A STUDY OF CIGUATERA FISH POISONING IN THE VIRGIN ISLANDS AREA

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YEAR END REPORT

A STUDY OF CIGUATERA FISH POISONING IN THE VIRGIN ISLANDS AREA

Robert W. Brody, Principal Investigator

October, 1973

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College of the Virgin Islands
St. Thomas, U.S. Virgin Islands 00801

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APPENDIX I

INFORMATION SUMMARY ON CIGUATERA

OR

TROPICAL FISH POISONING

A. R Teytaud and R. W. Brody
Illustrations by Lyn Slocum

October, 1971

FORWARD

"A Study of Ciguatera Poisoning in the Virgin Islands Area" was a project funded under the National Sea Grant Program, authorized by the National Sea Grant College & Program Act [PL89-688]. The project period began March 1, 1971 for a period of one year, but was extended to terminate June 30, 1972. Funding was authorized for the project in the amount of $50,300 from the National Oceanic & Atmospheric Administration [NOAA] with $28,993 in matching funds appropriated by the College of the Virgin Islands. In addition, support was given the project in the amount of $6,000 from the Dave Hokin Foundation Research Fellowship.

The purpose of the CRI project [#53] was to pursue "preliminary research in the study of the ecology and epidemiology of ciguatera fish poisoning in the Virgin Islands area to include the location of a dependable source of toxic fish, the establishment of a facility competent to run screening bioassays on a large-volume of samples on a dependable schedule, the establishment of accurate reporting systems, and accurate data about incidents of human intoxication, and to analyze and compare toxin."

Personnel engaged in the project were: Robert W. Brody, principal investigator and marine ecologist; Anton R. Teytaud, research assistant/technician; James Gordon Williams, technician.

INDEX

CHRONOLOGY

Chronologically, the Ciguatera Project made the following efforts during a 16-month period:

March 1971 - October 1971

1. Literature survey and preparation of two information summary brochures on ciguatera fish poisoning.
2. Construction of chemistry and animal facility and ecology laboratory-office complex.
3. Equipping and setting up chemical extraction and bioassay laboratory.

October 1971 - January 1972

1. Establishment of Ciguatera Case Repository and start-up of information dissemination program. Interview program with fishermen and fish poisoning victims.
2. Bioassay of United Nations Fisheries Development Program samples.
3. Bioassay of fish from local St. Thomas areas as collected by project fishing efforts and local fishermen-contributions to the program.
4. Preparation and presentation of "Fish Poisoning in the Eastern Caribbean," Proceedings, Gulf and Caribbean Fisheries Association, 24: 1971.
5. Quantitative bioassay of toxic fish samples recovered in screening program or from fish poisoning victims.

February 1972 - May 1972

1. Publication of "Information Summary on Ciguatera or Tropical Fish Poisoning" and "Information About

Fish Poisoning" brochures.

2. Start-up of physician and clinic contact program. Initiation of epidemiology reporting from V.I. Public Health, Drug and Poison Control Center, etc.
3. Continuation of bioassay programs mentioned in paragraphs above.
4. Experimental bioassay development including new method for preparation of ciguatoxin samples and protozoan-mouse series.
5. Preliminary survey of Ciguatera incidence St. Maarten south to Martinique; establishment of contacts with physicians, public health and fisheries personnel.

June 1972

1. Summation of Ciguatera Case Repository collections to date.
2. Preparation of proposals and cooperative programs for 1973 fiscal year program.
3. Preparation of final (year end) report.
4. Close-down of laboratory.

The project activities are described in detail in the text and results summarized. The output from the first year's project will be incorporated into several publications if a continuation of the project is arranged. Much of the data is not complete enough for publication at the present time.

PROJECT ACTIVITIES

Epidemiology Programs

Information summary brochures were designed to serve two distinct audiences. "An Information Summary on Ciguatera or Tropical Fish Poisoning" was required to summarize present scientific and medical data in a

form easily read and digested by physicians, public health and fisheries personnel in the Virgin Islands. Its language was necessarily technical and the project was thus not bound to oversimplification and generalization. An appendix was prepared with taxonomically correct line drawings of highly suspect fish species. This was designed to aid the physician in identifying the species of fish wherever possible with the aid of the fish poisoning victim himself. An additional appendix described other types of biointoxications from ingestion of fish and summarized suggested therapies. All recipients of this summary were provided with Ciguatera Case Repository forms to encourage reporting of ciguatera and other toxin cases.

Preparation of a second pamphlet, reduced in size and depth of coverage required a considerable amount of "predigestion" and simplification. This pamphlet, entitled "Information About Fish Poisoning" was aimed at the adult layman and particularly at the native Caribbean population. The same appendix of drawings was provided to aid identification. The preparation of these information summaries began in April, 1971 and they were complete with editorial changes and final drafts available for printing in mid-October [Appendix I & II].

Two "public information" radio spot announcements were written and broadcast about 4 times per day for one month on local A.M. and F.M. stations. In addition, a number of public appearances (Rotary Club, Fisherman's Association, Hotel Association, local radio and TV) were made to inform the public of the project's existence, its research goals and to present the basic facts of fish poisoning in the Virgin Islands. School children who visited the laboratory as part of the Environmental Studies Program were given a short tour of the facilities and information about the fish poisoning problem.

A summary of recent information and apparent patterns of ciguatera and other types of fish poisoning was prepared for presentation at the 24th Annual Session of the Gulf and Caribbean Fisheries Institute at Miami, Florida. A copy of that paper is appended as Appendix III.

A systematic method was required for recording information from fishermen, physicians and fish poisoning victims. Two basic methods of data collection on the clinical events of ciguatera poisoning were designed and one program initiated immediately. A number of persons came to us after reading newspaper accounts of the project and volunteered case histories. People called in to inquire about the validity of treatments, some prescribed by their physicians, some prescribed by friends or relatives. Wherever possible the callers were invited to the laboratory, otherwise, a staff member visited [See Appendix IV for questionnaire].

Upon receipt of the Information Summaries in February, several project epidemiology reporting programs moved forward. Contacts with local medical facilities and their administrators were made. The project prepared a "check list" type form for physicians' use particularly in the clinic and emergency room situations. Wherever possible, this form became part of the "chart" record of admitted hospital patients. The project found good cooperation with most physicians expressing interest in assisting the project and satisfaction with the data collection format. The project's frequency of reports of ciguatera outbreaks increased noticeably when this contact with local medical authorities was established.

Both the victim interviews and the physician's checklist forms are filed in the Ciguatera Case Repository; a tabular summary of the data appears as Appendix V. A total of 29 cases were recorded during the first six months of 1972 from a total population of about 70,000 [U.S. Gov't. census, 1970].

Interviews with local fishermen were sought to discover leads to fish poisoning outbreaks and to put together folklore and experimental data on the geographic, seasonal and biological factors of fish poisoning known to them. A straightforward interview form was often difficult to administer and no quantitative inferences should be drawn from the data. Wherever possible, the following information was requested:

1. What kinds of fish (species) are most likely to be poisonous?
2. What time of year is most likely to produce toxic fish, if any?
3. What specific place(s) are toxic fish likely to be caught?
4. When was the last time you caught a poisonous fish and sold it?
5. Have you ever been fish poisoned?
6. How can you tell if a fish is poisonous?
7. What makes fish get to be poisonous?
8. Is fish poisoning a problem in selling fish?

The majority of fishermen mention the following species as toxic, usually in this order:

barracuda	-	Sphyraena barracuda
amberjack	-	Seriola dumerili
dog snapper	-	Lutjanus jocu
horse-eye jack	-	Caranx latus
jack crevalle	-	Caranx hippos
yellowfin grouper	-	Mycteroperca venenosa

Several other species are mentioned as infrequent (but occasionally violent) offenders including:

king mackeral	-	Scomberomorus cavalla
red hind	-	Epinephelus guttatus
black grouper	-	Mycteroperca bonaci
old wife	-	Balistes vetula

The majority of fishermen expressed the opinion that fish were toxic year round. Some expressed the opinion that the months of April through June are more likely to bring toxic fish. Several fishermen believe that some species "become" toxic by annual migrations through toxic areas or by seasonal cycles (including breeding) which bring demersal fish into the reef zone.

Virgin Islands fishermen uniformly express the opinion that the south side of the Virgin Islands bank produces toxic fish and the north side does not. Some temper this by excepting certain species, others except certain defined northside areas. Some fishermen (generally those who pot fish on the south side) insist that only certain areas produce toxic fish and that other areas are "safe". Most fishermen cull only amberjack from their catch and this because large individuals are heavily parasitized. We are only aware of an occasional incident of ciguatera poisoning where incorrect recognition of the species was a factor.

Most fishermen state that they are not aware of many fish poisoning incidents but all recognize the problem. Most had either experienced fish poisoning or eaten fish which caused symptoms in his immediate household. All of the local fishermen who supply a specific clientele or restaurant express concern and expect the buyer to exercise the decision; only a very few cull out highly suspect species. All expressed some degree of concern that fish poisoning affected the fishermens' income, but none seem to feel that it is the most important factor.

Most fishermen tend to reject certain fish species on the basis of personal experience and folklore. None indicated that there was a really reliable test that they themselves use. Many fishermen place some credence in the presence (or absence) of parasites on fish but the interpretations vary. Some say a "jack with a cockroach in his mouth" is not toxic but a large grouper is. "An amberjack with worms is okay but poisonous"

without" is another opinion. Most fishermen are only a bit more certain when questioned as to the origin of ciguatera poisoning. Many insist that fish feed on "copper banks" offshore or on reef areas where "copper from wrecked ships" is found. A very few insist that the real culprit is a "sea moss" which grows on rocks, pot warp or buoys. Several specimens have been brought to us and future work will include analysis and bioassay of these samples.

Bioassay Programs

The project began with no physical plant facilities. During April, May and June a lease was negotiated for a waterfront property on Benner Bay, St. Thomas with one prefab shell building, docks and a marine railway. A laboratory trailer surplussed by NASA following the Tektite II Program was acquired, moved from Lameshur Bay and installed at the Benner Bay property. A second trailer (a former classroom shell) was acquired and placed adjacent to it.

Beginning in mid-June, project staff, aided by Summer Conservation Program students and part-time help from CVI maintenance rebuilt the classroom trailer and refitted the laboratory trailer. All service facilities and interior work were complete by mid-August; installation of the electrical service was not made until after September 15. The shell building which already existed on the property was wired, framed out for partitions and ceilings and panelled between early August and mid-October by the project staff, maintenance personnel and a few weeks of summer student help. The facility was occupied and ready for work during the last week in October.

Chemical supplies, equipment bioassay animals and furniture were ordered beginning in May. The bulk of laboratory equipment was delivered in late September due to shipping strikes, mis-addressed packages and misplaced cargo containers. The last essentials, the mice for bioassay, arrived two weeks before electricity was installed (two weeks after it should have been available). The bulk of the shipment escaped while in

temporary quarters at C.V.I. The chemistry laboratory and mouse bioassay facility were in operating condition during the last week of October.

Fish from the local Virgin Islands area were screened for ciguatoxin utilizing mongoose feeding techniques. The individual fish were selected according to several criteria. The information gained from the literature, from interviews with victims and fishermen and from previous research efforts was combined to select certain fishing grounds as "potentially toxic". Within these areas, the largest specimens of the most suspect species were chosen for bioassay. These fish were taken by the project staff on several collecting trips aboard R.V. Reef Sampler and CRI small boats.

Several local professional fishermen expressed interest in providing toxic fish and about six were regular contributors to the project. Local sport fishermen also provided several specimens for bioassay. In both cases, the largest specimens of the most suspect species were retained for testing.

The mongoose assay used for all screening efforts assigned an arbitrary number to the severity of the incident based upon the symptoms demonstrated as follows:

0 no noticeable symptoms
1 weakness, lascitude
2 partial paralysis
3 complete paralysis of limbs but recovery within one week
4 death within twenty-four hours
5 death within one week

Several sets of fish samples were received from exploratory fishing efforts of the United Nations Food and Agricultural Organization Fisheries Development Programme, headquartered in Barbados during the early months of the project. A number of the samples had been stored and undergone several thaw/refreeze cycles and were badly decomposed. A series of subsamples of the UN/FAO fish were screened with mongoose bioassay to deter-

mine the toxicity of the catch. The samples are too small to represent a valid statistical base, but a number of toxic individuals were discovered. In all, samples from 12 localities were screened; only 2 toxic individuals were detected from 19 assayed with mongoose.

Chemical extraction and bioassay of ciguatoxin was undertaken within the restrictions of the project's personnel and equipment limitations. An extraction method developed by Scheuerer at the University of Hawaii and modified in cooperation with project consultants was utilized. The extracted ciguatoxin was bioassayed via I.P. injection into young 15-20 gm. mice. Samples were selected from positive results in the mongoose screening program and from toxic fish samples supplied by victims. Mouse results were intended as a comparative technique to "calibrate" the laboratory and work out the problems in technique. Since complete chemical laboratory services are not available in this area, we felt that our laboratory should be equipped to bioassay and determine toxicity of local samples but not to directly pursue chemical research better done in a continental laboratory. To this end we worked towards a method which provided optimum storage and shipping. The chemical extraction technique proved extremely time consuming and productivity was not as high as had been hoped. A series of comparative bioassays utilizing a variety of extraction technqiues was unfinished at the termination of the project. A series of micro-bioassay experiments utilizing marine and fresh protozoans in cooperation with the late Dr. Paul Burkholder was likewise unfinished and resumption of the series will be difficult in his absence. Although data are incomplete, we have reason to believe that freeze-drying samples (up to 200 grams) is the best method for preservation for shipping. We can find no change in toxicity using a variety of extraction techniques with mouse bioassay. This general chemical procedure and large scale freeze drying apparatus should be part of a continuation of the program.

The abrupt termination of the program has prevented a complete, orderly summation of the project and a number of questions remain unanswered. It is hoped that a continuation of the ciguatera project's research goals can be arranged specifically to complete research in the following areas:

Epidemiology Survey:

1. Complete one-year cycle in Virgin Islands area.
2. Extend coverage to British Virgin Islands and Leeward Islands areas.

Chemistry Program:

1. To identify toxic fish for freeze drying and shipment to continental laboratories.
2. To improve bioassay reliability and increase ease of handling samples.
3. To provide support as necessary to ecology program.

Ecology Program:

1. To identify the food chains and toxic fish species, particularly at the lower trophic levels.
2. To discover the source of ciguatoxin in the ecosystem utilizing bioassay technqiues and directed screening of potentially toxic plants, fungi and invertebrates.

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October, 1971

St. Thomas, (U.S.)V.I.

TABLE OF CONTENTS

Note added in press:

The statement (on page 7) that one component of ciguatoxin acts as an irreversible cholinesterase inhibitor is based on evidence from several *in vitro* studies. However, more recent research (Rayner, M.D., *et al.* 1969. J. Fish. Res. Bd. Canada 26(8):2208-2210) demonstrates that "Although ciguatoxin, prepared from the liver and flesh of the moray eel *Gymnothorax javanicus* [a Pacific species], is an *in vitro* anticholinesterase, it has not been found to be an *in vivo* anticholinesterase. Respiratory failure and death due to ciguatoxin is not a function of cholinesterase inhibition as previously speculated, although the toxin may have a widespread action at cholinergic junctions." Rayner (1970. *In* Food - drugs from the sea, Proceedings, 1969. Marine Tech. Soc. Pp. 345-335) and Boyarsky and Rayner (1970. Proc. Soc. Exp. Biol. Med. 134:332-335) suggest that this widespread action involves changes in membrane excitability and membrane potential.

Setliff, *et al.* (1971. Toxicol. Applied Pharmacol. 18:676-684) conducted experiments on the effect of ciguatoxin on the sodium transport mechanism in frog skin. Their results suggest that ciguatoxin "primarily increases the passive permeability of the skin to Na, although an effect on the Na pump cannot be ruled out completely".

A.R.T. and R.W.B.

FOREWORD

THE CIGUATERA STUDIES PROJECT

OF

CARIBBEAN RESEARCH INSTITUTE

This information summary has been prepared by the staff of the Ciguatera Studies Project of the Caribbean Research Institute, College of the Virgin Islands, St. Thomas, U. S. V. I. The project seeks to carry forth research programs initiated in 1967 under Exploratory Fishing and Fisheries Development projects funded by the Bureau of Commercial Fisheries and the Bureau of Sport Fisheries and Wildlife under the Dingell/Johnson and Public Law 88-309 Programs with matching funds from the Government of the Virgin Islands. The present program is supported by a Sea Grant from the National Oceanographic and Atmospheric Agency of the U. S. Department of Commerce with matching funds from the United Nations Caribbean Fisheries Development Program, the National Marine Fisheries Service, the Hokin Foundation and the Caribbean Research Institute.

The Ciguatera Studies Project, with laboratory facilities at Benner Bay, St. Thomas, is concentrating on two important phases of research at this time. Suspect species are collected at various locations throughout the Virgin Islands and are subject to a bioassay procedure wherein the toxin is extracted from fish muscle tissue and purified with solvent and chromatographic techniques. The purified toxin is then injected into laboratory mice for bioassay. Through this procedure a species and geographic distribution record of the toxin may be obtained.

