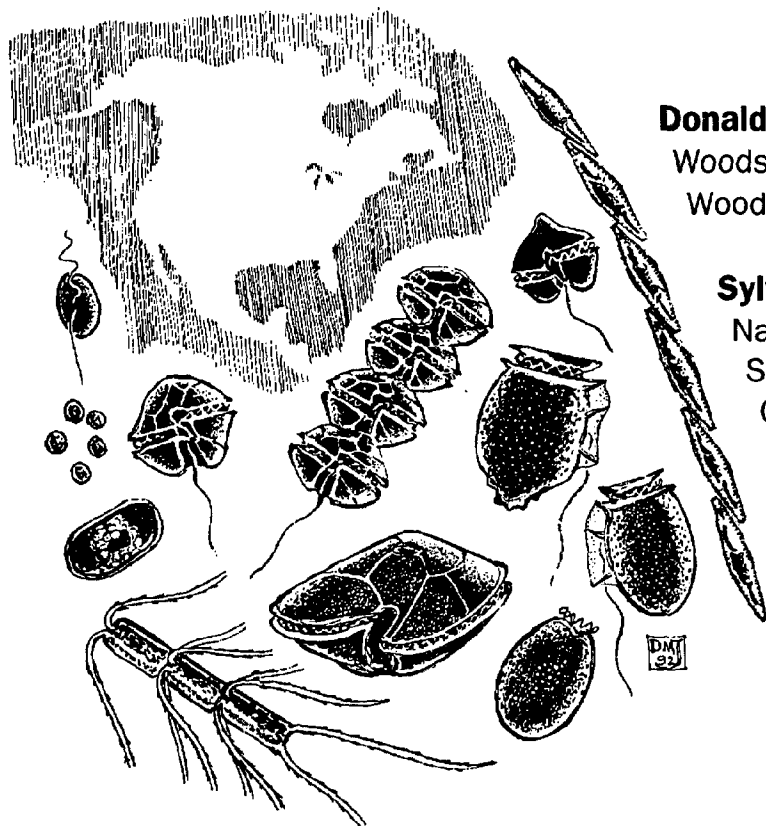




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Marine Biotoxins and Harmful Algae: A National Plan



Donald M. Anderson

Woods Hole Oceanographic Institution
Woods Hole MA 02543

Sylvia B. Galloway

National Marine Fisheries Service
Southeast Fisheries Science Center
Charleston, SC 29422

Jeanne D. Joseph

National Marine Fisheries Service
Southeast Fisheries Science Center
Charleston, SC 29422

January 1993

Woods Hole Oceanographic Institution

Technical Report

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A National Plan**

Donald M. Anderson
Biology Department
Woods Hole Oceanographic Institution
Woods Hole, Massachusetts 02543

Sylvia B. Galloway
National Marine Fisheries Service
Southeast Fisheries Science Center
Charleston, South Carolina 29422

Jeanne D. Joseph
National Marine Fisheries Service
Southeast Fisheries Science Center
Charleston, South Carolina 29422

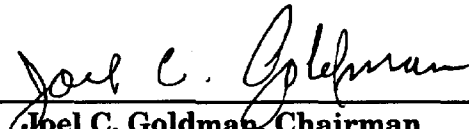
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Joel C. Goldman, Chairman
Biology Department

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MARINE BIOTOXINS AND HARMFUL ALGAE: A NATIONAL PLAN

EXECUTIVE SUMMARY

Marine biotoxins and harmful algae represent a significant and expanding threat to human health and fisheries resources throughout the United States. These phenomena take a variety of forms. From a public health standpoint, four human illnesses are associated with toxic algal blooms and consumption of toxin-contaminated shellfish in the United States: paralytic, neurotoxic, amnesic, and diarrhetic shellfish poisoning (called PSP, NSP, ASP, and DSP respectively). Except for ASP, all are caused by biotoxins synthesized by a class of marine algae called dinoflagellates. ASP is produced by another class of marine algae (diatoms) that until recently were thought to be harmless. A fifth human illness, ciguatera fish poisoning (CFP) is caused by biotoxins produced by epibenthic dinoflagellates attached to surfaces in many coral reef communities. Ciguatera toxins are transferred through the food chain from herbivorous reef fishes to larger carnivorous, commercially valuable finfish. In a similar manner, the viscera of other commercially important fish such as herring or sardines can contain PSP toxins, endangering human health following consumption of whole fish. Whales, porpoises, seabirds, and other animals can be victims as well, receiving toxins through the food chain via contaminated zooplankton or fish.

Marine fauna can be affected by a variety of algal species that release toxins or other compounds into the water or that kill by physically damaging gills. Problems associated with harmful algal species and farmed fish have increased considerably in recent years, due in part to the simultaneous expansion of the fish-farming industry. Furthermore, the death and decay of algal blooms can lead to anoxia through decompositional oxygen demand, resulting in widespread mortalities of fish, shellfish, and invertebrates. An additional problem is that of benthic or planktonic macroalgae that can proliferate in response to anthropogenic nutrient enrichment. This can lead to major negative impacts due to displacement of indigenous species, habitat alteration, or oxygen depletion.

