TOXICITY WITH LIVER TOXICITY IN EELS OF VARYING SPECIES AND ORIGIN

Species	Origin	Mongoose rating of eel fish	Toxicity of the liver in M.U.
G. javanicus	Popote Bay, Tahiti	4	500
G. javanicus	Popote Bay, Tahiti	3	500
G. javanicus	Popote Bay, Tahiti	2	80
G. javanicus	Teavaraa Pass, Tahiti	0	70
G. javanicus	Bora Bora	0	120
G. undulatus (Lacépède)	Kaneohe Bay, Hawaii	0	0
G. undulatus (Lacépède)	Okinawa	0*	2
G. flavirmarginatus Råppell	Okinawa	0*	15
G. meleagris Shaw	Okinawa	0*	0

*Cat screening.

eels whose flesh is rated zero or three on the mongoose scale. A zero rating may be the result of no toxicity or of toxicity in such low concentration that no ciguatera signs can be observed. We are inclined to think that flesh of zero eels is indeed marginally toxic, and that we are unable to detect it by mongoose screening.

Table 2 confirms these findings by showing that all eel specimens from the Society Islands (Tahiti and Bora Bora) bear toxic livers and that the level of toxicity falls off as we proceed downward on the mongoose scale. Hawaii, on the other hand, is an area where ciguatera is not known and no toxicity could be detected in the eel livers. The data from the three Okinawa eels belonging to three different species are of particular interest. All three eels were nontoxic by cat screening, and the livers were nontoxic within the limits of the experimental procedure. Ciguatera poisoning is well known in Okinawa, and HASHIMOTO and YASUMOTO (1965) surveyed several genera of fishes among the snappers, groupers, and eels which have had a reputation of producing ciguatera poisoning. The results of their screening (cat) indicated that the largest number of toxic specimens from Okinawa are groupers identified as *Epinephelus fuscoguttatus*. Extraction of a toxic grouper further revealed that the toxin—unlike ciguatoxin—is not extracted into the lipid solvent but remains in the aqueous phase. Hashimoto has named it ciguaterin. The Hashimoto survey, incidentally, produced no toxic eels from Okinawa and none were extracted. In a recent extension of their ciguatera research, HASHIMOTO and co-workers (1969*a*, *b*) extracted specimens of three *Gymnothorax* species and demonstrated the presence of the water soluble ciguaterin in the livers of all three species and in the flesh of *G. flavimarginatus*. No lipid soluble ciguatoxin was detected in the muscle of any of the eels, although its presence was demonstrated in the flesh of red snappers, *Lutjanus bohar*. Since our extraction procedure is designed solely for ciguatoxin, ciguaterin might well have been present but remained undetected. Hashimoto's and our findings are therefore in agreement. The questions which remain unanswered are, *inter alia*, why Okinawan eels elaborate ciguaterin and not ciguatoxin and what is the structural relationship between the two toxins.

Another question which remains obscure is the nature of the relationship between toxin levels in the flesh and liver of the eel. The liver might be the major reservoir of toxin in ciguateric fish or it might be the site where a biological precursor is modified, or it might serve in both capacities.

Acknowledgements—We are indebted to Professor Y. HASHIMOTO for the screened specimens from Okinawa; to Professor A. H. BANNER and Mr. WATARU TAKAHASHI for valuable discussions; to the U.S. Public Health Service for generous financial support through Research Grant UI-00216; and for additional financial support to the National Science Foundation (GF-244), the Atomic Energy Commission (AT(04-3)-235), and the Office of Naval Research (N00014-67-C-0127).

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Health Department Prob

Hawaii's toxic fish problems appear to be getting worse, with an increasing number of people and fish species affected by the poisoning, according to the state Health Department.

However, officials stress that the public should not panic and stop eating fish.

Only one, or none, out of 100 fish might contain what is known as ciguatera toxin, said Dr. Ned Wiebenga, chief of the Health Department's epidemiology branch.

BUT, HE SAID, officials are concerned because of a "significant increase" in the number of toxic cases being reported, from 15 episodes involving 42 persons in 1978 to 21 incidents involving 68 persons so far this year.

"What we don't know is the number of cases that have occurred that are not being reported," Wiebenga said.

"What's bothering us," he continued, "is that in previous years ciguatera was associated with a relatively limited number of species of fish and now, all of a sudden, it is spread across the board.

"We even have suspected cases from eel, which we never heard of before, and we don't know why."

scare-type situation because of lack of information."

He said considerable work is being done on research and surveys to try to answer the questions and alleviate the concern.

Hokama's laboratory has federal grants to study the distribution and chemistry of the toxin and effects on tissues and he is trying to devise techniques to detect the poison "so that, perhaps, we can protect the consumer."

He said more than 800 kahalas have been tested since April and 12 to 14 percent have been ruled out with suspected toxin.

HOWEVER, the research effort is hampered by lack of money, Hokama said.

Wiebenga confirmed this, saying, "They have done a lot of testing we haven't paid for yet."

He said the laboratory needs about $30,000 a year to sustain the testing program and another $90,000 just for research.

"We haven't been able to come up with those resources. . . . Our problem is that we don't have a simple, reliable, economical test to be able to tell somebody whether the fish they have in stock is poisonous or not," Wiebenga said.

produces the toxin that causes ciguatera," he said.

He said the flagellate was first found in the Gambier Islands about four years ago during a ciguatera epidemic there. Banner received a specimen from a Japanese scientist and said he was able to establish that it was producing ciguatoxin.

He said he found flagellate growing spontaneously near the University's marine laboratory on Coconut Island in Kaneohe Bay in January 1978. It was also found off the reef runway and in Pokai Bay.

IT PROBABLY has always been here, he said, but existing in such small numbers that only an occasional fish would become toxic.

But he said a sample tested in the laboratory had "literally billions of these little things. There is no way you can see them with the naked eye unless they are in aggregate."

The laboratory was able to start a culture of the microscopic plants last year and Banner said, "We did have ciguatoxin produced by them."

"Why they suddenly come up in heavy blooms no one knows," he said. "In some cases it seems to be coincidental with acts of

Banner said the ciguatera problem "has been going up and down for years" and that the phenomenon still isn't understood.

HE SPECULATED that the dinoflagellate bloom may result from some sort of subtle chemical change in the water or an increase in trace elements.

He said the same order of flagellates is responsible for a number of other toxins found in various marine foods, or even deaths of animals.

"For example, virtually all Alaskan clams are off the market because of shellfish poisoning caused by the bloom of a dinoflagellate," Banner said.

The famous red tide that killed fish by the millions off Florida was another dinoflagellate, he said. And a third type of the organism caused a number of serious outbreaks off Maine and New England in the past few years.

Banner said the ones in the temperate zones are planktonic — occuring in the open sea — but the type from the Gambier Islands lives on the ocean bottom, attaching itself to certain brown and red algae.

"So, we have the potential, if conditions turn up right, of having

es Fish Poisoning

a serious outbreak," Banner said.

HE NOTED that most cases in Hawaii have occurred at random over the last 10 years or so. A serious outbreak occurred in Haleiwa in the 1960s, resulting in several deaths. But the poisoning is rarely fatal, Banner said.

The most serious effects occur when people eat the viscera of the fish, especially the liver and gonads, where the toxin concentrates. This is what happened in the Haleiwa case.

A number of the recent cases occurred from fish caught in Pokai Bay, although "probably 99 percent of the fish out there are safe," Banner said.

HE SAID THE major concern is with kahala because of its popularity as a table fish and its economic importance.

"We also have to be concerned about mullet because a lot of residents use it," he added.

Kahalas sold through the United Fishing Agency have been screened since April by Yoshitsugi Hokama, pathologist at the University of Hawaii School of Medicine.

Only those considered safe have been released and no kahala poisoning cases have been reported since he began the testing program.

HOKAMA SAID fish poisoning isn't as serious as salmonella or staph poisoning but "the industry is concerned . . . It becomes more of a mystery or

"THE RESULT is that if a market person is prudent, he doesn't sell it any more, and this represents a big economic loss."

In many cases it may be an unncessary loss because the fish may not contain any toxin, he said.

Albert Banner, University zoologist who has studied the fish toxin problem throughout the Pacific for about 30 years, said, "It is getting worse . . . but it is certainly not a major catastrophe. It is a matter of degree."

He said it is believed now that the poisonous fish problem may be related to a microscopic plant animal called dinoflagellate.

"WE BELIEVE, although we haven't absolutely tied it down, that there is one that lives on coral reefs that

man, and in other cases they seem to come up sponatenously without acts of man."

Despite all the pollution and stress in Kaneohe Bay's history, Banner said no ciguatera cases were reported.

BUT HE SAID fish poisoning in the Tuamotu Islands appeared to be "coincidental with marine intrusion" which occurred when the French established an atom staging site on Hao Island.

The Tuamotus never had a case of ciguatera until 18 months after the staging base was built, he said.

Within two years, 45 percent of the population had suffered fish poisoning at least once, he said.

"They tell me today that people no longer are being poisoned out there — they have no cases . . ."

The Marine Advisory Program
MAKAI
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Taape--food bargain by another name?

Follow-up efforts are now being investigated for specific research and education programs to help make taape (blue-lined snapper) a more commercially attractive product for local fishermen and an inflation-answering dish for the consumer.

Concerted effort between the University of Hawaii, the UH Sea Grant College Marine Advisory Program, and state agencies was one recommendation arising out of a public workshop on taape to determine "what needs to be done" with the controversial blue-lined snapper first introduced to island waters in 1955.

Among the recommendations from the workshop were:

- Continuing research into the biology and feeding habits of taape and its effects on native fish
- Educational efforts to build acceptance of taape as a food fish, increase consumption, and encourage the fishermen to catch them
- Promotion of the underutilized fish for home consumption and restaurant use

In a three-hour workshop attended by more than 100 persons at the McCully-Moiliili Library November 7, the taape was described as everything from an "opportunistic predator" and "junk fish" to a boon to Hawaii's housewives. It was said that the taape has spread throughout the island's ocean waters and has even infiltrated Kauai's freshwater streams.

Howard Co of the Department of Planning and Economic Development pointed out that, to some, taape looks like a "pet fish" and that people are reluctant to try a yellow fish with blue stripes. However, he noted that many island people eat octopus, not exactly an attractive seafood.

Speaking in a more serious tone, Frank Farm, president of Alii Holo Kai diving club, said that based on his own experience, he believes the population of native fish preferred by local anglers is declining because of the taape.

However, Henry Sakuda of the State Division of Fish and Game, and University of Hawaii research scientists, said there is still a lack of evidence to show that taape are displacing preferred fish such as menpachi, kumu, and uku. Despite the differences in interpretation of existing counts it was agreed that a way must be found to make the best of the situation. Consumers and fishermen alike agreed that a stronger educational effort—one that is ongoing and widespread—is needed to make the public aware that the taape is a tasty, versatile, and economical fish. After filleting, it can be baked, fried, broiled, boiled, served as pupus, cooked in batter or with any

(Continued on page 6)

Try this fish dish—you'll like it!

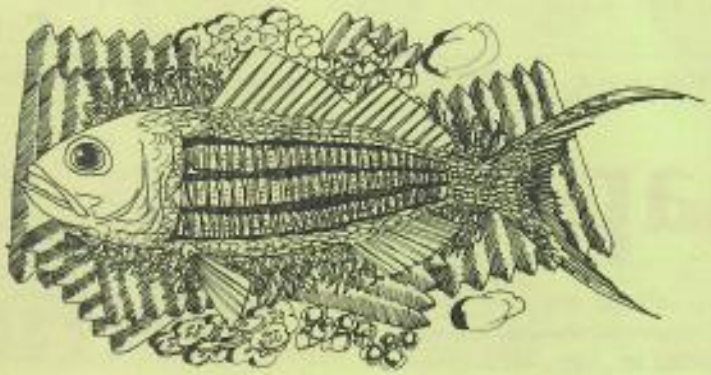
The UH Sea Grant College Marine Advisory Program has available recipes for taape (one copy per request, please), which can be obtained by writing to the program at 2540 Maile Way, 252 Spalding, University of Hawaii, Honolulu, HI 96822. Current retail prices for taape average between $1.40 and $1.80 per pound, a relatively inexpensive price for a meaty fish. Taape can be found in marketplaces and prepared like any other fish—fried, poached, steamed, broiled, and baked.

Some consumers who prefer to eat red and silver fish are reluctant to try the blue-lined, yellow taape. Experience and exposure can change that attitude, however. The recipes included in the MAP brochure include: baked taape with shoyu sauce, taape manauea, crackling taape, baked taape, sweet-sour lemon taape, Vietnamese-style taape, and taape canapes. □

Participants sample taape after the workshop.

Seafood of Hawaii

Onaga, the holiday favorite



Hawaii's unique ocean heritage has made fish the main course on festive holidays here in the islands. The sea has a direct influence on lifestyle, even crossing over ethnic boundaries. The celebration of holidays, especially the New Year, often involves generous settings of raw and cooked fish, seaweeds, and shellfish well known to island families of all backgrounds.

To the traditional Japanese community, fish are considered a symbol of joy, never to be served or eaten when sorrow has befallen a household. In Japan, among the most popular fish served on festive occasions is the sizable, brightly colored tai, a red snapper with a very pronounced belly. It is served both raw and steamed. But, tai is not found in local waters.

However, another red snapper, known locally as onaga *(Etelis carbunculus Cuvier)*, happens to be a perfect replacement for tai, although the latter is now flown in frozen from Japan. The onaga, or in Hawaiian the 'ula'ula, which means "very red" or "scarlet red," was used especially for sacrificial purposes in ancient Hawaii. Other Hawaiian fish species also bear a close resemblance to the onaga, such as the 'opakapaka *(Bowersia violescens)* and the uku *(Aprion virescens)*, but there are few fish that match the social and food value represented by the onaga.

In preparing the onaga, the entire fish is used intact and as fresh as possible to preserve as much of the red coloration. Depending on the occasion, it is served either raw (as sashimi) or steamed, but always after the stomach has been emptied with a deft, invisible cut in the abdomen.

The onaga is served with other holiday foods such as mochi (sweet rice flour balls with a bean filling), sushi (rice cakes), and shrimp tempura. Fresh from the fish market, raw onaga makes for a popular wedding gift. The fish is laid on its side with its mid-section opened and the meat within thinly sliced in three horizontal rows. The finishing touch is a "fishnet" made of stretched daikon (turnip) laid over the dish. But the real bonus is the perfect, moist, creamy-white (or pink) meat under the net.

Long, delicately filamentous caudal fins make the onaga a true show fish. The same raw fish can be served on New Year's or the Japanese holiday, Boy's Day, but without the fishnet. In its place, an elaborate green matting supports a fine daikon mesh, atop which lays the fish. Around it are piled an assortment of flower-shaped carrots, cucumbers, and turnips, and a special green mustard called wasabi.

An excellent soup can be made from the remaining parts of the onaga, or the whole fish can be served steamed in a soy sauce and laid over a bed of somen noodles and ice. Prepared in this manner, a special stand holds the head and tail up above the serving dish (ike-tsukuri) to help make the fish look "alive."

For those interested in a holiday treat, the following recipe is provided by Gasco, Inc.

SAKANA NO NITSUKE
(Fish Cooked in Shoyu)

1/3 cup shoyu
1/2 cup water
1 tablespoon sake or mirin
1/4 teaspoon monosodium glutamate
3 tablespoons sugar
1/2-inch piece ginger, crushed
1 1-1/2 to 2 pound size fish, scaled and cleaned

Combine first 6 ingredients in large saucepan and bring to a boil. Place fish in single layer in sauce, cover and simmer 8-12 minutes or until fish is cooked.

Pour spoonful of sauce over fish to serve and garnish with minced green onion, if desired. Yield: 4 servings. □

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Shorecasting—popular island pastime

Coastal census looks for fun fishers

A Honolulu research firm is completing a survey of Hawaii's fishing population as part of a one-year comprehensive government-sponsored recreational fishing study of Hawaii, Guam, and American Samoa. The survey is funded by the National Marine Fisheries Service (NMFS).

In addition to extensive telephone interviewing, the staff of Survey Marketing Services (SMS) is polling fishermen at piers and launching ramps and along coastlines on the time they spend fishing, fishing methods used, location of sites, size of their catch, and the length, weight, and species of the fish caught.

The information gathered will provide an estimate of total fishing activities by recreational fishermen, total catch by species, and total weight of fish caught.

The Western Pacific Regional Fishery Management Council has contracted with SMS to conduct 1,360 additional field interviews of boat fishermen in the survey areas. The information obtained will provide better catch estimates for offshore species of concern to the council, such as billfish.

The survey began on November 1, 1978 and is being extended for two months through December 31, 1979. The second survey, now being planned by NMFS, will begin in January 1980 and will also include the Mariana Islands. The NMFS study, which is expected to continue for several years, will provide data on recreational fishing in the survey areas for which there is no existing data available at present. □

Ciguater detection

There are few organisms in the ocean—plant or animal— which man cannot eat or touch safely. Of these, the fishes which are poisonous to eat are also limited in number.

One of the varieties of fish poisoning which occurs in Hawaii is "ciguatera."

RESEARCH

The word comes from the Spanish conquistadores in Cuba, who identified an illness apparently caused by eating a marine mollusk, *Turbo pica,* or "cigua." At present, the word ciguatera is used to describe a human disorder that occurs after eating an affected individual which belongs to one of several coral reef fish families, such as jacks, snappers, or surgeonfish. Pelagic fishes and coral reef invertebrates are rarely affected.

Clinical ciguatera symptoms in humans are neurological, gastrointestinal, and cardiovascular. The symptoms include tingling of the lips, mouth, and fingertips; itching of the skin; reversal of temperature sensation (hot feels cold and vice versa); loss of motor ability; vomit-

Boating safety pamphlets on board

The Marine Advisory Program Honolulu office has a limited supply of pamphlets on boating safety including:

- *Emergency Repairs Afloat*
- *Federal Marine Sanitation Device Regulations: Marine Toilets*
- *Federal Requirements for Recreational Boats*
- *Marine Communications for Recreational Boaters*
- *Minimum Equipment Requirements: Navigational Lights for Sailboats*
- *Minimum Equipment Requirements: Personal floatation devices*

The pamphlets may be obtained free of charge (one copy per request, please) from the MAP Resource Center in Spalding Hall 252-B, University of Hawaii, Manoa. The pamphlets are also available from the US Coast Guard and may be obtained by calling Commander Arthur Foster, 14th Coast Guard District Boating Safety Officer, 546-5575. □

From Emergency Repairs Afloat, *Department of Transportation, US Coast Guard, CG-151, October 1977*

a research seeks fast method

Technician at UH pathology laboratory holds snappers (Lutjanus bohar) *from Samoa which were tested for ciguatoxin.*

ing; and diarrhea. The disease is rarely fatal, but can be prolonged and disabling. It tends to recur upon eating seemingly nontoxic fish.

Fortunately, few outbreaks of ciguatera occur in Hawaii. Approximately 20 cases are reported each year. Incidences of ciguatera have been reported to occur from 35 degrees north latitude to 34 degrees south latitude, but island groups closer to the Equator, such as the Society Islands (Tahiti) have a significant number of cases every year.

It is now believed that ciguatoxin, the poisonous component of ciguatera, is not manufactured by the affected fish, but is accumulated through the food eaten by the fish. The toxin occurs in certain species of microscopic marine animals called dinoflagellates, which are found in certain bottom algae eaten by herbivorous reef fish. These fish are later eaten by larger carnivorous fish. The toxin accumulates in the system of the latter fish over time.

Most ciguatoxic fish feed on reef dwellers. The exception are pelagic fishes such as some mackerels, which spend part of their lives in schools close to shore, feeding on benthic fishes and organisms.

The toxin has been thought to accumulate in the larger individuals of fishes such as kahala (amberjack). Federal and state scientists have been studying the kahalas coming into the Honolulu market since April this year and have found that size or weight are not reliable indicators of toxicity.

Poisonous fishes of tropical coral reefs have presented a longtime health problem. No scientific cures such as an anti-toxin have been developed as yet. All that doctors can do at present is to treat the symptoms as they occur. However, certain Pacific island peoples use local remedies which are just now being studied by western science.

Ciguatera is the most common form of tropical fish poisoning. Fortunately, it is relatively rare in Hawaii. Last year there was a case on the Big Island in which several individuals were affected from eating po'ou *(Cheilinus rhodochrous)* caught in the Kawaihae area.

After gradually eating quantities of the toxin-bearing algae, the fish becomes toxic. The viscera, and especially the liver of reef fishes, should not be eaten, according to medical authorities. The least toxic part of a fish is the muscle tissue (flesh). Cooking has no apparent effect on the toxin, nor does washing.

The problem is complicated by the unpredictability of the occurrence of ciguatera. Some individuals of a species of fish on certain reefs will be poisonous, while others will not be. In some island groups nearer to the Equator, all fish on certain reefs are apparently affected, while fish on adjacent reefs are not. Ciguatoxic fish cannot be identified by sight.

At the University of Hawaii, zoology professor Dr. Albert Banner is working on an enzyme-linked colormetric test designed to be used in the field. Chemical processes involved in the test would show the presence of ciguatoxin by causing a color change in the testing compound. This method remains to be perfected.

Current technology requires that a sample of fish be sent for analysis to the University of Hawaii pathology laboratory in Honolulu, headed by Dr. Yoshitsugi Hokama.

Core samples of kahala tissue are taken in the early morning by National Marine Fisheries Service scientists from three locations in each fish brought to the Oahu fish market. The laboratory scientists use a radioimmunology assay test to reveal ciguatoxin levels in the samples, and, by the afternoon of the same day, can inform the fish market of its approval or rejection of the sampled fish. Since the testing process began this April, the lab has analyzed tissue from 750 kahala, rejecting an average 14 percent as unsafe. Of the remaining 86 percent sold, not a single case of ciguatera has resulted.

The lab will be studying tissue samples for one year to determine whether a cycle exists in which higher incidences of toxic levels in kahala are found. So far, the study has shown that toxicity is not related to size or weight of the fish. Rather, it may be that other factors are involved, such as environmental conditions or cyclical fish metabolism.

With increasing amounts of data now being collected by numerous study efforts on ciguatera, by the end of the study period, much old data may be invalidated. More importantly, with better understanding of this form of fish poisoning, controls and methods of prevention can be developed.

Now the problem basically rests with sports fishermen, who catch fish—particularly ulua—which do not get tested by the UH pathology laboratory. Fishermen should be aware of what areas have generated cases of toxic poisoning, such as near the reef runway or off Barbers Point on Oahu. Fishermen who are uncertain if the fish has ciguatoxin should call the Board of Health. The fish should be kept frozen until the UH laboratory can test samples of the tissue. *[Reprinted from the "Marine Advisor,"* West Hawaii Today.*]* □

MARINE MISCELLANY

The first issue of the new *Sea Grant Quarterly*, devoted to an analysis of "Whaling: Native Needs vs. Moratorium," has been published. Future issues of the *Sea Grant Quarterly* will present technical reports on the results of research projects conducted with Sea Grant funding and will also include scientific articles of interest to a technical audience. Editor **Rose Pfund** is requesting that persons interested in receiving the *Sea Grant Quarterly* send in their name and address to the UH Sea Grant College Program by December 31, 1979, to be included in the mailing list.

* * *

"Birds of Rabbit Island" is the title of an article featured in the October issue of *Sea Frontiers* magazine, written by **G. Causey Whittow**, professor of physiology at the UH School of Medicine. Whittow's research concerns the environmental physiology of marine mammals and oceanic birds. Whittow has done research in various parts of the world, including Scotland, Malaysia, and also Rabbit Island.

Rabbit Island

* * *

Scuba divers and medical personnel attended a free seminar on diving medicine held in Kona last month, sponsored by the UH Sea Grant College Marine Advisory Program and the State Office of the Marine Affairs Coordinator. Prevention of diving accidents and ways to enhance the enjoyment of the sport were the topics of the discussions, presented by a panel of diving physicians including **Dr. Ken Kizer, Dr. Jon Pegg, Dr. Jeffrey McDevitt, and Dr. Frank Ferren.**

* * *

Kauai's new recompression chamber staff, members of the Kauai Underwater Association, got some first-hand experience in treating diving decompression sickness in late October. The volunteer crews, freshly trained by **Dr. Ed Beckman**, physiology professor at the UH School of Medicine, and **Ed Hayashi**, diving safety officer, were just let out of class, when two Kauai divers having bends symptoms were brought in by ambulance attendants. The teams were quickly recalled to the Kauai Veterans Memorial Hospital and, supervised by Beckman and Hayashi and by **Dr. Robert Overlock**, supervisor of hyperbaric medicine, brought the divers through the recompression chamber treatment. The chamber functioned perfectly and the two divers were out in under five hours. One diver's pain returned later and he returned for another five-hour treatment the next day. Both divers were reported in good condition after treatment. The chamber was purchased and installed with funds obtained principally from the State Office of the Marine Affairs Coordinator.

* * *

"Na Wahine O Ke Kai," the first official Molokai to Oahu canoe race for women, took place October 14, with nearly 20 crews competing. The first crew to cross the finishing line off the Fort DeRussy beach was the Outrigger Canoe Club.

* * *

The first federal loan guarantee for Hawaii's developing aquaculture industry has been granted. Lowe Aquafarm of Kahuku has qualified to receive a $2 million line of credit from City Bank of Honolulu, authorized by the Farmers Home Administration of the US Department of Agriculture. Lowe Aquafarm plans to construct and operate a 160-acre farm to produce and market Malaysian prawns. It also expects to expand its workforce as production and marketing expand.

* * *

The Innerspace Pacifica Film Festival, which was to be held in Honolulu November 3, was cancelled after our press date due to the UPW strike. Apologies to *Makai Newsletter* readers who turned out, only be turned away.

* * *

Noted Hawaii botanist, **Dr. Otto Degener**, wrote in to correct an item in the October *Marine Advisory Program Newsletter* (now known as *Makai Newsletter*) in which the latest incidence of Portunid crabs washing ashore was given as 1930 on Oahu. Degener noted that a similar instance occurred off the beach at Mokuleia in 1977, in which megalops of the same Portunus family washed ashore in great numbers. This August, millions of tiny red Portunid crabs washed ashore on Kauai beaches. It is still not known why this has occurred. □

A guide to Hawaiian limu

"Seaweeds of Hawaii" is the title of a soon-to-be-published, easy-to-use identification guide to the seaweeds of the Hawaiian islands. Color photographs of 118 species taken in their natural habitats and short, non-technical descriptions are included to help limu (seaweed) eaters, beachcombers, scuba divers, students, teachers, and scientists alike to identify the common Hawaiian seaweeds. In addition to the photographs, there are informative chapters describing the ecology

EDUCATION

and reproduction of seaweeds, how to collect and press them for use as stationery or decoration, and how to prepare them in Hawaiian, Japanese, and Filipino recipes. Readers will also find Hawaiian names for the common edible species as well as scientific names for all 118 species.

The book, written by William H. Magruder and Jeffrey W. Hunt, is being published by the Oriental Publishing Co. Magruder is a marine biologist working on a PhD in marine seaweeds at the Hopkins Marine Station of Stanford University. Hunt is an instructor of marine biology and botany at the Windward Community College and coordinator of the Windward Community College Marine Option Program. He is working on a PhD on coralline algae at the University of Hawaii. □

Taape *(Continued from page 1)*

number of sauces, or served with limu.

Increased consumption, according to one of the panelists of the taape workshop, will eventually bring the price up, making taape catches more worthwhile to commercial fishermen. Increased fishing pressure may also trim down the numbers of taape.

Workshop panelists said all of these actions will require imaginative management, and studies will be needed of market economics, consumer psychology, and basic biology of the taape to understand its feeding habits, growth, and reproduction as well as its interaction with other fish and marine life. Future articles in the *Makai Newsletter* will report on the progress of such efforts. □

Marine world in sight and sound

Feel like raising prawns? Or learning a marine skill?

Want to catch aku with topminnow as bait?

Want to learn about identifying Hawaiian coastal plants? Or what precious corals abound in the deep?

Interested in knowing how ancient Hawaiians fished in harmony with the sea? Or what are some modern conservation principles?

Want to know about beach safety on Oahu, or about whales and dolphins? What about fishery potential in the Leeward Hawaiian islands?

If your answer is "yes!" to any of these questions, the UH Sea Grant College Marine Advisory Program has a slideshow or videotape for you. Audiovisual productions may be borrowed without charge by groups or individuals, or they may be purchased. For information, write to the UH Sea Grant College Marine Advisory Program, 2540 Maile Way, 252 Spalding, Honolulu, Hawaii 96822, or call the local offices: 948-8191 (Oahu); 244-4157 (Maui); 245-4471 (Kauai); 935-3830 (Hilo); 322-2577 (Kona).

The following titles are available:

Slideshows

- Aquaculture in Hawaii
- Getting Started with Prawns
- Marine Technology Program (Palau)
- Topminnows for Aku Fishing (16 mm film)
- Coastal Plants of Hawaii
- Ka'ena, O'ahu
- Precious Corals of Hawaii
- Ancient Hawai'i in Harmony
- I'a of Hawaii: Present Day Kapu
- Beach Safety on O'ahu
- UH Sea Grant College Marine Advisory Program 1979-80
- Aloha Tower Marine Information Education Center
- UH Sea Grant College Program Summary 1979
- Whales and Dolphins
- Leeward Islands
- Ecology of Coral Reef Fishes
- Kaloko, O'ahu

Videotapes

- Whales and Dolphins
- Beach Safety on O'ahu

Makai Newsletter
University of Hawaii Sea Grant College
Marine Advisory Program
2540 Maile Way, Spalding Hall 252B
Honolulu, Hawaii 96822

Application to mail at second-class postage rates pending at Honolulu, Hawaii

GEORGE H. BALAZS
JR. MARINE BIOLOGIST
HIMB
COCONUT ISLAND

...LLETIN, MONDAY, AUGUST 2, 1948

...Back at Annexation ...emony

Was Simple

...Julia never saw anything like this before."