The second phase of the present work attempts to gather valid public health records of ciguatera intoxications from physicians who are contacted by toxin victims and through a questionnaire program coupled with public awareness announcements in local media. Through these procedures a more precise estimate of the statistical magnitude of this public health problem may be constructed. The Project is also interested in other marine biotoxications; to this end

we have included a series of description of different types of poisoning incidents(Appendix II).

Cooperative research programs are under way to isolate and chemically characterize the toxin(s) which produce ciguatera poisoning and to more precisely describe its physiological mechanisms of action. These programs are based at the National Marine Fisheries Service laboratory in Seattle, Washington and, through the auspices of the United Nations' World Health Organization, at laboratories in Bilthaven, the Netherlands and San Juan, Puerto Rico.

All concerned individuals are invited to assist the Ciguatera Studies Program by providing data on intoxications known to them. If possible, the fish should be identified (utilizing the illustrations in Appendix I where possible) and other information (such as that in Appendix III) noted. If even a small sample of the toxic fish is recoverable (100 grams or more) the reporting individual is encouraged to make every effort to obtain a sample.

In the Virgin Islands please preserve the fish by freezing the sample and contact the Project at 775-2410 (call collect from the British Virgin Islands). At other localities in the Caribbean preservation for shipping (by air mail, collect) should be made in 85-95% ethyl or isopropyl alcohol. Our mailing address:

CIGUATERA STUDIES PROJECT
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This project is supported by a Sea Grant (1-35368) from the National Oceanic and Atmospheric Agency of the U.S. Department of Commerce.

INFORMATION SUMMARY ON CIGUATERA,

OR

TROPICAL FISH POISONING

Ciguatera is a type of poisoning which results from eating the flesh or viscera of a large variety of tropical marine fishes. While a number of different types of fish poisoning are known, ciguatera is the type most prevalent in the Virgin Islands. According to a recent survey (Halstead, 1970), fish poisoning is an important public health problem in the West Indies. There is evidence suggesting that the largest population of toxic fish in the Caribbean are found within the Virgin and northern Leeward Islands.

The following information summary has been extracted from the voluminous scientific literature on the subject, and is intended as a source of accurate information for physicians and members of the community concerned with prevention and treatment of ciguatera in this area. Illustrations of toxic species and a summary of other types of fish poisoning are given in the appendices.

NATURE & ORIGIN OF THE TOXIN

It is not known whether ciguatera is a single poison or a complex of poisons, but several investigators feel that it is probably a complex structure having several fractions. The chemical formula of the toxin and its biogenesis still remain a mystery, even after many years of research. Ciguatera is in no way associated with bacterial food poisonings (it can be caused by fresh fish) and its symptoms and treatment are entirely different from such cases. Neither cooking nor any common method of fish preparation such as drying or salting seems to have any appreciable effect in destroying ciguatoxin. Since the toxin is slightly water-soluble, soup is a particularly dangerous method of preparing toxic fish.

There is much evidence indicating that the source of the toxin is in the environment, rather than in the internal organs of the fish. Most investigators believe the toxin is derived from the food. The toxin is cumulative; that is, the concentration of toxin tends to increase as the fish gets older since it apparently is not broken down

or excreted. This accounts for the well-known fact that unusually large specimens of certain fishes are more apt to cause ciguatera than smaller individuals. The highest concentration of toxin is usually in the liver, followed by the intestines, gonads, and then the flesh.

Many theories have been proposed as to the source of the ciguatoxin and its place in the marine food web. Several of these theories are lacking in scientific support, including a most persistent and widespread belief in the role of copper as a causative factor in ciguatera outbreaks. It should be emphasized that no pharmacological or ecological study has implicated copper in ciguatera poisoning. Also arguing against this theory is the fact that many well-documented outbreaks of ciguatera have occurred on limestone atolls where no copper is present in the substrate and where there were no known shipwrecks. It is true that the site of an abandoned copper mine in Virgin Gorda is one of the most notoriously ciguatoxic areas in the Virgin Islands. However, this kind of circumstantial evidence does not demonstrate a direct connection between copper and ciguatoxin.

Most researchers at the present time subscribe to some variation of the algal food chain theory, which may be summarized as follows (modified from Randall, 1958):

1. Fishes become poisonous because of some factor in their environment.
2. The toxicity of fishes is associated with their food supply.
3. The basic poisonous organism is benthic.
4. Since obligate herbivorous and detritus-feeding fishes may be poisonous, the toxic organism would most likely be an alga, a fungus, a protozoan, or a bacterium.
5. Blue-green algae would be the most likely source for ciguatoxin since this same group is also involved in freshwater poisonings.
6. The most poisonous fishes are the large predacious species, especially those that feed on other fishes. When a predator eats an herbivore, detritus-feeding fish, invertebrate, or another carnivore, he acquires in one short period the toxin accumulated by

the animal over its lifetime.

7. The organisms producing ciguatoxin may be some of the first growing on new or denuded surfaces in tropical seas in the course of normal ecological succession. The availability of new surfaces may be the result of storms, hurricanes, earthquakes, dumping of war material, garbage, dredging operations, blasting, shipwrecks, or by the outflow of turbid fresh water which kills the normal marine flora.

CIGUATOXIC SPECIES IN THE VIRGIN ISLANDS

Apparently any tropical marine fish can become ciguatoxic under the right conditions. However, toxicity appears far more frequently in certain species, usually insular near-shore forms, although a few open-ocean species are often poisonous. Most ciguatoxic fishes are reef dwellers, living either on the bottom or feeding on bottom-dwelling fishes. They are usually found at depths of less than 300 feet.

Carnivores, benthic herbivores, and detritus feeders are more apt to become poisonous than plankton-feeding species which (with some exceptions) are generally safe to eat. Moderate and large-sized fishes are thought to be more often toxic than small ones, but there are many cases in which small species have proven toxic.

The following is a list of fishes known to be commonly ciguatoxic in the vicinity of the U. S. and British Virgin Islands; species at the top of the list are more frequently reported as toxic than those lower down.

Table 1

Common Name	Scientific Name
Barracuda (Barra)	Sphyraena barracuda
Amberjack	Seriola dumerili
Horse-eye Jack	Caranx latus

Table 1 (cont'd)

Common Name	Scientific Name
Bar Jack (Carang)	Caranx ruber
Crevalle Jack (Cavalli)	Caranx hippos
Dog Snapper (Dog Tooth)	Lutjanus jocu
Yellowfin Grouper (Gramminix)	Mycteroperca venenosa
Kingfish	Scomberomorus cavalla
Blue Runner (Hard Nose)	Caranx fusus
Conger, Congo, or Moray	(probably several species of Gymnothorax or other morays)
Rock Hind	Epinephelus adscensionis
Black Grouper	Mycteroperca bonaci
Cero (often erroneously referred to as "Spanish Mackerel")	Scomberomorus regalis
Black Jack	Caranx lugubris
Hogfish	Lachnolaimus maximus
Gray Snapper	Lutjanus griseus
Almaco Jack	Seriola rivoliana
Yellow Jack	Caranx bartholomaei
Red Hind	Epinephelus guttatus
Black Snapper	Apsilus dentatus
Blackfin Snapper	Lutjanus buccanella
Queen Triggerfish (Old Wife)	Balistes vetula
Misty Grouper (often assumed to be the "Warsaw Grouper" by V. I. fishermen who have seen that species in Florida).	Epinephelus mystacinus

None of the species listed above is always toxic, but it is well to regard them with suspicion (especially the first 14) and to avoid eating the larger individuals and those caught in known toxic localities. Line drawings and descriptions of the above species are presented in Appendix I as an aid to identification.

LOCATIONS REPORTED AS TOXIC IN THE V. I.

Some locations have been notorious for producing ciguatoxic fishes over a time span of several centuries. Other areas have

suddenly become toxic and then lost their toxicity within a few months. There is no known way of predicting ciguatoxic areas - one section of a reef may produce severe intoxications while an adjacent area of the same reef may be completely safe. Not all the fishes in a toxic locality may be poisonous; even within the same species some individuals may be poisonous while others are completely wholesome. Since new ciguatoxic areas may appear without warning, newcomers to the islands are advised to seek information from the local fishermen before consuming any fish.

The following areas have been mentioned in the literature as often producing toxic fish:

(1) St. Croix - Eastern tip, Lang Bank
(2) St. Thomas - entire South side, Smith Bay and Luongo Cay, Sail Rock
(3) St. John - South side, East End, Congo Cay
(4) Tortola and adjacent islands - islands and cays from Norman Island to Virgin Gorda, Necker Island Pass, Southeast side of Tortola, Anegada Reef near Roccos.

There is a common belief among the local fishermen that spring and early summer are the seasons when toxic fish are most common.

SYMPTOMS OF CIGUATERA POISONING (FROM HALSTEAD, 1967)

Ciguatera fish poisoning in its simplest uncomplicated form develops within 3 to 5 hours after the fish is eaten. There is a sudden onset of abdominal pain followed by nausea, vomiting, and a watery diarrhea. The gastrointestinal symptoms will occur in about 40 to 75 percent of the cases. The victim feels weak, generally ill, and may experience muscle aches throughout the back and thighs in about 10 percent or more of the cases. Soon after, the victim complains of numbness and tingling in and about the mouth which then extends to the extremities (present in about 50 percent or more of the

cases). Fever, headache, and rash are generally absent, and the patient has no desire for food. The acute symptoms usually subside in about 8 to 10 hours, and within 24 hours after onset most of the patient's symptoms will have completely subsided except for a feeling of weakness. However, the numbness and tingling may continue to a lesser extent for a period of 4 to 7 days. The foregoing resume is typical of the majority of uncomplicated ciguatoxications that are generally encountered by the practicing physician in an endemic ciguatoxic locality.

Ciguatera, like many other diseases, may vary greatly in its clinical manifestations depending upon the toxicity of the fish that is eaten, the individual's sensitivity to the poison, amount of fish ingested and other factors. In a broader sense ciguatera fish poisoning may be characterized as follows: the onset of symptoms may vary from almost immediately to within a period of 30 hours after ingestion of the fish, but is usually within a period of 6 hours. The initial symptoms in some cases are gastrointestinal in nature, consisting of nausea, vomiting, watery diarrhea, metallic taste, abdominal cramps, and tenesmus, whereas in other patients the initial symptoms consist of tingling and numbness about the lips, tongue, and throat. This may be accompanied by a sensation of dryness of the mouth. The muscles of the mouth, cheeks, and jaws may become drawn and spastic with an accompanying sensation of numbness throughout. Generalized symptoms of headache, anxiety, malaise, prostration, dizziness, pallor, cyanosis, insomnia, chilly sensations, fever, profuse sweating, rapid weak pulse, weight loss, myalgia, and back and joint aches may be present in varying degrees, or one or more of these symptoms may be entirely absent. The victims usually complain of a feeling of profound exhaustion and weakness. The feeling of weakness may become progressively worse until the patient is unable to walk. Muscle pains are generally described as a dull, heavy ache, or cramping sensations, but on occasion may be sharp, shooting, and affect particularly the arms and legs. Victims complain of their teeth feeling loose and painful in their sockets. Visual disturbances consisting of blurring, temporary blindness, photophobia, and scotoma are common. Pupils are usually dilated and the reflexes diminished. Skin disorders are frequently reported that are initiated by an intense generalized pruritus, accompanied by erythema, and maculopapular eruptions, blisters, ex-

tensive areas of desquamation - particularly of the hands and feet - and occasionally ulceration. There may also be a loss of hair and nails.

In severe intoxications the neurotoxic components are especially pronounced. Paresthesias involve the extremities, and paradoxical sensory disturbances may be present in which the victim interprets cold as a "tingling, burning, dry-ice or electric-shock sensation", or hot objects may give a feeling of cold. In regard to the paradoxical sensory disturbance (P. S. D.), a classic example is that of a naval officer who was poisoned by an amberjack. Four weeks later he was observed subconsciously blowing on his ice cream, which was "burning his tongue", in order to cool it. Ataxia and generalized motor incoordination may become progressively worse. The reflexes may be diminished, muscular paralyses may develop, accompanied by clonic and tonic convulsions, muscular twitchings, tremors, dysphonia, dysphagia, coma, and death by respiratory paralysis. The limited morbidity statistics show a case fatality rate of about 12 percent. Death may occur within 10 minutes, but generally requires several days.

TREATMENT

Treatment of ciguatera is mostly symptomatic at present. No immunity is conferred by an attack (in fact, sensitization may occur) and no specific antidote is in general use.

The stomach should be emptied by gastric lavage, emetics, or saline purges as soon as possible. Hyperventilation and shock associated with the acute cases of poisoning in lab animals suggest a profound alteration of electrolyte balance and blood pH which would be amenable to calcium therapy. Some severe human cases have responded well to intravenous 10 percent calcium gluconate, whereas others have not. It is recommended that calcium therapy be tried.

Preliminary studies have shown that at least one component of the toxin acts as an irreversible cholinesterase inhibitor, thus paralleling the action of organophosphorus compounds present in certain insecticides. One promising development has been the use of the oxime protopam chloride (2-formyl-1 methyl pyridinium

chloride oxime, distributed by Campbell Pharmaceutical Inc., New York) in severe ciguatoxications in both lab animals and humans. When given intravenously or intramuscularly together with atropine, there was a remarkable decrease in the symptoms of severe cases if treatment was administered early enough. Bagnis (1968) cites the following dosages used in his experiments:

Method 1: Very serious cases of immediate urgency, accompanied by a state of shock.

- 1 gramme of Protopam in an intravenous perfusion of 250 cc of salt serum to be given in the course of 30 minutes.
- 1/4 milligramme of atropine to be given subcutaneously and repeated within the following half-hour.
- These doses may be repeated once after two hours if the state of shock remains unchanged.

Method 2: Serious cases unaccompanied by a state of shock.

- 1 gramme of Protopam in 20 cc of distilled water given by means of a very slow intravenous injection (at least 2 minutes).
- For the atropine, same technique and dose as Method 1.

Method 3: Average cases.

- 1 gramme of Protopam in 10 cc of distilled water given by means of a slow intramuscular injection (one minute) either on its own or in conjunction with 1/4 of a mg of atropine given subcutaneously.

Supportive therapy consisting of intravenous glucose in normal saline and injections of vitamin B_6 have been used with varying degrees of success. Victims suffering from moray eel

poisoning appear to be particularly a susceptible to violent convulsions and may present difficult nursing problems. Rest, quiet, and sedation are essential because the convulsions may be precipitated by noise or commotion of almost any type. Paraldehyde and ether inhalations have been reported to be effective in controlling the convulsions. Nikethamide or one of the other respiratory stimulants are advisable in cases of respiratory depression. In patients where excessive production of mucus is a factor, aspiration and constant turning are essential. Atropine has been found to make the mucus more viscid and difficult to aspirate, and may be contraindicated. If laryngeal spasm is present, intubation and tracheotomy may be required. Oxygen inhalation may be necessary. If the pain is severe, opiates may be necessary. Morphine given in small divided doses has been recommended. During later stages of recovery cool showers may be useful in relieving the severe itching that is sometimes present. Fluids given to patients suffering from the paradoxical sensory disturbance (temperate sensation reversal) should be tepid rather than either hot or cold. Intravenous procaine hydrochloride has been used with reputed success in the treatment of a single case of barracuda poisoning (Halstead, 1967).

PREVENTION

At present no simple chemical field test exists to detect a ciguatoxic fish. The most reliable method is to test the fish by feeding flesh or viscera to a test animal (dog or cat) - if the animal does not become sick the fish is presumably safe.

There seems to be no value whatever in certain folklore methods which are commonly used by natives to detect poisonous fish. Some methods used in the islands are the following (Dammann, 1969):

1. Ciguatoxic fish have different coloration than normal fish (a) more yellow or brassy (b) stripes (c) darker.
2. Presence of isopod parasites indicates nonciguatoxic fish.
3. Raw flesh of ciguatoxic fish, especially the liver, tastes bitter or hot in the mouth.

4. Flies will not land on exposed flesh of ciguatoxic fish.
5. Silver turns black when boiled with ciguatoxic fish.
6. Sweet potato turns black when boiled with ciguatoxic fish.
7. Ciguatoxic fish have brassy or coppery odor.
8. Ciguatoxic fish have enlarged or bloated stomach.
9. Ciguatoxic fish have yellow mucous on inner linings of gullet.
10. Ciguatoxic fish have green tint to raw flesh.
11. Suspected specimen with roe is ciguatoxic.
12. Ants will not eat ciguatoxic fish.
13. Ciguatoxic fish have tiny black "veins" running through the flesh.