The National Academy of Sciences recently issued a report expressing serious concerns about the quality of the nation's seafood (Ahmed, 1991), emphasizing the need for attention to marine biotoxins and harmful algae. In response to this directive and to a heightened public and governmental awareness of the changing nature of the coastal marine environment, governmental funding is being targeted towards marine biotoxins, harmful algae, and their impacts. The optimum allocation of these resources can benefit greatly from scientific guidance as the new programs are formulated and implemented. A workshop on Marine Biotoxins and Harmful Algae was thus convened in Charleston, South Carolina, from 21-24 April 1992, to bring scientists and regulatory officials together to evaluate U.S. research knowledge and capabilities, and to identify areas where research funds should be directed for maximum benefit.

From a number of nationally recognized leaders in the areas of marine biotoxins, harmful algae, seafood safety, and public health, 24 participants were selected to represent the critical scientific disciplines and all regions of continental North America. Position papers on 12 relevant topics, written by the participants and distributed before the workshop, formed the basis for discussions within the three working groups. Conclusions of the working groups were presented to all participants in two plenary sessions.

Twenty-eight major impediments to progress were identified. These can be summarized as follows:

Deficiencies related to the biotoxins: Toxin standards are largely unavailable; standard sample preservation and handling protocols do not exist; existing assay methods are inadequate for monitoring and research; molecular pharmacology and pharmacokinetics of marine biotoxins are poorly understood; diminution or loss of toxin production can occur in laboratory algal cultures; mass culturing of most toxic species is difficult.

Lack of information on harmful algae: Algal bloom dynamics and species succession are complex and not yet predictable; the relative effects of natural versus anthropogenic influences on population size, species composition, bloom longevity, and toxin production are unknown; knowledge of the physiology of growth and toxin production is inadequate; toxin standards and rapid assay methods are lacking; availability of isolates of toxic or harmful algae is limited.

Lack of information on impacted fisheries resources and protected marine resources: Toxin uptake, metabolism, and depuration in shellfish, fish, and other marine animals is poorly known; toxin sensitivities of different life history stages, and long-term effects of algal metabolites on growth, reproductive success and recruitment are unknown; movement of toxins through the food web is poorly understood; databases are inadequate and not readily accessible to potential users; methods for rapid field assays of fish or shellfish are lacking; toxin standards are often unavailable; analytical methods for toxin detection in animal tissue need improvement.

Inadequate mechanisms and knowledge to protect public health fully: Early warnings of known and unknown toxins are required to protect consumers and industry; assay methods need improvement; toxin standards are not always available; sampling programs are inadequate for bloom detection or characterization; the extent of seafood poisonings is poorly documented; the fate and metabolism of toxins in humans is unknown.

In every category, the lack of sensitive, specific assays (for research and/or shipboard and dockside testing) and toxin standards were identified as major impediments.

The working groups met and proposed recommendations to address each of the impediments. These recommendations, too numerous to list here, are described in detail in the report. They are also embodied in the following goal and objectives (non-prioritized) of the National Plan for Marine Biotoxins and Harmful Algae.

GOAL: Effective management of fisheries, public health, and ecosystem problems related to marine biotoxins and harmful algae.

SPECIFIC OBJECTIVES:

- To isolate toxins and their natural derivatives, and characterize their chemical structures and pharmacological action.
- To develop specific detection methods based on the unique chemistry and/or pharmacology of individual toxins.
- To develop forecasting capabilities for the occurrence and impacts of harmful marine algal blooms.
- To determine the source, fate, and consequences of algal toxins in marine foodwebs and fisheries.
- To develop management and mitigation strategies to minimize impacts of marine biotoxins and harmful algae.
- To identify and improve access to databases for bloom incidence, toxin occurrence in shellfish, mass mortality events, and epidemiology.
- To develop communication programs that incorporate educational and public health materials, electronic communication, and on-site training.
- To provide for rapid response to toxic and otherwise harmful marine algal outbreaks.

PREFACE

Marine biotoxins and harmful algae represent a significant and expanding threat to human health and fisheries resources throughout the United States. This problem takes many forms, ranging from massive "red tides" or blooms of cells that discolor the water, to dilute, inconspicuous concentrations of cells that are noticed only because of the harm caused by the highly potent toxins these cells contain. The impacts of these phenomena include mass mortalities of wild and farmed fish and shellfish, human intoxications or even death from contaminated shellfish or fish, alterations of marine trophic structure through adverse effects on larvae and other life history stages of commercial fisheries species, and death of marine mammals, seabirds, and other animals.

The nature of the problem has changed considerably over the last two decades in the United States. Where formerly a few regions were affected in scattered locations, now virtually every coastal state is threatened, in many cases over large geographic areas and by more than one harmful or toxic algal species. There is a growing consensus in the scientific community that the number of harmful events and the economic costs associated with them have increased dramatically over the last several decades in the United States and around the world. The reasons for this expansion are the subject of considerable debate. Possible explanations include: the eutrophication of coastal waters by human activities, leading to a selection for, and proliferation of, harmful algae; increased aquaculture operations which, in other parts of the world at least, have been shown to enrich surrounding waters and stimulate algal growth, as well as to introduce fisheries resources which simply reveal the presence of previously undetected harmful algae; climatic changes; and increased scientific and regulatory scrutiny of coastal waters and fisheries products leading to the rapid discovery of toxic events.