* * *

President and Mrs. Dole presided at a state reception which began at 9 p. m. in the throne room of Iolani palace.

For an hour and a half, a line of people passed by to shake hands with the official party including Adm. Miller and Minister Sewall.

NEXT: The first congressional party.

REAR ADM. JOSEPH N. MILLER was commissioned by President McKinley as commander of the USS Philadelphia to officially raise the flag of the United States over Iolani palace. Adm. Miller arrived [illegible] published in the August 12, 1898 issue of the Pacific Commercial Advertiser.

Dumping of Ammunition In Ocean Seen as Cause of Poisonous Fish

The territorial health board has received a new explanation for the fact that fish from certain lagoons in the south Pacific are poisonous.

The explanation comes from Dr. S. K. Ross, a physician who kept careful records of fish poisonings on Fanning island for two years.

In a report received by the health department, Dr. Ross claims that the fish are unfit for human food because they feed on small floating plants permeated with poisonous metallic salts.

* * *

The metallic salts come, he says, from deteriorating ammunition which was dumped in the ocean at the beginning of the war.

Dr. Ross records 95 cases in which residents of Fanning island became ill after eating fish caught near an area where ammunition had dumped.

* * *

He claims that no illness from eating poisonous fish had been reported before 1946, when the ammunition was dropped into the sea.

The most recent case of fish poisoning in Honolulu was reported in April when persons became ill after eating fish given to them by crew members of a fishing vessel. The fish was not part of the commercial catch.

The health department has received no reports of food poisoning from commercial catches.

The Amazon received its name from a tribe of female warriors who were said to live on its banks.

Camels can travel three or four days in the desert without suffering hunger or thirst.

Coast Guard Day

WASHINGTON, Aug. 2 (AP)—President Truman has proclaimed August 4 as Coast Guard Day in recognition of the 158th anniversary of the revenue cutter service, which later became the coast guard.

* * *

"The services of the coast guard are invaluable to the people of this nation," Mr. Truman said, "and the work of its personnel is a vital contribution to the strength and stability of our economy and our military reserve power."

...Back at Annexation

...remony Was Simple

...AYLOR ... of 17 ... ve made ... the annexa... August 1... ppointed. Dole, Rear ...d Minister ...ed that the ...hort and ... making, no ... fireworks ...per, critical ... by Presi... m for the ... nsideration ... Hawaiian

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... of accep... sentences ... nal guard ... waiian flag ... vall Ponc... salute ...

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"The services of the coast ... are invaluable to the ... this nation," Mr. Truman ... "and the work of its ... a vital contribution to the ... and stability of our ... our military ...

US-B 2 AUG 1948 : 4

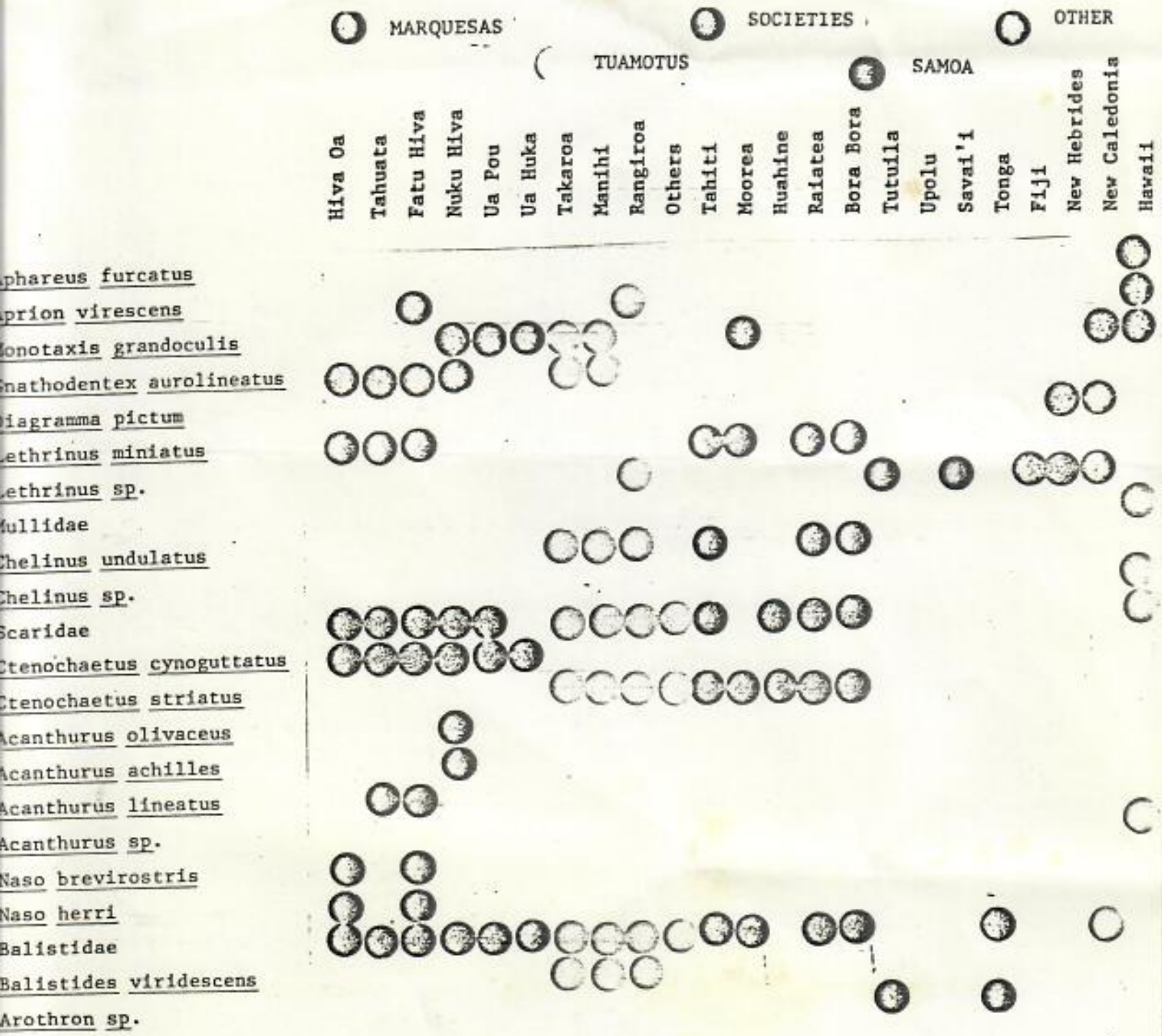
23 JAN 1981 Samoa - Nancy Lewis

ASSOCIATED WITH "Change in the marine environment"

LOCATIONS OF COMMONLY CIGUATOXIC SPECIES

MARQUESAS | TUAMOTUS | SOCIETIES | SAMOA | OTHER

SPECIES	Hiva Oa	Tahuata	Fatu Hiva	Nuku Hiva	Ua Pou	Ua Huka	Takaroa	Manihi	Rangiroa	Others	Tahiti	Moorea	Huahine	Raiatea	Bora Bora	Tutuila	Upolu	Savai'i	Tonga	Fiji	New Hebrides	New Caledonia	Hawaii
Carcharinidae																●	●	●	●				
Clupeidae																●	●	●	●	●	●	●	
Gymnothorax sp.	○	○	○	○	○	○	○				●				●	●	●	●	●	●	●	●	
Leptocephalidae																							
Sphyraena barracuda	●	●	○	○	○	○	○	○	○	○	○		●			●	●	●	●	●	●	●	
Sphyraena fosteri							○									●	●		●				
Gymnosarda unicolor		○	○																○	○	○		
Mugilidae																							
Scombermorus commersoni																				○	○		
Caranx sp.		●					○		○		●	●	●	●	●				○	○	○	○	
Seriola dumerlii																						○	
Plectropoma maculatum																					○		
Plectropoma leopardus							○	○	○	○	○		○	○									
Variola louti		●											●							○	○		
Serranidae																●	●						
Cephalopholis argus	●	○	○				○				○	○	○							○	○		
Epinephelus tauvina	●	○					○			○	○	○											
Epinephelus merra	●	○											○							○			
Epinephelus microdon											○									○	○		
Epinephelus fasciatus	○																						
Epinephelus sp.																●			●		○		
Lutjanus monostigma		○	○				○				○	○	○	○	○			○					
Lutjanus bohar	●	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○		
Lutjanus rivulatus											○							○	○				
Lutjanus argentimaculatus																●			○	○			
Lutjanus gibbus	●	○	○	○	○		○				○	○	○		○	○				○	○		
Lutjanus fluviflamma																					○		
Lutjanus sp.																							


MARQUESAS
TUAMOTUS
SOCIETIES
SAMOA
OTHER
Hiva Oa
Tahuata
Fatu Hiva
Nuku Hiva
Ua Pou
Ua Huka
Takaroa
Manihi
Rangiroa
Others
Tahiti
Moorea
Huahine
Raiatea
Bora Bora
Tutuila
Upolu
Savai'i
Tonga
Fiji
New Hebrides
New Caledonia
Hawaii
Aphareus furcatus
Aprion virescens
Monotaxis grandoculis
Gnathodentex aurolineatus
Diagramma pictum
Lethrinus miniatus
Lethrinus sp.
Mullidae
Chelinus undulatus
Chelinus sp.
Scaridae
Ctenochaetus cynoguttatus
Ctenochaetus striatus
Acanthurus olivaceus
Acanthurus achilles
Acanthurus lineatus
Acanthurus sp.
Naso brevirostris
Naso herri
Balistidae
Balistides viridescens
Arothron sp.

FAMLIES RESPONSIBLE FOR CIGUATOXIC EPISODES (%)

	Marquesas		Society Is.	Am. Samoa	Fiji
	1973	1977	1977	1970-5	1974-5
Lamniformes	0	0	0	19.0%	0
Clupeidae	0	0	0	0	1.1%
Muraenidae	0	0	.6%	4.0%	0
Holocentridae	0	0	0	1.0%	0
Sphyraenidae (cheap)	0	6.0%	.6%	9.0%	40.5%
Mugilidae	1.9%	0	5.2%	0	1.1%
Scombroidei	.09%	3.0%	.3%	0	4.9%
Carangidae	5.1%	3.0%	10.7%	2.0%	3.2%
Serranidae	16.0%	0	10.1%	6.0%	3.8%
Lutjanidae	26.6%	37.0%	9.8%	31.0%	40.7%
Lethrininae	10.5%	10.0%	9.3%	3.0%	3.7%
Labridae	0	0	2.9%	0	0
Scaridae	13.6%	13.0%	16.5%	0	0
Acanthuridae	14.1%	10.0%	30.1%	0	0
Tetrodontidae	0	0	.6%	0	0
Balastidae	.09%	3.0%	3.5%	0	0
Misc.	12.02%	15.0%	.3%	25.0%	1.0%

CIGUATERA MORBIDITY RATE AS REPORTED TO S.P.C. 1973-1977

Territory	'73	'74	'75	'76	'77
American Samoa (31,500)	.18	-	-	-	-
Cook Islands (18,500)	-	-	-	-	-
Fiji (607,000)	.01	.05	.22		.12
French Polynesia (141,000)	4.94	6.72	5.69		3.66
Gilbert Islands (56,000)	2.58	3.33	2.34		.77
Guam (90,000)	.05	-	.22		.07
Nauru (7,000)	-	-	-		-
New Caledonia (138,000)	-	1.53	4.07		3.58
New Hebrides (101,500)	-	-	.10		.50
Niue (3,700)	2.95	.25	-		-
Norfolk Island (1,900)	-	-	-		-
Papua New Guinea (2,990,000)	-	-	.006		-
Pitcairn Island (100)	-	-	-		-
Solomon Islands (214,000)	.01	.04	-		.03
Tokelau (1,609)	-	-	-		-
Tonga (93,000)	.08	.66	.16		.48
Trust Territory of the Pacific Islands (133,000)	2.06	2.24	2.07		2.53
Tuvalu (7,400)	-	-	-		5.87
Wallis & Futuna (10,000)	-	-	-		-
Western Samoa (153,000)	.45	.60	.13		.53

FISH CONSUMPTION STATISTICS

(kg./person/year)

COUNTRY	TOTAL	LOCAL CATCH	IMPORTED
Japan	74.7	-	-
Norway	47.2	-	-
U.S.A.(all States)	5.8	-	-
Hawaii	10.3	-	-
French Polynesia	28.9	21.9	7.0
Rarotonga	13.3	2.5	10.8
Aitutaki	56.0	45.2	10.8
Western Samoa	16.4	7.1	9.3
American Samoa	67.3	11.4	55.9
Tonga	13.0	11.7	1.3
Fiji	24.8	8.3	16.5

FISH IMPORTS 1977

Country	Exports ($A'000)	Imports ($A'000)	Food/% Imports	Fish/% Imports
Am. Samoa	70,636	47,776	16.2%	1.6%
Cook Islands	2,093	15,348	21.3%	1.7%
Fiji	144,300	275,451	19.2%	2.6%
New Hebrides	28,750	35,744	15.2%	1.1%
New Caledonia	280,795	262,954	16.3%	.8%
French Polynesia	19,303	259,586	17.5%	1.0%
Tonga	6,207	17,697	26.8%	.5%
Western Samoa	13,321	37,088	27.7%	2.2%

CIGUATERA MORBIDITY IN TAHITI 1976

267 cases reported

3 mortalities

5% hospitalizations

ORIGIN OF FISH:

30% fish caught in Tahiti
52% fish caught outside of Tahiti
18% unknown origin

OF THE 52% OF FISH CAUGHT OUTSIDE OF TAHITI:

73% TUAMOTUS
21% MOOREA
6% UNKNOWN

PLACE OF PURCHASE:

65% Municipal Marketplace
25% Shops
10% Resturants

Table 4 : Total and Per Capita Trade, 1977[1]

Country	Trade Total ($A'000) Exports	Imports	Balance of Trade	Per Capita Trade ($A) Exports	Imports	Balance
American Samoa	70,636	47,776	22,860	2,242	1,517	725
Cook Islands	2,093	15,348	-13,255	113	830	-717
Fiji	144,300	275,451	-131,151	238	454	-216
French Polynesia[2]	19,303	259,586	-240,283	137	1,841	-1,704
Gilbert Islands	18,212	11,693	6,519	325	209	116
Guam[3]	25,180	267,592	-242,412	252	2,674	-2,422
Nauru[3]	37,324	13,834	23,490	5,332	1,976	3,356
New Caledonia	280,795	262,954	17,841	2,035	1,906	129
New Hebrides[4]	28,750	35,744	-6,994	283	352	-69
Niue	223	1,850	-1,627	60	500	-440
Norfolk Island	801	6,926	-6,125	422	3,645	-3,223
Papua New Guinea[4]	548,835	492,648	56,187	184	165	19
Solomon Islands	29,614	25,753	3,861	138	120	18
Tonga	6,207	17,697	-11,490	67	190	-123
Trust Territory of the Pacific Islands[5]	10,528	34,947	-24,419	79	376	-297
Tuvalu	n.a.	1,237	n.a.	n.a.	167	n.a.
Western Samoa	13,321	37,088	-23,767	87	242	-155
South Pacific Region[6] Total						
1970	430,000	835,000	-405,000	109	211	-102
1971	465,000	930,000	-465,000	115	230	-115
1972	540,000	940,000	-400,000	130	226	- 96
1973	720,000	960,000	-240,000	169	225	- 56
1974	1,040,000	1,240,000	-200,000	239	284	- 45
1975	980,000	1,470,000	-490,000	220	329	-109
1976	990,000	1,450,000	-460,000	216	317	-101
1977	1,274,000	1,805,000	-531,000	270	383	-113

From: South Pacific Commission (1979) South Pacific Economies: Statistical Summary, Occasional Paper No. 15, Noumea, 1979.

In a given species toxicity often depends on the size and weight of the fish. In some places, fishermen systematically discard groupers, merous, *C. undulatus*, barracudas and sea perches over 60 centimetres. Jacks are often considered dangerous for human consumption when they weigh more than six or seven kilos. The increase in toxicity with size is not as evident in surgeon fish, tetrodons, *clupeidae* and parrot fish, amongst others.

For a given fish, the viscera (the intestine, the nervous system, the ovaries, the liver) are often more harmful than the muscle, but the degree of toxicity varies according to species. In moray eels, groupers, merous and other trigger fish, the liver, in particular, is far more rich in poison than the muscle. The ovaries of tetrodons and similar fish have a highly dangerous concentration of toxins.

Lastly, all individuals of one species are not necessarily or equally toxic; but in a given batch of fish originating from any one place, the percentage of specimens harmful to man is not constant.

THE SEASON

During the spawning period, at the time of maximum gonad activity, the concentration of toxins seems to be greatest in the ovaries and the liver of tetrodons. This period of maximum sexual activity occurs in May and June in the South Japan islands. In the Pacific, with a few exceptions, spawning takes place in November/December.

Outbreaks of clupeoid poisoning are also seasonal. In Fiji, outbreaks occur mostly during the hot season when the shoals of sardines, herrings or mackerel come close to the coast in search of food.

In Polynesia, ciguatera outbreaks caused by various species occur throughout the year. Though surgeon fish are reputed locally to have a seasonal toxicity, this is not borne out by statistics.

THE TIME FACTOR

Fluctuations in intensity in time show clearly the cyclic character of the phenomenon and its evolution by periodic outbreaks whose frequency and seriousness vary.

Thus the endemic rate seems to have decreased during the last 20 or 30 years in some islands; it seems to be increasing in others, and to be stationary elsewhere.

THE HUMAN IMPACT

In some islands and atolls, outbreaks of ichthyosarcotoxism broke out or occurred again two or three years after a start was made on major works such as dredging, improvement of harbour channels, breakwater building, dumping of materials and where sewers are discharged into the sea. In some areas, there was a definite causation relationship between the incidence of fish poisoning and the increase in human interference with the marine environment. In other areas, ciguateric endemicity was not influenced.

THE SPECIES OF FISH IN AN AREA AT A GIVEN MOMENT OF THE EVOLUTION OF THE OUTBREAK

In a given area surgeon fishes and parrot fishes are the first to be affected by the ciguateric phenomenon. No carnivorous species is affected for at least several weeks. They become gradually toxic when a sufficient quantity of crude toxic product accumulates in their liver and muscles.

Yet, at a given time, in a given place, any species of benthic fish, herbivorous or carnivorous, may become a vector of ciguateric poisoning.

PREPARATION OF THE FISH FOR EATING

Cooking affects fish toxicity most when cooking tetrodon. Most cases of fish poisoning occur after ingestion of a fish soup of puffer fish (muscle, viscera and skin).

FISH POISONING
IN THE
SOUTH PACIFIC

L'ICHTYOSARCOTOXISME
DANS LE
PACIFIQUE SUD

by

par le

Dr R. Bagnis
Médecin de 2ème classe
du Service de santé des Armées

SOUTH PACIFIC COMMISSION
NOUMEA, NEW CALEDONIA

COMMISSION DU PACIFIQUE SUD
NOUMEA, NOUVELLE-CALEDONIE

672

CYTOPLASMIC INCOMPATIBILITY IN INTRASPECIFIC CELLULAR FUSIONS OF THE FILAMENTOUS RED ALGA, GRIFFITHSIA PACIFICA Kyl. D. J. Koslowsky* and S. D. Waaland. Univ. of Washington, Seattle.

Somatic cellular fusion between two different isolates of Griffithsia pacifica Kyl. results in a cytoplasmic incompatibility reaction. The reaction begins in the fusion cell and travels from cell to cell along the filament. It is characterized by a rounding and agglutination of chloroplasts into long catenulate patterns. Subsequent chloroplast fusions result in a greater than 50% decrease in chloroplast number by the eighth hour after the start of the reaction. Total clearing of the cells occurs in three to five weeks. This report is a cytological characterization of the reaction at the light and ultrastructural levels.

673

FEMALE GAMETOGENESIS IN CYSTOSEIRA AND HESPEROPHYCUS (FUCALES, PHAEOPHYCOPHYTA). R.S. SETZER, UNIV. SO. CALIFORNIA, L.A.

I re-examined nuclear distribution ("nuclear extrusion") in oogonia of C. osmundacea and H. harveyanus. Hesperophycus oogonia behave mostly as previously described. In Cystoseira, after oogonial release, a large egg and 7 small cells are retained by a mucilaginous stalk on the receptacle, where fertilization of the egg and early germling development occur. The 7 small cells, previously known as "extruded nuclei" contain a nucleus, golgi, mitochondria, and vesicles, but no plastids. The cells persist with the germling within the mucilage, but their contents degenerate. In Hesperophycus the deposition of wall layers coincident with nuclear division is interpreted in support that the Fucales possess an alternation of heteromorphic generations. The exochite of the "oogonium" is interpreted to represent a megasporangium wall. The mesochite is thought to represent coalesced walls of four meiospores; consistent release of fucoid reproductive bodies in mucilage of mesochite origin suggests a delayed spore release. At the 8-nucleate stage, formation of endochite and walls partitioning the eggs, a 2-step process, may represent the formation of gametophyte and gametangial walls.

674

REPRODUCTION OF AGGREGATIVE VALONIA SPECIES IN HAWAII. P. K. Reese. Jackson Estuarine Laboratory, Durham, N. H.

Aggregatious (clump-forming) Valonia species are predominant and conspicuous algae in intertidal and shallow subtidal habitats in Hawaii. Relative frequency of swarmer formation, one aspect of the reproductive biology, was quantified for a population in Waikiki. Collections were made over an 11 month period. The number of cells becoming reproductive during a 2 week observation period was recorded. On two occasions the population was surveyed for a 24 hour period. In the laboratory swarmers were produced within 48 hours of collection and during early morning hours. In the field, reproduction took place during daylight hours for the two instances in which it was detected. Reproductive cells, although a small percentage of the total number of cells, were observed in all but 5 of the 37 collections. The potential to form swarmers is present throughout the year but is realized in relatively few cells at any one time.

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GROWTH AND TOXICITY OF THE DINOFLAGELLATE GAMBIERDISCUS TOXICUS FROM HAWAIIAN WATERS. N. W. Withers, R. H. York* and A. H. Banner*. University of Hawaii, Honolulu.

An investigation of the physical and chemical parameters which affect growth rates and toxin production in laboratory cultures of the Kaneohe Bay isolate of the toxigenic dinoflagellate Gambierdiscus toxicus Adachi and Fukuyo revealed that the organism is photoautotrophic. An optimum growth rate (1.2 divisions/day) occurred in f medium with 100% seawater. Media supplemented with aqueous extracts of a mixture of macroalgae (common substrates for G. toxicus) enhanced yields (cells/ml) 20-fold that of control cultures at peak population density. Of the ten species tested, extracts of Acanthopora spicifera (Vahl) Boergensen, and Sargassum polyphyllum J. Agardh gave greatest enhancement. Mouse intraperitoneal injections of G. toxicus extracts revealed three toxins, identified as ciguatoxin, maitotoxin, and an unidentified "acetone-soluble" toxin, based on molecular polarity and characteristic symptomology. The levels of these toxins varied greatly in the cultures tested.

Gambierdiscus toxicus, *a dinoflagellate implicated in ciguatera fish poisoning.*

New tests seek out seafood toxins

According to an old adage, a diner does well to avoid seafood during months lacking "r," when humans are most apt to fall prey to the chemical warfare of the sea. Last week, at the meeting of the American Chemical Society in Washington, D.C., government officials joined scientists to discuss the newest findings in the distribution and chemistry of seafood toxins. In particular, two researchers announced the development of kits that commercial and recreational fishermen could use to test seafood for toxins at the site of the catch.

The main plagues of the seafood industry — ciguatera fish poisoning and paralytic seafood poisoning (PSP)—stem from dinoflagellates, single-celled organisms that make up the dreaded red tide. Summer upwellings bring nutrients to the ocean's surface, providing a fertile home for the tiny poison-producers, upon which bony fish and shellfish feed.

Though many marine organisms can eat and accumulate the toxins without ill effect, humans are not so lucky. Both ciguatoxin and saxitoxin (the main poison implicated in PSP) interrupt transmission of nerve impulses by keeping sodium ions from moving through cell membranes.

Edward P. Ragelis of the Food and Drug Administration (FDA) estimates that every year, between 10,000 and 50,000 fish eaters get ciguatera, an often chronic, occasionally fatal disease characterized by diarrhea and temperature reversal (patients perceive cold as hot and vice versa). PSP, while less common, is more deadly; its victims suffer usually fatal paralysis. Both toxins lack effective antidotes.

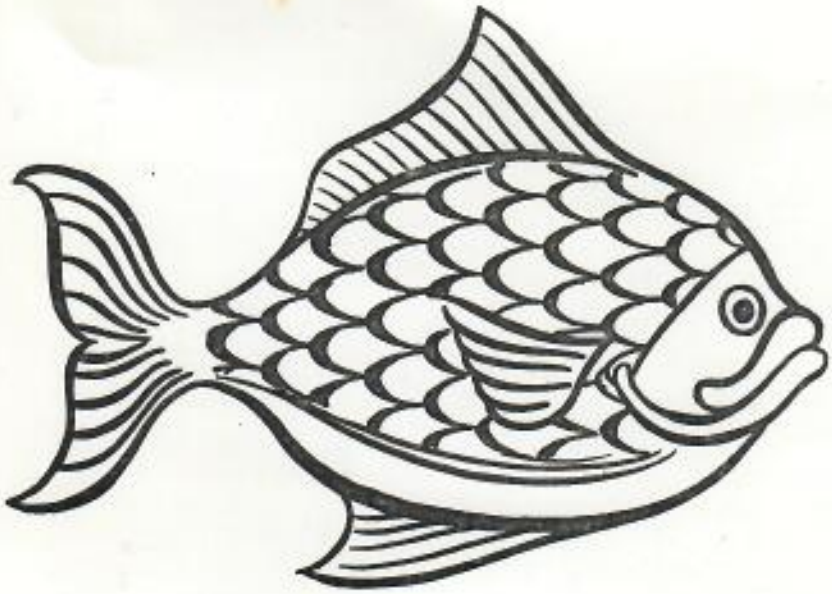
Currently, the only way to prevent harvesting of poisonous seafood is to close catch areas seasonally or in response to an outbreak of a toxin-related disease. "Ciguatera and PSP are major impediments to the development of the U.S. fishing industry," says Ragelis. "They also present a serious threat to communities on small isolated islands, where people depend on seafood for protein and bulk."

But quarantines do not prevent contaminated seafood from being caught. Improved methods of preserving and transporting foods have turned the problem of seafood toxicity into an epidemiologist's nightmare, and precipitated complicated legal liability battles. "It's like not having a traffic light at a dangerous intersection," says Ragelis. "We have no way to monitor the harvesting of toxic seafood, so people just keep getting hit."

But now, using the relatively new technique of enzyme immunoassay (EIA), two scientists are creating methods to screen seafood for toxins as it is caught. "We're working with the FDA to develop a poke-stick for fish," says Yoshitsugi Hokama, a ciguatera researcher at the University of Hawaii in Honolulu. "Basically, a fisherman could poke a fish and dip the stick in a series of reagents. If the stick turns blue, you don't eat the fish." Meanwhile, Patrick E. Guire of Bio-Metric Systems, Inc. in Eden Prairie, Minn., has begun field-testing a dip stick that works on a similar principle to detect saxitoxin in pulverized samples of shellfish.

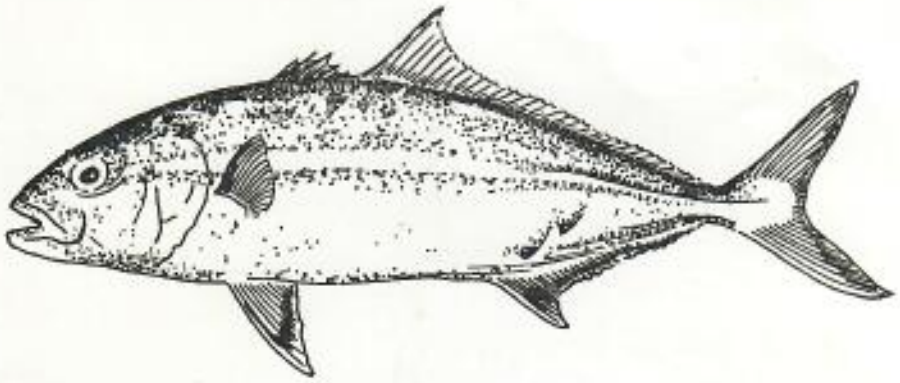
EIA utilizes enzymes and monoclonal antibodies, which react with the toxin, to turn color. While researchers have found this assay just as sensitive as more commonly used assays, they say that EIA provides a cheaper and easier way to detect the concentration of single, specific toxins in solution. However, scientists now believe that ciguatera and PSP are not caused by single toxins, but by several closely related poisons. To be effective, the chemical assay kit must be specific enough to detect only toxic chemicals, but not so specific that it misses potential poisons. Both Guire and Hokama hope to overcome the problem of specificity soon, and to have their kits ready for market within the next few years. — *S. Steinberg*

WHAT YOU SHOULD KNOW ABOUT CIGUATERA POISONING



WHICH FISHES MAY CAUSE CIGUATERA POISONING?