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APPENDIX I

ILLUSTRATIONS AND DESCRIPTIONS

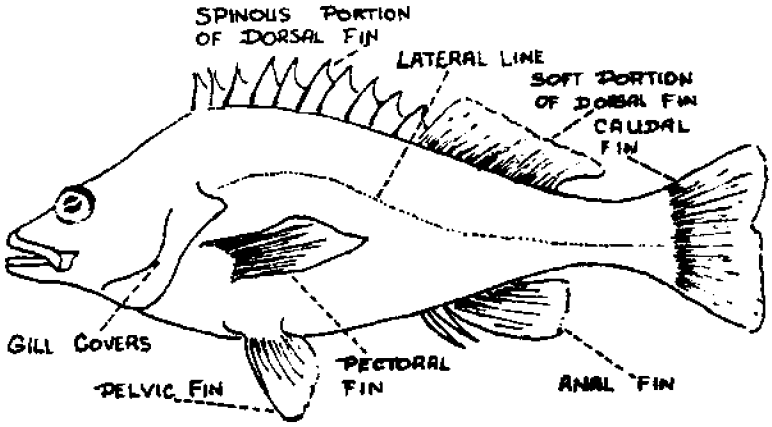
OF

COMMONLY CIGUATOXIC FISHES

APPENDIX I

ILLUSTRATIONS AND DESCRIPTIONS OF COMMONLY CIGUATOXIC FISHES

The fishes illustrated are presented in approximate groupings by family and genus to facilitate comparison between closely related species. The illustrations are taxonomically correct with the distinguishing features emphasized. The generalized fish (Illustration 0) will be of assistance in utilizing the descriptive data which appears opposite each illustration. For precise taxonomic data the reader is referred to Randall (1968), Bohlke and Chaplin (1968) or Nichols (1930). For the working physician or Public Health official the likelihood of precise species identification is doubtful since the fish implicated has most likely been mutilated in cooking. If at all possible the ciguatoxicated patient might be shown the pictures to aid in identification.



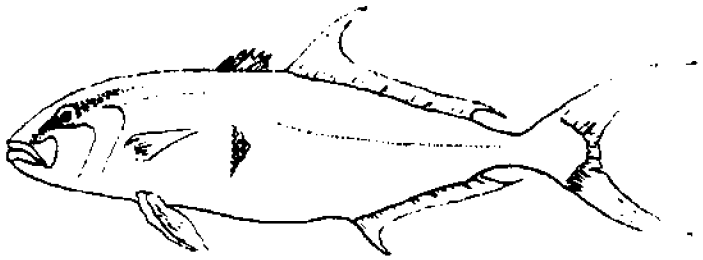
0.

(1) Amberjack (Seriola dumerili)- In adults the body is dark on the back (steely blue, brownish, or olive), silvery white on the belly, and intermediate on the sides. Sometimes there is a lengthwise amber stripe from the eye to the tail. A diagonal dark band runs from the snout through the eye to the neck. May reach 6 ft.; most individuals are 3-4 ft., occurs in schools over shallow reefs in the evening and at night.

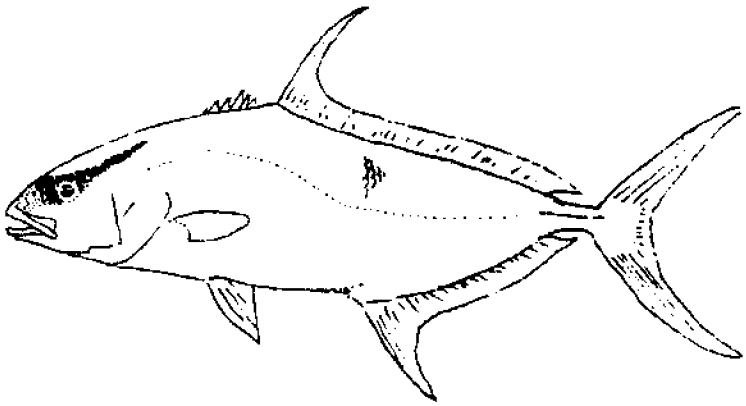
(2) Almaco Jack (Seriola rivoliana) - Similar to the Amberjack in coloration but has a deeper body and a longer lobe at the front of the second dorsal fin. The band through the eye is darker than in the Amberjack, and there is no lengthwise stripe on the sides. Probably attains at least 50 lbs.

(3) Bar Jack or Carang (Caranx ruber) - Very similar in body shape to the Yellow Jack and the Blue Runner, but differs in coloration. The distinguishing mark is a blackish stripe which runs along the upper back just below the soft dorsal fin, crosses the base of the tail and extends onto the lower lobe of the tail fin. A brilliant blue stripe runs below the dark stripe from the base of the tail to the tip of the snout in fresh specimens. The body is otherwise silvery all over. Grows to 2 ft. in length.

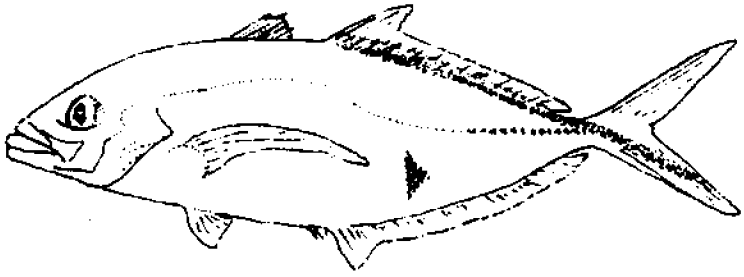
(4) Blue Runner (Caranx fusus) - Has a black spot on the rear of the gill covers, which is not present in either the Bar Jack or the Yellow Jack. The back is irridescent blue, but there is no black stripe as in the Bar Jack. Sides and belly silvery grey to golden. Grows to 2.5 ft. and 4 lbs.



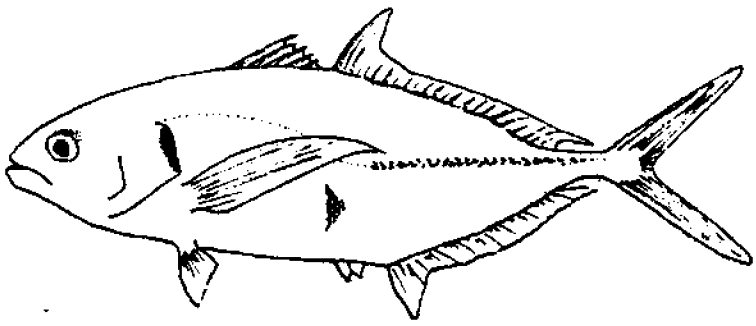
1. Amberjack (Seriola dumerili)



2. Almaco Jack (Seriola rivoliana)



3. Bar Jack or Carang (Caranx ruber)



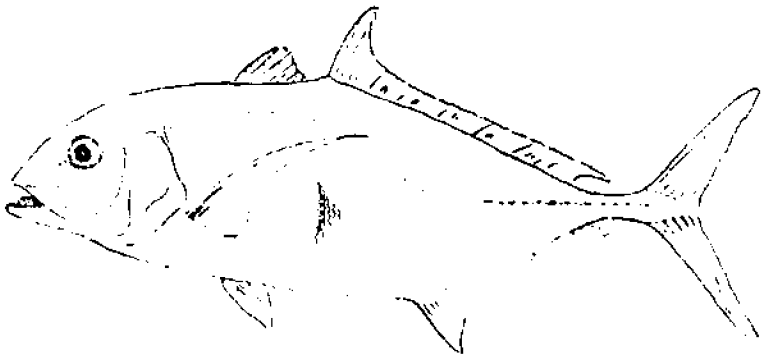
4. Blue Runner (Caranx fusus)

(5) Yellow Jack (Caranx bartholommaei) - Back irridescent blue, side silvery with a yellow cast. There is no black stripe on the back as in the Bar Jack, and no black spot on the gill covers as in the Blue Runner. Attains at least 3 ft. 3 in. and 17 lbs.

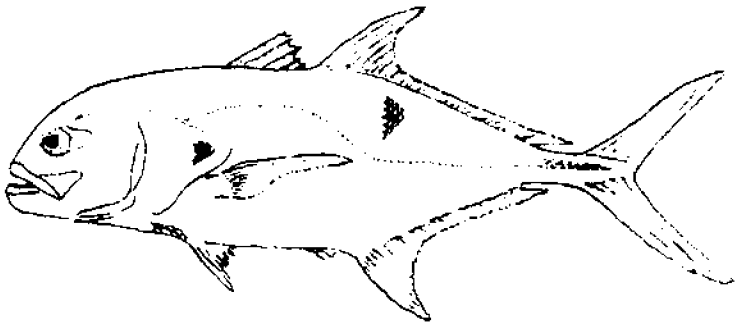
(6) Crevalle Jack or Cavalli (Caranx hippos) - Has a steep head profile like the Black Jack and Horse-eye Jack. Greenish to bluish on back, silvery or yellowish on belly and sides. Has only a small patch of scales on the chest region in front of the ventral fins, which distinguishes it from the Horse-eye. Attains more than 3 1/2 ft.; 55 lbs is the record.

(7) Horse-eye Jack (Caranx latus) - Very similar in coloration to the Crevalle. Distinguishing characteristics are a larger eye than the Crevalle, and the presence of scales on the entire chest region. Grows to at least 30 in.

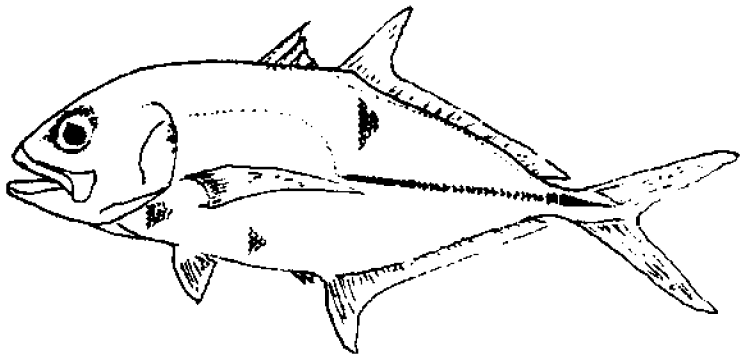
(8) Black Jack (Caranx lugubris) - Body is uniformly dark brown to black. The coloration distinguishes this fish from any other jack in this area. Attains 3 ft. 3 in. and 15 lbs.



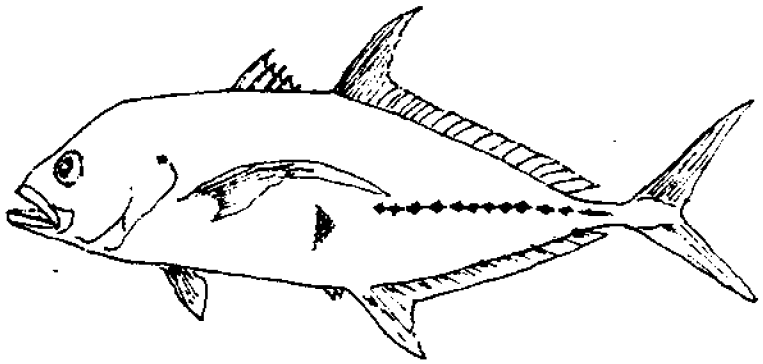
5. Yellow Jack (Caranx bartholomaei)



6. Crevalle Jack or Cavalli (Caranx hippos)



7. Horse-eye Jack (Caranx latus)



8. Black Jack (Caranx lugubris)

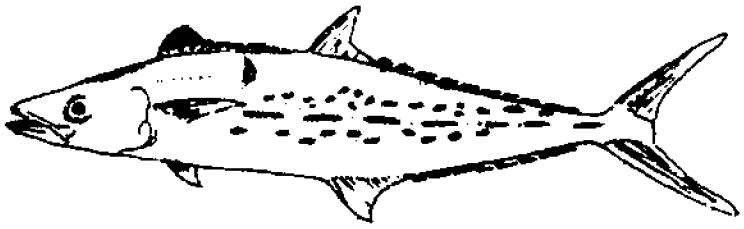
(9) Great Barracuda or Barra (Sphyraena barracuda) - Dark green to gray on back, silvery on sides, with several black blotches on lower sides which vary in size and position. White on belly. May exceed 6 ft., but 3 1/2 ft. is considered large. No similar fish in region.

(10) Cero or "Spanish Mackerel" (Scomberomorus regalis) - Sides silvery, back bluish-green, a row of yellow or brown longitudinal streaks in the middle of the side, with small yellow spots above and below this row. There is a large black area at the front of the first dorsal fin. There are scales on the pectoral fins. Attains 4 ft. and 26 lbs.

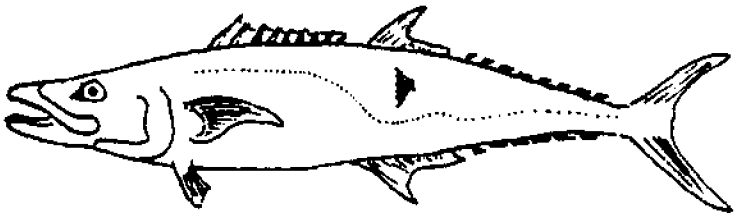
(11) Kingfish (Scomberomorus cavalla) - bluish-green on back, sides plain silvery (no streaks or spots as in the Cero), no black area in the first dorsal fin. There are no scales on the pectoral fins. The lateral line takes a much sharper downward curve below the front of the second dorsal fin than it does in the Cero. Grows to at least 5 1/2 ft. and 80 lbs.

(12) Dog Snapper or Dog Tooth (Lutjanus jocu) - Body dark olive to brown on the upper parts and generally reddish with a coppery cast elsewhere. Narrow pale cross-bars on side. The pale triangular-shaped bar beneath the eye is the most characteristic color feature, but this is not always present. There is a row of blue spots below the eye and across the gill cover. Large specimens reach 30 in and 20 lbs.

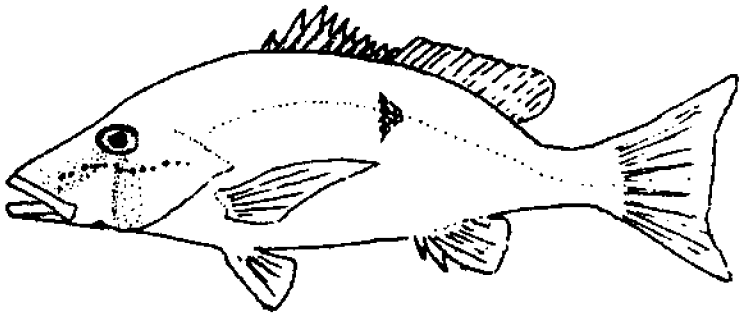
9. Great Barracuda or Barra (Sphyraena barracuda)



10. Cero or "Spanish Mackerel" (Scomberomorus regalis)



11. Kingfish (Scomberomorus cavalla)



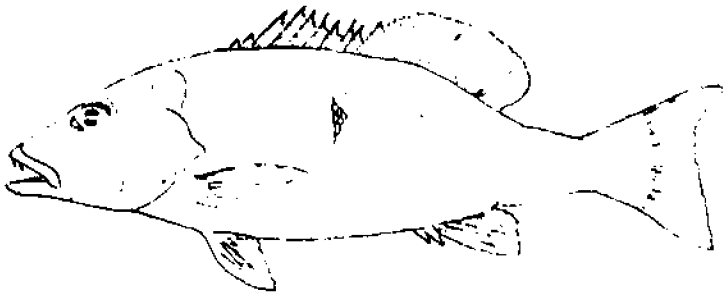
12. Dog Snapper or Dog Tooth (Lutjanus jocu)

(13) Gray Snapper (Lutjanus griseus) - Generally grayish with reddish tinges, but coloration is highly variable. Commonly there are orange to rusty spots on the centers of the scales on the sides, forming rows. Distinct dark diagonal bar through eye often present. Resembles the Dog Snapper, but differs in having a proportionately shorter pectoral fin and shallower body. Reaches at least 3 ft. and 20 lbs.

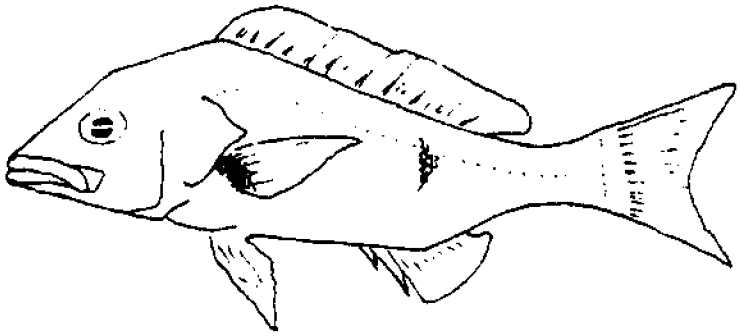
(14) Blackfin Snapper (Lutjanus buccanella) - Red above, silvery with red below, tail fin bright yellow, other fins yellowish. Distinguishing mark is a dark blotch at base of pectoral fins. Primarily a deepwater species. Attains 20 in. in length.

(15) Black Snapper (Apsilus dentatus) - Entirely brown, darker on the back than below. Has no scales on posterior dorsal and anal fins. Rarely exceeds 8 lbs. Generally deepwater.

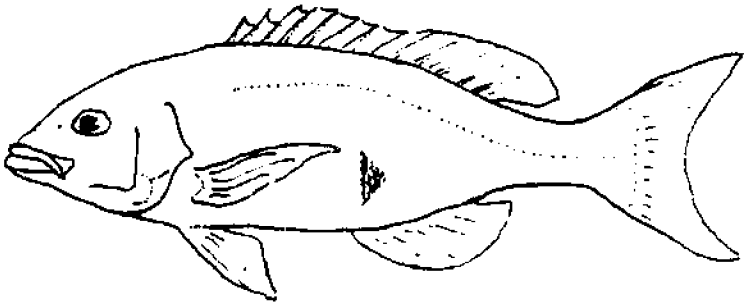
(16) Hogfish (Lachnolaimus maximus) - Highly variable in color pattern, depending on size, sex and environmental conditions. Distinguishing characters are the body shape and the three elongated, streamer-like rays at the front of the dorsal fin. Large males like the one illustrated have a long, pig-like snout and buck teeth. Grows to almost 3 ft.