- Many species of reef fishes (perhaps over 500 species worldwide) may cause ciguatera poisoning.
- It was previously believed that because Hawaii lies in the sub-tropics, ciguatera was not present. Indeed, most earlier reported cases in Hawaii involved the eating of fishes brought in from other Pacific islands.
- A survey of reported ciguatera-poisoning cases in Hawaii between the years 1900 to 1979 (a period of 80 years) revealed a total of 123 incidents with at least 600 people being affected. About 91 of these incidents involved fishes caught in Hawaiian waters.
- For comparison, of 33,085 individuals interviewed in the rural part of Tahiti, about 2795 persons (8.45%) were poisoned during the year 1966, alone.
- Ciguatera poisoning in Hawaiian waters has been implicated most often in the kahala (amberjack; 37 incidents) and the ulua (jackfish; 19 incidents).



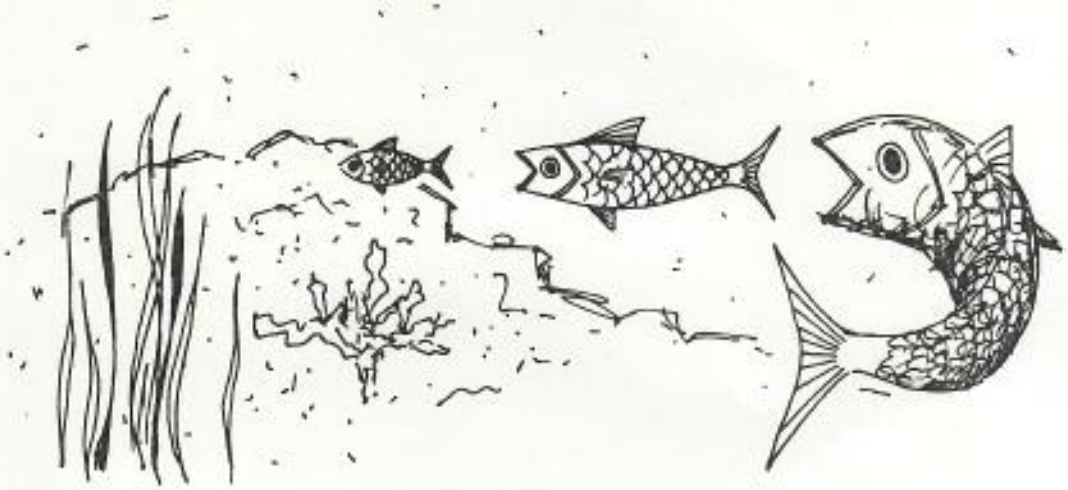
kahala

WHAT IS CIGUATERA POISONING?

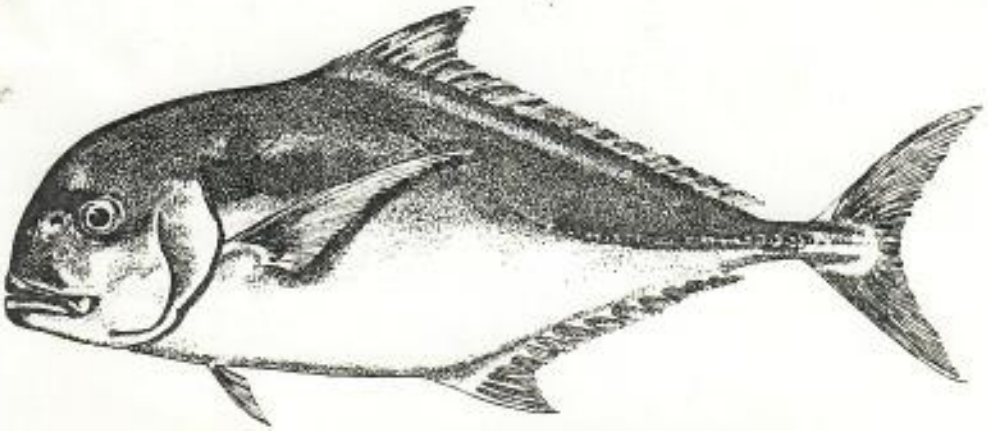
- Ciguatera (sē'-gwäh-tā'-rä̈h) is a type of food poisoning caused by the eating of various toxic fishes associated with coral reefs.

- Ciguatera poisoning is widespread in warmer temperate and tropical regions and has been present in various waters of the Caribbean and Pacific for centuries.

- The poison is produced by at least one kind of dino-flagellate (a microscopic marine organism). Fish eat this dinoflagellate(s) without any apparent harm. Other fish consume these fishes and may store or concentrate the toxin.



- The environmental conditions (sunlight, salinity, nutrients, etc.) which help the dinoflagellate(s) to grow, multiply, and produce toxin are not well understood. Under certain conditions, various fishes may become toxic or decrease in toxicity. In other cases, fishes may be poisonous only in certain areas.



ulua

- Certain other reef fishes have been implicated sporadically in ciguatera poisoning (fewer than eight reported incidents per fish species over an 80-year period).

- Ahi, aku, marlin, mahimahi, and other deep-sea fishes are not affected because the dinoflagellate is confined to shallower waters.

- Ciguatera poisoning in Hawaii is difficult to predict or control due to the generally small number of incidents recorded, the lack of knowledge of the dinoflagellate(s) and the environmental factors which influence its growth, the lack of seasonality when incidents have occurred, and the mobile nature of most fishes.

HOW CAN YOU TELL IF YOU ARE POISONED?

- Symptoms may appear from a few minutes to ten hours or more after eating a toxic fish.

- Symptoms are varied and may consist of:
  - Nausea, often followed by vomiting
  - Abdominal pain and cramps
  - Diarrhea or painful defecation
  - Slowed pulse
  - Lowered blood pressure
  - Tingling and numbness about the lips, tongue, throat, and extremities
  - Confusion of temperature sensation where touching a cold object gives the sensation of burning or tingling, and hot objects feel cold
  - Muscular weakness and incoordination
  - Headache
  - Nightmares
  - Dilation of the pupils
  - Shock, convulsions, muscular paralysis
  - Rarely death

- In most cases recovery is within a few days, but occasionally symptoms may persist for several weeks.

- Treatment recommended for ciguatera poisoning consists initially of emptying the digestive tract.

- Other treatments are symptomatic and supportive; vitamins, glucose, calcium, and other medications are used with varied success.

IS THERE A METHOD OF DETECTION?

- You cannot detect toxic from non-toxic fishes by their appearance.

TOXIC

NON-TOXIC

- You cannot destroy the toxin in the fish by freezing, frying, baking, broiling, boiling, stewing, steaming, drying, or salting.

- Generally, the larger fishes of a species are involved, especially the carnivores which feed on reef fishes.

- A method of possibly determining the presence of toxin is to feed a portion of the fish to test animals. However, this method is not always reliable, and test animals may not be readily available.

- Presently, studies are being conducted at the University of Hawaii School of Medicine to develop a simple test to determine toxic levels of ciguatera in fish.

WHAT SHOULD I DO?

- Do not eat the brain, spinal cord, intestines, gonads, and liver of all reef fishes since the toxin, if present, accumulates in these organs.

- Do not consume reef fishes from areas where ciguatera poisoning cases have occurred in recent months.

- To avoid possible severe ciguatera poisoning reaction, do not eat large quantities of potential ciguatoxin-bearing fishes in one sitting.

- Avoid eating unusually large reef fishes without prior testing.

- If symptoms appear after the ingestion of fish, you should see your physician immediately for treatment. If your physician is unavailable, contact the POISON CONTROL CENTER. Also notify the State Department of Health Epidemiology Branch. Wrap and freeze any leftover portion of fish for future analysis.

- If afflicted, avoid eating reef fishes implicated in ciguatera poisoning for at least three months after the symptoms have disappeared.

- Call the State Department of Health Epidemiology Branch (548-5985) or Food and Drug Branch (548-3280) if you have further questions.

Pamphlet Prepared By:
Ciguatera Advisory Committee
c/o HAWAII STATE DEPARTMENT OF HEALTH

Health Education Office
HAWAII STATE DEPARTMENT OF HEALTH
1250 Punchbowl Street
Honolulu, Hawaii 96813

Picture of kahala obtained from National Marine Fisheries Service

Picture of ulua obtained from: Tinker; Spencer Wilkie. 1978. Fishes of Hawaii, A Handbook of the Marine Fishes of Hawaii and the Central Pacific Ocean. Hawaiian Service, Inc. Honolulu, HI 532 pp.

EQUAL RIGHT TO HEALTH SERVICES

The Hawaii State Health Department provides services and opportunities to participate in its programs and activities without regard to race, color, national origin, age, or handicap, as required by Titles VI of the Civil Right Act of 1964, the Age Discrimination Act of 1975, and Section 504 of the Rehabilitation Act of 1973 and federal regulations. Anyone who believes that he or she is being discriminated against on the basis of race, color, national origin, age or handicap has a right to file a complaint with the State Department of Health or the Office for Civil Rights of the Department of Health, Education, and Welfare.

George Yuen, Director
George Ariyoshi, Governor

Produced by
Health Education Office
9/80; 1000

Anon. (1908) Poisoned fish may bring on cholera. The Pacific Commercial Advertiser, Honolulu 17 January: 4.

B.D. Mitchell, an old-timer here and a graduate of a College of Physicians and Surgeons, thinks that cholera may be traced to the eating raw of fish which, at certain times of the year consume a poisonous variety of limo.

"The first case of cholera here in 1895 appeared among the feasters at a raw fish luau. Years ago when I taught school on one of the other islands, the natives who ate the limo-fed fish uncooked came down with something very much like cholera if not the real thing itself. I don't know about the recent case on River street but I should like to know what the man had been eating before he was taken down.

HAWAII CLIPPING SERVICE
P.O. Box 10242-Honolulu, Hawaii
PHONE: 734-8124
Victoria Custer Elaine Stroup
HONOLULU ADVERTISER

DEC 8 1984

Toxic fish found off Big Isle

United Press International

HILO — The state health department says there have been nine cases of fish poisoning on the Big Island in recent months.

The department said this week a variety of reef fish caught along the South Kona coast was the source of the ciguatera poisoning from April through October.

The poisoning is caused by a substance the fish stores in its body, mostly in the internal organs. There is no way to tell a toxic fish from its appearance, smell or taste. The toxin cannot be destroyed by freezing, cooking, drying or salting.

There have been no reports of the problem arising from deep-sea fish such as ahi, aku, marlin, mahimahi or ono. On the other hand, almost any reef fish can carry the toxin.

Health officials advise minimizing the risk of ciguatera poisoning by eating deep-sea fish and avoiding eating large amounts of large reef fish at one sitting.

Symptoms of ciguatera poisoning vary, but most patients usually experience nausea followed by tingling and numbness around the lips, tongue, and throat.

One of the classic symptoms is temperature reversal, feeling a sensation of heat when touching a cold object and feeling cold when touching a hot object. To the patient, tap water feels like it has been carbonated.

Fish blamed for illness of 13 on Kauai

By Jan TenBruggencate
Advertiser Kauai Bureau

LIHUE — The Kauai office of the state Department of Health issued a warning against eating a small fish caught last week off the Na Pali Coast when 13 people who ate the fish became ill and one was hospitalized with suspected ciguatera poisoning.

The fish are kole, the gold ring surgeonfish, a small reef fish that is mostly brown with a yellow ring around the eye. They were netted near Nualolo Valley and distributed on the west side of Kauai to individuals and stores.

The poison is strongest in the organs of the fish, the head and guts, but can also be found in the flesh, said district health officer Theodore Inouye.

Anyone who bought or received the fish should destroy it. It should not be fed to pets, since they can be poisoned also.

Ciguatera is a kind of food poisoning caused by eating reef fishes that get it from microorganisms in algae they eat. It doesn't hurt the fish, but is stored in their bodies.

Epidemiological specialist Steven Terrell-Perica said the symptoms of poisoning can begin 10 minutes to 10 hours after eating. The symptoms can include stomach pain; tingling sensations around the mouth, hands and feet; nausea and diarrhea; headache and dizziness; muscle weakness and clumsiness; hot things feeling cold, and cold things feeling hot; lowered pulse and blood pressure and, sometimes, shock; and other foods tasting strange or metallic.

Persons feeling such symptoms after eating the fish should call the Department of Health. They should freeze fish remains for study.

Kahala are biting — but

from the sea

mike markrich

KAILUA-KONA — "People tell me that if I could offer this kind of fishing action in California, I'd be a millionaire," says Kona charter boat skipper Peter Hoogs.

At a time when many charter boats in the state have been struggling, Hoogs says he has been getting calls from the Mainland and all over the state from people who want him to take them kahala fishing. Although kahala, or amberjack, has suffered from a reputation for carrying ciguatera fish poisoning in recent years, it has become the focus of a small fishing boom on the Big Island.

"This is pretty hot fishing compared to what they get in San Diego," said Hoogs, who offers California-style stand-up fishing from his 40-foot charter boat, the Pamela. He supplies special long fishing poles and harnesses similar to those used on "party boats" in California.

He said amberjack is one of the most popular California game fishes and his customers are constantly amazed at the large fish they bring up.

The large number of available kahala is partially due to the publicity generated about the fish during the mid-1970s when it became associated with several cases of ciguatera on Oahu and Kauai. Although once a highly popular local fish (in Japan it is known as hamachi or buri and commonly sold), it was withdrawn voluntarily from local fish markets in 1979 and many people stopped fishing for it altogether.

Since then, many people have been unsure about what to do with the kahala they catch.

"A lot of fishermen call me up about eating kahala," says Henry Okimoto of the Division of Aquatic Resources. "I tell them not to take a chance. Our guess is that 80 to 90 percent of the fish is OK.

"But it's because of that last 10 or 20 percent that the (fish) market people don't want to to sell it — because if they sell something questionable, they become liable for any kind of suit."

He also suggests that people who choose to eat kahala not invite a lot of guests for dinner. (Two months ago on Kauai, 15 people from three families ended up sick after eating a 60-pound kahala.)

According to state epidemiologist Bruce Anderson, kahala made up 17 to 21 percent of the 81 cases of ciguatera reported from 1975 and 1981. (Papio and ulua combined accounted for 25 percent, the wrasse poau was third at 12 percent and a number of other reef fishes made up the remainder.)

Yoshisugi Hokama of the University of Hawaii's John A. Burns School of Medicine tested 5,500 kahala between 1978 and 1981 to trace fish poison. He found 15 percent of the fish toxic and the remainder completely safe. The safe fish were sold in Honolulu markets without incident until 1979, when it was decided that, at $25 per fish, the joint federal/state-sponsored tests were too expensive for use on a commercial basis.

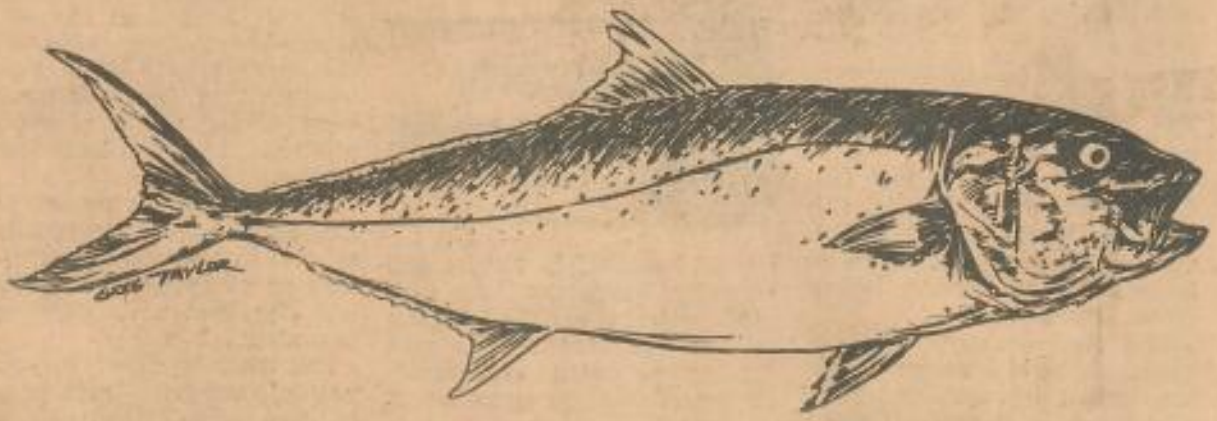
(According to one longtime Hawaiian fisherman who asked not to named, prior to the tests it was common to feed a small piece of kahala liver — the organ where toxin concentrates most — to a cat before eating the fish. If the cat did not become ill soon after, the fish was considered safe. Not surprisingly, this method of testing fish has been discouraged by the Hawaiian Humane Society.)

Hokama is particularly concerned about kahala being shipped to the Mainland by fish wholesalers. He says that six Mainland poisoning cases involving several different kinds of fish have been traced back to Hawaii in the last year and it is unwise to risk the reputation of the local fishing industry by sending fish without testing it.

"I'd like to see them clamp down on that," agrees Bruce Johnson, a Maui fish exporter. . . . There is enough information. I don't want a guy sitting here looking over my shoulder all day but we (fish wholesalers) should have some kind of standard."

Those who export kahala or serve it in Big Island restaurants oppose a ban. They say it unfair to ban Big Island kahala because it is the fish from other areas that are toxic. Their argument is bolstered by statistics from the state Department of Health that indicate 17 of the 18 kahala fish poisoning incidents since 1975 were caused by fish caught off Oahu or Kauai and the

should you take a bite?



Advertiser sketch by Greg Taylor

The kahala — good fishing, sometimes dangerous eating.

other was traced to fish from off Maui. None came from Big Island waters.

But Hokama warns that there are no guarantees. He said research indicates that large kahala of 75 pounds or more seem to be safer than small ones but there are no firm rules as to why kahala from one area are relatively safe while those from other areas are more dangerous.

Hokama adds that testing the fish will be easier after a new inexpensive ciguatera kit he has developed becomes commonly available. In the meantime, he advises caution.

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GARDEN ISLE
SEP 1 1985

North Shore ciguatera

Fish poison hits a dozen

by Mak Mock

Twelve Northshore residents were stricken this past week with fish poisoning. All received mild doses of ciguatera toxin from eating kole and ulua, according to the state Department of Health.

State health officer, Dr. Jeffry Smith, said none needed hospitalization. "The group should be symptom-free in a few weeks."

Symptoms of fish poisoning include nausea, cramps, diarrhea and, since the toxin affects the nervous system, a sensation of temperature reversal, in which hot feels cold and cold feels hot. "A really heavy dose can put you in ICU and the effects on your nervous system can last for months," said Smith.

Earlier this year there were seven cases of ciguatera poisoning involving hospitalization of the victims. But this time only about half the victims sought medical attention, Smith said.

The toxin concentrates in the head and internal organs of the fish; the current group ate only the flesh so they received a mild dose.

The ciguatera toxin comes from a marine plankton called Gambierdiscus toxicus that reef fish ingest when they eat certain seaweeds. Larger fish then eat the smaller fish and absorb the toxin. Humans get the toxin by eating a fish so poisoned.

There is no way to inactivate the toxin," said Smith. "You can't cook it out, you can't freeze it out, nor can you detect it from the appearance or odor of the fish. It takes a lab test to find it." he said.

He also mentioned that certain foods containing a high concentration of certain fatty acids can exacerbate the problems and recommended that the victims avoid fish, seeds and nuts for the next few weeks.

One traditional way to check a suspected fish is to rub a piece of raw fish liver on the gums. If there's a tingling sensation, it is likely that the fish contains high levels of toxin. But, Smith warns, this does not always give the tingling even when the fish is toxic.

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Sunday
Star-Bulletin & Advertiser
June 21, 1987 J:1

Poisoning from fish still stumps biologists

By Jan TenBruggencate
Advertiser Kauai Bureau

Hawaii residents almost every year suffer from the form of fish poisoning called ciguatera, but biologists are still not sure what the disease is or how it is caused.

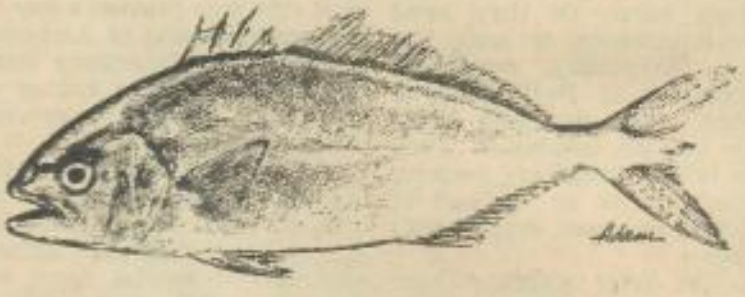
Biologists Donald M. Anderson and Phillip S. Lobel, of Woods Hole Oceanographic Institution in Massachusetts, said in a recent Biological Bulletin that broad advances in study on ciguatera have occurred, but much is still needed.

"Despite this surge in activity, the general state of knowledge on ciguatera remains relatively poor . . . Some important generalizations are gaining acceptance, but discrepancies and disagreements abound," they said.

Many reef fishes are capable of carrying the poison, which they apparently get from ingesting microorganisms that grow among seaweeds. Recent Kauai poisonings have involved kole and mullet caught near Milolii on Kauai's Na Pali Coast.

The poisoning causes nervous system symptoms like tingling, numbness and the sensation that hot things are cold and cold things hot. Diarrhea is also sometimes present.

The poison, generally stronger in the organs than in the muscle tissue, does not appear to hurt the fish, and can be passed from a grazing fish to its predator without hurting the predator.



The kahala fish has been implicated in causing the form of fish poisoning called ciguatera.

Ulua and kahala are such predators which have been implicated. Kahala has received the worse reputation of the two, and many commercial anglers now ignore it because of the danger.

Scientists 10 years ago linked the poisoning to a microscopic organism called *Gamberdiscus toxicus*. But more recent studies have shown many other marine microorganisms on seaweeds also contain poisons. The findings further confused the issue, and led to the conclusion that multiple poisons may be involved in ciguatera cases.

Laboratory studies on the poisons are going well, but are still incomplete, and there is even some question whether the major poison in ciguatera cases actually comes from the *Gambierdiscus toxicus*.

A chemical test that tells whether the poison or related compounds are present in a fish still isn't accurate enough to tell which is present. If a non-poisonous, but chemically similar compound is there, the test says the fish is bad.

Still, though you might throw out a good fish with this test, if it continues to perform as it has, at least you won't end up keeping a bad one.

Some studies suggest links between the way a fish eats and the amount of poison that is likely to end up in its tissues. Such studies might be able to steer people away from certain fish with more hazardous habits, and toward safer varieties.

Poison! Don't Eat Kole, Weke Caught Off the Reef Runway

3/19/87 HSB A9

Associated Press

It's dangerous to eat two varieties of reef fish caught off Honolulu Airport's reef runway.

Seven people became ill after eating kole caught off the runway Feb. 24 and March 8, the state Health Department said. The symptoms were of ciguatera fish-poisoning.

The department said another illness, hallucinogenic fish-poisoning, was reported after people ate white weke, or goatfish, from the same area.

Symptoms of ciguatera vary from mild to severe, occurring within three to five hours of eating a toxic fish.

Common symptoms are general weakness, diarrhea, muscle pain, joint aches, numbness and tingling around the mouth, hands and feet and reversal temperature sensation. The illness may last for weeks or months.

Hallucinogenic fish-poisoning is characterized by hallucinations, sensation of tight constriction around the chest, insomnia, intense dreaming, weakness and dizziness.

Symptoms occur soon after eating the fish and can include frightening nightmares, the department said.

The toxins that cause the illnesses cannot be detected by appearance, smell, taste or freshness and are not affected by cooking, drying, salting or freezing fish, the department said.

3/11/87 HSB

A Gem from the Deep

Star-Bulletin Staff

Black coral — it may not be a girl's best friend. But it looks like it's about to become the official state gem.

A bill designating it as the state gem has passed the Senate and moved to the House.

The black coral was picked because it's indigenous to Hawaii, is a product of the sea, and would stimulate local jewelry manufacturing and sales. Discovered in local waters, Hawaii divers began harvesting the gem in 1957.

CapitolCALENDAR

Today is the 34th day of the 60-day legislative session.

Committee hearings are as follows:

HOUSE

Tomorrow

8:30 a.m. — Planning, Energy, and Environmental Protection Committee hearing on resolutions to request further development of alternate energy sources in Hawaii and to request the Legislative Reference Bureau to make recommendations for legislation that would require electric utilities to provide financing for alternate energy and conservation measures for consumers. Room 328.

9 a.m. — Finance Committee hearing on bills relating to the state budget, judiciary, Office of Hawaiian Affairs and state bonds. Room 307.

1:30 p.m. — Judiciary Committee hearing on bills relating to the right to sue by native Hawaiians and child support. Room 328.

SENATE

Tomorrow

1:30 p.m. — Health Committee hearing on a bill requiring applicants for a marriage license to take the AIDS HTLV-III test. Room 3.

3 p.m. — Education Committee hearing on a resolution to improve the preparation of teachers and to make teaching a more rewarding and respected profession. Room 6.

Cause of Dead Fish Eludes Investigators

Star-Bulletin Staff

State health investigators say they were unable to determine the cause of a fish-kill that occurred at Waikiki beach Sunday.

The dead fish, tilapia, were confined to the Kapahulu Avenue storm drainage system that empties into the beach at Kalakaua Avenue.

A-2 Honolulu Star-Bulletin Monday, January 18, 1988

Ciguatera, poison that lives on the reefs

Second in a series about fish poisoning in Hawaii

By Susan Scott
Special to the Star-Bulletin

The word ciguatera was probably first used by the Spanish conquistadores in Cuba to describe the illness caused by eating a marine snail.

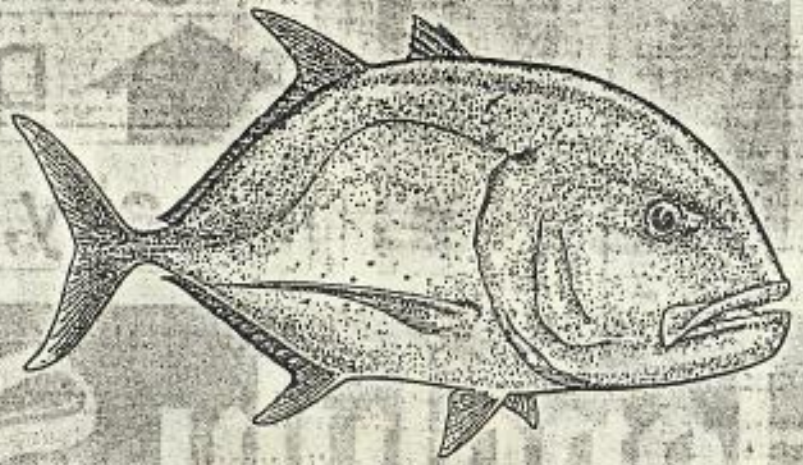
Captain Cook described ciguatera poisoning among his crew members after a meal of red snapper in the New Hebrides in 1774.

Today ciguatera is all too familiar to tropical islanders as the serious illness one gets from eating certain reef fish.

Ciguatera poisoning is caused by a specific type of dinoflagellate that grows in abundance under certain conditions. Dinoflagellates are planktonic organisms that lie somewhere between the plant and animal kingdoms since they have characteristics of both.

THE CIRCUMSTANCES that cause these organisms to flourish are poorly understood, but when they do grow, small reef fish eat them.

Larger fish then eat the small fish, and in this way the poison is passed up the food chain. The ciguatera toxin, which doesn't seem to affect the fish at all, gets stored in the animals' liver, brain and gonads.



Hawaii Fishing News

POISON FIN PAL—As a reef-fish eater, the *ulua* can be a source of ciguatera poisoning.

More than 500 species of reef fish can carry the ciguatera toxin because its source is so low in the food web of the reef animals. Reef invertebrates, however, are not affected, nor are offshore fish who don't eat reef fish.

Since Hawaii lies in the subtropics, it was once believed that ciguatera was not present on our reefs. However, a survey of ciguatera-poisoning cases from 1900 to 1979 revealed a total of 123 incidents, with about 600 people affected. About 91 of the incidents came from fish caught in Hawaiian waters.

Our subtropical status, however, probably helps keep the numbers down. In 1966 alone, 2,795 people in Tahiti were poisoned out of a relatively small sample group of 35,000.

In Hawaii, the fish most often implicated in ciguatera poisoning are the *kahala* (amberjack) and the *ulua* (jackfish), both predators of reef fish.

How can you tell if you are poisoned? Symptoms start from a few minutes to 10 hours after eating a toxic fish and resemble food poisoning at first. Nausea, vomiting, diarrhea and abdominal cramps will tell you that you have eaten something bad.

AFTER THAT, ciguatera poisoning differs from other food poisonings by affecting the central nervous system. Tingling and numbness of arms and legs, dilation of pupils, nightmares, and muscle weakness may all be experienced by the poisoned person.

Confusion of temperature sensation is a classic ciguatera symptom — a hot object will feel cold and cold will give a burning sensation. If you go to an emergency facility, which you should if you have ciguatera symptoms, you will probably be given some ice for a touch test.

Treatment of ciguatera is similar to any poisoning. The digestive tract should be emptied immediately. Further treatment depends on particular symptoms.

Severe poisoning can cause shock, convulsions and occasionally death. Usually the symptoms go away after a few days, but they can last for weeks.

There is no reliable method of detecting ciguatera in a fish, although folk tales claim otherwise. Each locality has its own special method of detection, from cooking a coin in the fish to checking the backbone for a black line, but none is foolproof.

Susan Scott is a marine biologist and free-lance writer. Her OceanWatch column appears Monday in the Star-Bulletin. Next week, the poisonous puffers.

George FYI
→ WGG

CF

Marshalls' M.D. Curing Fish Poison

By Giff Johnson

Ciguatera fish poisoning is a major problem in the Pacific. The symptoms may range from a slight numbness of the lips to loss of muscle coordination and even death. But mild or severe, there has never been a cure for the debilitating effects of fish poisoning that thousands of people experience each year around the Pacific.

A Marshall Islands-based medical doctor, however, may have discovered the cure for fish poisoning. Nobody has ever come up with a reliable treatment for fish poisoning—until a 1983 discovery by Dr. Luis Jain, a surgeon, who came to the Marshall Islands in 1979 from his home in the Philippines.

Jain cautions that the cure needs more exhaustive study to prove its effectiveness and to avoid it being given in the wrong dose.

Like many scientific firsts, Jain's discovery came about by accident.

The Majuro hospital often receives emergency radio calls from the outer ring atolls asking for assistance for fish poisoning victims. In 1983, the health assistant on Jaluit Atoll called with an emergency case, and Jain flew to Jaluit on a special medical mission to treat two patients who had been unconscious and in critical condition for two days as a result of severe fish poisoning.

The fish poisoning had caused severe muscle spasms for both patients: their legs and arms were as stiff as a board, Jain said. After examining the two comatose Marshall Islanders, Jain thought they might be suffering from swelling of the brain as an allergic reaction to eating the poisoned fish.

■ **Mannitol:** The two patients were loaded onto the plane on stretchers and just before take off for the return to Majuro hospital, Jain began intravenous injection of mannitol, a drug used to relieve brain swelling. The impact was quick and startling.

"Within 10 to 15 minutes, just after take off, one of the patients woke up and began asking 'where am I'," said Jain. A few minutes later the other patient also came out of his coma.

The first man's condition improved so much during the 45 minute flight that he got up and helped the other patient into a wheel chair on arrival at Majuro airport.

Jain related this development and the quick recovery of the patients after using mannitol but his colleagues at the hospital were skeptical. "Nobody would believe me that mannitol worked," said Jain. Soon after he began trying it out on others who experienced severe fish poisoning symptoms.

In 1986, another critically ill fish poisoning victim from Jaluit arrived at the hospital. Dr. Neal Palafox, a graduate of the University of Hawaii Medical School, was the patients doctor, and Jain suggested he try mannitol on the patient who was conscious but could not move because of severe muscle contractions.

He agreed, and five minutes after administering the drug, the patient stood up, Jain said. Since then, Jain and Palafox, now medical director of public health for the Marshall Islands, have been administering the drug to people with severe cases of fish poisoning with success.

"So far I haven't seen any side effects," said Jain. It has been used effectively on more than 50 patients, although only 20 cases have been documented, he said.

Palafox and Jain have nearly completed a 'protocol' of clinical standards so that they can systematically study the results over a two to three year period.

"We want to run a study simultaneously on Majuro, Ebeye and Kwajalein," said Palafox.

The Majuro doctors have received a number of inquiries from doctors in other parts of the Pacific who have heard of the developments. Both say that they use low dosages of the drug, which is very inexpensive and readily available in most countries.

However, they urged health officials interested in the treatment to contact them before experimenting with it to avoid the risk of giving wrong dosages to patients.

■ **Symptoms:** Majuro hospital alone reports an average of more than five cases of fish poisoning a month—a figure that is probably far below the actual number of cases as Majuro has only 40% of the nation's population of 40,000. Fish poisoning symptoms range from numbness of lips and a reversal of temperature feelings (hot feels like ice, cold like heat) to more severe cases that may include diarrhea and nausea, muscle pains, unconsciousness and death.

Jain observed that three or four patients with severe cases of fish poisoning had died here before the mannitol discovery in 1983. In December, 15 crew aboard a Marshall Islands government field trip vessel were struck with fish poisoning symptoms after eating a large fish caught near Ebon Atoll in the south. Mannitol was used successfully to relieve the symptoms in the most serious cases, the hospital reported.

KOUSEI SHIMADA

Dr Luis Jain: administering Mannitol to relieve brain swelling

Ciguatera fish poisoning is caused by a toxin in fish known as ciguatoxin. The fish with ciguatoxin do not appear any different than safe fish, and the poison cannot be destroyed by cooking or freezing the fish.

Jain and Palafox are working to get the discovery published in a major medical journal. A paper describing the mannitol cure is now with the Journal of the American Medical Association for consideration. They are hopeful that publication will stimulate increased interest in studying the use of mannitol for curing the symptoms of fish poisoning.

If the cure can be proven through medical study, then it would represent a breakthrough for the Pacific region where fish represent the main source of protein. Many thousands of cases—the exact number is unknown because of poor statistics—occur annually. ■

CTX Treatment Under Study by UH Researchers

by Priscilla Billig

Recent newspaper accounts touting the curative effects of the drug, mannitol, as an antidote for ciguatera fish poisoning are being questioned by Dr. Yoshitsugi Hokama, professor of Pathology at the University of Hawaii. Ciguatera, a major concern in the rising U.S. consumption rate of seafood and in the fishing industries of endemic areas throughout the Pacific and the Caribbean, has been the focus of research by Hokama for a number of years. In a June interview, he said newspaper reports, which called mannitol a "miracle drug" and a "complication-free antidote" for fish poisoning, were misleading and discussed the treatment's scientific report published this May in the *Journal of the American Medical Association.*

Hokama, refuting *The Honolulu Advertiser* and *Honolulu Star-Bulletin* articles stated that use of mannitol "would be a useful treatment for an acute situation, but, until more studies are done, to suggest that it is an antidote is premature." According to Hokama, an antidote, by definition, would neutralize the ciguatoxin but the misconception in terminology ignores the fact that the specific mechanism by which the drug acts is still unknown. Therefore, mannitol cannot be effectively termed an antidote. The *Advertiser* article, Hokama notes, also mistakenly states that the drug is injected when it is actually infused intravenously during treatment. Hokama points out that treatment by infusion of a mannitol solution should be carried out even though the mechanism of its action is yet unknown.

Authors of the original JAMA scientific article do not claim to have found a cure for ciguatera fish poisoning but instead report that the administration of mannitol shortens the neurologic and muscular dysfunction period in each patient infused. Hokama questions whether the mannitol itself is relieving symptoms or simply acting as a dilution of the ciguatoxin within the system since the reason for the drug's apparent effectiveness remains unclear. A similar type of treatment was reported almost 25 years ago by Dr. Raymond Bagnis, a physician with the Institut de Recherches Medicales in French Polynesia who had treated patients suffering from CTX poisoning by infusing them with calcium gluconate. Structurally, this drug is very similar to mannitol and is presently one of the therapies recommended to local doctors treating cases of CTX poisoning.

Mannitol was first discovered to relieve symptoms of CTX poisoning in 1986 when administered to two comatose fishermen in the Marshall Islands diagnosed as suffering from cerebral edema. Use of the drug is standard procedure when the brain accumulates fluid. Upon recovery, it was found that the men had eaten fish tainted with ciguatoxin. Hokama suggests that, since the major clinical symptoms of CTX poisoning are primarily gastrointestinal, involving vomiting and diarrhea which cause dehydration, mannitol infusion may be merely rehydrating the patient. Again, the basic mechanism must be discovered before mannitol can be clearly claimed as an antidote.

The JAMA article reports speculation that the mechanism of action in mannitol indicates the possible absorption of ciguatoxin from the gastrointestinal tract and that the ciguatoxin is thus displaced. Hokama theorizes that the mannitol, which also functions as a diuretic, is not displacing ciguatoxin but rather is dispersing or diluting the ciguatoxin, thereby diminishing its activity. UH researchers are presently conducting studies in this area.

According to Hokama, the most significant problem resulting from recent newspaper reports which claim mannitol as an antidote is the danger this type of misinformation poses to the public. Importers of fish from areas known to have high toxicity rates may feel freer to sell questionable fish on the open market now that an antidote to ciguatera is thought to be available. An example of this confusion was indicated by a fish importer from the Marshall Islands who commented to Hokama, "Now that they have a cure I can bring those fish in and it doesn't matter if people get sick." By all indications, this attitude ignores safety. Hokama warns that it does matter and that irresponsible behavior should not be tolerated when dealing with the public's health. □

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Jack R. Davidson, Director, Sea Grant College Program

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More information offered on ciguatera fish poisoning

OCEAN WATCH
By Susan Scott HSB 11/11/91 A2

LAST week I wrote about some concerns that fishermen and fish market owners have about ciguatera, a type of fish poisoning that has sharply increased in Hawaii since 1984.

I learned that some new test kits, which are dipsticks in a solution, are for recreational fishermen only. These kits were never intended for commercial use.

So how do commercial fishermen and fish markets know if their reef fish are OK?

The men I talked to, who asked to remain anonymous, said that certain areas such as Honolulu Harbor, Pearl Harbor and the Waianae Coast have had a high incidence of ciguatoxin and are therefore off limits. Markets won't buy fish that come from there.

And what keeps fishermen honest about this?

The markets periodically have Dr. Y. Hokama, a UH ciguatera researcher, test batches of reef fish for safety.

If they're toxic, then the market won't buy fish caught from that area again.

This means that if a fisherman fibs and says, for instance, he caught fish in Hawaii Kai when they're really from Waianae, then Hawaii Kai is falsely ruined for fishing if the tests are positive.

"If we lied about where we catch our fish, we'd be cutting our own throats," the fisherman told me.

Nevertheless, some fishermen have lied about location and are now paying for it. The market owners told me that they no longer buy any reef fish from fishermen that they don't know well.

But that doesn't mean that honest reef fishermen are getting more business.

They're getting less.

Because of the increase in ciguatera poisoning in the past decade, the business is in trouble. Many markets have drastically decreased their supplies of reef fish.

Although no one knows what's causing the recent increase of this type of fish poisoning in Hawaii, researchers do know how it makes people sick.

Ciguatera poisoning is caused by a microscopic dinoflagellate called toxicus.

Dinoflagellates, which have chlorophyll like plants but swim like animals, are common plankton in the world's oceans.

Gambeierdiscus toxicus is always common in some areas; in other places it occurs only occasionally, blooming for unknown reasons among the seaweeds that reef fish graze on.

The toxin doesn't hurt the fish but can pack a punch to humans who eat those fish, especially the internal organs where the poison is concentrated.

No one knows how much ciguatoxin a person must eat to become sick, but it's possible that it may accumulate in the human body. This means that eating a small amount time and again could eventually make a person sick.

Death is rare from ciguatera fish poisoning but misery is common and can go on for months.

Because the toxin affects the nerve and muscle cells, the symptoms can be varied and bizarre.

The list of possible symptoms is long but reversal of hot and cold sensation, muscles aches, joint pain, vomiting, diarrhea and irregular heartbeat are among them. To avoid ciguatera fish poisoning:

- Know the fish most associated with ciguatera. Ulua, wahanui (black snapper), roi (argus grouper) and weke (goatfish) are the highest among 18 species looked at in a study from 1987-1989.
- Avoid eating large amounts of ciguatera-associated fish at one meal.
- Avoid eating repeated meals of the same reef fish.

There is no cure or antidote for ciguatera poisoning but some medicines may help the symptoms.

If you suspect fish poisoning:

- Call your doctor or go to an emergency department immediately.
- Don't take any drugs or eat any more fish.
- Freeze the rest of the fish for testing.
- Call the Department of Health and report your case.
- Avoid eating fish from areas associated with toxicity.

For information about ciguatera test kits, call the Department of Health at 586-4400.

For information about ciguatera and which fish have caused poisonings (and how often) in Hawaii, read "Seafood Poisoning: Ciguatera" by J.L. Shirai, L.K. Shirai and Y. Hokama available in local bookstores. (Published in 1991 by Yosh Hokama Family Trust, Gardena, CA, $16.95).

Susan Scott is a marine science writer and author of Oceanwatcher, a guide to Hawaii's marine animals. Her Oceanwatch column appears Monday in the Star-Bulletin.

SPC/Fisheries 20/ WP. 19
26 July 1988

ORIGINAL: ENGLISH

SOUTH PACIFIC COMMISSION

TWENTIETH REGIONAL TECHNICAL MEETING ON FISHERIES
(Noumea, New Caledonia, 1 - 5 August 1988)

FISH POISONING IN TOKELAU

by
Mose Pelasio
Institute of Marine Resources
University of the South Pacific

Fish Poisoning in Tokelau

Mose Pelasio
Institute of Marine Resources
University of the South Pacific

In Tokelau ciguatera is not a major problem in a commercial sense; few fish are exported or even sold domestically because of the subsistence nature of the fishery. Fish poisoning is, however, a local public health problem.

Tokelau, with a population of approximately 1600, has had only a few cases of fish poisoning recently. Apart from ciguatera, no other cases of seafood toxicity, such as that from gastropods, crabs, lobsters, turtles, or bivalves have been reported.

Fish poisoning in Tokelau is not common; SPC reported eight cases in 1976, fourteen in 1979, and three in 1981. Since then, there has not been any other fish poisoning reported. This may be an artifact of the reporting system.

From the above 25 cases reported in the 3 years, there was one case in which a life was claimed. The victim was reported to have eaten the liver and the mature eggs of a Pseudobalistes flavimarginatus. He was reported to have suffered for several days, while in the other cases they were sick for a much shorter period.

Scarus longiceps, Pseudobalistes flavimarginatus, Lutjanus bohar, Epinephelus fuscoguttatus, Sphyraena sp. (cf S. picuda, cf S. barracuda.) and Melichthys sp. (cf M. niger, cf M. vidua.) are thought to have caused the cases of fish poisoning. As there has been no formal studies on fish poisoning in Tokelau, much of the available information is anecdotal in nature. The exact identification of potentially toxic fish is therefore difficult.

In 1977 a Korean longliner was wrecked on one of the Tokelau Islands. Shortly after the grounding on the reef, there was a substantial rise in the incidence of fish poisoning. The local people believed that either the constant smashing of the reef or the resultant oil spill may have been responsible for the fish poisoning. It is interesting to note that only the fish caught in the immediate area of the wreck were poisonous; the same species on the other side of the island remained unaffected.

A factor which may reduce the incidence of fish poisoning, is the abundance of fish in Tokelau. Compared to some neighboring countries, there is no shortage of fish for consumption on the three atolls. Consumers have a wide variety of fish species to chose from and due to the subsistence nature of the fishery there is little cost differential between species. Those marginal species which may cause fish poisoning can therefore be avoided without much effort.

In February 1987 Fakaofo Atoll was hit by a huge surf. In the storm, waves washed completely over the major inhabited islet and destroyed most of the living coral on the west side of the atoll. Tokelau officials, fearing a major increase in the incidence of fish poisoning, asked the SPC to investigate the fishery implications of the massive coral destruction. A South Pacific Regional Environment Programme consultant was dispatched to investigate the situation. He concluded that, although the coral damage was heavy, it was similar to that of the Tuamotu Group in the early 1980s where there was not a subsequent increase in fish poisoning. The consultant recommended that because the conditions may be conducive to the development algae responsible for fish poisoning, the situation should be closely monitored.

Successful Treatment of Ciguatera Fish Poisoning With Intravenous Mannitol

Neal A. Palafox, MD; Luis G. Jain, MD; Alexander Z. Pinano, MD; Tod M. Gulick, MD; Robert K. Williams, MD; Irwin J. Schatz, MD

Twenty-four patients with acute ciguatera fish poisoning were treated with intravenous mannitol, and each patient's condition improved dramatically. All exhibited marked lessening of neurologic and muscular dysfunction within minutes of the administration of mannitol. Gastrointestinal symptoms disappeared more slowly. Two patients in coma and one in shock responded within minutes, with full recovery after infusion. Although these observations were empiric and uncontrolled and the mechanism of action of mannitol in this disease is unclear, mannitol should be considered for initial use in patients with significant illness and morbidity from ciguatera fish poisoning.

(*JAMA* 1988;259:2740-2742)

CIGUATERA fish poisoning is caused by a common ichthyosarcotoxin endemic throughout the Caribbean and Indo-Pacific islands; it has a reported annual incidence of between 100 and 300 per 100 000 population.[1-3] Since it is the most frequently reported seafood-related disease in the United States,[3] its adverse impact on the fishing industry is a major economic problem (report of the Expert Committee on Ciguatera, South Pacific Commission, Noumea, New Caledonia, 1981).

From the Armer Ishoda Memorial Hospital, Majuro, Marshall Islands (Drs Palafox, Jain, Pinano, Gulick, and Williams); and the Department of Medicine, John A. Burns School of Medicine, University of Hawaii, Honolulu (Drs Palafox, Gulick, and Schatz). Drs Palafox and Williams are employees of the Public Health Service assigned through the National Health Service Corps to the Department of Health of the Republic of the Marshall Islands.

The opinions and assertions contained herein are those of the authors and are not to be construed as official or reflective of the views of the Public Health Service.

Reprint requests to Box 1047, Majuro, Marshall Islands 96960 (Dr Palafox).

Ciguatoxin is a heat-stable, lipid-soluble compound originating from a dinoflagellate named *Gambierdiscus toxicus*.[4,5] The toxin is passed through the food chain and is concentrated in certain fish, such as red snapper (*Lutjanus bohar*), amberjack (*Seriola dumerili*), barracuda (sphyraeniadae), and surgeonfish (acanthuridae).[4] Consumption of affected fish, whether it is cooked, raw, or frozen, may result in poisoning.[7]

Symptoms usually appear within 12 hours,[8-10] but sometimes they appear within minutes after the ingestion of ciguatoxin. Clinical presentation varies from minor complaints to coma and death.[1,8,11] Paresthesias, burning or pain when cold water is touched, arthralgias, and myalgias are common.[1,8,9,11] Abdominal pain, cramping, diarrhea, nausea, and vomiting are less prominent. Paresis, hypotension, and shock occur in some victims. Coma, paralysis, respiratory depression, and death have all been reported.[8-10] The duration of illness averages 8.5 days but may be prolonged[10]; one study reported a median hospital stay of six days.[10]

Laboratory data are nonspecific.[1,8] Several bioassays for identification of the toxin from the fish source exist, including an enzyme immunoassay and a radioimmunoassay.[12-14] Unfortunately, the availability of these tests is limited, particularly in those areas of the world where ciguatera fish poisoning is a significant clinical problem. Less elegant methods of detection include feeding contaminated fish to laboratory or domestic animals and observing them for clinical outcome. The mainstay of diagnosis remains history and clinical correlation.[1,8,9,11]

Treatment remains supportive,[2,9,11,15,16] since most attempts at specific therapy are unsatisfactory and have no proved efficacy. These have included calcium gluconate, pyridoxine hydrochloride, pralidoxime chloride, corticosteroids, atropine sulfate, vitamin B complex, and amitriptyline hydrochloride.[8,11,15,16]

Methods

One of us (L.G.J.) suspected cerebral edema in two patients who were comatose from ciguatera fish poisoning. Both patients' conditions improved suddenly and dramatically after mannitol infusion. As far as we know, these were the first two cases of ciguatera fish poisoning successfully treated with mannitol.

As a consequence of this success, we administered mannitol to patients with

clearly defined ciguatera fish poisoning with significant morbidity. All suspected cases were evaluated and defined as ciguatera fish poisoning if the following criteria were met: (1) if there was a history of consumption of fish within 1½ hours after it was cooked (it is commonly known to the physicians and people of the Marshall Islands that this produces ciguatera fish poisoning) and (2) if clinical signs and symptoms of the disease developed within 24 hours after the fish was consumed. Each patient was carefully evaluated for the following conditions: circumoral or facial paresthesias or numbness, paresthesias in the extremities, paresis, coma, reversal of temperature sensation (cold to hot), abdominal pain or cramping, watery diarrhea without blood, nausea, vomiting, muscle pain, arthralgia, and hypotension. The presence of at least one neurologic sign and one other sign was considered diagnostic of ciguatera fish poisoning.

Mannitol was administered to patients over the age of 5 years if one or more of the following conditions was present: paresis, vomiting severe enough to warrant fluid replacement, diarrhea at a frequency greater than one time per 1½ hours, muscle pain severely restricting ambulation, and hypotension requiring therapy.

An intravenous infusion of 5% dextrose in Ringer's or saline solution was established at a rate of 30 mL/h unless more copious fluid replacement was necessary. Mannitol (20%) was then given in a piggyback manner. The dose was calculated to a maximum of 1 g/kg at the rate of 500 mL/h. The total dose did not exceed 250 mL in any six-hour period. Careful monitoring for any change in symptomatology and vital sign examination were performed every five minutes. Infusion was immediately stopped if the systolic or diastolic blood pressure dropped more than 15 mm Hg from the initial blood pressure or as soon as the symptoms and signs disappeared.

Report of Cases

CASES 1 AND 2.—Two Marshallese men (aged 30 and 31 years) had been in good health until circumoral numbness and tingling in their fingers developed approximately two hours after they ingested cooked "Jalia" (long-nosed snapper [*Lethrinus miniatus*]) within two hours of capture. Within three hours, both noted a burning sensation on drinking or touching water; marked weakness of all extremities then developed, followed by joint pains and lethargy. After six hours they were unconscious, responded only to pain, and had flaccid paralysis with down-going plantar responses.

The reflexes were 1/5+ throughout, and they were symmetrical. Vital signs were normal, and no meningeal signs were apparent. Administration of lactated Ringer's solution was started at a rate of 125 mL/h; medical evacuation by airplane from their outlying atoll occurred approximately 72 hours later. Results of physical examination by the physician on the aircraft confirmed the above findings.

Due to the critical condition of the patients and the possibility that cerebral edema might be present, 250 mL of 20% mannitol solution was administered intravenously in a piggyback manner at a rate of 500 mL/h while the patients were in the aircraft. One patient stood up and asked for orientation within ten minutes after mannitol therapy was started; the other patient, though confused, sat up after five minutes. Both were brought to Armer Ishoda Memorial Hospital in Majuro and were discharged fully recovered in 48 hours.

CASE 3.—A 29-year-old Marshallese man presented to the emergency department with a 20-hour history of watery, nonbloody, hourly diarrhea not associated with tenesmus. These symptoms occurred after he ingested a large portion of cooked red snapper (*L bohar*). He had circumoral tingling and a burning sensation in his throat when he drank water and burning in his hands when he touched water, but there were no other neurosensory complaints. Similar symptoms developed in two other members of his household, but they were not seriously ill.

His vital signs were normal, but dizziness and profuse diaphoresis occurred ten minutes after the initial examination. Blood pressure was 90/60 mm Hg, with a thready pulse rate of 120 beats per minute. Lactated Ringer's solution was given intravenously in large amounts. The patient's blood pressure dropped to 70/40 mm Hg, and, because vasopressors were unavailable, 0.5 mL of 1:1000 norepinephrine was given intravenously. His systolic blood pressure remained at 70 mm Hg at first, but his blood pressure then dropped to 60/0 mm Hg. Because we understood the potential danger of giving a potent diuretic to a patient in shock and because we attributed the cause of the fall in blood pressure to ciguatoxin, mannitol was given intravenously at the prescribed rate.

The patient's blood pressure rose to 70/40 mm Hg within one minute of infusion, was 90/60 mm Hg in five minutes, and stabilized at 110/60 mm Hg in 15 minutes. Administration of all other fluids was stopped when the systolic blood pressure reached 100 mm Hg. The circumoral tingling disappeared. The frequency of diarrhea decreased to three episodes in 24 hours, and the burning sensation on touching water ceased. He was observed for 24 hours and then left the hospital against medical advice. He received a total of 250 mL of 20% mannitol and 1750 mL of intravenous fluids during this event.

Clinical Features of 24 Patients With Ciguatera Fish Poisoning

Sign or Symptom	No. of Patients
Neurologic or neurosensory	
Circumoral or facial paresthesias and numbness	23/24
Paresthesias in the extremities	23/24
Reversal of temperature sensation (cold to hot)	19/24
Paresis	4/24
Coma	2/24
Gastrointestinal	
Abdominal pain or cramping	10/24
Watery diarrhea without blood	9/24
Nausea	8/24
Vomiting	8/24
Musculoskeletal	
Muscle pain in the extremities	18/24
Arthralgia	18/24
Cardiovascular	
Hypotension	3/24

Results

To date, of a total of 38 patients evaluated, 24 patients with ciguatera fish poisoning have been treated with intravenous mannitol. They ranged from 6 to 66 years old, with nine women and 15 men. Twenty-two were Marshallese or part Marshallese. One was of Japanese ancestry, and one was of Filipino ancestry.

Of these patients, ten were admitted to the hospital; 14 patients were treated in the emergency department and then released after a period of observation and stabilization. The clinical features of the patients are given in the Table. Eighteen of the 24 patients were released from the emergency department or the hospital within 24 hours. Four were hospitalized for 48 hours and two for seven days.

The mean time from the onset of symptoms to the time mannitol was administered was ten hours in the 21 patients who lived in the center of the district. The three patients from the outlying atolls were treated with mannitol within 82 hours after the onset of symptoms.

All neurologic and neurosensory manifestations responded to mannitol within ten minutes, with complete resolution within 48 hours in 17 of 23 cases. Recurrent circumoral paresthesias after discharge occurred in one patient and lasted approximately 36 hours. The

mean time for total resolution of neurologic and neurosensory symptoms in all cases was ten hours.

In three cases, nausea, vomiting, and abdominal pain resolved within ten minutes. Diarrhea decreased in frequency in all nine cases over the 24 hours following treatment. Total resolution of the diarrhea occurred within 96 hours in all cases.

Neurologic symptoms recurred in five of 23 cases after discharge. One of these patients required re-treatment. Watery diarrhea persisted in three cases at a much decreased frequency of no more than three loose stools per day.

One case of clinical shock stabilized within minutes after the patient was given 250 mL of mannitol.

There were no apparent complications from using mannitol. Hypotension or significant diuresis did not occur.

There were no significant correlations between any of the clinical variables and the rapidity of the patients' response to therapy, nor did the severity of the illness relate to the ultimate outcome. Neurologic symptoms and signs seemed to reverse more quickly than did gastrointestinal complaints. Identifiable fish were those found commonly in the Marshall Islands; no preponderance of any particular fish was observed.

Comment

Ciguatoxin belongs to a newly described class of toxins that act on sodium channels[4,8,11] and cause changes in the electrical potential and permeability of cells. This may explain many of the clinical manifestations of the disease.[4,5] Although ciguatoxin is the predominant toxin in ciguatera fish poisoning, other compounds have been associated with the disease; this may account for the variability in the clinical presentation. Scaritoxin and maitotoxin produce both peripheral and central effects as well as cholinergic and α-adrenergic actions in animal models. Low doses cause hyperventilation, bradycardia, atrioventricular conduction defects, tachycardia, and transient hypertension.[5]

Our index patients were given mannitol because cerebral edema was suspected. The subsequent dramatic clinical improvement suggests that mannitol may have a valuable therapeutic effect on this disease. Each patient treated with mannitol experienced sudden, marked improvement in clinical status. In each patient, it was not likely that any other agent was responsible for the beneficial change in clinical course.

The mechanism of action of mannitol is obscure, but two attractive possibilities are that competitive inhibition occurs at the cell membrane or that one or more of the involved toxins is rendered inert. Since some of the symptoms return after treatment, it is possible that there is further ciguatoxin absorption from the gastrointestinal tract and that the mannitol molecule is displaced. Treatment with mannitol, even if patients were severely debilitated and comatose, shortened the time of significant morbidity to less than 48 hours and also shortened hospital stays significantly compared with our experience with patients not treated with mannitol. Eighteen of 24 patients deemed to have significant morbidity were discharged within 24 hours. Both comatose patients were discharged in 48 hours.

Mannitol is both inexpensive and safe. None of our patients required more than 250 mL of 20% mannitol given intravenously; the rate of infusion in all cases was 1 g/kg given over 30 minutes in a piggyback manner. Since many endemic areas of the world are remote, with only minimal medical care facilities, this form of treatment is both practical and realistic.

We recognize that our assessment of mannitol in ciguatera fish poisoning is observational and empiric. It has not been scientifically controlled by a randomized prospective study. Thus, there is a need for considerable caution in recommending any new therapy for this distressingly common problem. A controlled assessment of intravenous mannitol with precisely defined end points clearly is necessary. Until such a study is available, however, we believe that mannitol should be considered for initial use in patients with significant clinical signs or symptoms of ciguatera fish poisoning.

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12 November 1990

PLEASE ADDRESS REPLY TO
THE SECRETARY-GENERAL

BALAZS G.H.
NATIONAL MARINE FISHERIES SERVICE
2570 DOLE ST. - HONOLULU - HAWAII 96822-2396
USA

Dear Sir/Madam

Several previous SPC Regional Technical Meeting on Fisheries have noted the importance of ciguatera fish poisoning to countries of the region. This importance appears to be growing. Coastal development programmes that result in environmental damage or change (causeways, reef blasting), pollution (poisoning of corals, eutrophication caused by excess nutrients), destructive fishing methods (bleach and dynamite fishing) and natural disasters (cyclones, El-Nino-associated changes in water temperature) and accidents such as shipwrecks continue to provoke sporadic ciguatera outbreaks or chronic ciguatoxicity in an apparently increasing number of incidents.

Consequently the Fisheries Programme will accelerate the establishment of a Special Interest Group (SIG) on ciguatera, and with the support of all other sections, will prepare an Information Bulletin on the topic.

Following the SPC Workshop on Inshore Fishery Resources, we sent a questionnaire reflecting the subject areas covered by the Workshop. You marked with an **A**,**B** or **C** the topic ciguatera, showing your interest in participating to the elaboration of this bulletin.