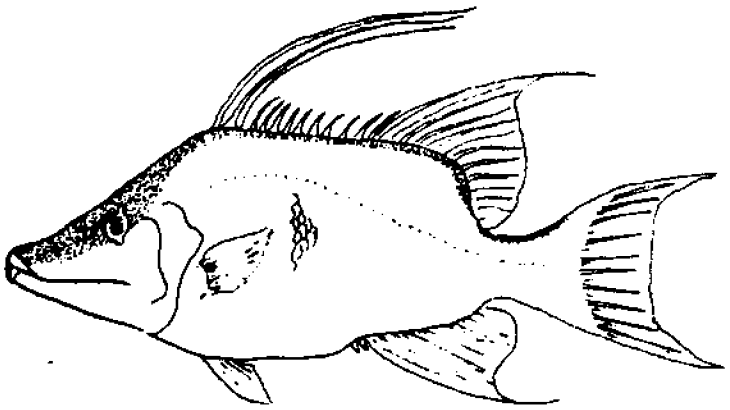
13. Gray Snapper (Lutjanus griseus)



14. Blackfin Snapper (Lutjanus buccanella)



15. Black Snapper (Apsilus dentatus)



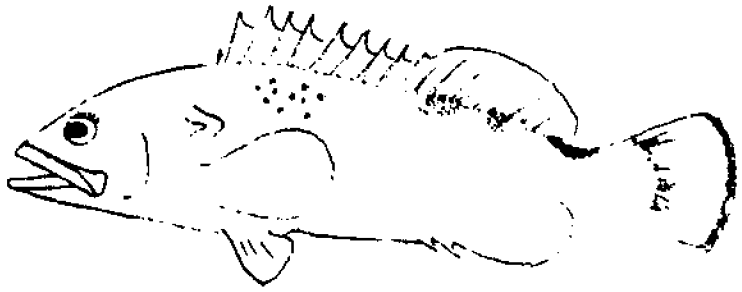
16. Hogfish (Lachnolaimus maximus)

(17) Rock Hind (Epinephelus adscensionis) - Head and body have a ground color of pale olive or blue on the back, and white on the lower half. Entire body and fins covered with spots which are red with darker, brownish centers. Has large black blotches along the base of the dorsal fin which are not continuous with any black areas on the dorsal fin. A dark brown blotch on the upper part of tail base. Grows to 18 in. or longer.

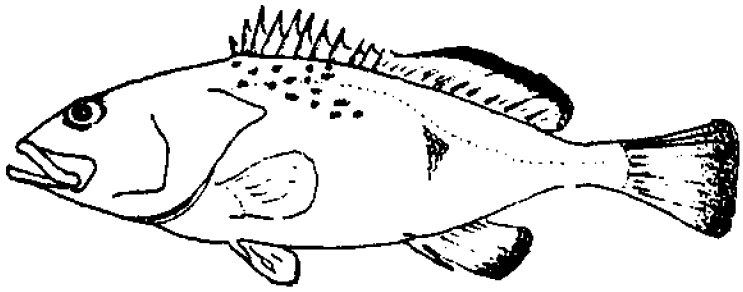
(18) Red Hind (Epinephelus guttatus) - Similar to Rock Hind, but differs in coloration. Ground color of head and body is variable, but reddish spots with darker centers are always present. The rear portion of the dorsal fin, the anal fin, and the tail fin have broad margins of black, with a thin white line along the extreme edges - these dark margins on the fins are not present in the Rock Hind. If there are any dark blotches along the base of the dorsal fin, then they are connected with black areas on the fin itself. Grows to 18 in. or longer.

(19) Yellowfin Grouper or Gramminix (Mycteroperca venenosa) - Highly variable in color, but body usually has a gray to olive ground color with many dark-brown blotches arranged in approximate lengthwise rows. Numerous small, dark reddish or orange spots. Distinguished from the black grouper by the broad yellow margin on the pectoral fins (margin is about 1/3 the length of the fin in width) which is sharply marked off from the rest of the fin. Grows to at least 3 ft.

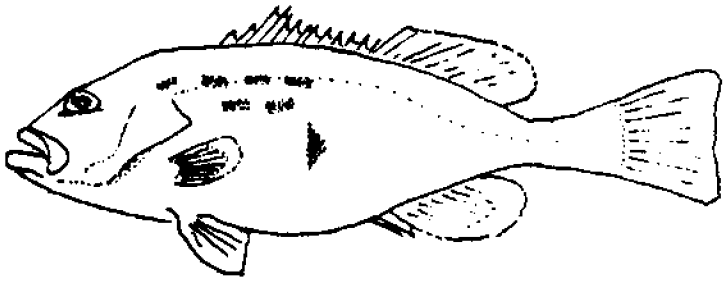
(20) Black Grouper (Mycteroperca bonaci) - Closely resembles the Yellowfin. Distinguished from the Yellowfin by the narrow orange margin on the pectoral fins (margin is about 1/5 the length of the fin in width) that grades into the darker basal portion of the fin. The dark blotches on the body are larger, more rectangular, and more regularly aligned than the Yellowfin. Recorded to a length of 4 ft. and 100 lbs. wt.



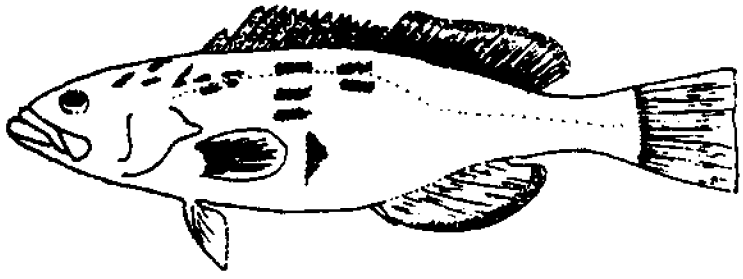
17. Rock Hind (Epinephelus adscensionis)



18. Red Hind (Epinephelus guttatus)



19. Yellowfin Grouper or Gramminix (Mycteroperca venenosa)

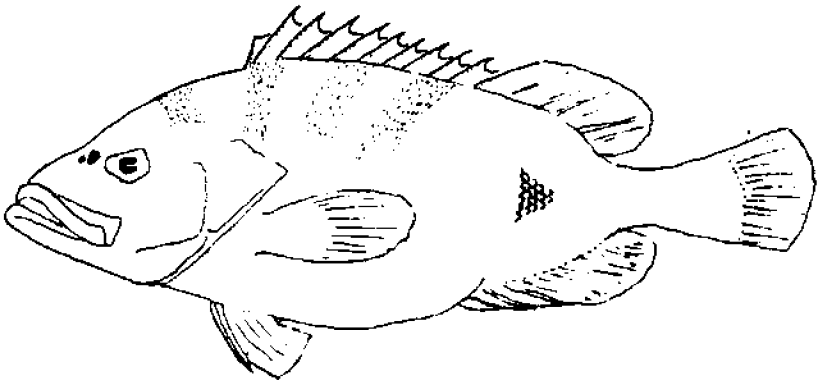


20. Black Grouper (Mycteroperca bonaci)

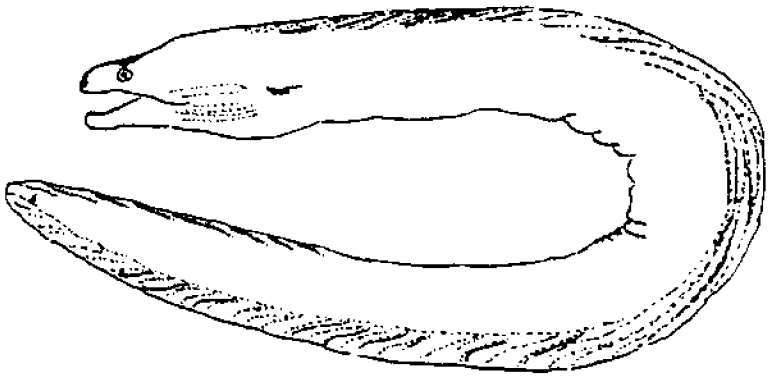
(21) Misty Grouper (Epinephelus mystacinus) - Body dull brownish or greyish brown, crossed by bands of darker olive brown. A deep-water species. Grows to 3 ft. and 120 lbs wt. Generally resembles a dark Nassau but lacks the distinct black blotch at the base of the tail.

(22) Conger, Congo, or Moray (Gymnothorax funebris)
There are many species of morays in this area. Although color patterns vary widely, all have the same distinctive body shape. The species illustrated in the Green Moray (Gymnothorax funebris) which may attain a length of more than 6 ft. Morays can cause a particularly violent form of ciguatera.

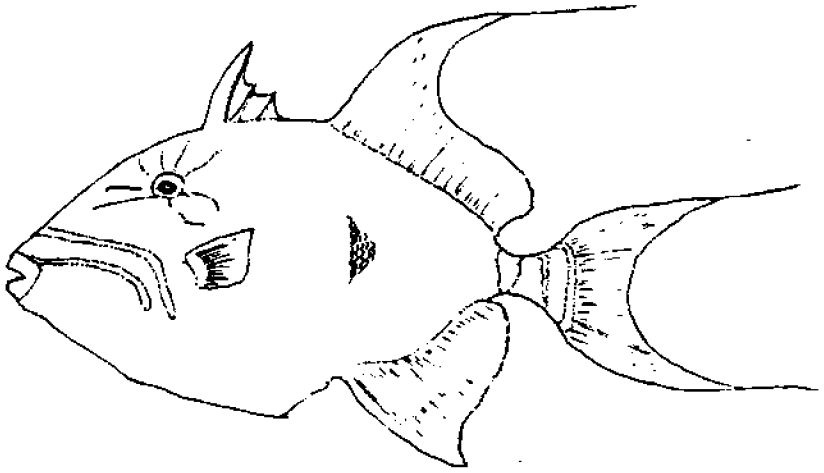
(23) Queen triggerfish or Old Wife (Balistes vetula)- Body greenish or bluish gray on back, orange-yellow on lower part of head and belly. The color pattern and the long filamentous fin lobes distinguish the Old Wife from the other species of trigerfishes in the Virgin Islands. The two diagonal bright blue stripes running from the snout to below the pectoral fins are the most characteristic color marks and are always present. Dark blue lines with yellow edges radiate from the eye, and there is usually a broad blue bar across the base of the tail. Attains at least 22 inches (not including the long tail filaments.)



21. Misty Grouper (Epinephelus mystacinus)



22. Conger, Congo, or Moray (Gymnothorax funebris)



23. Queen Triggerfish or Old Wife (Balistes vetula)

APPENDIX II

OTHER TYPES OF FISH POISONING

Brief descriptions of other types of fish poisonings are given in this section as an aid in distinguishing them from ciguatera.

Elasmobranch Poisoning - caused by the ingestion of the liver or flesh of some species of sharks and rays. Gastrointestinal symptoms predominate in mild cases, but ingestion of toxic livers may have severe effect, with onset of symptoms in less than 30 minutes. "Nausea, vomiting, diarrhea, abdominal pain, headache, weak pulse, malaise, cold sweats, oral paraesthesia, and a burning sensation of the tongue, throat, and esophagus may be present. Neurological symptoms develop later and include extreme weakness, trismus, muscular cramps, a sensation of heaviness of the limbs, loss of superficial reflexes, ataxia, delirium, incontinence, respiratory distress, visual disturbances, convulsions and death" (Bagnis, et al, 1970). The absence of the paradoxical sensory disturbance supports the belief that elasmobranch toxin is different from ciguatoxin. The nature of the poison is not known, but it is not destroyed by heat or gastric juices. Treatment is symptomatic only. The mortality rate is thought to be relatively high.

Puffer or Tetraodontoid Poisoning - caused by eating the liver, gonads, intestines or skin of puffer-like fishes, including the ocean sunfishes, sharp-nosed puffers, porcupine fishes, and puffers proper. The flesh of these fishes is usually safe to eat, but the viscera and skin contains a potent neurotoxin capable of producing rapid death. "The biotoxication is characterized by paraesthesias of the lips and tongue which gradually spreads to include the extremeties and later develops into severe numbness. The numbness may later involve the entire body. Gastrointestinal disturbances may or may not be present. Respiratory distress is a prominent part of the clinical picture, and the victim later becomes intensely cyanotic. Petechial haemorrhages, blistering, and severe desquamation may develop. Ataxia, aphonia, dysphagia, muscular twitchings, tremors, incoordination, paralysis, and convulsions are frequently present. The victim may become comatose, but in most instances remains conscious until shortly before death. Treatment is symptomatic. The case fatality rate is 61%. If death occurs, it usually

takes place within the first 24 hours" (Bagnis, et al, 1970). This type of fish poisoning is not a common public health problem in the West Indies, as puffers are not held in high esteem as food fish.

Clupeoid fish poisoning - caused by herrings, anchovies, tarpons, and bonefishes, which are most likely to be toxic during the summer. The viscera are the most toxic parts, but the nature of the toxin is not known. Clupeotoxism has only recently been recognized as a distinct clinical entity from ciguatera, based upon the symptomology. "The clinical characteristics of clupeotoxism are distinct and usually violent. The first indication of poisoning is a sharp metallic taste which may be present immediately after ingestion of the fish. This is rapidly followed by a severe gastrointestinal upset which may be accompanied by a drop in blood pressure, cyanosis, and other evidence of a vascular collapse. Concurrently, or within a short period, a variety of neurological disturbances develop - nervousness, dilated pupils, violent headaches, numbness, tingling, hyperventilation, muscular cramps, respiratory distress, paralysis, convulsions, coma, and death. Death may occur in less than 15 minutes." (Bagnis, et al, 1970). The mortality rate is very high. Treatment is symptomatic.

Scombroid fish poisoning - caused by eating improperly preserved tunas, mackerels, bonitos, skipjacks, cero, or kingfish. Scombroid poisoning is probably as prevalent in the Virgin Islands area as ciguatera poisoning. Bacterial decomposition produces saurine, a histamine-like substance which is not destroyed by cooking. "The symptoms of scombroid poisoning resemble those of histamine intoxication. Symptoms usually develop within a few minutes after ingestion of the toxic fish and are intense headache, dizziness, throbbing of the cartoid and temporal vessels, epigastric pain, burning of the throat, cardiac palpitation, rapid weak pulse, dryness of the mouth, thirst, inability to swallow, gastrointestinal upset, diarrhea, abdominal pain, generalized erythema, urticarial eruptions, severe pruritis, swelling and flushing of the face, bronchospasm, suffocation and severe respiratory distress. There is danger of shock, and deaths have been reported. In rare instances, scombroid fishes have been involved in both scombroid and ciguatera

poisoning in the same individual. The victim usually recovers within a period of one or several days. Treatment requires the use of antihistamine drugs" (Bagnis, et al, 1970).

Hallucinatory fish poisoning - caused by eating the head or brains of certain reef fishes This type of fish poisoning is more common in the tropical Pacific and Indian oceans than in the tropical Atlantic. In the West Indies certain species of fishes are suspected of causing this type of intoxication, but very little information is available. The toxin is not destroyed by ordinary cooking procedures, and its nature is unknown. "This biotoxication is unpredictable and sporadic in its occurence. The poison affects primarily the central nervous system. The symptoms may develop within a few minutes to 2 hours and persist for 24 hours or longer. Symptoms are dizziness, loss of equilibrium, lack of motor coordination, hallucinations and mental depressions. A common complaint of the victim is that 'someone is sitting on my chest,' or there is a sensation of tight constriction around the chest. The conviction that he is going to die, or some other frightening phantasy, is a characteristic part of the clinical picture. Other complaints consist of itching, burning of the throat, muscular weakness and abdominal distress. No fatalities have been reported and in comparison with other forms of (fish poisoning) hallucinogenic fish poisoning is relatively mild" (Bagnis, et al, 1970).

The public and medical authorities are encouraged to report any instances of these non-ciguatoxic fish poisonings to the staff of the Ciguatera Studies Program at the Benner Bay Laboratory, St. Thomas.

Lobster poisoning - although no statistics are available, it is known that in the Virgin Islands a number of cases of severe intoxication have been attributed to ingestion of "spoiled" or "poisonous " spiny lobsters. Whether this is a result of bacterial food spoilage due to improper preservation, or whether it is due to a toxic substance present in the flesh of some lobsters is not known. However, it is probable that some of the local spiny lobsters may contain a toxic substance, since recent research has shown this to be true in the Pacific. Cases of severe crab or lobster poisoning, some of them lethal, have been reported from several tropical

Pacific Islands. Any local incidents of lobster poisoning should also be reported to the staff of the Ciguatera Studies Project. Data about the lobster responsible (size, weight, sex, where caught, etc.), as well as a complete record of the poisoning incident (date, kind of symptoms experienced and their sequence, time of onset, etc.) would be extremely valuable.