To make this bulletin worthwhile, we would stress that it is **extremely important** for members to keep us informed of their activities with the aim to supply items such as: research activities, export marketing problems...

I thank you for any assistance you can provide, I look forward to hearing from you.

Best regards

Jean-Paul Gaudechoux
Fisheries Information Officer

The Health State

HAWAI'I DEPARTMENT OF HEALTH

Health Promotion & Education Branch
Epidemiology Branch

Photos of Wahanui & Mullet
© Kathie Mullins-Rutt.
Special thanks to
Randy Honebrink &
Annette Tagawa
Department of Land and
Natural Resources,
Division of Aquatic Resources
for use of all other photos.

John Waihee, *Governor*
John C. Lewin, M.D., *Director of Health*

Form CH10/92 • Printed on Recycled Paper

Ciguatera
FISH POISONING

Ciguatera fish poisoning is a serious problem in Hawai'i. The poison is found in many reef fish. Poisoning is caused by a toxin found in certain algae eaten by reef fish. The poison accumulates when reef fish are eaten by larger fish, and then by humans.

Hawai'i Department of Health

To avoid ciguatera fish poisoning you should:

- know the most common types of reef fish that carry the poison
- know that any reef fish can carry the poison
- know where cases of ciguatera fish poisoning have been reported
- clean the fish very well
- eat only small portions of large fish which might have the poison
- not eat the roe (eggs), liver, head or guts because they have higher levels of the poison

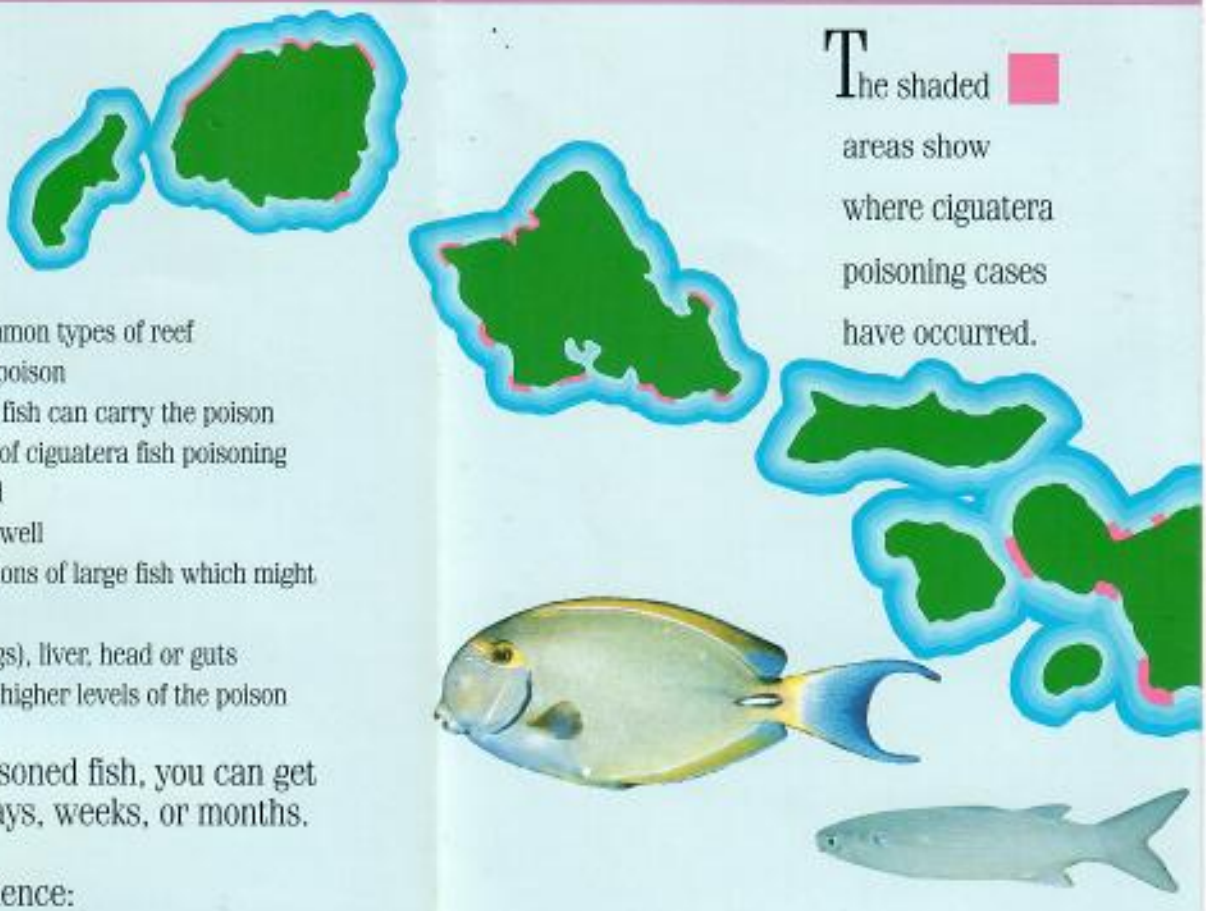
If you eat a poisoned fish, you can get very sick for days, weeks, or months.

You may experience:

- weakness
- diarrhea
- muscle pain, joint aches
- numbness and tingling around your mouth, hands, and feet
- temperature reversal (cold feels hot; hot feels cold)
- nausea, chills, itching, headache, sweating, and dizziness

If ciguatera fish poisoning is suspected, contact your doctor and the Hawai'i Department of Health immediately. Save any uneaten portions of the fish for testing.

The shaded ■ areas show where ciguatera poisoning cases have occurred.



Always report a case of suspected ciguatera fish poisoning to the Hawai'i Department of Health, Epidemiology Branch.

O'ahu	586-4586
Maui	243-5288
Hawai'i	933-4539
Kaua'i	241-3495

None of the deep-sea fish such as Ahi and Aku (Tuna), Kajiki (Marlin), Mahimahi (Dolphinfish), and Ono (Wahoo) have been found to carry the ciguatera fish poison.

Ciguatera

FISH POISONING

These fish have been found to carry the ciguatera poison:

Palani
Surgeonfish
length up to 18 i
weight up to 3 p

Wahanui
Snapper
length up to 2 feet,
weight up to 2 pounds.

Kole
Surgeonfish
length up to 7 inches,
weight up to 1/2 pound.

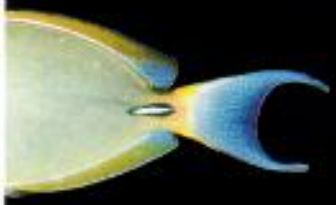
'Ū'ū
Menpachi
length up to 14 inches,
weight up to 1 pound.

Uku
Snapper
length up to 24 inches,
weight up to 50 pounds.

Puhi
Moray Eel
length up to 6 feet,
most under 2 feet,
weight up to 70 pounds.

Weke
Goatfish
length up to 18 inches,
weight up to 2 pounds.

Kākū
Barracuda
length up to 6 feet,
weight up to 70 pounds.



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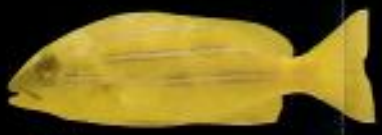


Ta'ape
Snapper
length up to 15 inches,
weight up to 1-1/2 pounds.

Po'ou
Wrasse
length up to 2 feet,
weight up to 2 pounds.

Ulua
Mature Jack
ength over 5 feet,
eight up to 120 pounds.

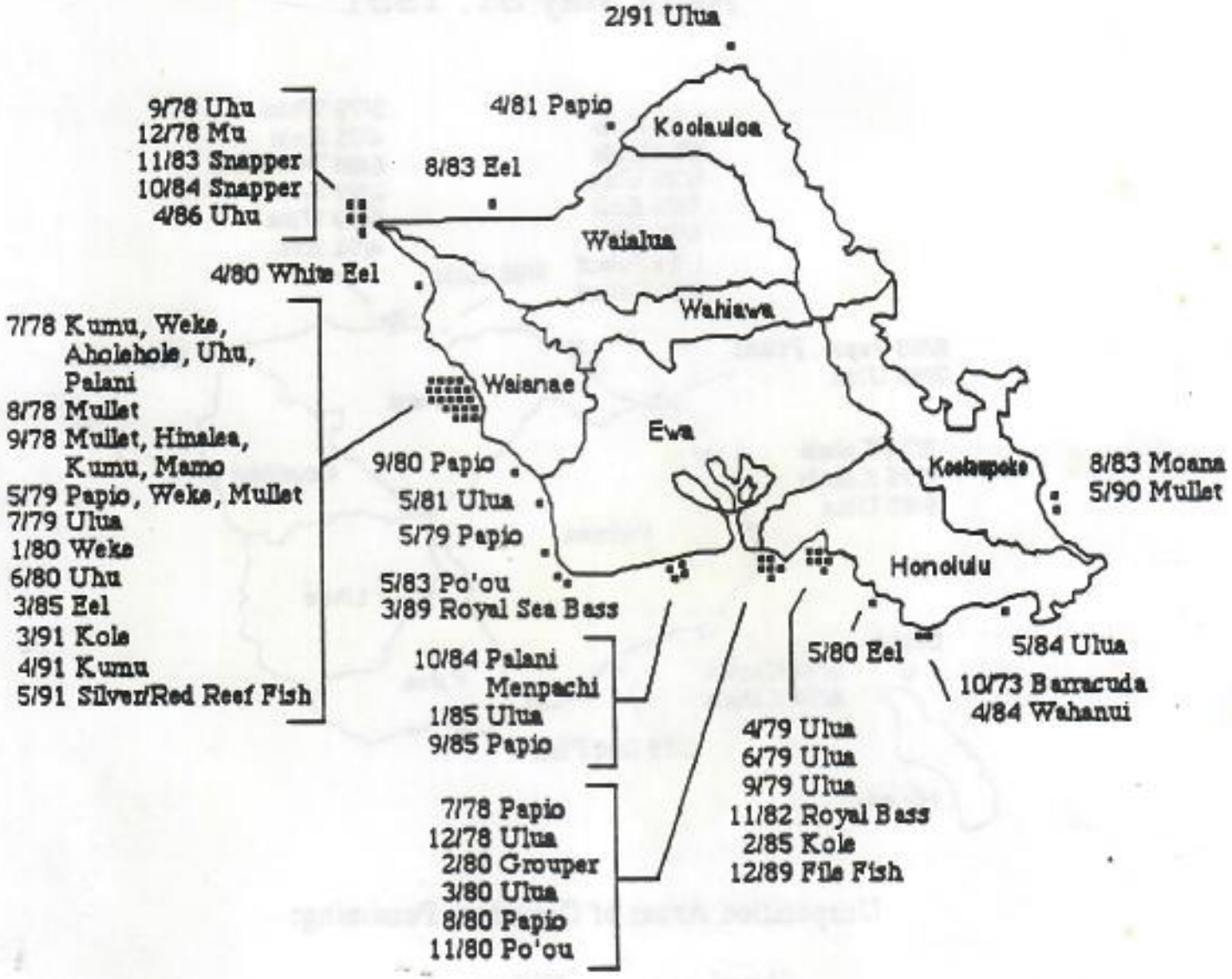
Pāpio
uvenile Jack
ength up to 4-8 inches,
eight up to 10 pounds.

'Ama 'Ama
Mullet
length up to 2 feet,
weight up to 5 pounds.

Kāhala
Amberjack
length up to 6 feet,
weight up to 120 pounds.

Roi
Grouper
length up to 20 inches,
weight up to 5 pounds.

Incidents of Ciguatera Poisoning from 1973 - 1991 Islands of Oahu As of May 31, 1991

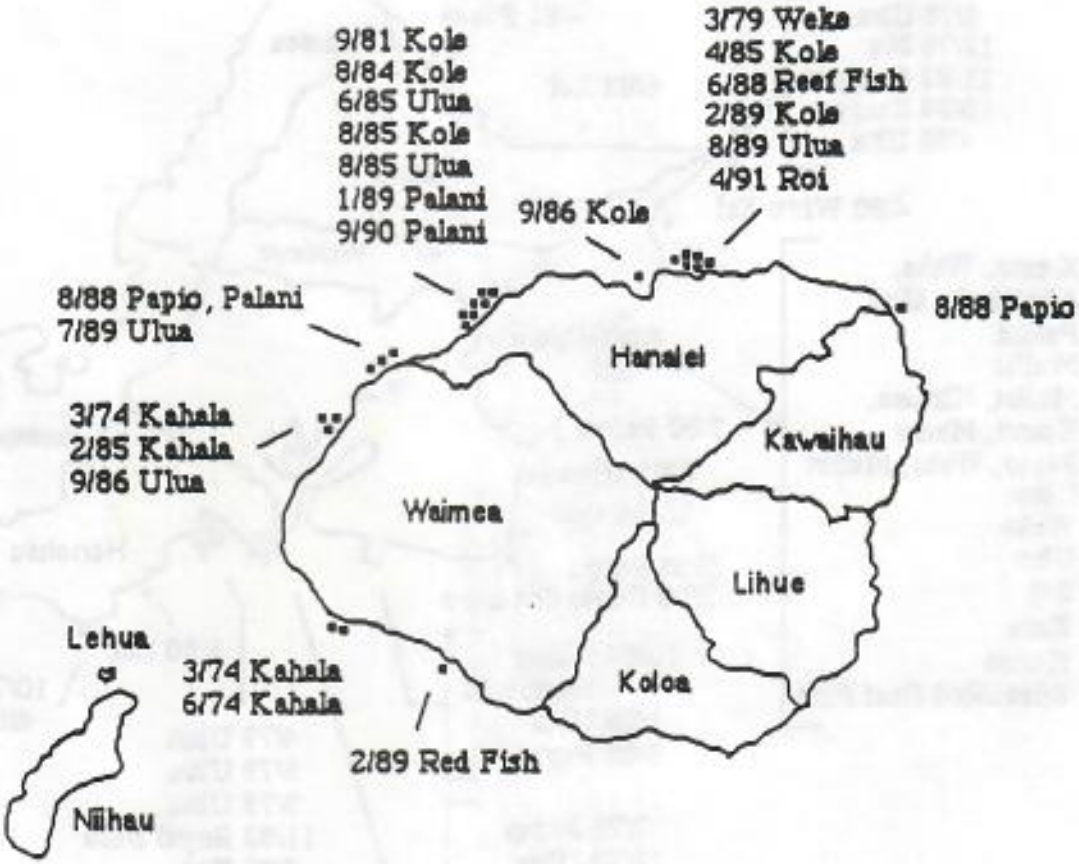


Unspecified Areas of Ciguatera Poisoning in Waianae:

1/79 Palani	2/85 Weke
8/79 Papio	3/85 Eel
10/79 Ulua	9/86 Snapper
7/82 Snapper	2/91 Mullet
8/84 Palani	3/91 Palani

• Represents 1 fish

Incidents of Ciguatera Poisoning from 1971 - 1991 Islands of Kauai and Niihau As of May 31, 1991

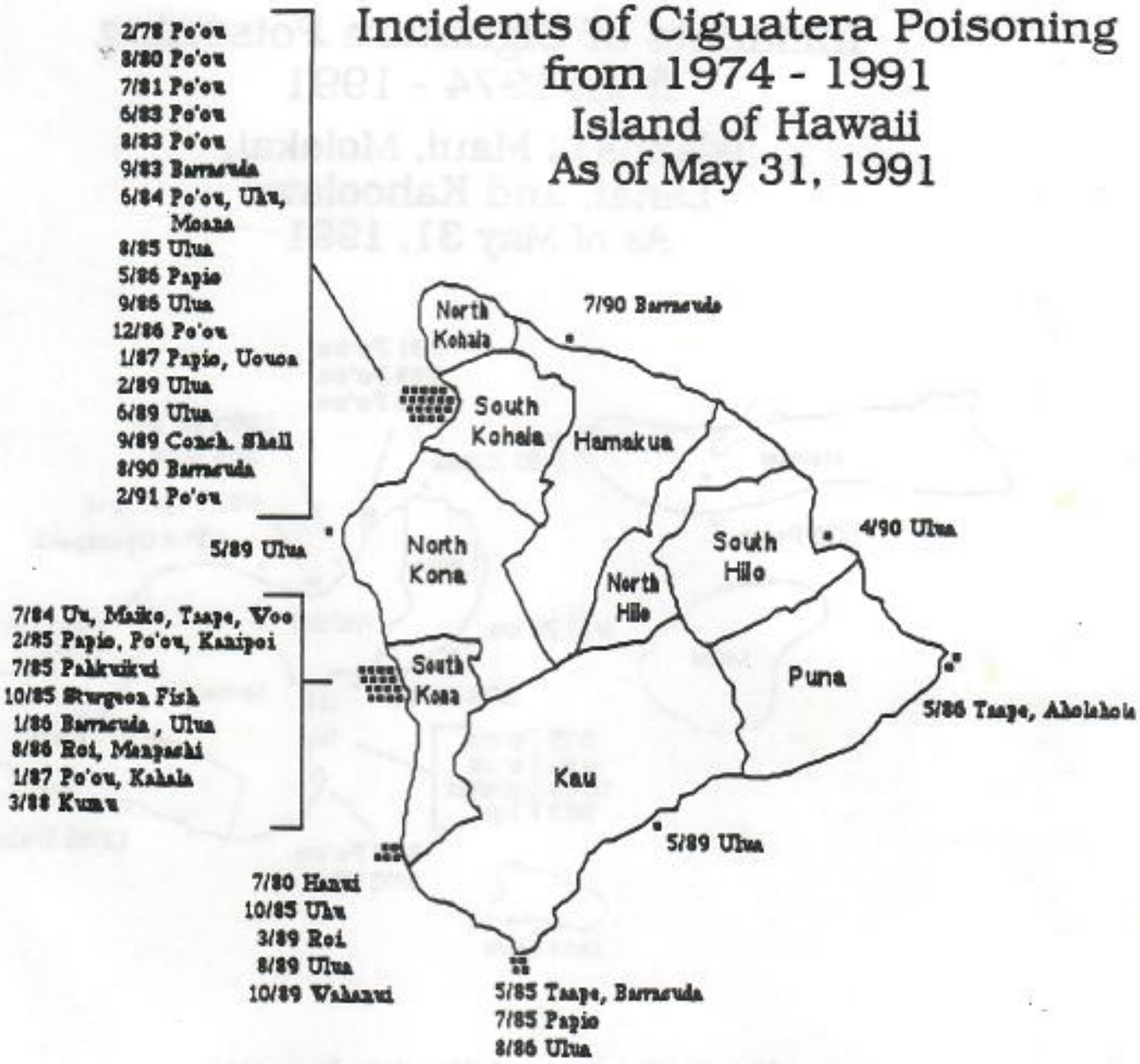


Unspecified Areas of Ciguatera Poisoning:

Kauai	Niihau
4/74 Weke	5/71 Kahala
11/77 Kahala	4/79 Kahala
1/79 Kahala	12/85 Weke Ule

▪ Represents 1 fish

Incidents of Ciguatera Poisoning from 1974 - 1991 Island of Hawaii As of May 31, 1991



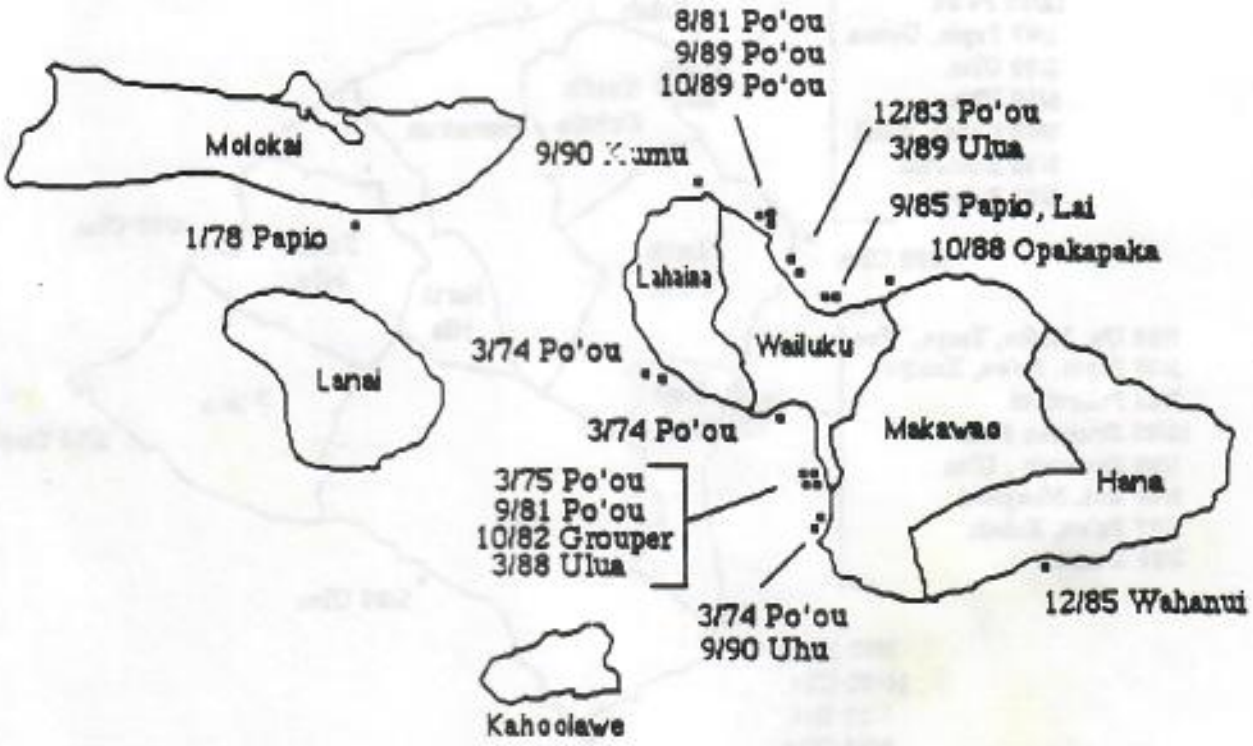
Unspecified Areas of Ciguatera Poisoning:

North Kohala	South Kohala	North Kona	South Kona	Kona	Puna	Hawaii
6/87 Wahanui	3/87 Ulua	6/87 Papio	11/83 Kole	3/74 Kahala	6/83 Ulua	11/89 Grouper
8/87 Kawelas		4/88 Ulua	4/84 Omilu	8/84 Papio, Mu		
10/87 Kole			9/84 Po'ou, Roi	7/85 Sea Bass, Manpachi		
			10/84 Kole	1/86 Roi		
			12/84 Manini	4/91 Sea Bass, Kole		
			1/85 Po'ou			
			1/87 Kahala			
			4/87 Uhu, Manpahi			
			6/87 Kole, Maiko			
			7/87 Roi			
			11/87 Weke			

▪ Represents 1 fish

Incidents of Ciguatera Poisoning from 1974 - 1991

Islands of Maui, Molokai, Lanai, and Kahoolawe As of May 31, 1991



Unspecified Areas of Ciguatera Poisoning:

Molokai	Lanai	Kahoolawe	Hana	Maui
4/80 Ulua	11/90 Roi, Po'ou	4/75 Po'ou	10/84 Po'ou	8/87 Tako
7/81 Weke		2/91 Roi		
8/82 Barracuda				
3/84 Papio				
6/89 Sea Bass				
7/89 Ulua				
9/90 Kole				
10/90 Uhu				

• Represents 1 fish

Communicable Disease Report

SEPTEMBER 1988

CIGUATERA POISONING IN HAWAII

Ciguatera poisoning is an illness which is transmitted by the consumption of fish contaminated with ciguatoxin or CTX. The toxin originates in a small dinoflagellate, *Gambierdiscus toxicus*. These dinoflagellates grow on the surface of macro-algae which are eaten by herbivorous fish. The toxin becomes concentrated in the viscera of the herbivorous fish; these fish are then eaten by carnivorous fish and eventually the toxin travels up the food chain to humans.

Ciguatera poisoning has been noted to occur throughout the Caribbean and the South Pacific (primarily in the latitudes south of Hawaii). It is endemic to Hawaii and Florida. Other cases of ciguatera poisoning have occurred in the Mainland, U.S., but these cases are attributed to fish that were imported from areas where ciguatoxin is endemic. Ciguatera poisoning is not a new disease; it has been described as early as the 17th century and was known to have affected the crew of Captain James Cook in 1774. The term ciguatera was first used in 1787 in a technical treatise published in Cuba by Don Antonio Parra, a Portuguese biologist. The name was derived from "cigua," a local name in the Caribbean for the marine snail *Turbo pica*, which causes similar symptoms to ciguatera poisoning when it is ingested.[1]

The reef fish in Hawaii (both omnivorous and carnivorous) such as the jacks, wrasses, barracudas, groupers, and eels are most frequently implicated in outbreaks of ciguatera poisoning. Locally, these reef fish are known as ulua, papio, kahala, po'ou, kaku, opakapaka, puhe, tohei, and hupu upuu. The deep water fish, ahi, aku, and mahi mahi (the tunas and dolphinfish) have not been implicated in outbreaks of ciguatera poisoning.

In addition to the above more commonly encountered fish, cases of ciguatera poisoning have occurred from consumption of palani, mu, ta'ape, weke, waha nui, moano, maiko, menpachi, roi, kole, lai, ala'ihi, and paku'i ku'i..

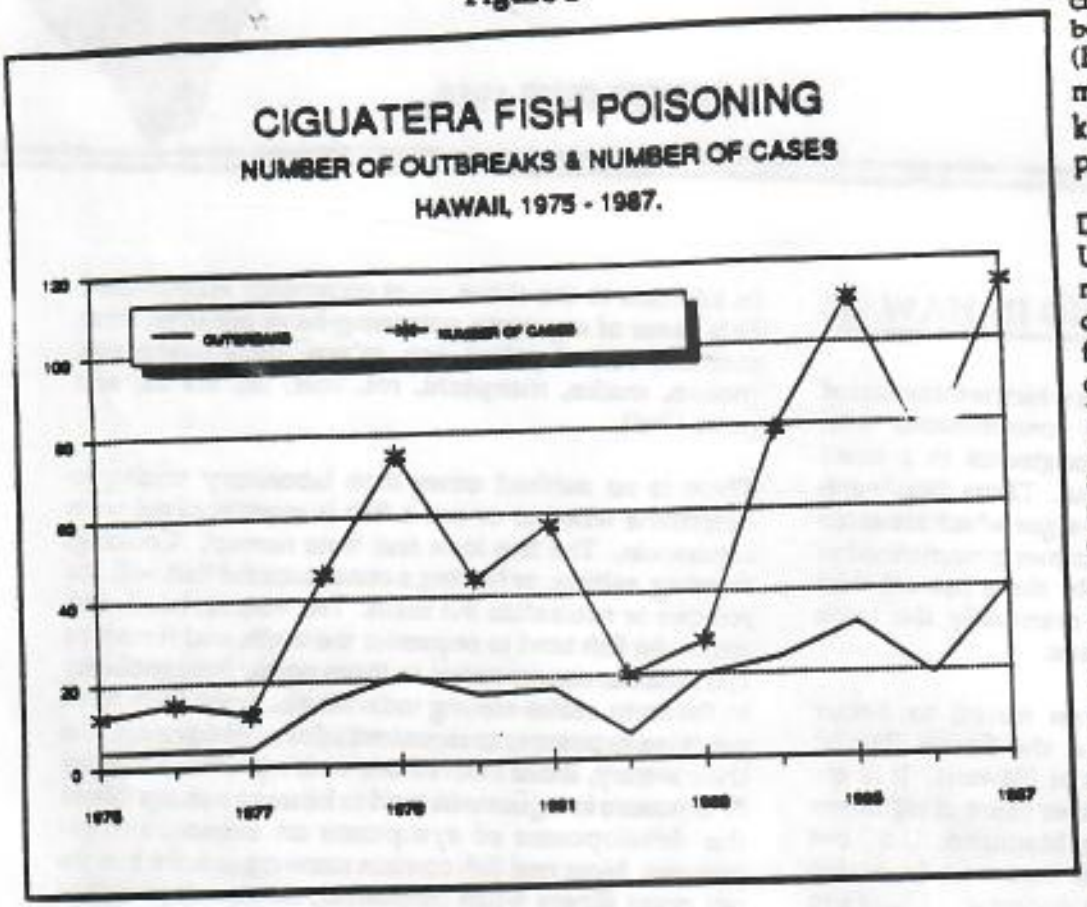
There is no method other than laboratory testing to determine whether or not a fish is contaminated with ciguatoxin. The fish look and taste normal. Cooking, freezing, salting, or boiling a contaminated fish will not remove or neutralize the toxin. The viscera, head, and roe of the fish tend to sequester the toxin, and it may be 100 times as concentrated in these parts. Susceptibility to the toxin varies among individuals. Immunity from previous exposures to ciguatoxin does not develop. On the contrary, those individuals with a previous history of exposure to ciguatoxin tend to be more susceptible to the development of symptoms on subsequent exposures. Most reef fish contain some ciguatoxin but do not cause illness when consumed; the development of symptoms is a factor of both host susceptibility and the amount of ciguatoxin that is consumed.