CARIBBEAN RESEARCH INSTITUTE

CIGUATERA STUDIES PROGRAM

CIGUATERA CASE REPOSITORY

CIGUATERA OUTBREAK REPORT FORM P-2# _____
(Physician's contact)

1. PATIENT'S NAME _____
Last First Middle
AGE _____ SEX _____ WEIGHT _____

2. ADMISSION DATE (or clinic visit) _____
SYMPTOMOLOGY
ONSET (check and indicate order with numerals)
HOURS AFTER INGESTION _____
NAUSEA _____ ABDOMINAL CRAMPS _____
VOMITING _____ METALLIC TASTE IN MOUTH _____
DIARRHEA _____ P. S. D. _____
NUMBNESS AND TINGLING _____ WHERE _____

3. LABORATORY STUDIES (if made):
URINE ANALYSIS _____
HEMATOCRIT _____
HEMOGLOBIN _____
WHITE COUNT _____
DIFFERENTIAL _____
ELECTROLYTES _____
OTHER STUDIES: _____
_____
_____

4. TREATMENT:
DRUGS PRESCRIBED: _____
_____
DIET: _____
_____

5. FINAL DIAGNOSIS: _____

6. a) MAY WE (CIGUATERA STUDIES PROJECT) CONTACT THE PATIENT? _____
b) ANY OTHER COMMENTS? _____
_____
_____
_____

_____ _____ _____
DATE PHYSICIAN TELEPHONE #

ST. THOMAS • VIRGIN ISLANDS 00801 • CABLES: COVI • TEL. 809-774-1252

Dear Doctor,

We have prepared the enclosed information summary to provide an up-to-date and accurate summary of the present literature of ciguatera poisoning as we know it from the Virgin Islands area. We hope that the information is of use to you in the diagnosis and treatment of fish poisoning incidents.

We have also included several copies of an epidemiology form which we use in conjunction with our research program. It would be a great help if you could find the time to fill out the questionaires and return them to us whenever you treat a fish poisoning case. We will be glad to pick up the completed form at your office if you prefer - our telephone number is (809) 775-2410.

We hope to prepare an expanded version of this information summary to cover the entire Caribbean area. To this end, we would welcome your comments and suggestions. If you have any questions about our research program or our plans, we would welcome the opportunity to talk with you.

Sincerely yours,

ROBERT W. BRODY
Project Director
Ciguatera Studies Program

APPENDIX II

INFORMATION ABOUT FISH POISONING

A. R. Teytaud and R. W. Brody

October, 1971

APPENDIX II
Year End Report
A Study of Ciguatera Fish Poisoning
in the Virgin Islands Area
Robert W. Brody
May, 1973

INFORMATION ABOUT FISH POISONING

A. R. Teytaud and R. W. Brody

Illustrations by Lyn Slocum

October, 1971

St. Thomas, (U.S.)V.I.

INFORMATION ABOUT FISH POISONING

Every year, many people in the Virgin Islands suffer from a type of fish poisoning called Ciguatera. This pamphlet gives a short description of Ciguatera and provides an appendix with clear drawings of fishes which are likely to be poisonous in this area.

What is Ciguatera?

Ciguatera is a common illness which results from eating certain fishes in the Virgin Islands and other tropical areas, even fishes which are perfectly fresh and which come from clean water. Ciguatera is caused by a poisonous substance in the flesh and internal organs of these fishes. Scientists do not know exactly what this substance is or how it finds its way into the food chain of the fish, but there is not much evidence to back up the belief of many people that copper is in some way involved in fish poisoning.

How Ciguatera Affects you.

The most common symptoms of ciguatera are sudden abdominal pain, nausea, vomiting, and a watery diarrhea, which begins within a few minutes to 6 hours after the fish is eaten. There is generally a tingling sensation and numbness in the mouth area which later spreads to the arms and legs. The victim may complain of weakness and pains in joints and muscles which may continue for days or weeks after the other symptoms have passed away. Often there is a reversal of temperature sensations, in which cold objects seem hot and hot things seem cold.

In most cases, the symptoms of ciguatera pass away in several days to two weeks. However, symptoms sometimes last much longer, and may be serious enough to cause death. Because people vary widely in their reaction to

ciguatera, it is always wise to consult a doctor in any case of fish poisoning.

Which Fishes are Poisonous?

Certain kinds of fishes are more likely to cause ciguatera than others, but at present there is no way to tell whether a particular fish is poisonous except by eating it or feeding a part to a dog or cat (if the animal gets sick the fish is poisonous). Many people believe that you can tell a poisonous fish by cooking it with a silver spoon or looking at the color of the flesh, but all such methods are old wives' tales and are absolutely not true.

It is well known that certain areas in the sea produce many more poisonous fishes than other areas. Some of these poisonous areas are:

(1) St. Croix - Eastern tip, Lang Bank
(2) St. Thomas - entire South side, Smith Bay, Luongo Cay, Sail Rock
(3) St. John - South side, East End, Congo Cay
(4) Tortola and adjacent islands - islands and cays from Norman Island to Virgin Gorda, Necker Island Pass, Southeast side of Tortola, Anegada Reef near Roccos.

However, fishes which are poisonous in one area may be perfectly safe to eat in other areas.

The following is a list of fishes which have frequently caused ciguatera in the Virgin Islands; the fishes towards the top of the list are more likely to be poisonous than those lower down. Drawings and descriptions of these fishes are included in the appendix to help you in identifying them.

Table 1

Common Name	Scientific Name
Barracuda (Barra)	Sphyraena barracuda
Amberjack	Seriola dumerili

Table 1 (Cont'd)

Common Name	Scientific Name
Horse-eye Jack	Caranx latus
Bar Jack (Carang)	Caranx ruber
Crevalle Jack (Cavalli)	Caranx hippos
Dog Snapper (Dog Tooth)	Lutjanus jocu
Yellowfin Grouper (Gramminix)	Mycteroperca venenosa
Kingfish	Scomberomorus cavalla
Blue Runner (Hard Nose)	Caranx fusus
Conger, Congo, or Moray	(probably several species of Gymnothorax or other morays)
Rock Hind	Epinephelus adscensionis
Black Grouper	Mycteroperca bonaci
Cero (often erroneously referred to as "Spanish Mackerel")	Scomberomorus regalis
Black Jack	Caranx lugubris
Hogfish	Lachnolaimus maximus
Gray Snapper	Lutjanus griseus
Almaco Jack	Seriola rivoliana
Yellow Jack	Caranx bartholomaei
Red Hind	Epinephelus guttatus
Black Snapper	Apsilus dentatus
Blackfin Snapper	Lutjanus buccanella
Queen Triggerfish (Old Wife)	Balistes vetula
Misty Grouper (often assumed to be the "Warsaw Grouper" by V. I. fishermen who have seen that species in Florida).	Epinephelus mystacinus

Hints to Avoid being Poisoned

1. Avoid eating the fishes in table 1 unless you know that they were caught in a safe area; even then, you should not eat the larger individuals.

2. Never eat the liver or other internal organs of the fishes in table 1, as these parts contain more of the poisonous substance than the flesh does (it would be better not to eat the internal organs of any local fish).

3. Neither cooking, drying, salting, or any other common method of preparation will destroy ciguatera poison. If you have reason to suspect that a fish may be poisonous, do not eat it.

4. Since the poison dissolves slightly in water, soup is a very dangerous way of preparing a fish which may be poisonous.

What to do if you are Poisoned

1. The stomach should be emptied as soon as possible. Except in very mild cases, a physician's help should be sought.

2. Save as much as possible of the fish which caused the poisoning. Make a note of what kind of fish it was, and where it was caught (if known).

3. Please call the Ciguatera Studies Project at 775-2410, and tell us about the incident.

The Ciguatera Studies Project

This information summary has been prepared by the staff of the Ciguatera Studies Project of the Caribbean Research Institute, College of the Virgin Islands, St. Thomas, U. S. V. I.

The Project is collecting data on fish poisoning in the Virgin Islands and we encourage the cooperation of the public in immediately reporting all incidents of fish poisoning to us. We will send an interviewer to contact the victim and obtain a sample of the poisonous fish from him. Therefore, it is important that the reporting individual make every effort to preserve at least a part of the fish responsible for the poisoning. This sample will be used in an attempt to develop a reliable test for ciguatera.

NOTE:

"Illustrations and Descriptions of Commonly Ciguatoxic Fishes" [See Year End Report Appendix I] was included here.

APPENDIX III

FISH POISONING IN THE EASTERN CARIBBEAN

Robert W. Brody

November, 1971

Paper presented at the
Gulf and Caribbean Fisheries Institute
Twenty-fourth Annual Session, November, 1971

APPENDIX III
Year End Report
A Study of Ciguatera Fish Poisoning
in the Virgin Islands Area
Robert W. Brody
May, 1973

FISH POISONING IN THE EASTERN CARIBBEAN

Paper presented at the
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Twenty-fourth Annual Session, November, 1971

Robert W. Brody

CARIBBEAN RESEARCH INSTITUTE
COLLEGE OF THE VIRGIN ISLANDS
St. Thomas, U.S. Virgin Islands 00801

CONTENTS

I. INTRODUCTION

"When I first got here I thought that I would be able to practically exist on fish....."; "I was poisoned fifteen times before I left my father's house. He always liked to buy big fish because there was more meat....."; "I can't get a contract for grouper or snapper with any of the hotels. They buy the same fish from Santo Domingo for ten cents a pound more....."; "Where are all the seafood restaurants? I thought this was an island!"

These comments and others like them represent a biotoxicological problem which has always plagued the eastern Caribbean. Ciguatera fish poisoning, scombroid poisoning, and to a lesser extent "clupeoid", tetraodontoid and elasmobranch poisoning have been reported since pre-Columbian time and the problem shows no sign of lessening.

This paper will present the basic facts of fish poisoning in the eastern Caribbean area as we presently know them. It will not attempt to review the voluminous literature from the Pacific; the reader is referred to excellent summaries of

Halstead (1967) and Banner (1971). At the present time we have very little "hard data" on the chemistry, biogenesis and biology and pharmacology of the fish poisoning problem in the area. We have yet to confirm that the most important type of poisoning, ciguatera, is in fact identical to the toxin from the Pacific. The sections on ciguatera are therefore based on our local observations and inferences from studies in the Pacific are noted.

II. TYPES OF FISH POISONING

Fish poisoning in the eastern Caribbean can be broken down into three major groupings. The endotoxins from the puffer-like fishes with the additional rarely reported cases of clupeoid, elasmobranch and hallucinogenic fish poisoning form the first group. These incidents represent a very small percentage of the total number of cases reported; several years of casual data-gathering and six months of active research have only produced two accounts in the last four years, both of clupeiotoxism.

The biology, chemistry, and pharmacology of puffer fish poisoning have been accurately summarized elsewhere (Bagnis, 1970). Although the world-wide fatality rate is high (61%), very few cases of tetraodon poisoning are reported in the Caribbean, probably because the puffers are not highly esteemed as a food fish. This may also be the reason for the low incidence of reports of elasmobranch poisoning in the area. Shark has been harvested recently in an effort to produce a packaged seafood product ("Sea Flake") with generally good customer acceptance; there have been no instances of elasmobranch poisoning brought to our attention as a result. Hallu-

cinatory fish poisoning (icthyoallyeinotoxism) is also an apparently rare event in the eastern Caribbean; it has been described to us by an herbalist on St. Thomas but an actual case history has not been received.

Cleupeoid poisoning is a form of icthyosarcotoxism caused by ingestion of the flesh of fishes of the order Clupeiformes. The symptoms are frequently violent with an extremely high case fatality rate. The onset of symptoms is noted with a sharp, metallic taste in the mouth followed rapidly by severe gastrointestinal upset with distinct indications of vascular collapse (drop in blood pressure, cyanosis). This may be accompanied or rapidly followed by neurological disturbances nervousness, dilated pupils, violent headache, tingling, and in severe cases respiratory distress, convulsions, coma, and death. The toxin is apparently particularly virulent: death may occur within fifteen minutes. The literature reports that persons have died while in the act of eating the fish - "part of the fish was still in the victim's mouth at the time of death" (Halstead, 1967).

Fish in the families Clupeidae (herrings), Engraulidae (anchovies), Albulidae (bonefishes), and Elopidae (tarpons)

have been reported as toxic in the Caribbean; our experience has been limited to two cases involving the clupeoid fishes Herengula humeralis (yellow-billed sprat) and Opisthonema oglinum (thread herring). One case is reported from a fisherman on Tortola, B. V. I. who (with his family of five) ate "yellow bill sprat" (probably Opisthonema oglinum from his description of a "spine on the back fin") caught in Great Harbor, Peter Island, B. V. I. He reported "bad fish poisoning" symptoms generally similar to ciguatera poisoning; the rapid onset reported above was present. He forced all of the members of his family to vomit and all took a "heavy dose of sulphur" (a patent medicine). He did not contact public health authorities. The family recovered from acute symptoms within 36 hours. (This incident took place in late 1967 and was reported in April 1971; the interview was thus clouded by time and at least two subsequent incidents of ciguatera poisoning.) The second case is reported by Halstead (1970) from Antigua, W. I. from 1968 (?) when "some small surface-feeding 'herring-like' fish were eaten". Two people died in this outbreak. Halstead suggests that local terminology of "yellow-billed sprat" is applicable to Herengula humeralis. The violence of the episode suggests that this case was an example of classic clupeiotoxism while the Tortola case is not definitely separable from ciguatoxism.

The chemical nature and biogenesis of clupeiotoxism is not presently known. Numerous authors have suggested that fish caught during the summer months are more likely to be toxic. All reported incidents are from fish caught close to tropical islands. There are several references in the literature to planktonic blooms as the causative organism, specifically a "monad" (dinoflagellate ?) (Halstead, 1967), Skujaella, (Trichodesmium) (Randall, 1958) or to "swarming of palolo worms" (Halstead, 1967). It is probable that the toxin is produced by some planktonic form as the clupeoid fishes are for the most part plankton feeders, this may add strength to the position that clupeiotoxism is a separate entity from ciguatera poisoning. Clupeiotoxin is not thought to be degraded by normal cooking and the degree of freshness does not seem to have effect on the toxicity.

Clupeiotoxism may pose a wider threat to public health than the occasional locally consumed fish. If the toxin is indeed caused by "blooming" plankton organism the likelihood of a large school of toxic fish cannot be overlooked. The sardine and anchovy groups are frequently thought of as ideal fish for Fish Protein Concentrate production; we have no data on the ability of current FPC technology to eliminate the toxin from

the raw fish.

The second major group of poisonings experienced in the eastern Caribbean is the result of bacterial decomposition of fresh fish. In the Virgin Islands and throughout the northern Leeward Islands fish are typically sold "fresh" from the boat. Very few fishermen use ice or gut the fish before sale, in fact there is a strong feeling among older citizens that such preservation is used to camouflage the true "freshness". Eastern Caribbean fishermen do not use live-wells and fish frequently spend a good part of the day in the sun. These conditions obviously tend to promote bacterial decomposition of the fish and the consequent toxicity problem; it is interesting that the younger people in St. Thomas, U. S. V. I. who tend to buy fish in the supermarket report a significantly lower incidence of fish poisoning attributable to scombroids.

We presume that an undetermined proportion of the poisonings reported are the result of some sort of bacterial decomposition. In many "mild" cases the only complaint is of gastrointestinal distress; the neurological symptoms specific to ciguatera and ciguatera-like toxins are not noted. Many of these cases may be attributable to scombroid poisoning but the symptoms may not be sufficiently pronounced for a proper

diagnosis to be made.

The distinct histamine-like toxicity caused by bacterial degradation of the flesh of fish of the family Scombridae is a relatively common type of fish poisoning throughout the Caribbean. The symptomology includes a distinct "sharp" or "peppery" taste upon eating the fish followed by intense headache, dizziness, a variety of circulatory disfunctions, gastrointestinal distress, dryness of the mouth, and inability to swallow. These symptoms are followed by generalized erythema, the face becomes swollen and flushed, eyes are sunken, and an urticarial eruption may develop covering the entire body. In severe cases there may be additional complications of shock and respiratory distress. Death has been reported in a few cases but acute symptoms generally dissipate in eight to twelve hours. This toxic reaction is brought about by the bacterial degradation of histidine in scombroid muscle tissue which produces a substance designated as scombrotoxin. Scombrotoxin probably has a combination of chemical constituents including saurine, histidine, and possibly other toxic compounds. The disease responds well to treatment with antihistaminic drugs; this specific treatment has mitigated the severity of scombroid poisoning as a public health problem in recent years.

In the eastern Caribbean scombroid poisoning has been reported from Acanthocybium solandri (wahoo) Scomberomorus cavalla (kingfish or king mackerel). Scomberomorus regalis (spanish mackerel), and S. maculatus (cero). We are not aware of cases reported recently from the eastern Caribbean in the tunas (Auxis, Euthynnus, Sarda, Scomber, Thunnus) but these genera may also be incriminated. There probably is no true seasonality to scombroid poisoning although the incidence in any one area can be correlated with local "runs" of the particular species involved. Thus there seem to be more poisonings during the tourist season when sport fishing pressure is high.