There are a multitude of symptoms and symptom complexes which occur from the ingestion of ciguatoxin. Typically, they involve the nervous system and the gastro-intestinal tract. The incubation period ranges from 2 to 30 hours with a mean of 6 hours. Usually, vomiting and diarrhea are the first symptoms, followed by myalgia and weakness. Approximately 18 hours after ingestion of the ciguatoxin, paresthesias and dysesthesias of the perioral region and extremities develop. Intense pruritus often occurs 24 hours after the onset of the first symptoms. The duration of GI symptoms is usually 24 hours. Two to five days after the initial onset of symptoms, temperature reversal may occur where cold objects feel warm and vice-versa. Hypotension, hypovolemia, coma, or death occur rarely.[2] The case fatality rate is approximately 0.1% but

Hawaii State Dept. of Health — Epidemiology Branch

P.O. Box 3378 Honolulu, Hi. 96801 (808) 548-6986

Figure 1



In Hawaii, the incidence of ciguatera fish poisoning has been gradually increasing (Figure 1). The most commonly implicated fish are the kahala, ulua, papio, and po'ou.

Dr. Yoshitsugi Hokama of the University of Hawaii, Department of Pathology has developed an ELISA stick-test for the assessment of the ciguatoxin content of fish.[5] This test is not available for commercial use, however, sport fishermen may submit fish to Dr. Hokama for ciguatoxin testing.

Routine testing of commercial fish does not appear to be cost-effective. The occurrence of fish contaminated with ciguatoxin is sporadic; a single toxic fish may be in close proximity to other non-toxic fish which would make accurate sampling difficult.

varies in different regions. Symptoms usually persist for at least one week and may last as long as several months in some individuals.

Tricyclic antidepressants, atropine, calcium, and non-steroidal anti-inflammatory drugs have been shown to be beneficial in treating symptoms of ciguatera poisoning[3]; there is no known antitoxin. In a recent study by Palafox et. al., remarkable recovery was observed in 24 patients affected by ciguatoxin who received intravenous mannitol.[4]

Consumption of sea foods, nuts, alcoholic beverages, and sesame oil has been known to exacerbate symptoms of ciguatera poisoning and prolong the duration of illness.

The Department of Health, Epidemiology Branch is rently involved in surveillance of ciguatera fish poi ing outbreaks. Please report all suspected case ciguatera fish poisoning to the Epidemiology Brancn at 548-5986.

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ROBERT M. WORTH, MD, PhD, Chief, Communicable Disease Division
EUGENE PON, MD, MPH, Chief, Epidemiology Branch
RICHARD I. FRANKEL, MD, Chief, Tuberculosis Branch
ERIDA REICHERT, MD, PATRICK HARTMAN, MD, and RICHARD I. FRANKEL, MD, Directors, Hansen's Disease Branches
DEWEY V. STURGES, PhD, Director, AIDS/STD Project

Submitted by James H. Collop, MD, Preventive Medicine Resident, Communicable Disease Division.

State of Hawaii

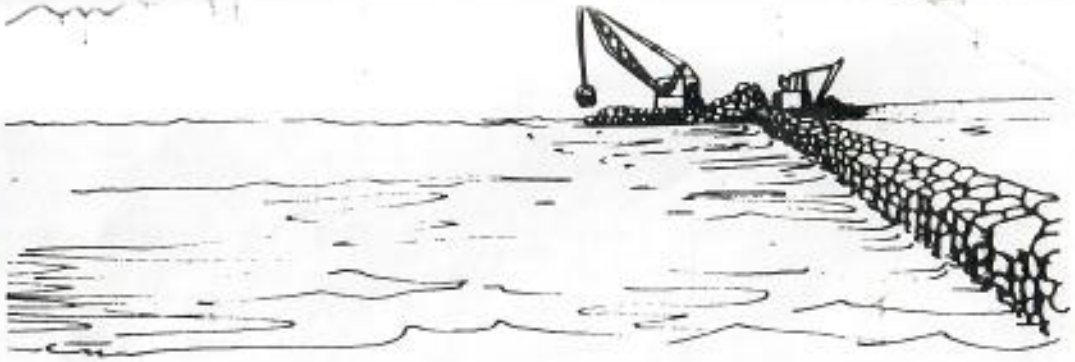
John Waihee, *Governor*

John C. Lewin, M.D., *Director of Health*

Prepared by

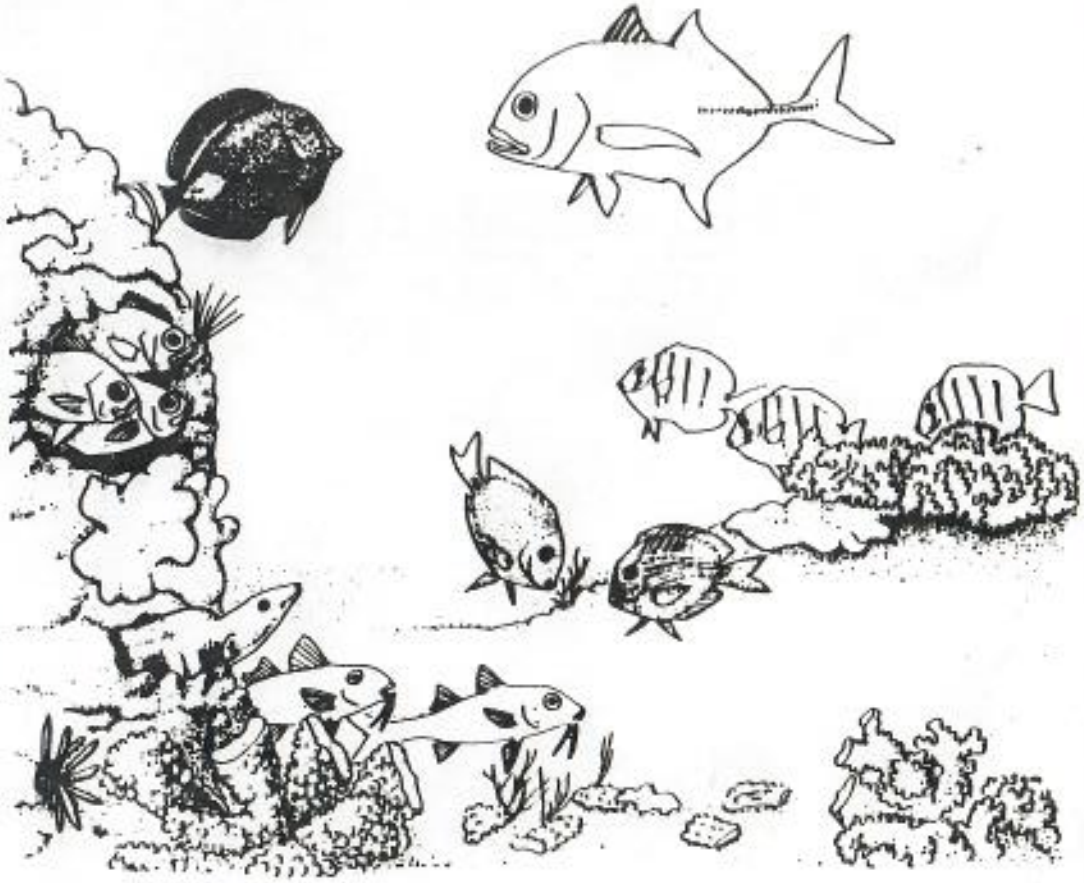
The Environmental Epidemiology Program

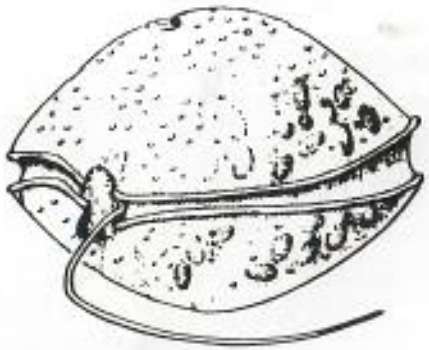
April 1, 1987



Fish Poisoning

in Hawai'i





Gambierdiscus toxicus
(Magnified 450 times)

t Fish poisoning can result from eating spoiled fish r from eating fresh fish containing toxic substances. here are various types of fish poisoning. Some types re associated with specific fish, for example hal- cinogenic fish poisoning (mullet, manini, nenue or eke) and puffer fish poisoning (puffer fish or mola). combroid fish poisoning occurs when certain fish nahimahi, 'ahi or aku) are eaten after they have poiled, usually because of improper refrigeration or torage. Ciguatera fish poisoning is a natural occur- nce still not well understood.

Ciguatera Poisoning

Ciguatera poisoning is thought to have been unknown to early Hawaiian fishermen. Until recently most cases of ciguatera fish poisoning involved fish from other areas of the Pacific, such as Midway or Johnston Island. Today in Hawai'i, ciguatera fish poisoning is a serious problem for recreational fishermen and the fishing industry.

Many species of fish that are highly esteemed as food have been implicated in causing serious illness. Ulua (jack), kahala (amberjack), kole (surgeon fish) and po'ou (wrasse) have been most often responsible for ciguatera poisoning in Hawai'i. Many other reef fish have been found to be toxic.

The toxin(s) is produced by microscopic marine organisms, a dinoflagellate called *Gambierdiscus toxicus*. These organisms, which grow on the surface of marine algae, are eaten by herbivorous fish. Since the toxin accululates in fish, any fish up the food chain can become toxic. The kole and palani (surgeon fish), uhu (parrotfish) and others that feed directly on the algae or coral reefs upon which the algae grows may be toxic. Fish that prey on these herbivores, ulua, kahala, uku (snapper) and eel may be even more toxic because of the cumulative effect. None of the deep-sea fish, 'ahi and aku (tuna), marlin, mahimahi and ono (wahoo), have been found to be ciguatoxic in Hawai'i to date.

Unfortunately, there is no way of detecting a ciguatoxic fish from its appearance, smell or taste. The fish seem to be unaffected by ciguatoxin. The freshness of a fish has no relationship to its toxicity. Furthermore, the toxin is not altered by cooking, drying, salting or freezing the fish.

However, to minimize the risk of being poisoned, **do not eat the roe (eggs), liver or guts of any reef fish.** The toxin is concentrated up to 100 times more in these parts of the fish. In Hawai'i, several deaths resulted from eating the viscera of toxic fish. For this reason it is recommended that all reef fish be promptly and thoroughly cleaned.

Symptoms of ciguatera poisoning vary greatly from one individual to another, even when the same amounts of the same fish have been eaten. Illness usually occurs within three to five hours after eating a toxic fish.

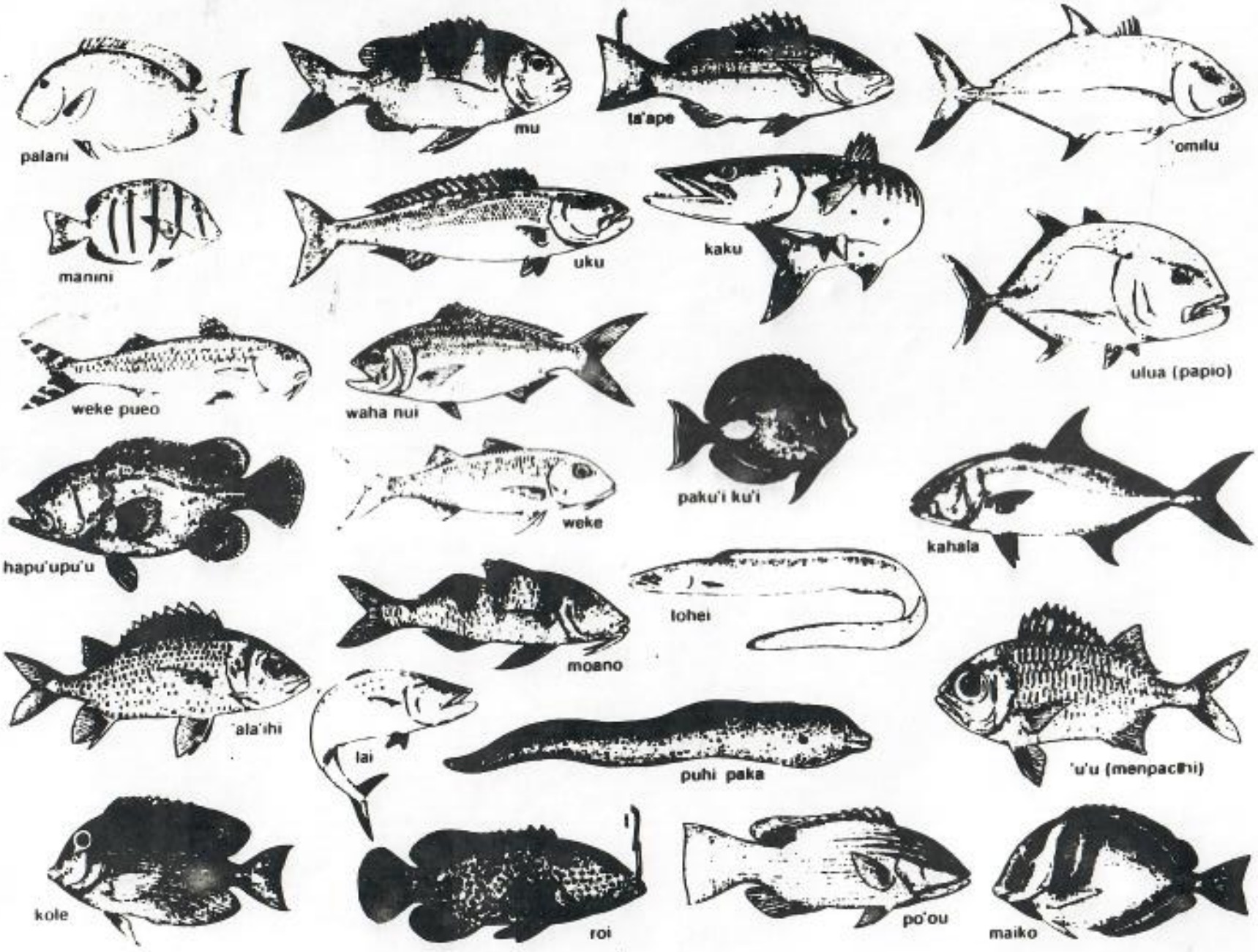
Common symptoms of illness may include the following:

- **general weakness**
- **diarrhea**
- **muscle pain, joint aches**
- **numbness and tingling around the mouth, hands and feet**
- **reversal of temperature sensation, where cold objects feel hot (a burning or tingling sensation may also be felt) and hot objects feel cold**
- **others, such as nausea, vomiting, chills, itching, headache, sweating and dizziness.**

The temperature reversal sensation phenomenon usually does not develop until two to five days after eating a toxic fish. Illness may last for weeks or months, rarely years.

When ciguatera poisoning occurs, the affected individual should avoid consuming fish or shellfish products, alcoholic beverages and nut or seed products. These may increase the severity and/or duration of illness.

HAWAIIAN WATERS FISH SPECIES IMPLICATED IN CIGUATERA FISH POISONING INCIDENTS



Scombroid Fish Poisoning

Scombroid poisoning results from eating spoiled fish, primarily tuna, mahimahi and related species. Imported mahimahi has been most often associated with scombroid poisoning in Hawai'i. The disease is sometimes misdiagnosed as "fish allergy."

Inadequate or delayed refrigeration at sea results in the overgrowth of various bacteria normally found in these fish. The natural action of these bacteria in the fish flesh produces histamine, saurine and possibly other toxic substances. The fish may not have a foul odor. However, some victims have reported a sharp "metallic" or "peppery" taste while consuming the fish, believed to be due to the presence of histamine.

Symptoms may appear within a few minutes to several hours, usually within an hour after eating a spoiled fish. Symptoms vary widely between individuals but may include the following:

- flushing of the face, resembling a sunburn, sometimes involving the neck, arms, and upper part of the trunk
- severe throbbing headache
- palpitations of the heart
- abdominal cramps
- diarrhea

Other symptoms may include itching on the face or around the mouth, a burning sensation in the throat, dryness of the mouth, difficulty in swallowing and/or breathing, nausea (rarely vomiting) and weakness.

Symptoms usually last for eight to 12 hours, after which rapid recovery is expected. No fatalities have been recorded from scombroid poisoning to date in Hawai'i, and they appear to be extremely rare worldwide. Scombroid fish poisoning has been successfully treated with antihistamines.

Other Types of Fish Poisoning

Other types of fish poisoning occur less frequently than ciguatera or scombroid poisoning. These include intoxications usually associated with specific types of fish.

"Hallucinogenic fish poisoning" is associated with mullet and a number of other species of fish including weke (goatfish), weke 'ula, weke puaa, nenue (rudderfish) and manini (surgeon fish). It is seasonal, occurring usually in the summer months from the islands of Kaua'i, O'ahu and Moloka'i. Hallucinations, insomnia, intense dreaming, weakness, general malaise, dizziness, itching and burning of the throat and other symptoms are common soon after ingestion. Terrifying nightmares have been reported when the onset of symptoms occurs while asleep. Constrictive chest pains can also occur.

Puffer Fish Implicated in Seven Fatalities in Hawai'i

"Puffer fish" poisoning or "tetrodotoxication" may result from eating puffers, blowfish, balloon fish, 'o'opuhue, makimaki, keke and others. Many species of puffer fish contain at least one very potent toxin, tetrodotoxin. Initial symptoms may occur within minutes and include sweating and tingling of the lips, tongue and finger tips, followed by numbness that may spread throughout the body. Extreme weakness associated with nausea, vomiting, headache, profuse sweating and other symptoms have also been reported. In severe cases, respiratory and muscular paralysis may ensue and may be followed by death. The most common puffer fish, or "fugu" (*Arothron hispidus*), has been implicated in at least seven fatalities in Hawai'i.

If fish poisoning is suspected:

- **CALL YOUR PHYSICIAN IMMEDIATELY FOR TREATMENT. If your physician is unavailable, call Hawai'i Poison Center at 941-4411. A physician's care is needed to remove unabsorbed toxins from gastrointestinal tract and to treat the manifestations of illness.**
- **Do not take any drug or medication without your physician's advice.**
- **Do not eat any remaining fish that could be toxic.**
- **Save the remainder of the fish (including the head and guts) in your refrigerator or freezer to be given later to the Department of Health. The Department of Health can assist in arranging for laboratory confirmation of suspected fish poisoning cases.**
- **Call the DEPARTMENT OF HEALTH to report a case of suspected fish poisoning.**

On O'ahu, call the Epidemiology Branch 548-5886

On the neighbor islands, call

Maui District Health Office 244-4288

Hawaii District Health Office 961-7278

Kauai District Health Office 245-4495

Your kokua in reporting cases will help to prevent further illnesses, and possibly deaths from occurring.

The Health State
HAWAII STATE DEPARTMENT OF HEALTH

Communicable Disease Report

FEBRUARY/MARCH 1991

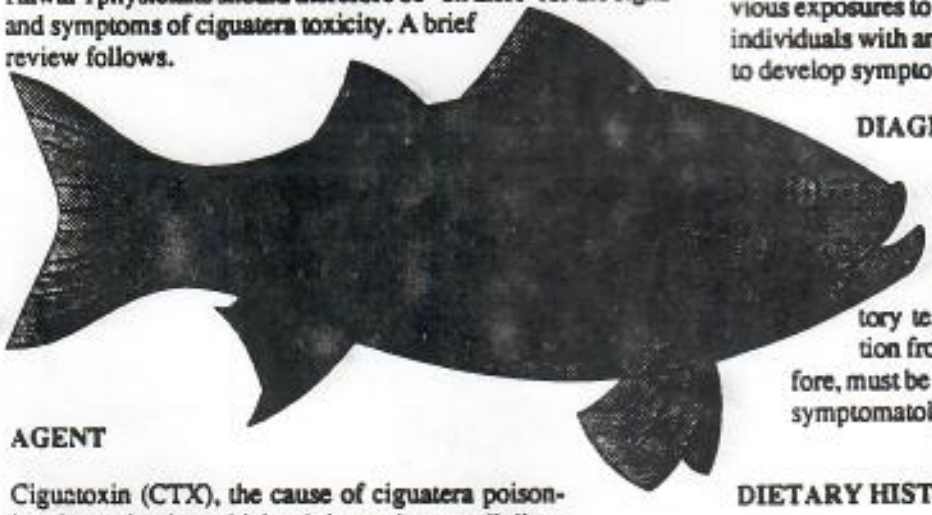
CIGUATERA ALERT

OUTBREAK SUMMARY

Between February 8 and 23, 1991, 35 reports of ciguatera fish poisoning were received at the Epidemiology Branch. Twelve separate incidents involved fish served at meals in private homes and local restaurants. Nine of the incidents were linked to po'ou (wrasse) caught off the Big Island by commercial fishermen. The others involved ulua (jack or crevally) and 'ama'ama (mullet) harvested by recreational fishermen off the Waianae coast.

Of the 35 cases, 14 were hospitalized due to the severity of illness, 11 of whom were placed in intensive care for monitoring dysrhythmias and hypotension. Ages of cases ranged from 1 to 69 years, with sexes equally distributed.

Ciguatera is the commonest cause of marine poisoning worldwide, with the highest incidence occuring in the tropics.[1,2] Hawai'i physicians should therefore be "on alert" for the signs and symptoms of ciguatera toxicity. A brief review follows.



AGENT

Ciguatoxin (CTX), the cause of ciguatera poisoning, is a polyether which originates in a small dinoflagellate, Gambierdiscus toxicus. It is extremely toxic (LD50 .45ug/kg in experimental animals).[3] The apparent mechanism of action is by the opening of voltage dependent sodium channels in mammalian nerve cells.

TRANSMISSION

CTX-producing dinoflagelates grow on the surface of microalgae which are eaten by fish. This toxin becomes sequestered in these fish, being concentrated up to 100 times in the head, viscera and roe. As these herbivores are eaten by carnivorous fish, the toxin travels up the food chain to humans. Illness occurs after the consumption of contaminated fish. These fish look, smell and taste normal. Laboratory testing is the only way to determine whether a fish is contaminated with CTX. Cooking, salting, boiling, or freezing the fish will not remove or neutralize the toxin.

SUSCEPTIBILITY

Most reef fish contain some ciguatoxin but at levels which do not usually cause illness when consumed. The development of symptoms is a function both of host susceptibility and of the amount of CTX ingested. Immunity, however, from previous exposures to ciguatoxin does not develop. In fact, those individuals with an earlier history of exposure are more likely to develop symptomatic illness with subsequent exposure.

DIAGNOSIS

CTX fish poisoning should be a consideration when presented with any episode of acute gastroenteritis or neurologic abnormality. Unfortunately, there is no laboratory test available to confirm ciguatera intoxication from human specimens. The diagnosis, therefore, must be made on a dietary history and corroborating symptomatology.

DIETARY HISTORY

A history of fish consumption within 2-48 hours before the onset of symptoms should be sought in all instances. If there is a history of fish consumption, the patient should be advised

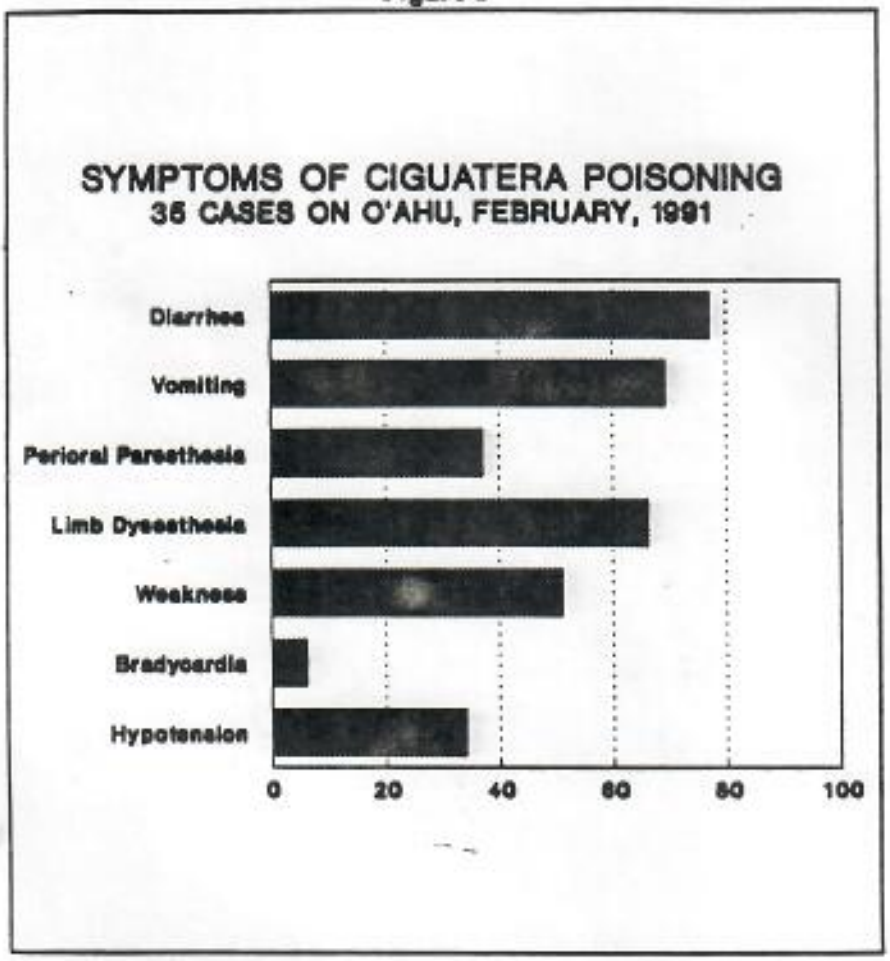
Hawaii State Department of Health, Epidemiology Branch
P.O. Box 3378 Honolulu, HI 96801 (808) 548-5986

to freeze and hold any remaining suspect fish. It may be possible to test the remnants for CTX.

HISTORY OF SYMPTOMS

The symptom complexes which occur from the ingestion of ciguatoxin typically involve the nervous system and the gastro-intestinal tract (Figure 1). The incubation period ranges from two to 30 hours with a mean of six hours. Usually, vomiting and diarrhea are the first symptoms, followed by myalgia and weakness. Approximately 18 hours after ingestion of the ciguatoxin, paresthesias and dysesthesias of the perioral region and extremities develop. Intense pruritus often occurs 24 hours after the onset of the first symptoms. The duration of GI symptoms is usually 24 hours. Two to five days after the initial onset of symptoms, temperature reversal may occur, where cold objects feel hot and vice-versa. Bradycardia, hypotension, or hypovolemia occur rarely but may be severe enough to warrant intensive care management. Symptoms usually persist for at least one week and may last as long as several months in some individuals. The case fatality rate is approximately 0.1%.[4]

Figure 1



TREATMENT

There is no definitive therapy for ciguatera intoxication at present. Management of cases remains supportive in nature. Tricyclic antidepressants, atropine, calcium and nonsteroidal anti-inflammatories have all been purported to alleviate various symptoms caused by CTX poisoning.[5]

Recently, several investigators have reported dramatic improvement in patients with CTX poisoning following administration of intravenous mannitol. After any necessary volume replacement, mannitol (1g/kg) was administered over a 30-60 minute interval. However, no placebo-controlled trials have been published to date which unequivocally document the efficacy of this new therapy.[6,7,8]

ADVICE TO PATIENTS

Following the resolution of an acute CTX poisoning some patients may experience an exacerbation of symptoms upon ingestion of certain food items. These dietary 'triggers' vary from patient to patient; but on the whole, patients should be advised to avoid the following for several months:

- fish and fish sauces
- shellfish
- alcoholic beverages
- nuts and nut oils

CONTROL and PREVENTION

Because the occurrence of fish contaminated with CTX is sporadic and somewhat unpredictable, routine testing of commercial fish is not cost effective. However, individual fish specimens can be tested with a Stick-Enzyme Immunoassay (S-EIA) at the University of Hawai'i to confirm the presence of ciguatoxin.[9]

ROBERT M. WORTH, MD, PhD, Chief, Communicable Disease Division
EUGENE PON, MD, MPH, Chief, Epidemiology Branch
RICHARD I. FRANKEL, MD, MPH Chief, TB/H.D. Control Branch
ERIDA REICHERT, MD and PATRICK HARTMAN, MD, Directors, Hansen's Disease Inpatient Programs
DEWEY V. STURGES, PhD, Director, STD/AIDS Prevention Branch

STD reporting: 735-5304
AIDS reporting: 735-5304 (Rodrigo Gillara)
Information and disease reporting: 548-5986
After-hours emergency reporting: 247-2191 (state operator)
Editor: Alix Gillam, MPH
Production: Audrey Arakawa and Alix Gillam, MPH

Because CTX accumulates in the head, viscera and roe of reef fish, consumption of these parts should be discouraged as a general preventive measure.

The Epidemiology Branch actively investigates reported cases of CTX poisoning to determine the species of fish responsible and the site of harvest. From this information, fishers can be warned of areas to avoid and types of fish likely to be toxic. Reports from physicians concerning potential cases of ciguatera are an essential component of the Health Department's efforts to control this serious health hazard. To report suspected fish poisoning cases call the Epidemiology Branch on O'ahu at 548-5986, Hawai'i at 933-4539, Maui at 243-5288 or Kaua'i at 241-3495.

Special thanks to the Queen's Medical Center Emergency Room Staff for their prompt reporting of suspect ciguatera fish poisoning cases. Their prompt reporting prevented the occurrence of additional cases in the above mentioned outbreak.

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Submitted by Dr. Paul Effler, Preventive Medicine Resident, Epidemiology Branch.

UPDATE: Malaria Prophylaxis with Mefloquine

CDC recommends mefloquine (Lariam®) alone as the drug of choice for malaria prevention for travelers to areas with drug-resistant *Plasmodium falciparum* malaria. Based on accumulating experience with this drug, the prophylactic dosing regimen has been revised to a single dose of mefloquine to be taken every week. The first dose should be taken one week before travel. It should be continued weekly during the entire period of travel in malarious areas and for four weeks after departure from such areas.

All studies confirm that mefloquine is well tolerated when used for prophylaxis. No serious adverse reactions to mefloquine prophylaxis (i.e., psychoses and convulsions) have been observed among Peace Corps volunteers or among 18,462 persons enrolled in prophylactic drug trials and surveys of travelers who were taking mefloquine weekly. However, serious adverse reactions have been reported, especially when mefloquine was used for treatment of patients with malaria. Because mefloquine has been used in the United States for only 18 months, monitoring of adverse reactions remains important. Physicians are encouraged to report serious adverse reactions in persons using mefloquine to CDC's Malaria Branch, Division of Parasitic Diseases, Center for Infectious Diseases; telephone (404) 488-4046.

Consistent with previous guidelines, mefloquine is not recommended for use by travelers with known hypersensitivity to mefloquine; children < 15 kg (30 lbs); pregnant women; travelers using beta blockers; travelers involved in tasks requiring fine coordination and spatial discrimination, such as airplane pilots; and travelers with histories of epilepsy or psychiatric disorder.

Travelers to areas of risk where chloroquine-resistant *P. falciparum* is endemic and for whom mefloquine is contraindicated, may elect to use daily doxycycline alone or chloroquine alone. If chloroquine is used, the traveler needs to be aware of the need to seek medical attention for febrile episodes and to carry a treatment dose of pyrimethamine-sulfadoxine (Fansidar®) to be used if medical care is not available within 24 hours of onset.

The CDC publication Health Information for International Travel (2) provides detailed information on the risk for malaria to travelers and on the presence of drug-resistant *P. falciparum* for each country. Health information for travelers is available 24 hours a day from the CDC automated telephone system at (404) 332-4555.

Periodic shortages of mefloquine have occurred in the United States. Travelers who cannot obtain mefloquine before departure may be able to purchase it in Europe while in transit to countries with endemic malaria. Prescriptions written in the United States are accepted at airport pharmacies in Frankfurt and Paris (both Charles de Gaulle and Orly airports). The pharmacy at Heathrow in London requires prescriptions written in Great Britain. At the airport pharmacy in Brussels, a prescription from the airport physician is required. Mefloquine is not available at the airports in Amsterdam and Rome and at Gatwick (London).

In some countries, a fixed combination of mefloquine and pyrimethamine-sulfadoxine marketed under the name Fansimef® should not be confused with mefloquine, and it is not recommended for prophylaxis of malaria.

FISHERIES INFORMATION PROJECT
SOUTH PACIFIC COMMISSION
PO BOX D5 - NOUMEA
NEW CALEDONIA

CIGUATERA

INFORMATION BULLETIN Number 1 - May 1991

Group Co-ordinator: Richard Lewis, Queensland Dept. of Primary Industries, Southern Fisheries Centre, P.O. Box 76, Deception Bay, Qld 4508, Australia.

NOTE FROM THE CO-ORDINATOR

The Ciguatera Information Bulletin is an important new initiative of the South Pacific Commission. This bulletin recognises the significance of the problem of ciguatera to many Pacific Island countries. It is hoped the bulletin will: (i) increase awareness and understanding of ciguatera in Pacific Basin countries; (ii) provide a forum for exchange of ideas on how ciguatera can be managed; (iii) facilitate more accurate reporting of outbreaks of ciguatera in the region; (iv) provide a vehicle for the documentation and dissemination of results of studies on ciguatera in the region; and (v) highlight important research findings forthcoming from laboratories investigating ciguatera throughout the world.

The first bulletin contains a wealth of practical information clearly describing ciguatera, reports on research findings and directions for two groups in the field. To those interested in ciguatera, please become a member of the Special Interest Group. Literature in the field (particularly 'grey literature') can be sent to SPC for inclusion in its already extensive bibliographic database and library.

Finally I want to encourage the submission of articles related to ciguatera to SPC (at the above address) for inclusion in forthcoming bulletins.

Richard J. Lewis

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PIMRIS is a joint project of 4 international organisations concerned with fisheries and marine resource development in the Pacific Islands region. The project is executed by the South Pacific Commission (SPC), the South Pacific Forum Fisheries Agency (FFA), the University of the South Pacific's Pacific Information Centre (USP-PIC), and the South Pacific Applied Geoscience Commission (SOPAC). Funding is provided by the International Centre for Ocean Development (ICOD) and the Government of France. This bulletin is produced by SPC as part of its commitment to PIMRIS. The aim of PIMRIS is to improve the availability of information on marine resources to users in the region, so as to support their national development and management. PIMRIS activities include: the active collection, cataloguing and archiving of technical documents, especially ephemera ('grey literature'); evaluation, repackaging and dissemination of information; provision of literature searches, question-and-answer services and bibliographic support; and assistance with the development of in-country reference collections and databases on marine resources.

PIMRIS

Pacific Islands Marine Resources Information System

Ciguatera fish poisoning in the Pacific

by SPC Health Programme Staff
(SPC Information Circular # 115)

This Information Circular was prepared and produced by the South Pacific Epidemiological and Health Information Service in 1988. Because of the specific information contained in the circular, we thought it appropriate to reproduce this document, outlining the main points of interest with the aim of broadening awareness of the ciguatera fish poisoning problem in the Pacific.

Introduction

Ciguatera fish poisoning is a significant public health problem in the Pacific and a potential barrier to further development of small-scale commercial fisheries in the Pacific Islands. In recent years, over 3,000 cases of fish poisoning have been reported annually to the South Pacific Epidemiological and Health Information Service (SPEHIS). It is estimated that this total represents only 10-20% of the actual number of cases of fish poisoning, which would therefore be in the order of 15,000 to 30,000 cases annually. Though death from ciguatera fish poisoning is rare, illness can be severe and recovery slow. Publicity on cases of fish poisoning can result in a decline in the consumption of fresh fish in Island communities and problems in exporting fresh fish to foreign markets.

Fish poisoning can result from eating spoiled fish or from eating fresh or processed fish containing naturally occurring toxic substances. Ciguatera fish poisoning results from eating reef fish that have previously consumed toxic dinoflagellates (microscopic marine organisms) or from eating predators of these reef fish. There are several types of fish poisoning in addition to ciguatera. Some are associated with specific fish, for example clupeoid poisoning (sardines, anchovies or herring) and puffer fish poisoning. Scombroid fish poisoning occurs when certain types of fish (mackerels and tunas) are eaten after they have produced toxins through spoilage, usually because of inadequate chilling and refrigeration.

Clinical symptoms

The major clinical symptoms that have been associated with ciguatera fish poisoning are described in Table 1. Symptoms usually appear within two to thirty hours (with an average of about six hours) after the consumption of toxic fish, and may vary with the individual and the species, the quantity and parts of the fish consumed. Usually the first symptoms to appear are numbness with a prickling sensation around the lips, tongue and throat, and general weakness and nausea.

The usual progression of the illness is shown in Figure 1. There seems to be a dose-response relationship in ciguatera fish poisoning, with increased ingestion of toxic fish causing more severe symptoms. The illness may last for weeks or months, and occasionally years, depending on the severity of the symptoms. Repeat cases are usually more severe.

Death from ciguatera fish poisoning occurs in less than one per cent of the cases and is usually associated with consumption of the most toxic parts of fish (liver, viscera, organs, roe, etc.). Reported causes of death include respiratory and heart failure and shock from severe dehydration due to vomiting and diarrhoea.

Table 1: Clinical symptoms associated with ciguatera fish poisoning (adapted from Hokama, 1988)

Digestive:
Nausea, often followed by symptoms of watery diarrhoea, abdominal cramps and sometimes vomiting that usually subside within 24 hours. Symptoms may cause dehydration.
Neurological :
Initially, sensitivity disturbances such as reversal of temperature sensation, where cold feels hot (a burning or tingling sensation may also be felt) and hot feels cold, intense itching and numbness with tingling in the limbs. Severe cases may exhibit partial paralysis, convulsions, shaking and spasms. Neurological symptoms may persist from weeks to months (rarely years).
Cardiovascular:
Slow or accelerated pulse rate that is often irregular. Reduced blood pressure. Heart beats may be slightly muffled. These symptoms generally disappear in 2-3 days.
General:
General weakness, joint pain, muscle pain (especially of the legs), headache, chills, sweating and dizziness.

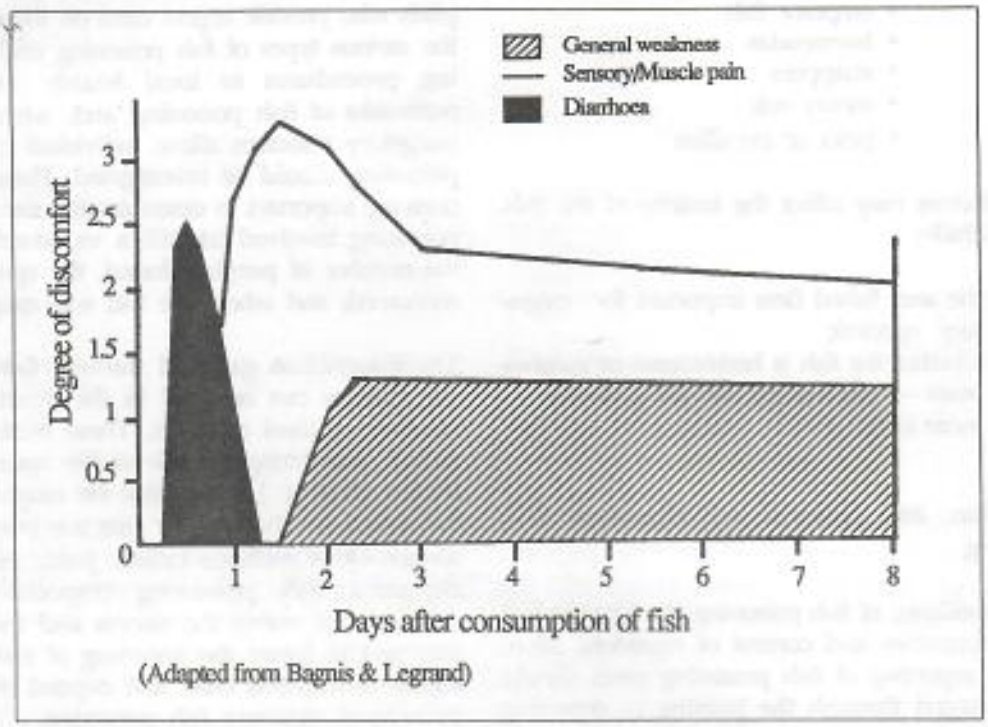


Figure 1: Severity and duration of ciguatera symptoms

Treatment

In most cases, people with ciguatera fish poisoning recover completely, and treatment of the symptoms is usually sufficient to ensure recovery. Individuals with ciguatera fish poisoning should avoid consuming any fresh or saltwater fish or shellfish products, alcoholic beverages and nut or seed products. Consumption of these foods can cause a relapse in symptoms, and increase the severity and/or duration of the illness. This observed syndrome may be due to chemical substances in these foods which mimic the fish poisoning toxins, thus causing a type of allergic reaction.

Efforts are being made to develop a possible curative treatment for ciguatera fish poisoning. A recent article by Palafox et al., in the *Journal of the American Medical Association*, reports that mannitol, an inexpensive sugar compound widely used to help the flow of urine, may be an effective treatment for acute ciguatera fish poisoning. Mannitol may work by flushing out fish poisoning toxins from the body through the urine. Studies to confirm the effectiveness of mannitol are in progress.

Causative agent of ciguatera fish poisoning

Ciguatera fish poisoning is associated with several polyether toxins, of which ciguatoxin is probably the most important, especially in carnivorous fish and in toxic fish found to cause ciguatera. Toxic *Gambierdiscus toxicus* is the source of introduction of ciguatoxin into the food chain, leading to fish poisoning inhumans. These dinoflagellates attach themselves to marine algae and are then passed up the food chain by being consumed by small herbivorous fish, which are then consumed by carnivorous fish. Humans are poisoned after consumption of either type of toxic fish.

Ciguatoxin is one of the most potent and stable marine toxins known. It is resistant to heat and acid, and cannot be destroyed by cooking, smoking, marinating, freezing, The higher fish are in the food chain, the more concentrated is the toxin in their tissues and the more severe the symptoms from eating the fish. drying, salting or freeze-drying.

Ciguatoxin and other related toxins do not alter the smell, taste or coloration of the toxic fish tissues. The cigua-toxin concentrates in the liver, viscera, organs, roe and head of the fish, which feel no ill effects from the toxin

Fish species implicated in ciguatera fish poisoning

In theory, almost any reef fish or predator to reef fish could become ciguatoxic under the right conditions. In the Pacific the majority of the implicated fish are carnivorous and include:

- groupers or rock cods
- mullets
- parrot fish
- trigger fish
- surgeon fish
- wrasses

- emperor fish
- barracudas
- snappers
- moray eels
- jacks or trevallies

Several factors may affect the toxicity of the fish. These include:

- the area fished (less important for migratory species);
- whether the fish is herbivorous or carnivorous — carnivorous fish are generally more toxic.

Prevention and control of ciguatera fish poisoning

Local surveillance of fish poisoning is a key element in the prevention and control of ciguatera. More complete reporting of fish poisoning cases should be encouraged through the training of reporting sources (such as medical staff at clinics and hospitals who provide urgent care) on the diagnosis of the various types of fish poisoning and the reporting procedures to local health officials. All outbreaks of fish poisoning and, when staff and budgetary resources allow, individual cases of fish poisoning should be investigated. These investigations are important in determinating the type of fish poisoning involved (ciguatera vs. scombroid, etc.), the number of people affected, the species of fish consumed, and where the fish was caught.

The information gathered through fish poisoning surveillance can be used in the development of ciguatera control methods. These methods might include prohibiting the sale of fish species that are known often to be toxic, that are caught in known 'hot spots' on the reef, or that are over a certain weight. Other methods include public education on ciguatera fish poisoning (especially in the avoidance of eating the viscera and roe (eggs) of fish) and in future, the screening of toxic fish. The choice of methods used will depend on the local patterns of ciguatera fish poisoning.

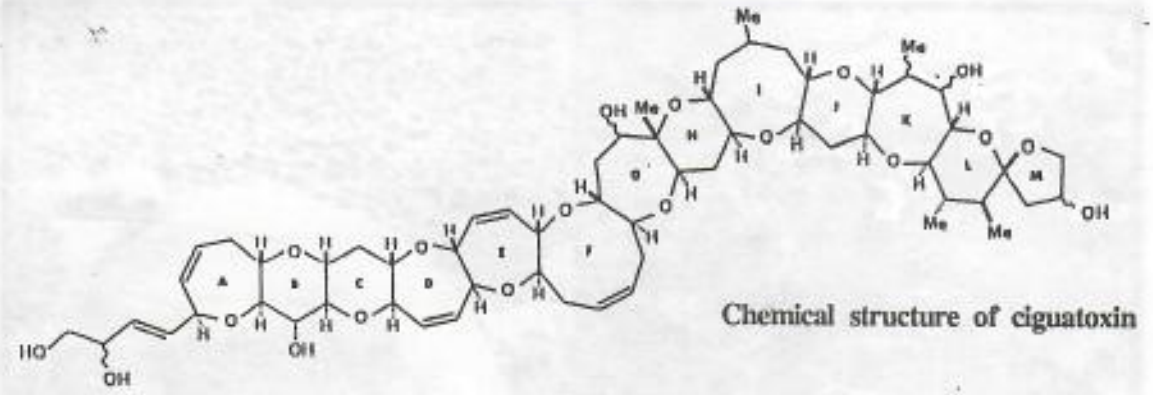
Ciguatera research in French Polynesia

by A.M. Legrand
Institut de Recherches Médicales Louis Malardé
Papeete, French Polynesia

View of the Institut de recherches médicales Louis Malardé

Ciguatera fish poisoning is an important problem in French Polynesia as in the whole South Pacific. About 800 to 1000 cases are declared and documented every year.

The Ciguatera Unit of the Institut de recherches médicales Louis Malardé was created in 1967 by Dr R. Bagnis to study the complex situation of the various seafood poisonings observed in the Pacific Islands.



Chemical structure of ciguatoxin

Until 1979 mainly ecological research was conducted in the field in the Tuamotu, Marquesas and Gambier Islands. The purpose was to try to understand the original source of the fish toxins and the reasons for the presence of hazardous fish in the lagoons.

Based on the diversity of fish species affected, it had long been thought that the origin of the ciguatera toxins was exogenous (the toxin was not produced by the fish). First, field research focused on the algae colonising coral beds. Then, in 1977, the discovery of aboundant amounts of the dinoflagellate *Gambierdiscus toxicus* on coral beds in a ciguatera endemic area of the Gambier Island of Mangareva provided the first evidence for the unicellular origin of the toxins, ecologically related to climatic and man-made disturbances induced in coral reefs.

This important step is one of the very successful results obtained by the Institut Malardé which was associated during that period with some of the field experiments of Yasumoto's group (Tohoku University, Sendai, Japan).

Thereafter, a research programme was developed at the laboratory, the purpose of which was to establish a reliable and sensitive method to detect hazardous reef fish.

During several years this programme was hampered by insufficient knowledge about the chemical nature of the toxins involved in human intoxications.

In 1986, supplementary financial support allowed the acquisition of modern chromatographic equipment so that complete purification of the toxins was hence forth possible.

A chemical research program for identification and characterisation of the fish toxins led to a recent success in 1989: the chemical structure of ciguatoxin is now well-known. This important new result is the outcome of close collaboration between our laboratory and Professor Yasumoto's group.

As a consequence of this important progress and thanks to both scientific and financial support provided by the Pasteur Institute of Paris, a new research programme is on course to develop the reliable and sensitive assay to detect hazardous fish that every fishery service needs. We hope to succeed in that project within three years.

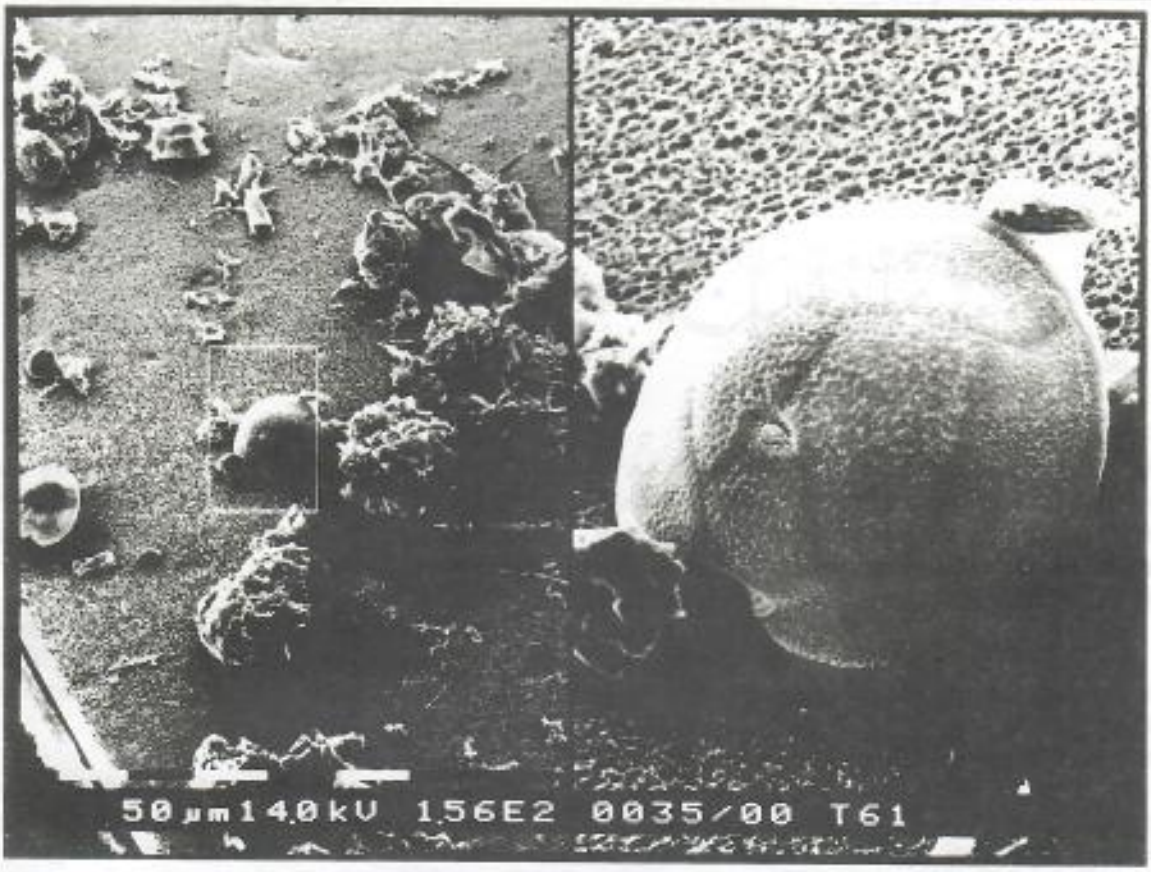
QDPI research on ciguatera

by R.J. Lewis
Queensland Dept of Primary Industries
Deception Bay, Australia

The Queensland Department of Primary Industries (QDPI) has been researching the ciguatera problem in Australia for over 10 years. A recent review of ciguatera provides a background to this work (Gillespie et al., 1986).The research group comprises myself (Richard Lewis), Michael Holmes, Ashley Hoy and Michelle Sellin. The goal of our present research is to minimise the adverse impact of ciguatera on the fishing industry and on public health. We are investigating three main areas.

Production of antibodies to ciguatera

The goal of this project is to produce monoclonal antibodies to ciguatoxin (the toxin that causes ciguatera). These antibodies will be used to develop a test kit to detect if fish are toxic or safe to eat. We have now purified sufficient ciguatoxin (mainly from moray eels from Tarawa) for the antibody production phase of this work to commence. This material will first be coupled to a carrier protein prior to immunisation, as ciguatoxin on its own



View of *Gambierdiscus toxicus*

will not induce an immune response. A success in this project will have regional and international significance.

Origin of ciguatoxin

The goal of this project is to determine how ciguatoxin enters the food chain and if environmental factors influence the quantity of toxin entering. This work has focused on culturing *Gambierdiscus toxicus*. We discovered that only two of thirteen strains of *G. toxicus* in culture actually produced ciguatoxin precursors (gambiertoxins) and no ciguatoxin was detected (Holmes et al., in press). The strain-dependent production of ciguatoxin precursors may explain why ciguatera incidence correlates with *G. toxicus* numbers in some areas but not in others.

Treatment of ciguatera

The goal of this project is to establish new treatments for ciguatera. This work has provided independent confirmation in Australia (Pearn et al., 1989) of the use-fulness of mannitol in the treatment of ciguatera. Experimental studies show mannitol is likely to reverse an oedema of Schwann cells seen during ciguatera. We are also investigating the potential of local anaesthetics as treatments and are collaborating with P. Amade and D. Laurent at ORSTOM Noumea, on a study looking at the potential of traditional remedies as treatments. Development of an orally effective treatment for ciguatera is our long-term goal.

Details of each of these projects will be presented in articles in future issues of the bulletin. In addition to these studies, we have a watching brief on other tropical marine food toxins, particularly dinoflagellate-borne toxins such as those causing paralytic shellfish poisoning.

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Holmes, M.J., Lewis, R.J., Poli, M.A. and Gillespie, N.C. (1991). Strain dependent production of ciguatoxin precursors (gambiertoxins) by *Gambierdiscus toxicus* (Dinophyceae) in culture. *Toxicon* (in press).

Pearn, J.H., Lewis, R.J., Ruff, T., Tait, M., Quinn, J., Murtha, W., King, G., Mallet, A. and Gillespie, N.C. (1989) Ciguatera and mannitol: Experience with a new treatment regimen. *Med. J. Aust.* 151, 77-80.

Excretion of ciguatoxin from fishes

by R.J. Lewis
Queensland Dept of Primary Industries
Deception Bay, Australia

Humans who eat fish in tropical and sub-tropical areas are at risk from ciguatera. This risk stems from the ability of otherwise edible fish in tropical and sub-tropical areas to accumulate ciguatoxin through the marine food chain. Species of herbivorous, detritivorous, omnivorous and carnivorous fishes have been implicated with ciguatera.

A long-standing dogma is that, once a fish becomes contaminated with ciguatoxin, it retains this quantity of toxin over its life time. However, in a recent report from our group (Lewis et al., 1991), we indicate that a population of moray eels became progressively less toxic over time. This article summarises some of the findings of this study.

The moray eels (*Lycodontis javanicus*) were collected from Tarawa, Republic of Kiribati, in an area where ciguatera has been endemic for several decades. Moray eels were captured in fish-baited cage traps set at various locations on the ocean reefs adjacent to Teaoraereke with the assistance of T. Tebano. The viscera (including liver) of each eel were removed and stored frozen prior to air dispatch to Brisbane, Australia. Viscera were pooled to a convenient sample weight for extraction (0.3 to 1.0 kg). The lipid-soluble fractions were then bioassayed in mice to quantify the toxin content of each sample of eels. During this study, eels captured from the ocean reef adjacent to the villages of Bikenibeu, Bairiki and Betio (Figure 1) were also found to be similarly toxic to the eels from Teaoraereke. However, a pooled sample of viscera from five eels from the ocean reef adjacent to Tanaea did not contain detectable ciguatoxin.

A total of 217 eels was obtained from nine collections over a 500-day period, commencing September 1987. These eels yielded a total of 35.9 kg of viscera from which were extracted 99,200 mouse units (m.u.) of ciguatoxin. The average toxicity was 2.43 $\pm$ 1.69 m.u. per g viscera and ranged from 0.59 to 7.3 m.u. per g. Interestingly, no significant regression was found between toxicity and average viscera weight, indicating that these eels did not become more toxic the larger they grew. However, the toxicity of viscera was found to decline significantly over the 500-day period of the collections (Figure 2). An exponential relationship fitted this decrease in toxicity.

The slope of the regression estimated that the half-life for the loss of ciguatoxin from the population of eels was 264 days. All eels contained detectable ciguatoxin and no seasonal fluctuations in toxicity were evident.

We proposed that this loss of ciguatoxin from eels stems from the excretion and/or decay of ciguatoxin. Excretion (depuration) comprises the loss from eels of ciguatoxin *per se*. Decay comprises the metabolism of ciguatoxin to less toxic moieties within eels.

Case history data on fish poisoning (including ciguatera) in the Republic of Kiribati collected by the South Pacific Epidemiological and Health Information Service from 1982 to 1989 (Figure 3) indicate an upsurge in poisoning in 1986/87. The upsurge was followed by a decrease in the incidence of poisoning in 1988 and 1989. This decrease coincides with the period when eels were declining in toxicity. We suggest that moray eels could be a good indicator species for assessing ciguatera levels in an area. The upsurge in fish poisoning coincides with reef disturbance associated with the Dai Nippon causeway project several kilometres to the west of Teaoraereke (Tebano and Lewis, 1990).

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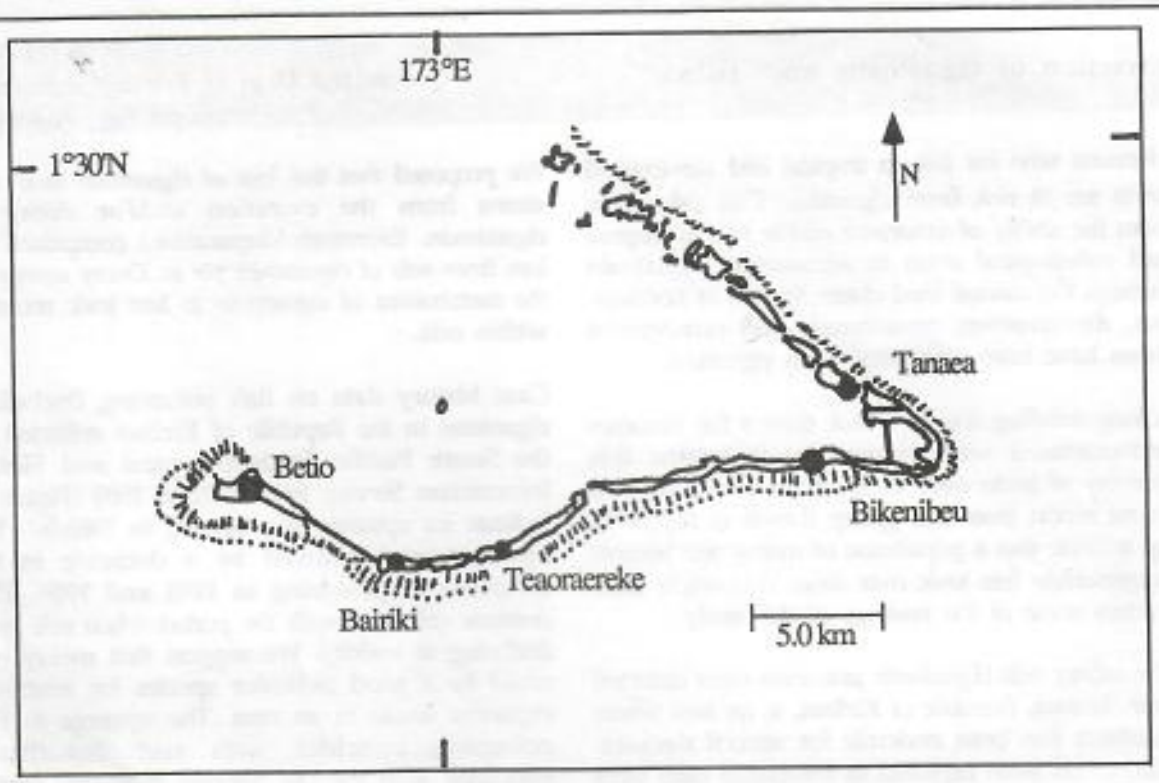


Figure 1. Map of the southern half of Tarawa, Republic of Kiribati. The dashing indicates the outer barrier reef. The areas reported toxic in 1983 are indicated by the dotted line. Eels were collected on outer reefs adjacent to Teaoraereke from September 1987 to January 1989.