The third general type of fish poisoning is described as ciguatera fish poisoning. Evidence from the Pacific suggests that there are at least three (probably more) distinct toxins capable of producing the ciguatera syndrome. Many authors (and many physicians in the eastern Caribbean area) have not separated the diagnosis or treatment of ciguatera from that of scombroid poisoning and some confusion has resulted. Both ciguatera and scombroid poisoning have been occasionally reported from the same fish in the Pacific (Halstead, 1967); we have no such report from the Caribbean in recent years.

III. SYMPTOMOLOGY AND PUBLIC HEALTH ASPECTS OF CIGUATERA

In the absence of precise chemical and biogenic data we have defined ciguatera in terms of its symptomology. The following symptoms are extracted from Halstead (1967), Bagnis (1970), and Banner (1971) and are quoted as they appear in Teytaud and Brody (1971):

> "Ciguatera fish poisoning in its simplest uncomplicated form develops within 3 to 5 hours after the fish is eaten. There is a sudden onset of abdominal pain followed by nausea, vomiting, and a watery diarrhea. The gastrointestinal symptoms will occur in about 40 to 75 percent of the cases. The victim feels weak, generally ill, and may experience muscle aches throughout the back and thighs in about 10 percent or more of the cases. Soon after, the victim complains of numbness and tingling in and about the mouth which then extends to the extremities (present in about 50 percent or more of the cases). Fever, headache, and rash are generally absent, and the patient has no desire for food. The acute symptoms usually subside in about 8 to 10 hours, and within 24 hours after onset most of the patient's symptoms will have completely subsided except for a feeling of weakness. However, the numbness and tingling may continue to a lesser extent for a period of 4 to 7 days. The foregoing resume is typical of the majority of uncomplicated ciguatoxications that are generally encountered by the practicing physician in an endemic ciguatoxic locality.
>
> Ciguatera, like many other diseases, may vary greatly in its clinical manifestations depending upon the toxicity of the fish that is eaten, the individual's sensitivity to the poison, amount of fish ingested, and other factors. In a broader sense ciguatera fish poisoning may be characterized as follows: the onset

of symptoms may vary from almost immediately to within a period of 30 hours after ingestion of the fish, but is usually within a period of 6 hours. The initial symptoms in some cases are gastrointestinal in nature, consisting of nausea, vomiting, watery diarrhea, metallic taste, abdominal cramps, and tenesmus, whereas in other patients the initial symptoms consist of tingling and numbness about the lips, tongue, and throat. This may be accompanied by a sensation of dryness of the mouth. The muscles of the mouth, cheeks, and jaws may become drawn and spastic with an accompanying sensation of numbness throughout. Generalized symptoms of headache, anxiety, malaise, prostration, dizziness, pallor, cyanosis, insomnia, chilly sensations, fever, profuse sweating, rapid weak pulse, weight loss, myalgia, and back and joint aches may be present in varying degrees, or one or more of the symptoms may be entirely absent. The victims usually complain of a feeling of profound exhaustion and weakness. The feeling of weakness may become progressively worse until the patient is unable to walk. Muscle pain are generally described as a dull, heavy ache, or cramping sensation, but on occasion may be sharp, shooting, and affect particularly the arms and legs. Victims complain of their teeth feeling loose and painful in their sockets. Visual disturbances consisting of blurring, temporary blindness, photophobia, and scotoma are common. Pupils are usually dilated and the reflexes diminished. Skin disorders are frequently reported that are usually initiated by an intense generalized pruritus, accompanied by erythema, and maculopapular eruptions, blisters, extensive areas of desquamation - particularly of the hands and feet - and occasionally ulceration. There may also be a loss of hair and nails.

In severe intoxications the neurotoxic components are especially pronounced. Paresthesias involve the extremities, and paradoxical sensory disturbances may be present in which the victim interprets cold as a "tingling, burning, dry-ice or electric-shock sensation", or hot objects may give a feeling of cold. In regard to the paradoxical sensory disturbance (P. S. D.), a classic example is that of a naval officer who was poisoned by an amberjack. Four weeks later he

was observed subconsciously blowing on his ice cream, which was "burning his tongue", in order to cool it. Ataxis and generalized motor incoordination may become progressively worse. The reflexes may be diminished, muscular paralyses may develop, accompanied by clonic and tonic convulsions, muscular twitchings, tremors, dysphonia, dysphagia, coma, and death by respiratory paralysis. The limited morbidity statistics show a case fatality rate of about 12 percent. Death may occur within 10 minutes, but generally require several days."

Table 1 summarizes the symptoms occuring during the first 24 hours after ingestion as they were reported by 25 persons who were interviewed following ciguatoxications of minor to moderate severity in St. Thomas U.S. V.I. during 1971. Several of these reports represent the symptoms produced in different individuals by a single fish; they therefore do not represent 25 separate outbreaks.

TABLE I. Summary of Symptoms

Symptom	Percent Reporting
abdominal pain	96 (96)
nausea	88 (92)
vomiting	68 (68)
diarrhea	96 (96)
numbness, tingling about mouth	56 (64)
headache	48 (48)
numbness in extremeties	48 (56)
metallic taste	24 (36)
weakness	96 (96)
muscle aches	40 (48)
paradoxical sensory disturbance	32 (32)
itching	64 (68)

Summary of symptoms manifested by 25 ciguatoxicated individuals during first 24 hours after ingestion of fish. (Percentages in parentheses represent change in original descriptions following questions by the interviewer.)

A-III

Several additional symptoms were reported by three or fewer (less than 12%) of the persons interviewed. These symptoms include lack of coordination, muscle spasm, high fever, visual disturbances, diminished reflexes, and skin rash. It is notable that none of these persons required hospitalization and only three reported visiting a physician (several others contacted a physician by telephone during the time period 24 - 72 hours after ingestion).

Virtually all of the persons interviewed reported noticeable symptoms of ciguatoxication for several days after the onset of the incident. Most commonly reported was extreme weakness and lethargy lasting up to two weeks. Many victims reported gastrointestinal symptoms well into the third day along with itching and/or skin rash. Those persons reporting the paradoxical sensory disturbance stated that it persisted for at least three days, in some cases ten days or two weeks. The bulk of the other symptoms noted were reported as having dissipated within the first 24 hours.

We are currently undertaking a more extensive epidemiology reporting program in cooperation with local media, physicians, and public health authorities. Data from this survey combining questionnaire and interview procedures should

be available early in 1972. At this time we have no accurate estimate of the magnitude of the ciguatera poisoning problem in the Virgin Islands or for that matter anywhere in the eastern Caribbean. Outbreaks in Puerto Rico are sufficiently notable to receive coverage in the major English-language media which suggests that they are infrequent. The officials responsible for public health record-keeping in St. Thomas, on the other hand, estimate three or four cases per week are seen in the emergency room; if our 25 cases reported in table 1 represent typical reporting ratios there may be as many as 30 cases per week in St. Thomas. These figures probably represent the maxima however and cannot be confirmed. Reports of ciguatoxication in the British Virgin Islands have stated that virtually "everybody" has been poisoned at least once (some as many as fifteen times) but medical advice is almost never sought. Interestingly the British Virgin Islands are the only demographic unit mentioned by Halstead (1970) where fish poisoning is "not regarded as deterrent to the development of the fisheries programme".

The pattern of sporadic reporting of ciguatera poisoning despite the relatively high incidence of the disease is common throughout the northern Leeward Islands. Information

from Halstead (1970) and our own contacts with fishermen, inter-island traders, charterboat operators and fishery personnel in the region suggests that the problem is indeed severe. Virtually every person contacted from St. Kitts, St. Maarten, St. Eustatius, Anguilla, and Montserrat had either been a victim himself or knew of a close friend or relative who had been poisoned within the last five years. Very few of these cases were brought to the attention of a physician; most public health officials believe that "only the very severe cases are brought to the attention of the medical authorities" (Antigua); Halstead,(1970). One long-time resident of St. Kitts estimates only about one case in ten receives medical attention; as might be expected the bulk of the cases reported involve tourists and non-native residents.

IV. GEOGRAPHIC DISTRIBUTION

The geographic distribution of ciguatoxic fish in the Virgin Islands is shown in Figure 1. The island of St. Croix, on a separate geologic platform, is not reported as producing ciguatoxic fish in any appreciable quantity and has been omitted from this figure. A large number of the areas indicated have been reported as producing toxic fish for centuries. It cannot be presumed that these are the only localities; toxic fish are frequently caught in other areas.

In the Virgin Islands there is an extremely strong feeling among the fishermen that the south side of the Virgin Bank from Sail Rock east to Peter Island consistently produces toxic fish. Other fishermen would extend this area east and north to include most of the coastline of Virgin Gorda, some would include the Horseshoe Reef and Anegada. Still others (particularly those who regularly fish this southern Bank) state that only specific locations in this area produce toxic fish and that reef areas or "banks" only a few miles away are free of ciguatera. Virtually all fishermen feel that the entire north side (the Atlantic side) of the Bank is free of toxicity with the exception of a very few species. This pattern of geographic

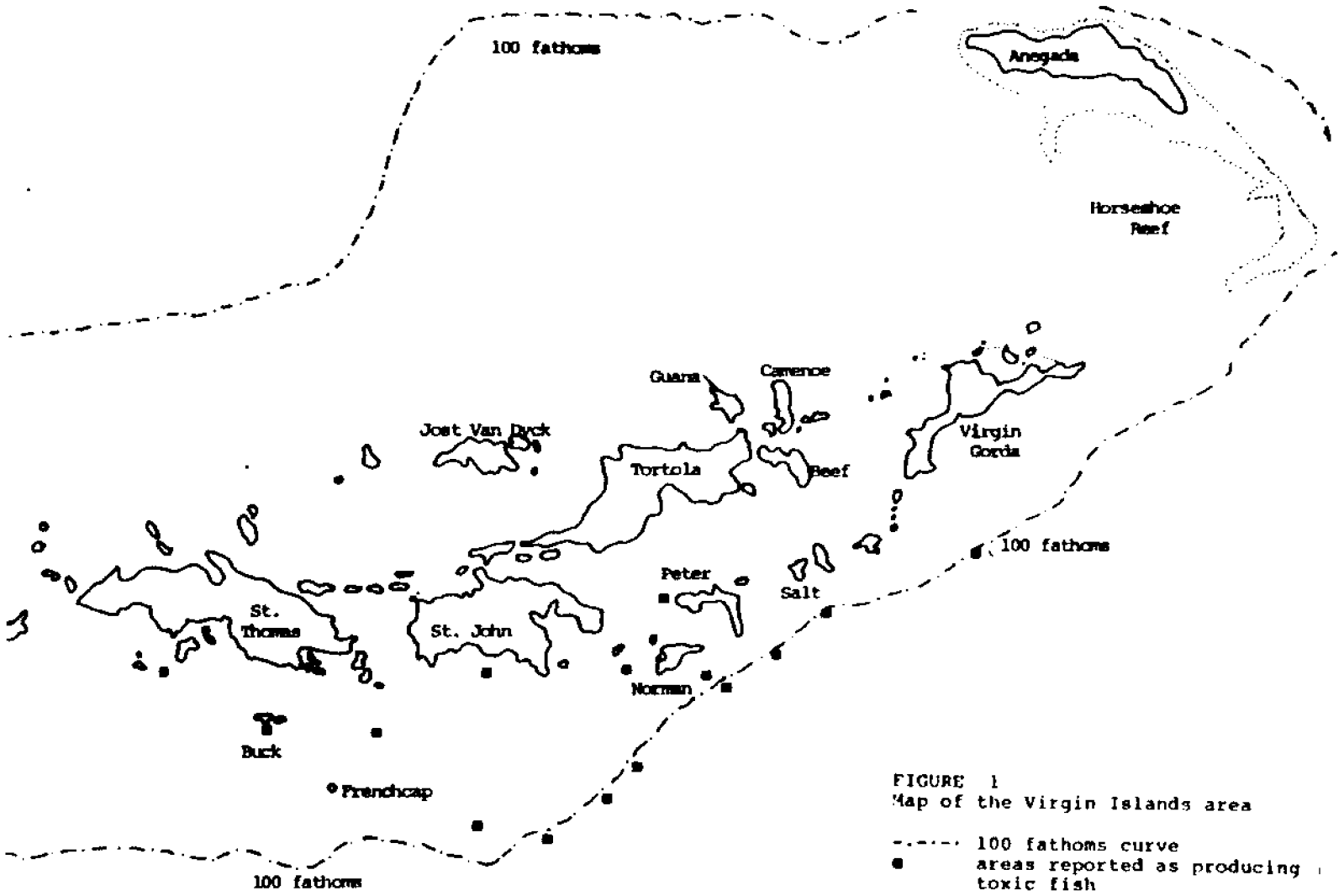


FIGURE 1
Map of the Virgin Islands area

------ 100 fathoms curve
● areas reported as producing toxic fish

distribution of ciguatoxic fishes seems consistent with those areas reported by earlier authors (Walker, 1922; Arcisz, 1950; Brown, 1945; de Sylva, 1956, Mann, 1938). Other writers quoted in Halstead (1967) notably Hill (1868) Rogers (1899) and Gilman (1942), are contradicted by local fishermen, at least for the bulk of the species implicated elsewhere in the Virgin Islands.

In almost all reports on the geographic locality where toxic fish are caught the interviewee was referring to depths of 30 fathoms or less; the bulk of reports refer to reef areas in eight - fifteen fathoms, but this may be an artifact of fishing methods rather than a biological distribution. The relationship of depth to capture of ciguatoxic fishes is discussed in a later section.

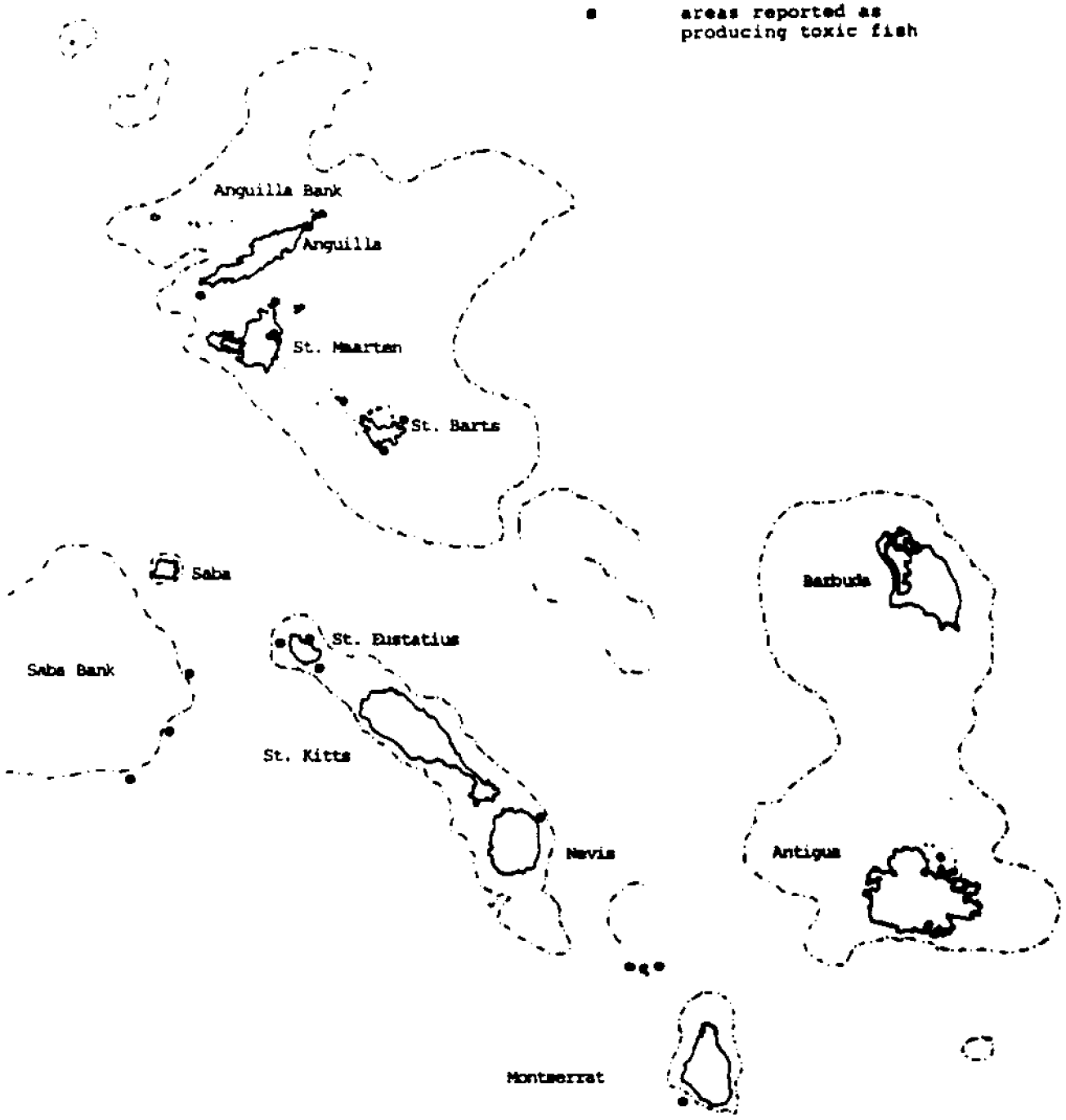
The areas reported (by our contacts and by Halstead, 1970) as producing toxic fish in the northern Leeward Islands are shown in Figure 2. These data are plotted along with areas mentioned as toxic by various authors in Halstead (1967) and do not represent an intensive survey. More specific data will be presented in a later paper. The more southerly group of islands in the eastern Caribbean (Martinique south to Trinidad: the Windward Islands) have not commonly been reported as producing ciguatoxic fish in this century. Earlier authors make

reference to a variety of species and locations but this is not confirmed by present residents.

Although no quantitative data are available it seems clear that the majority of ciguatera poisoning outbreaks in the eastern Caribbean occur in a rather small area from Montserrat north to the Virgin Islands including all of the northern Leeward Islands and portions of Saba and Anguilla Banks. Beyond this area ciguatera poisoning is limited to sporadic outbreaks which generally involve large specimens of only a few species.

FIGURE 2
Map of the Leeward Islands

-.-.-. 100 fathoms curve
• areas reported as producing toxic fish



V. SPECIES REPORTED AS CIGUATOXIC

More than 400 species of fish have been implicated in ciguatera poisoning on a global basis (Bagnis, 1970). Of these 400, 91 species could conceivably be found in the eastern Caribbean. It is possible that an even larger number could be associated with ciguatoxications if they were desirable as food fish. There are also a number of reports of molluscs, crabs, and lobsters producing the disease. Appendix I lists the 24 fish most frequently reported as toxic in the Virgin Islands. All of these species are valued as food fish with the exception of barracuda and amberjack; these two species have such a bad reputation as ciguateric that only the smallest specimens can be sold. It is somewhat surprising that the moray eel is as highly esteemed as it is, considering reports of toxicity from the Pacific. In St. Thomas eels are typically purchased by individuals from Spanish Caribbean cultures (Puerto Rico, Dominican Republic, Cuba), areas where ciguatera is less frequently reported. The fish at the top of the list tend to be reported as toxic more frequently than those lower down.

In general the larger specimens of these species are frequently incriminated in ciguatera poisoning incidents. The fish generally can be considered "shore-fishes" or "reef-

fishes" and for the most part conform to the pattern noted by other authors (Randall. 1958; Halstead, 1967; Banner, 1971): toxic fishes are not common at depths greater than 80 - 100 fathoms. Two notable exceptions in the Virgin Islands are the blackfin snapper, Lutjanus buccanella and misty grouper, Epinephalus mystacinus. We have data on two outbreaks affecting five people from blackfin snapper caught in deep water and three additional outbreaks implicating misty grouper (which has not been taken at depths of less than 55 fathoms) involving at least ten persons during mid-1971. These data appear in Appendix II. Additional data on ciguatera from deep-living species is noted in a later section of this report. Although Banner (1971) states "true ciguateric fishes appear to be only those fishes tied directly to the flora and fauna of coral reef..." there is excellent clinical data to support these outbreak reports; several members of our staff were among the victims. Samples have been retained for extraction and bioassay to quantify the toxicity of these fish (see also section on fisheries development).

VI. CHEMISTRY AND PHARMACOLOGY

Our knowledge of ciguatera poisoning in the eastern Caribbean is presently based upon clinical reports and is only beginning to be quantified by chemical and biological assays. On the basis of symptomology and from the species implicated it is highly probable that ciguatera poisoning in the eastern Caribbean is produced by very similar (if not identical) compounds to those known from the Pacific. Scheuer and other workers at the University of Hawaii have isolated what they consider to be the primary toxin and, in cooperation with Hashimoto and his colleagues at the University of Tokyo; several secondary toxins. The primary toxin (deemed ciguatoxin) is insoluable in water, soluable in polar organic solvents, heat stable to 100°C, stable below 0°C as a crude toxin but unstable in the semipurified or purified form unless extracted, purified, and stored in an inert atmosphere at low temperatures. The non-crystalline product has the empirical formula $(C_{35}H_{65}NO_3)_n$ and the molecule has indications of a quaternary nitrogen atom, one or more hydroxyl groups and a carbonyl function. It is not a phospholipid. A crystalline product currently undergoing analysis to determine its structural formula (Banner 1971).

Present evidence (again from the University of Hawaii group) suggests that ciguatoxin acts upon excitable membranes to increase the permeability of Na + ions, upsetting the ionic balance of the membrane. Ciguatoxin is not an anticholinesterase in vivo, despite earlier papers and various therapies for ciguatoxications based on stimulation of cholinesterase cannot be endorsed at the present time. The toxin is active at the level of 0.025 mg/kg when injected into mice with a toxin yield of 5 - 10 mg/kg from highly toxic flesh. The toxin is carried at a uniform level throughout the musculature of toxic fish but may be 50 to 100 times as concentrated in the viscera, particularly the liver.

Our laboratory in St. Thomas is currently using an acetone - diethyl ether extraction with purification by column chromatography developed by Scheuer (per. comm.) and bioassay using intraperitoneal injection into 20± gram Charles River CD-1 mice. We have previously used other extraction techniques including crude aqueous extraction with emulsifiers and a variety of experimental bioassay techniques. Our conclusions are basically the same as the workers in Hawaii although based on much less experience: careful solvent extraction and purification are necessary, rigidly controlled bioassay procedures are required, and experienced laboratory

personnel are an absolute requirement. To date there has been no rapid, simple colorimetric or other chemical test for ciguatoxin developed. Screening programs are exceedingly expensive and difficult to manage and are only in operation in Japan on a limited basis for selected samples from highly suspect areas. As much as we might desire it, we are not very close to a rapid means of identifying ciguatoxic fish in the laboratory and even further from a simple test which might be part of a housewife's shopping kit.

The traditional West Indian methods of determining if a fish is ciguatoxic have been discussed at length by previous authors. Appendix III lists these methods as reported to us by natives of the Virgin and Leeward Islands. Many housewives swear by some particular method utilizing visual inspection of external characters of the fish. Most admit that in practice both the visual methods and those requiring addition of some indicator are unreliable. We have submitted each of these methods to an assay with at least two known toxic fish and two non-toxic fish and have not found them reliable.

VII. BIOGENESIS AND TRANSMISSION OF CIGUATOXIN

At the present time we have no accurate data on the mechanisms of biogenesis of ciguatoxin (or its related compounds) nor information on its transmission through the food chain. Banner, Helfrich, Randall, and others at the University of Hawaii have concentrated a good deal of effort on these problems in the Pacific and their findings to date are summarized below (from Banner, 1971):

1. No causative agent or organism has yet been identified as producing toxins similar to ciguatoxin.

2. No definite evidence has been found to suggest that: a) copper or other metallic ions act as chelators, trace minerals or catalysts in the formation of the toxin; b) no demonstrable increase in ciguateric fishes was noted in areas where "new surfaces" were exposed by natural disasters, dredging, blasting, predation by Acanthaster; c) contamination of the marine environment by pollutants (specifically wax esters at Wake Island) have no effect on ciguatera.

3. Normally non-toxic omnivores can be made toxic when fed small amounts of toxic fish over a period of time.

4. Toxic Lutjanus bohar retain toxicity for up to 30 months when fed a non-toxic diet.

5. A detrital feeding acanthurid (Ctenochaetus striatus) has demonstrable ciguatoxin in the flesh, viscera, and gut contents.

6. Most carnivores seem to carry the same toxin (ciguatoxin) although additional toxins (eg: Aluterin, ciguaterin) may also be present.

It should be noted that Dr. Banner will present a paper entitled "Biological Origin and Transmission of Ciguatoxin" tomorrow (18 November 1971), which could shed some new light on this subject.

Given the similar symptoms and species distribution reported in Pacific and eastern Caribbean ciguatera poisonings it is reasonable to assume that similar biogenesis and transmission of the toxin can be expected. There are several persistent beliefs among eastern Caribbean fishermen which will be repeated here although we have been unable to confirm them.

1. Ciguatera is produced by fish which eat the fruit or leaves of manchineel (Hippomane mancinella). This theory has been proposed since 1511 (by Peter Martyr of Anghera;

Halstead, 1967) and is probably based on advise to early explorers from Caribbean Indians. Hippomane is certainly toxic but its pharmacological action is quite different. It is doubtful that this theory is correct.

2. The most persistent theory in the Virgin and Leeward Islands involves copper. Natural copper deposits ("copper banks") are presumed to exist and fish which feed on these banks become toxic. Some of the more sophisticated fishermen suggest that it is not actually the copper metal but a small "sea moss" (which grown in areas where copper concentrations are high) which actually manufactures the toxin or a precursor. The "sea moss" responsible has been pointed out to us by several fishermen (actually) three species: Enteromorphia lingulata from shallow water at Buck Island, St. Thomas; Cladophora sp. from fish pot warps of Flanagan Island, U. S. V. I.; Chaetomorphia sp. from rocky subtidal at Buck Island, St. Thomas). None of these algae showed toxic activity when extracted with Tween and injected I. P. into mice; we plan to repeat this experiment with solvent - solvent extraction and column chromatography when these algae can be collected from historically toxic areas. Most fishermen suggest that the production of toxic "sea moss" is seasonal

with peak growth in late spring or early summer. The association of ciguatoxin with copper is not limited to copper banks by fishermen; it is proposed that shipwrecks (particularly older wrecks with copper-sheathed bottoms) and copper antifouling paints supply all of the copper needed. We can in no way confirm the theory of copper-induced ciguatoxin at this time.

3. A theory proposed by a few fishermen and completely unproven at this time attempts to explain the high concentrations of toxic fish on the south of the Virgin Bank (with the lower toxicity reported from the same species on the north side of the Bank) and in the area from Antigua north to the Anegada Passage. It is proposed that the toxin is produced by some organism (presumably a primary producer) which is found only in areas where deep, cold, nutrient-laden water is upwelling. The theory is reasonable when applied to the southern Virgin Bank and the southeastern portions of Saba Bank, both noted as producing toxic fish, as there is good evidence that upwelling does indeed occur in the Anegada Passage. The upwelling process cannot be confirmed in the St. Kitts - St. Eustatius - Redondo area at the present time due to lack of data. The specific organisms(s) responsible and the mechanism of toxin production are not known by the proponents of this theory.

We are therefore no closer to an accurate description of the biogenesis or transmission mechanisms of ciguatoxin than purely theoretical considerations. We have proposed a series of studies similar to those undertaken by the University of Hawaii group including chemical, ecological, and epidemiological programs for the next three years to attempt a better understanding of this problem.

VIII. EFFECTS OF CIGUATERA POISONING ON THE DEVELOPMENT OF FISHERIES

We have every reason to believe that ciguatera poisoning is a major impediment to the sale of local finfish in the Virgin Islands and thus is a strong deterrent to expansion of the commercial fisheries. Interviews by Halstead (1970) suggest that this is true throughout the Leeward Islands too, although residents of Antigua, St. Kitts, and St. Maarten express the opinion that there is little alternative to continued buying of local fish and risk of intoxication.

In Dammann's 1967-68 survey of commercial fisheries of the Virgin Islands slightly more than half of total finfish consumption was from local (U.S. and British V.I.) sources (1,672,400 of 3,084,373). We have no data on how much of the fish imports could be replaced by local production if ciguatera were not a problem. Interviews with fishermen suggest that very few hotels and restaurants catering to the tourist trade would purchase locally caught grouper, snapper, jacks, and kingfish because of fear of fish poisoning. Dammann's Table 9 "Fisherman-reported problems in the Virgin Islands commercial fishery" does not include any data on this subject, however Table 12 indicates that only two percent (two of 79) of the fishermen

contacted felt that there were "no fish" (commonly reported as ciguatoxic) so one might assume that ciguatera was indeed considered a problem.

Two investigations of fisheries development potential in the Virgin Islands area have recently been completed. The first (Dammann et al., 1970) developed lines of approach carried out in the second project (Brownell and Rainey, 1971) for expanding the Virgin Islands fisheries through exploitation of deep water stocks. This effort was motivated by several natural limiting factors on the shallow water fish populations, among them the risk of ciguatera poisoning. It now appears that even species previously considered non-toxic because their normal depth-range is greater than 100 fathoms are implicated in ciguatoxications. Brownell and Rainey (1971) report three outbreaks from misty grouper Epinephelus mystacinus taken at 130-135 fathoms and the only documented case of ciguatera from a silk snapper Lutjanus vivanus from 110 fathoms. Two questionable outbreaks are reported by Dammann et al. (1970) from Epinephelus nigritus (actually E. mystacinus). In addition to the outbreaks reported for E. mystacinus and Lutjanus buccanella in Appendix II, we are aware of several outbreaks from L. buccanella attributed to fish caught during the exploratory

fishing projects of the UN/FAO Caribbean Fisheries Development Programme (CFDP) in 1970 and 71. We are currently extracting and bioassaying about two tons of fish caught on UN/FAO cruises from areas where toxicity is reported. These data will be reported in early 1972. Although these data will provide us with a more precise estimate of the proportion of ciguatoxic fishes in the deep shelf - shelf-slope-populations, we have already ascertained that this resource is not free of ciguatoxin.

The toxic blackfin snappers caught by the CFDP came from Saba and Anguilla Banks; fishermen in Montserrat report that most known poisonous fish had been captured in deep water - up to 250 fathoms. It is highly probable that the abrupt drop-off to depths of 200 fathoms or more surrounding many of the Leeward Islands harbor excellent stocks of food fish but it is quite likely that some of these species carry ciguatera poisoning.

Halstead's 1970 survey found that fishermen, fisheries officers, and public health officials were almost unanimous that ciguatera was a deterrent to development of the commercial fisheries. Most Islands reported ciguatera in fish from depths of 0-60 fathoms and the most frequently toxic are all among the first ten species listed in Appendix I. At least two large commercial fishing operations in St. Maarten have given up shallow

water fishing because of repeated ciguatoxications by their catch; several fishermen have reported having to discard large catches of jacks and grouper because their previous catches had caused poisoning. In the small communities of the Virgin and Leeward Islands an individual fishermen is occasionally completely boycotted because of his reputation for regularly landing toxic fish. Fishermen are expected to be able to determine whether or not a particular fish is toxic; an occasional instance is accepted, however.

The fisheries of the Virgin and Leeward Islands do not lend themselves to exploitation by large vessels with modern ground fishing gear. The pelagic stocks are apparently not sufficient to support a much larger fishing effort than is currently in progress. There are probably not sufficient stocks in the shelf-edge populations to withstand intensive fishing pressure equivalent to the Gulf of Mexico - Florida Straits snapper industry. The majority of fishermen in the eastern Caribbean are owner-operators of small boats (20 feet or less) who rarely go more than ten miles from their home port. These fishermen could be trained and proper gear could be utilized for exploiting the area's natural stocks both in shallow and deep water but fish poisoning cases would be likely to increase. A thorough under-

standing of the ciguatera problem must be developed before expansion of the fishery can be effectively accomplished.

IX. SUMMARY AND CONCLUSIONS

1. Fish poisoning in the eastern Caribbean is reported from all of the islands of the northern Virgin and Leeward Islands group. Puerto Rico, Hispaniola and St. Croix have a much lower incidence rate as do the Windward Islands (Trinidad to Martinique).

2. Although clupeoid, elasmobranch, tetraodontoid, and hallucinatory fish poisoning are reported from the eastern Caribbean, scombroid poisoning and ciguatera poisoning are considered to be most important. Because scombroid poisoning can be prevented by modern preservation techniques and treatment of the disease is specific and effective, it is considered a less severe problem than ciguatera poisoning.

3. Epidemiological reporting of ciguatoxications has only been begun in the last month throughout the Virgin Islands and a careful survey of the Leeward Islands must await additional funding. Ciguatera is presently reported as a severe public health problem with only a fraction of the cases reaching medical attention. The problem seems most severe in the area from Montserrat north to the British Virgin Islands including the southeast portion of Saba Bank and the southern shelf of the Virgin Islands plateau.

4. The chemistry, pharmacology, and ecology of ciguatoxin and closely allied compounds are at present poorly understood. The symptomology and species distribution of the toxins in the eastern Caribbean strongly suggest that a situation exists which is very similar to that described from the Pacific islands by the Marine Biotoxin group at the University of Hawaii over the past sixteen years.

5. Toxicity in eastern Caribbean fishes seems to be more prevalent among the large carnivores of reef or reef-related habitats. There are a number of data which suggest that ciguatoxin(s) are produced by some organism in the reef food web and that the toxin is passed through the food web without a significant modification and concentrated by the larger carnivores.

6. Development of the commercial fisheries in the eastern Caribbean is severely impeded by the prevalence of ciguatoxin in commercially desirable species. There is good evidence that the shelf-edge stocks of snapper and grouper are not free from ciguatera poisoning as previously proposed and that exploitation of this presently underutilized resource may be impeded by this toxicity.