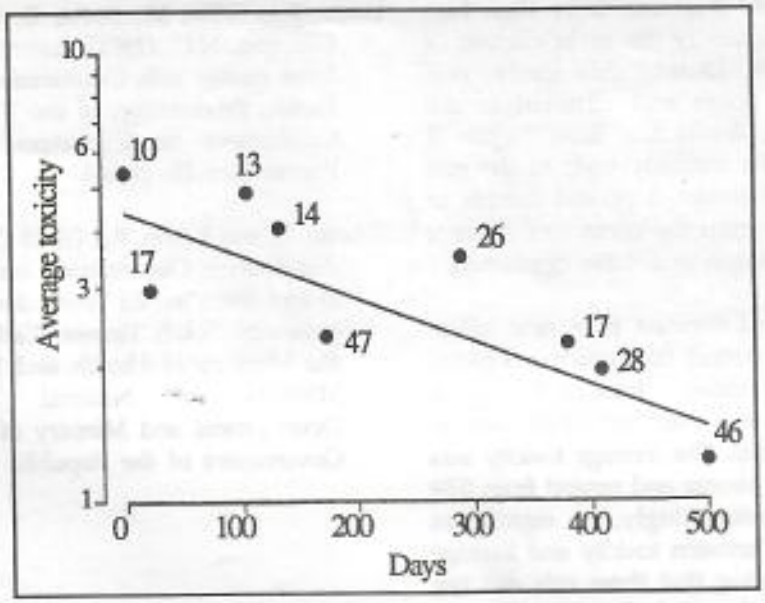


Figure 2. Toxicity (mouse units per g viscera) of eel viscera over a 500-day period. Note log scale for y axis. Numbers adjacent to each data point indicate the number of fish pooled for that collection. Toxicity declined significantly over the 500-day period.

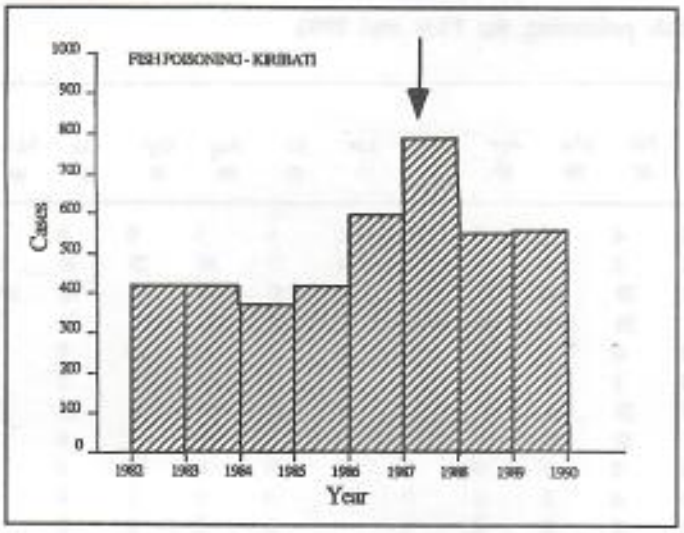


Figure 3. Annual incidence of fish poisoning in the Republic of Kiribati, 1982 to 1989. Data provided by the South Pacific Commission Epidemiological and Health Information Services and includes mostly cases of ciguatera fish poisoning. The arrow indicates the start of moray eel collections.

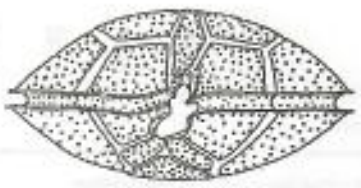
Improvement of ciguatera case history reporting

by P. Dalzell
South Pacific Commission
Noumea, New Caledonia

Outbreaks of ciguatera can have a deleterious effect on fisheries development, since fishing grounds may be closed and certain fish species prohibited for sale. This is particularly critical in the small islands and atolls of the South Pacific where economic opportunities are limited and fresh fish comprises a substantial portion of the national diet.

The South Pacific Epidemiological and Health Information Service (SPEHIS), based at the SPC, records between 3,400 and 4,700 cases of fish poisoning each year (see tables on p. 10), although not all of these are due to ciguatera intoxication. However, at present the number of ciguatera cases reported throughout the region is thought to comprise only between 10 and 20 per cent of actual poisoning incidence. That there is a need for some form of initiative on ciguatera in the South Pacific is evident. However, the effect that this fish poisoning has on island societies is largely unknown due to the poor reporting of case histories. A first step to improve the current under-reporting is to encourage both health and fisheries workers in the region to record case histories on a standard ciguatera reporting form, and to send them to SPC where they can be collated in a database. The SPC Health Programme has circulated the form attached to this bulletin to regional health workers via the SPEHIS monthly news sheet.

The form is reproduced here for fisheries workers in the region to record cases of ciguatera poisoning that they encounter. The copy (in English and in French) attached with this bulletin can be used as a template for making multiple copies, or, where copying facilities are unavailable, the SPC Inshore Fisheries Research Project will be happy to supply copies. As this form is still undergoing trials in the field, we would be glad to hear from persons who have criticisms or suggestions for improving the form. Finally, we would encourage fisheries workers in the region to work in co-operation with their colleagues in their health departments to record all incidents of ciguatera that they hear about. Only with your help can we gauge the true extent of this problem and plan and co-ordinate future work accordingly.



Monthly summary for fish poisoning for 1989 and 1990.

SPC Island member countries	Jan 89	Feb 89	Mar 89	Apr 89	May 89	Jun 89	Jul 89	Aug 89	Sep 89	Oct 89	Nov 89	Dec 89	Cum. Total 1/89 - 12/89 Cases	Rate*
American Samoa	0	4	1	2	0	0	0	5	0	0	0	0	12	0.3
Cook Islands	30	5	3	5	6	14	13	16	25	10	12	7	146	8.5
Fiji	100	79	54	37	9	52	89	74	138	65	249	79	1025	1.4
French Polynesia	81	54	55	41	73	95	89	101	88	81	54	48	860	4.9
Fed. St. of Micronesia	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Guam	0	1	0	0	0	1	3	1	28	2	2	0	38	0.3
Kiribati	35	29	33	26	95	39	43	28	58	73	72	22	553	8.2
Marshall Islands	11	12	13	5	6	7	5	15	2	6	7	12	101	2.7
Nauru	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
New Caledonia	0	0	0	0	0	26	0	0	0	0	0	154	180	1.1
Niue	0	0	0	0	0	0	0	0	0	0	4	0	4	1.6
North. Mariana Isl.	3	2	4	0	1	1	3	1	4	1	1	3	24	1.2
Palau	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Pitcairn Isl.	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Papua New Guinea	10	11	8	10	0	41	37	41	30	8	0	0	196	0.1
Solomon Islands													0	0.0
Tokelau	0	7	0	0	2	0	3	0	0	7	3	0	22	13.8
Tonga	0	0	0	1	0	0	0	0	0	1	0	2	4	0.0
Tuvalu	1	0	0	7	3	15	0	9	9	8	13	0	65	7.6
Vanuatu	93	54	129	75	95	80	65	45	71	87	60	34	888	6.1
Wallis and Futuna	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Western Samoa	2	1	1	0	4	4	3	4	3	4	3	4	33	0.2

* Number of active cases per 1,000 population

SPC Island member countries	Jan 90	Feb 90	Mar 90	Apr 90	May 90	Jun 90	Jul 90	Aug 90	Sep 90	Oct 90	Nov 90	Dec 90	Cum. Total 1/90 - 12/90 Cases	Rate*
American Samoa	0	0	0	0	0	0	0	0	0	0	0		0	0.0
Cook Islands	8	2	11	0	0	0	0	6	9	3			39	2.3
Fiji	17	19	62	4	68	62	39	57	64	84			476	0.7
French Polynesia	130	79	62	60	60	54	60	117	97	83	62	35	899	5.1
Fed. St. of Micronesia	1	0	0	0	0	0	0	0	0				1	0.0
Guam	0	4	0	24	0	7	0	0	2	6	2		45	0.4
Kiribati	71	224	93	93	83	235	41	100	142	144	37		1263	18.7
Marshall Islands	22	15	14	11	14	10	13						99	2.6
Nauru	0	0	0	0	0	0	0	3	0	0			3	0.3
New Caledonia	11	13	15	13	36	16	6	9	7				126	0.8
Niue	0	0	0	0	0	0	0	0	0	0	0		0	0.0
North. Mariana Isl.	2	1	1	0	0	2	10	4	3	7	2	0	32	1.6
Palau	0	0	0	0	0	0	0	0	0	0	0		0	0.0
Pitcairn Isl.	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Papua New Guinea	0	0	0	0									0	0.0
Solomon Islands													0	0.0
Tokelau	0	1	4	2	1	0							8	5.0
Tonga													0	0.0
Tuvalu	14	16	12	10	15	34	2	10	0				113	13.3
Vanuatu	46	77	11	13	82	13	27	49	38	55	23	14	448	3.1
Wallis and Futuna	0	0	0	0	0	0	0	0	38	0	0		38	2.6
Western Samoa	4	1	2	3	6								16	0.1

* Number of active cases per 1,000 population

Definition of fish poisoning: Vomiting, diarrhoea, sensory changes and/or rash or itching following consumption of fish (may be ciguatera, scombroid, or other)

(Source: SPEHIS — unpublished data)

Members of the Ciguatera Special Interest Group

We had received completed questionnaires from the individuals listed below at 28 February 1991. If you are on the list and your name and address is wrong, please send us a correction. If you are not on the list and want to be, fill in the form enclosed with this bulletin or write us for a new form.

Bremner A.
CSIRO - Division of Fisheries
Stowell Ave. - Hobart - Tasmania 7004
Australia

Capra M.F
Fac. of Science - Qld Univ. of Technology
GPO Box 2434 - Brisbane - Qld 4001
Australia

Hallegraeff G.
CSIRO - Marine Laboratories
GPO Box 1530 - Hobart - Tasmania 7001
Australia

Hunter G.D.
AIDAB
Middle Head Rd - Mosman - NSW 2090
Australia

Knight C.M
Fisheries Division - DPIF
PO Box 990 - Darwin
Australia

Preker M.
University of Queensland
Heron Isl. Res. Stat. Via Gladstone - Qld 4680
Australia

Trendall J.T
CSIRO
Div. of Fish. Res.-bungalow PO - Cairns - 4870 Qld-
Australia

Williams D.
Australian Institute of Marine Science
PMB 3 - Townsville - Qld 4810
Australia

Hinchberger B.
Third World Magazine
Rua Da Gloria 122, Sale 105 - Rio De Janeiro
Brazil

Archer R.
ICOD
5670 Spring Garden Rd. - Halifax - N. Scotia
Canada B3J 1H6

McConnell B.
ICOD
5670 Spring Garden Rd. - Halifax - N. Scotia
Canada B3J 1H6

Argue A.w.
Dept. of Fisheries and Oceans - Policy Plan. Branch
4050 Lockehaven Drive - Victoria - BC
Canada V8N 455

Howard N.T.
Ministry of Marine Resources
PO Box 85 - Rarotonga
Cook Islands

Neale A.F.
Tairoto Pearls Ltd.
Tauhunu -Manihiki Isl.
Cook Islands

Passfield K.
Ministry of Marine Resources
PO Box 85 -Rarotonga
Cook Islands

Zoutendyk D.A.
Ministry of Marine Resources
PO Box 85 - Rarotonga
Cook Islands

Fagolimul J.O.
Marine Resources Management Division
Dept. of Res. & Develop - PO Box 251 - Colonia - Yap
Federated States of Micronesia 96943

Asher E.
Community College Of Micronesia
PO Box 159 - Kolonia - Pohnpei 96941
Federated States of Micronesia

Falanruw M.c.
Institute Of Natural Science
PO Box 215 - Yap 96943
Federated States of Micronesia

Maito M.M.
Truk Maritime Authority
PO Box 789 - Truk 96942
Federated States of Micronesia

Pekalibe P.
Yap Fishing Authority
PO Box 338 - Colonia - Yap 96943
Federated States of Micronesia

Smith A.J.
Marine Resources Management Division
PO Box 251 - Yap 96943
Federated States of Micronesia

Curren E.F.
Pohnpei Marine Resources Division
PO Box B - Kolonia - Pohnpei 96941
Federated States of Micronesia

Dahl C.R.
Community College of Micronesia
PO Box 159 - Kolonia - Pohnpei 96941
Federated States of Micronesia

Gawel M.
Federated States of Micronesia Government
FSM Marine Resources - PO Box 383 - Pohnpei 96941
Federated States of Micronesia

Paatmag P.N.
Marine Resources Management Division
PO Box 251- Colonia - Yap 96941
Federated States of Micronesia

Syne D.S.
Community College of Micronesia
Lrc/library - PO Box 159 - Pohnpei 96941
Federated States Of Micronesia

Friberg C.S.
Yap Fishing Authority
PO Box 338 - Colonia - Yap 96943
Federated States of Micronesia

Iou J.B.
Marine Resources Management Division
PO Box 251 - Yap 96943
Federated States of Micronesia

Adams T.J.H.
Fisheries Division
PO Box 358 - Suva
Fiji

Alatoa H.G
Commonwealth Youth Programme
USP - PO Box 1168 - Suva
Fiji

Naqasima M.
SPAS - University of the South Pacific
PO Box 1168 - Suva
Fiji

Saunders P.
ICOD - Canadian Cooperation Office
Private Mail Bag - Suva
Fiji

Soeto J.
Institute of Marine Resources - USP
PO Box 1168 - Suva
Fiji

Sharma S.P
Fisheries Division - Lami
PO Box 358 - Suva
Fiji

Tanaka H.
South Pacific Aquaculture Dev. Project
UNDP - Private Mail Bag - Suva
Fiji

King Obed W.N.
University of The South Pacific
PO Box 1168 - Suva
Fiji

Bruslé J.
Labo. de Biologie Marine - Univ. Perpignan
Av. de Villeneuve - 66860 Perpignan
France

Durand Clément M.
INSERM
U 303 - BP 3 - Villefranche/mer
France

Bagnis R.
Université Française du Pacifique
BP 4685 - Papeete - Tahiti
French Polynesia

Fougerouse A.
EVAAM
BP 20 -Papeete - Tahiti
French Polynesia

Grand S.
Service de la Mer et de l'aquaculture
BP 20704 - Papeete - Tahiti
French Polynesia

Legrand A.M.
Institut de Recherches Médicales L. Malardé
33, rue l'Artémise - Papeete - Tahiti
French Polynesia

Wrobel L.
EVAAM
BP 20 - Papeete - Tahiti
French Polynesia

Yen S.
EVAAM
BP 20 - Papeete - Tahiti
French Polynesia

Lujan R.J.
Division of Aquatic & Wildlife Resources
Dept. of Agriculture - PO Box 2950 - Agana
Guam 96910 - USA

Balazs G.H
National Marine Fisheries Service
2570 Dole St. - Honolulu -
Hawaii 96822-2396 - USA

Brock R.E.
Hawaii Institute of Marine Biology
Univ. of Hawaii - 1000 Pope Rd - Honolulu
Hawaii 96822 - USA

Harman R.F.
West. Pacif. Reg. Fishery Management Council
1164 Bishop St #1405 - Honolulu
Hawaii 96813 - USA

Cates D.E.
National Marine Fisheries Service - SW Region
1320 Aupapaohe St. - Kailua
Hawaii 96734 - USA

Naughton J.
National Marine Fisheries Service
2570 Dole St. - Room 106 - Honolulu
Hawaii 96822 - USA

Nitta E.T
NOAA Fisheries - SW Region
2570 Dole Street - Honolulu -
Hawaii 96822 - USA

Wiedemeyer W.
University of The Ryukyus - Dept. of Marine Science
Senbaru 1 - Nishihara - Okinawa 903-01
Japan

Yamaguchi M.
University of The Ryukyus - Dept. of Marine Science
Senbaru 1 - Nishihara - Okinawa 903-01
Japan

Corbett T.
Island Trading Co.
PO Box 8 - Bairiki - Tarawa
Kiribati

Day C.M.
ODA c/o Fisheries Division
Fisheries Division - PO Box 276 - Bikenibeu - Tarawa
Kiribati

Tekinaiti T.
Fisheries Division
PO Box 276 - Bikenibeu - Tarawa
Kiribati

Uau L.
Fisheries Division
PO Box 276 - Bikenibeu - Tarawa
Kiribati

Yeeting B.
Fisheries Division
Po Box 276 - Bikenibeu - Tarawa
Kiribati

Vanderbilt C.F.
International Ocean Institute
PO Box 524 - Valletta
Malta

Zingmark R.
College of Micronesia
PO Box 1772 - Majuro 96960
Marshall Islands

Bungitak J.
PO Box 1184
Majuro 96960
Marshall Islands

Amade P.
ORSTOM
BP A5 - Noumea
New Caledonia

Grandperrin R.
ORSTOM
BP A5 - Noumea
New Caledonia

Laurent D.
ORSTOM
BP A5 - Noumea
New Caledonia

Joannot P.
Mairie de Noumea
BP 395 - Noumea
New Caledonia

De C. Cook S.
C/- PO Box 226
Thames
New Zealand

Kaly U.
University of Auckland
Zoology Dept. - Private Bag - Auckland
New Zealand

MacDonald J.A.
University of Auckland
Dept of Zoology - Private Bag - Auckland
New Zealand

Pollock N.J.
Victoria University
PO Box 600 - Wellington
New Zealand

Coffen-Smout S.
Dept. of Agriculture And Fisheries
PO Box 74
Niue

Leolahi S.h.
Fisheries Division Government of Niue
Dept of Agr. Forest.and Fish. - PO Box 74 - Alofi
Niue

Asigau W.
Department of Environment and Conservation
PO Box 6601 - Boroko
Papua New Guinea

Hill L.
Biology Department
University of PNG- PO Box 320 - Waigani
Papua New Guinea

Kalowai M.
Div. of Agriculture, Stock & Fisheries
PO Box 1 - Esa'ala - Milne Bay Province
Papua New Guinea

Kuk R.
Dept. of Fisheries & Marine Resources
PO Box 1343 - Port Moresby
Papua New Guinea

Lokani P.
Department of Fisheries & Marine Resources
Fisheries Research Station - PO Box 337 - Kavieng
Papua New Guinea

Mobiha A.
Dept. Of Fisheries & Marine Resources
Fish. Research Lab- PO Box 54 - Daru
Papua New Guinea

Omeri N.
Dept of Fisheries & Marine Resources
Library - PO Box 165 - Konedobu
Papua New Guinea

Rajeswaran N.
Dept of Fisheries & Marine Resources
PO Box 165 - Konedobu
Papua New Guinea

Tenakanai C.D.
Dept of Fisheries & Marine Resources
Library - PO Box 165 - Konedobu
Papua New Guinea

Ule N.
National Library of Papua New Guinea
PO Box 5770 - Boroko
Papua New Guinea

Wangi P.K.
MLRV - Faculty Of Medicine
PO Box 5623 - Boroko
Papua New Guinea

Wararu W.
Dept of Fisheries & Marine Resources
PO Box 165 - Konedobu
Papua New Guinea

Ahukela A.
Ministry of Natural Resources
Fisheries Division - PO Box G 24 - Honiara
Solomon Islands

Diake S.
Fisheries Div.- Ministry of Nat. Resources
PO Box G 24 - Honiara
Solomon Islands

Rawlinson N.J.F.
Commonwealth Scient. & Indust. Research Org.
Ministry of Nat. Res.- PO Box G 24 - Honiara
Solomon Islands

Saeve H.M.
Fisheries Division
PO Box 77 - Gizo - Western Province
Solomon Islands

Kimura T.
Ministry of Agriculture, Forestry & Fisheries
Fisheries Division - PO Box 14 - Nuku'alofa
Tonga

Koloa T.
Ministry of Agriculture, Forestry & Fisheries
Fisheries Division - PO Box 14 - Nuku'alofa
Tonga

Langi S.
Ministry of Agriculture, Forestry & Fisheries
Fisheries Division - PO Box 14 - Nuku'alofa
Tonga

Langi V.
Ministry of Agricult., Forestry & Fisheries
Fisheries Division - PO Box 14 - Nukualofa
Tonga

Gentle T.
PO Box 90
Funafuti
Tuvalu

Wells S.M.
IUCN - The World Conservation Unit
56 Oxford Road - Cambridge CB4 3PW
United Kingdom

Glucksman J.
2401 Wendy Rd
Alva - Fl 33920
USA

Cillaurren E.
ORSTOM
BP 76 - Port Vila
Vanuatu

Maeva S.M.
Fisheries Department
PO Box 129 - Port Vila
Vanuatu

Toloa F.
Ofice For Foreign Affairs
PO Box 865 - Apia
Western Samoa

Zann L.P.
FAO/UNDP
Private Bag - Apia
Western Samoa

Bibliographic references on ciguatera

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

All the documents in the list below are catalogued in the Fisheries Information Project's ciguatera bibliographic database and are held in the SPC library. Some are publications, but many are internal documents, mimeo reports and other forms of ephemera. In some cases we can provide single photocopies of references free of charge to SIG members or bona fide fisheries officers in Pacific Island countries. In other cases, where confidentiality requirements or copyright restrictions apply, we may be limited to advising enquirers of contact addresses through which they may be able to obtain the document in question.

If there are documents that you feel should be added to the database, please send us a copy, or, if this is not possible, a photocopy of the cover page. Documents do not need to be formal publications - many of those in the list are not and we are keen to archive as much 'grey literature', internal reports, correspondence, unpublished data, etc. as possible.

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FOURTH INTERNATIONAL CONFERENCE ON CIGUATERA FISH POISONING

INSTITUT TERRITORIAL DE RECHERCHES MEDICALES LOUIS MALARDE
B.P. 30 PAPEETE - TAHITI - POLYNESIE FRANCAISE
Tél. : (689) 41.64.64 Fax : (689) 43.15.90

December 10, 1990

FIRST ANNOUNCEMENT

Madam,
Sir,

The IIIrd International Conference on Ciguatera Fish Poisoning in Puerto Rico (May 1990) was a success.

Dr. Tosteson and ourselves have projected to organize the

IVth INTERNATIONAL CONFERENCE

ON CIGUATERA FISH POISONING

in

TAHITI, likely 4-8 May 1992

Please, keep in mind these dates if you intend to participate to the Conference.

Yours sincerely,

The Organizing Committee

Please adress your mail to : Dorothea CHEOU, Secretarial office
4th International Conference on Ciguatera Fish Poisoning

South Pacific Commission
SEAFOOD POISONING REPORT FORM

Please fill in the answers to the questions completely. Tick the boxes where appropriate.

Details of person filling in report form:

Name _____ Job/ Position _____

Contact address _____

Date: _____ Signature _____

Poisoned person's details:

Name _____ Sex (M/F) _____ Age (yrs) _____

Address _____

Details of the seafood that caused the poisoning: *(tick all the boxes that apply)*

Type of food	Where caught	How preserved	What eaten	How eaten
Fish ____ O	River ____ O	Fresh, no ice ____ O	Head ____ O	Unprepared (raw) ____ O
Crab ____ O	Mangrove ____ O	Fresh, iced ____ O	Flesh ____ O	Marinated ____ O
Lobster ____ O	Beach ____ O	Frozen ____ O	Skin ____ O	Cooked ____ O
Other crustacean ____ O	Reef patch ____ O	Salted ____ O	Liver ____ O	
Gastropod* ____ O	Lagoon ____ O	Dried ____ O	Roe ____ O	
Bivalve* ____ O	Outer reef ____ O	Smoked ____ O	Other organs	How many others
Other mollusc ____ O	Open sea ____ O	Pickled ____ O	(specify)	ate this meal? ____
Other (specify)	Other (specify)	Other (specify)	____ O	felt sick? ____
____ O	____ O	____ O	____ O	were admitted ____
Unknown ____ O	Unknown ____ O	Unknown ____ O	Unknown ____ O	to hospital? ____

What is the local name of the seafood? _____

What is the English name of the seafood? _____

Name of vendor or restaurant (if bought) _____

Name of place it was caught (if known) _____

When was the food eaten? Date _____ Time _____

When did you first feel sick? Date _____ Time _____

* *Gastropods are one-shelled seafoods like snails, trochus, conches, etc.*
Bivalves are two-shelled seafoods like clams, mussels, cockles, oysters, etc.

Symptoms: *(tick all the boxes that apply)*

Burning or pain when touching cold water ____ O	Pin pricking sensation on touching water ____ O	
Tingling or numbness sensations ____ O	Strange taste in mouth ____ O	
Difficulty or pain in urinating ____ O	Skin itching or redness ____ O	
Difficulty in breathing ____ O	Excessive salivation ____ O	Fever or chills ____ O
Difficulty in walking ____ O	Excessive sweating ____ O	Headache ____ O
Difficulty in talking ____ O	Diarrhoea ____ O	Joint aches ____ O
Eye irritation ____ O	Vomiting ____ O	Muscle cramps ____ O

Medical data:

Pulse _____ Blood pressure ____ / ____ Pupils _____

In case of death:

Date of death _____ Autopsy findings _____

Other information _____

Please return this form to: *South Pacific Commission, P. O. Box D5, Nouméa CEDEX, New Caledonia*

THANK YOU

Commission du Pacifique Sud

FORMULAIRE DE DECLARATION D'UNE INTOXICATION DUE A LA CONSOMMATION DE PRODUITS DE LA MER

Veuillez répondre en détail à toutes les questions. Cochez la case appropriée.

Identité du déclarant

Nom _____ Profession/ titre _____

Adresse _____

Date _____ Signature _____

Identité de la personne intoxiquée

Nom _____ Sexe (M/F) _____ Age _____ ans

Adresse _____

Renseignements sur le produit de la mer qui a provoqué cette intoxication *(Veuillez cocher les cases appropriées)*

Type de produit	Lieu de capture	Mode de conservation	Morceau consommé	Méthode de préparation culinaire
Poisson _____ O	Rivière _____ O	Frais, sans glace O	Tête _____ O	Sans préparation (cru) O
Crabe _____ O	Mangrove _____ O	Frais, sur glace _O	Chair _____ O	Mariné _____ O
Langouste _____ O	Plage _____ O	Congelé _____ O	Peau _____ O	Cuit _____ O
Autres crustacés __ O	Paté corallien __O	Salé _____ O	Foie _____ O	
Gastéropode* ____ O	Lagon _____ O	Séché _____ O	Oeufs ____ O	**Combien d'autres**
Bivalve* _____ O	Récif-barrière __O	Fumé _____ O	Autres organes	**personnes** _____
Autres mollusques O	Pleine mer ____O	Saumuré _____ O	(précisez)	ont mangé cet aliment? __
Autres (précisez)	Autres (précisez)	Autres (précisez)	_____ O	ont été malades? _____
_____ O	_____ O	_____ O	_____ O	ont été admises
Inconnu _____ O	Inconnu _____ O	Inconnu _____ O	Inconnu __ O	à l'hôpital? _____

Nom local de cet aliment? _____

Nom français _____

Nom du vendeur ou du restaurant (en cas d'achat) _____

Nom du lieu de pêche (si possible) _____

Aliment consommé le _____ à _____ heures

Apparition des premiers symptômes (date) _____ à _____ heures

**Les gastéropodes sont des fruits de mer coquille simple comme les escargots, les trocas, les strombes, etc.*
Les bivalves sont des fruits de mer à deux coquilles comme les palourdes, les moules, les coques, les huîtres, etc.

Symptômes *(Veuillez cocher les cases appropriées)*

Brûlure ou douleur au contact de l'eau froide _____ O	Picotements au contact d'eau _____ O	
Fourmillements ou engourdissement _____ O	Goût bizarre dans la bouche _____ O	
Miction difficile ou douloureuse _____ O	Démangeaisons ou rougeurs _____ O	
Respiration difficile _____ O	Salivation excessive _____ O	Fièvre ou frissons _____ O
Marche difficile _____ O	Transpiration excessive _____ O	Maux de tête _____ O
Difficulté d'élocution _____ O	Diarrhées _____ O	Douleurs articulaires _____ O
Irritation des yeux _____ O	Vomissements _____ O	Crampes _____ O

Renseignements médicaux

Pouls _____ Tension artérielle ___ / _____ Pupilles _____

En cas de décès

Date du décès _____ Conclusions de l'autopsie _____

Autres renseignements _____

Veuillez renvoyer ce formulaire à la *Commission du Pacifique Sud, B. P. D5, Nouméa CEDEX, Nouvelle-Calédonie*

MERCI

Ciguatera

FISH POISONING

These fish have been found to carry the ciguatera poison:

Palani
Surgeonfish
length up to 18 i
weight up to 3 p

Wahanui
Snapper
length up to 2 feet,
weight up to 2 pounds.

Kole
Surgeonfish
length up to 7 inches,
weight up to 1/2 pound.

ʻŪʻū
Menpachi
length up to 14 inches,
weight up to 1 pound.

Uku
Snapper
length up to 24 inches,
weight up to 50 pounds.

Puhi
Moray Eel
length up to 6 feet,
most under 2 feet,
weight up to 70 pounds.