X. ACKNOWLEDGEMENTS

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APPENDIX I

Species Most Commonly Reported as Toxic in the Virgin Islands Area

Species	Common Name
Sphyraena barracuda	Great barracuda; barra
Seriola dumerili	Amberjack; amber
Caranx latus	Horse-eye jack
Caranx ruber	Bar jack; carang
Caranx hippos	Crevalle jack
Lutjanus jocu	Dog Snapper; dog tooth
Mycteroperca venemosa	Yellowfin grouper; gramminix
Scomberomorus cavalla	Kingfish; king mackerel
Caranx fusus	Blue runner; hard nose
Gymnothorax funebris	Green moray (probably Conger or Congo eel)
Epinephelus adscensionis	Rock hind
Mycteroperca bonaci	Black grouper
Scomberomorus regalis	Cero (often mistaken for "spanish mackerel")
Caranx lugubris	Black jack
Lutjanus griseus	Grey snapper
Lutjanus buccanella	Blackfin snapper
Epinephelus mystacinus	Misty grouper (often erroneously called Warsaw grouper)
Epinephelus guttatus	Red hind
Seriola rivoliana	Almaco jack
Caranx bartholomaei	Yellow jack
Apsilus dentatus	Black snapper
Epinephelus morio	Red grouper
Lachinolaimus maximus	Hogfish
Belistes vetula	Queen triggerfish; old wife

APPENDIX II

Outbreak reports from fishes caught at depths greater than 500 feet during 1970-71 (data from interviews: Ciguatera Case Repository numbers refer to CRI files)

CCR-71-011. Blackfin snapper - Lutjanus buccanella - about four lbs. Fish caught south of Frenchcap Cay, U. S. V. I. at about 40 fathoms by local fishermen. Fish was eaten by three people all of whom reported abdominal pain, bowels, nausea, vomiting in that order. Onset between three and six hours after ingestion. Secondary symptoms included extreme weakness, listlessness, itching which developed 12-24 hours after ingestion and lasted for several five - seven days. None of the victims reported previous intoxication.

CCR-71-018. Blackfin snapper - Lutjanus buccanella - about three lbs. Fish was purchased on the waterfront at St. Thomas by young couple visiting relatives on the island. Fish was broiled with sauce, no symptoms developed until about eight hours after ingestion. Vomiting, diarrhea, weakness in the knees, dull headache persisted all the following day. Late afternoon produced P. S. D. for man but not wife. Returned to mainland three days after ingestion, no followup available.

CCR-71-008. Misty grouper - Epinephelus mystacinus - 36 lbs. Fish purchased at the dock - caught (apparently) south of St. John. Victims brought five lbs. (two large steaks); refrigerated them and cooked fish next evening. Four persons had dinner of this fish; three young men and a young lady - one man and the lady reported nausea, vomiting, and weakness within six hours; headache, nausea, weakness persisted for "three or four days". The third victim had no violent symptoms of gastrointestinal origin but was lethargic and felt "weak in the joints" next day. The fourth person did not report any illness.

CCR-71-021. Misty grouper - Epinephelus mystacinus - 56 lbs. Fish caught by local sport-commercial fishermen at the "Warsaw Pocket" (misnamed since the area produce misty groupers) - about 3-1/2 miles south of Norman Island, B. V. I. at depth of about 120 fathoms. Fish was filleted and headed; at least six persons ate fillets with no ill effects. Two more people made soup of the head; they reported some intestinal discomfort and weakness, tingling sensations and lethargy the following day. Five other persons fried a small section of the liver: each reported eating "not more than a few bites" that night. All awoke within three hours with violent abdominal cramps, vomiting, and violent headache. Severity of gastrointestinal symptoms continued for six hours or so, then extreme weakness, sinus-like headache, and watery bowels persisted for two - four days. P. S. D. and tingling and numbness in the lips were reported about sixteen hours after ingestion by three of the five. All reported persistent symptoms of weakness and soreness in all body joints for seven - ten days. P. S. D. persisted for at least a week in two victims.

CCR-71-023. Misty grouper - Epinephelus mystacinus - about 30 lbs. Steaks were sold to about four persons none of who apparently developed ciguatera symptoms. A soup was made of the head and eaten by three persons. All described gastrointestinal distress, diarrhea, and nausea within three - six hours; apparently the symptoms disappeared within about 24 hours for two of the victims; the third reported listlessness, weakness, and achy joints which lessened by the third day after ingestion.

APPENDIX III

Methods for identifying ciguatoxic fish as described in the folklore of the Virgin and Leeward Islands (from interviews; Dammann et al., 1969; Halstead, 1967)

I. External characters of the fish or fish flesh which indicate toxicity:

-More yellow or brassy color, especially about the head
-Stripes (in species where they are not normally obvious)
-Darker coloration
-Red coloration to the eyes
-Yellow mucus on inner lining of gullet
-Green tint to raw flesh
-Tiny black "veins" running through the musculature
-Brassy or coppery odor to the flesh
-Teeth are black
-Suspect specifies with roe is toxic
-Enlarged or bloated stomach
-Flesh tastes bitter or hot in mouth

II. Indicator organisms which suggest toxicity:

-Worms in the flesh (particularly jacks and mackerel)
(Worms in the stomach indicate a non-toxic fish)
-Isopod ectoparasites ("cockroach") are not found on toxic fish (jacks)
-Flies will not land on flesh
-Ants will not eat

III. Methods employing an indicator:

-Silver turns black when boiled with toxic fish
-Sweet potato turns black when boiled with toxic fish

APPENDIX IV

CIGUATERA STUDIES PROGRAM - CIGUATERA CASE REPOSITORY

APPENDIX IV

CARIBBEAN RESEARCH INSTITUTE

CIGUATERA STUDIES PROGRAM - CIGUATERA CASE REPOSITORY

CIGUATERA OUTBREAK REPORT FORM P-1 #_____
(Questionaire format, interviewer administered)

PATIENT'S NAME_____
LAST FIRST MIDDLE

AGE_____ SEX_____WEIGHT_____

RESIDENCE:_____

PREVIOUS INTOXICATIONS: YES_____ NO_____

DETAILS: LAST INCIDENT: DATE_____SPECIES_____SEVERITY___

PREVIOUS INCIDENTS:_____

_____

_____

_____

SPECIES OF FISH:_____

WHERE OBTAINED:_____

_____

SIZE:_____DATE BOUGHT:_____DATE COOKED:_____

HOW STORED:_____HOW COOKED:_____

PORTION EATEN_____SAMPLE OBTAINED?_____SAMPLE #_____

SYMTOMOLOGY:

ONSET: (check and place in order) HOURS AFTER INGESTION_____

NAUSEA_____VOMITING_____DIARRHEA_____

ABDOMINAL CRAMPS_____NUMBNESS & TINGLING_____WHERE?_____

ST. THOMAS • VIRGIN ISLANDS 00801 • CABLES: COVI • TEL. 809-774-1252

METALLIC TASTE _____ P.S.D. _____

OTHER SYMPTOMS: _____

24 HOURS AFTER INGESTION

SYMPTOMS: _____

_____

_____

ONE WEEK AFTER INGESTION: _____

_____

_____

_____

LONG TERM EFFECTS: _____

_____

_____

_____

TREATMENT

PHYSICIAN CONSULTED? _____ NAME: _____

ADDRESS: _____
(See physician contact form)

REMEDIES: 1. _____

EFFECTIVENESS: _____

2. _____

EFFECTIVENESS: _____

RECURRENCE DATA: _____

_____

_____

COMMENTS: _____

_____

INTERVIEWER: DATE:

APPENDIX V

SUMMARY OF INTERVIEW

AND

CLINIC VISIT REPORTS

December, 1971 - June, 1972

	Age	Sex	Previous Intoxications	Species involved in present intoxication (*1=same fish)	Size	Portion Eaten	Sample Obtained
(1)	36	M	No	Scomberomorus cavalla (*1)	4 ft.	head middle	Yes
(2)	26	M	No	Scomberomorus cavalla	4 ft.	middle	Yes
(3)	35	M	Yes	Balistes vetula (*2)	2½ lbs.	tail	Yes
(4)	45	M	Yes	Balistes Vetula (*2)	2½ "	head	Yes
(5)	51	M	No	Caranx lugubris	4 lbs.	-	No
(6)	26	M	No	Scomberomorus cavalla (*3)	36 lbs.	tail	No
(7)	28	M	No	Lutjanus vivanus	6 lbs.	1.5 lbs.	Yes
(8)	31	M	Yes	Scomberomorus cavalla (*3)	36 lbs.	middle	No
(9)	14	M	No	Scomberomorus cavalla (*3)	36 lbs.	middle	No
(10)	23	F	No	Scomberomorus cavalla (*4)	-	middle	Yes
(11)	29	M	No	Scomberomorus cavalla (*4)	-	middle	Yes
(12)	38	F	Yes	Lutjanus analis	2½ ft.	head	No
(13)	36	M	No	Sphyraena barracuda (*5)	18 lbs.	middle	Yes
(14)	31	M	No	Sphyraena barracuda (*5)	18 lbs.	middle	Yes
(15)	39	M	Yes	Sphyraena barracuda (*5)	18 lbs.	middle	Yes
(16)	57	F	No	Caranx sp. (*6)	10 in.	tail	No
(17)	40	F	No	Caranx sp.	10 in.	all	No
(18)	48	F	No	Sphyraena barracuda (*7)	large	head	No
(19)	51	M	-	Sphyraena barracuda (*7)	large	–	No
(20)	63	M	No	Caranx sp. (*6)	10 in.	–	No
(21)	26	F		Caranx Sp.	small	–	No
(22)	26	M		Caranx Sp. (*21)	small	–	No
(23)	22	F		Caranx Sp. (*21)	small	–	No
(24)	24	F		Caranx Sp. (*21)	small	–	No
(25)	24	M		Caranx Sp. (*21)	small	–	No
(26)	28	F		Scomberomorus cavalla	28 lbs.	steak	Yes
(27)	29	M		(*26)	28 lbs.	steak	Yes
(28)	22	M		Lutjanus	4 lbs.	all	No
(29)	48	F		Sphyraena barracuda	small		No

[Cont.]

	How Stored	How Cooked	Onset (hrs.)	Nausea	Vomiting	Diarrhea	Abdominal Cramps
(1)	fridge	boiled fried	10	+	+	+	+
(2)	fridge	fried	15	–	–	–	–
(3)	fresh	boiled	0	–	–	–	–
(4)	fresh	boiled	0	+	–	+	–
(5)	freezer	-	-	–	–	+	+
(6)	freezer	broiled	12	+	–	–	–
(7)	fridge	broiled	-	+	+	+	+
(8)	freezer	broiled	31	+	+	+	–
(9)	fresh	fried	½	+	–	+	+
(10)	freezer	fried	-	–	–	+	+
(11)	freezer	fried	4	+	–	+	+
(12)	freezer	boiled	5	+	+	+	+
(13)	freezer	fried	7	–	–	+	+
(14)	freezer	fried	7	+	–	+	+
(15)	freezer	fried	8	+	+	+	+
(16)	freezer	boiled	3	+	+	+	+
(17)	freezer	fried	-	+	+	+	+
(18)	fresh	boiled	-				
(19)	freezer	boiled	-	+	+	+	–
(20)	freezer	boiled	-	–	–	+	–
(21)	-	-	24 hrs.	+	+	+	+
(22)			24 hrs.	+	+	+	–
(23)	-	-	24 hrs.	+	+	+	–
(24)	-	-	24 hrs.	+	–	+	+
(25)	-	-	24 hrs.	+	–	+	+
(26)	fresh	broil	6 hrs.	+	+	+	+
(27)	fresh	broil	8 hrs.	+	+	+	+
(28)	fresh	boil	12 hrs.	+	+	+	+
(29)	fresh		8 hrs.	+	+	+	+

[Cont.]

	Numbness, Tingling	Metallic Taste	P.S.D.	Other Symptoms	Doctor Consulted?
(1)	–	–	–	itching, water burns, weak	Yes
(2)	–	–	–	weakness in knees	No
(3)	+	–	–		No
(4)	+	–	–	aching joints, dry mouth	Yes
(5)	+	–	+	weak, dizzy	Yes
(6)	+	–	–	weak, listless	No
(7)	+	–	+	burning nostrils	No
(8)	–	+	–		No
(9)	+	–	+	teeth hurt	Yes
(10)	–	–	–	weak legs; itching cold scratchy throat	Yes
(11)	+	–	+	itchy throat, weak knee socks	No
(12)	+	–	+	tired, aching joints, eyes burn	No
(13)	–	–	–		No
(14)	–	–	–	headache, burning nose	No
(15)	+	+	–	weak knees, urine burns	No
(16)	–	–	–	weak knees, itching	Yes
(17)	–	–	+	weak aching knees	No
(18)	+	+	+	itching, tired	Yes
(19)				died, possibly from other causes	Yes
(20)	–	–	–	itching	No
(21)	–	–	–	headache	Yes
(22)	–	–	–		Yes
(23)	–	–	–		Yes
(24)	–	–	–	headache	Yes
(25)	–	–	–		Yes
(26)	–	–	+	temp to 103° F weakness	Yes
(27)	+	+	+	weakness for 2 weeks	Yes
(28)	+	+	–	weakness, itching	Yes
(29)	+	–	+	headache, weakness	Yes

APPENDIX VI

MONGOOSE BIOASSAY DATA

Robert W. Brody

May, 1973

MONGOOSE BIOASSAY
DATA

APPENDIX VI
Year End Report
A Study of Ciguatera Fish Poisoning in the Virgin Islands Area
Robert W. Brody
May, 1973

Mongoose #	Fish Species	Size (lb)	Where Caught	Depth	Source	Mongoose Result
1	Barracuda	55	S. Anegada	50 ft.	CIG	+2
2	Barracuda	55	*#1	50 ft.	CIG	2
3	Barracuda	55	*#1	50 ft.	CIG	2
4	Yellowfin grouper	12	W. Saba Is.	70 fm.	UN	0
5	Almaco jack	12	W. of Saba	pot	UN	0
6	Red grouper	15	W. of Saba	65 fm.	UN	0
7	Horse-eye jack	7	N. of Anguilla	110 fm.	UN	0
8	Yellowfin grouper	11	W. of Bermuda	30 fm.	UN	0
9	Barracuda	18	E. of Savanna	trolling	CIG	2
10	Yellowfin grouper	8	N. of Anguilla	110 fm.	UN	0
11	Amberjack	4 1/2	W. of Saba	65 fm.	UN	0
12	Yellowfin grouper	12	N. of Anguilla Bk.	-	UN	0
13	Yellowfin grouper	8	W. of Barbuda	30 fm.	UN	0
14	Barracuda	18	*#9	-	CIG	2
15	Barracuda	18	*#9	-	CIG	4
16	Zulu snapper	2 1/2	off Flanagan Is.	100 fm.	UN	0
17	Queen snapper	9 1/2	off Flanagan Is.	100 fm.	UN	0
18	Red grouper	18	W. of Saba	65 fm.	UN	0
19	Queen snapper	6	off Flanagan Is.	100 fm.	UN	0
20	Yellowfin grouper	10	W. of Saba	60 fm.	UN	3
21	Yellowfin grouper	12	N. of Anguilla	-	UN	0
22	Queen snapper	8 3/4	Flanagan Is.	100 fm.	UN	0
23	Yellowfin grouper	10	*#20	60 fm.	UN	2
24	Black snapper	6	Flanagan Is.	100 fm.	UN	1
25	Barracuda	6	S. of St. John	trolling	donor	0
26	Yellowfin grouper	12	N. of Anguilla Bk.	-	UN	0
27	Kingfish	46	off St. Thomas Harbor	trolling	donor	2
28	Yellowfin grouper	11	S. of St. John	15 fm.	CIG	0

APPENDIX VI - Contd.

Mongoose #	Fish Species	Size (lb)	Where Caught	Depth	Source	Mongoose Result
29	Yellowfin grouper	3 1/2	S. of St. John	15 fm	CIG	0
30	Yellowfin grouper	13 1/4	S. of St. Thomas	-	donor	0
31	Yellowfin grouper	5 1/4	S. of St. Thomas	-	donor	0
32	Kingfish	46	*#24	-	donor	5
33	Yellowfin grouper	5	S. of St. Thomas	-	donor	0
34	Yellowfin grouper	3	S. of St. Thomas	-	donor	0
35	Yellowfin grouper	5 3/4	S. of St. Thomas	100 fm	CIG	0
36	Yellowfin grouper	5	S. of St. Thomas	100 fm	CIG	0
37	Almaco jack	13 3/4	Flanagan Is	130 fm	UN	0
38	Yellowfin grouper	11	S. of St. Thomas	15 fm	CIG	0
39	Moray eel	23 1/2	N. of St. Thomas	15 fm	donor	0
40	*#39	-	-	-	-	0
41	*#39	-	-	-	-	0
42	*#39(liver)	-	-	-	-	0
43	*#39(liver)	-	-	-	-	0
44	Yellowfin grouper	19 1/2	S. of St. John	15 fm	CIG	0
45	*#44	-	-	-	-	0
46	Yellowfin grouper	3 1/2	S. of St. John	15 fm	CIG	0
47	Yellowfin grouper	6	S. of St. John	15 fm	CIG	0
48	Dog snapper	8 3/4	S. of St. John	15 fm	CIG	3
49	*#48	-	-	-	-	2
50	Dog snapper	7	S. of St. John	15 fm	CIG	2
51	*#50	-	-	-	-	2
52	Yellowfin grouper	10	S. of St. John	15 fm	CIG	1
53	*#52	-	-	-	-	2
54	Yellowfin grouper	12	S. of St. John	15 fm	CIG	0
55	Yellowfin grouper	7 3/4	S. of St. John	15 fm	CIG	0
56	*#23	-	-	-	-	2
57	*#24	-	-	-	-	0