The United States research, monitoring, and regulatory infrastructure is not adequately prepared to meet this expanding threat. The present approach is to manage threatened fisheries resources using state-run monitoring programs and harvesting restrictions. When unexpected outbreaks occur, the response has often been confused, uncoordinated, and slow. This approach has, nevertheless, provided a reasonable level of protection to the seafood consumer, but illnesses and deaths from marine biotoxins have still occurred, and public confidence in seafood safety continues to erode. In addition to these public health concerns, other impacts of harmful algae can be significant, including the loss of marketable resources because of fish and shellfish mortalities, loss of income for fishermen during outbreaks, or unseen and potentially significant effects on marine trophic structure. No single federal agency has assumed a leadership role in coordinating and supporting the studies needed to optimize management and mitigation strategies. Research funding has always been sporadic and limited.

In an effort to surmount these problems, a workshop, supported by Saltonstall-Kennedy funds, was convened at the NMFS Charleston Laboratory to formulate a National Plan for the prediction, control, and mitigation of the effects of harmful algal blooms on marine biota of the

United States and to promote the safe consumption of seafoods. Participants were selected to represent all critical scientific disciplines and all geographic regions of continental North America. These individuals were selected from the ranks of academia, the Food and Drug Administration, state public health services, the fishing industry, NOAA/NMFS and the National Sea Grant Program. Attendance was limited to promote close working relationships during the workshop.

Prior to the workshop, 12 topics of importance were identified:

- Toxin pharmacology/epidemiology
- Toxin analysis/assays/chemistry/standards
- Bloom biology/ecology
- Remote sensing
- Nutrient/pollution effects
- Taxonomy/genetics/population biology of harmful microalgae
- Hydrography/physical oceanography
- Phytoplankton monitoring
- Shellfish monitoring
- Foodweb effects
- Shellfish depuration/physiology
- Fish mortalities

Workshop participants were assigned topics to review according to their areas of expertise. A group leader was selected for each topic and requested to prepare a position paper with the assistance of others in each group. The position papers, addressing background, current state of knowledge, impediments limiting progress, and prioritized research topics, were distributed to all participants prior to the workshop.

At the beginning of the workshop three working groups were formed. The *Toxins* working group was assigned the first two topics identified above, the *Bloom Biology and Ecology* working group the next six topics, and the *Fisheries and Food Webs* working group the last four topics. During individual working group deliberations, modified lists of impediments and recommendations were prepared. These lists were presented to all participants in two plenary sessions for further discussion and modification.

Given this procedure and the related nature of the issues discussed by the different working groups, it is not surprising that some issues appear several times in the lists of impediments and recommendations that follow. Rather than arbitrarily removing these common issues from subsequent sections after their first mention, the lists were left unchanged so as to emphasize the cross-disciplinary importance of certain issues.

There was considerable discussion on the need to prioritize the recommendations. Attempts to do this in some of the working groups were not successful, as most participants felt that the list of recommendations had already been distilled from a much larger list generated in the position

papers, and thus reflected priority issues. Another concern was that it was difficult to establish absolute priorities between very different topics (e.g. toxin chemistry issues versus bloom dynamics), since such decisions would vary dramatically among individuals or agencies with different responsibilities or interests. **Accordingly, the recommendations in this National Plan are not prioritized.** They are grouped by topic, so that agencies developing research or monitoring programs can work from the lists that most closely match their purview. All are deemed of high (and equal) priority, though it should be recognized that several recommendations appear in each of the three topic areas and thus have general applicability across all disciplines.

I. INTRODUCTION

In recent years, the incidence of harmful algal blooms (sometimes called "red tides") has increased in frequency, severity, and duration, both nationally and globally (Anderson, 1989; Smayda, 1990). These episodes are not attributable to a single algal class but rather to a variety of physiologically diverse species. Some have long been recognized as problem species, others have previously been considered harmless, and still others were unknown to science until their initial outbreaks. The causes for this apparent expansion are unknown, but some believe that human alteration of the water quality of the coastal zone is an important factor (Smayda, 1990).

Economic losses in the United States total millions of dollars per year, and include the cost of toxin monitoring programs, closures of harvestable shellfish resources, mortalities of wild and farmed fish and shellfish, and the value of resources that are not exploited or developed because of the presence or threat of toxic outbreaks. The United States has managed these threatened resources through state-run toxin monitoring programs and harvesting restrictions. Federal agencies have provided relatively minor and often unsustained research support for local or regional studies, and state support has been even smaller and more sporadic. Research teams have made some progress in developing methodologies for toxin analysis, in understanding the structure and pharmacology of certain toxins, in investigating the physiology of toxin production in algae and depuration from shellfish, and in documenting the abundance and distribution of certain harmful species during blooms. Despite these efforts, however, we remain woefully ignorant of the complex mechanisms underlying the growth and accumulation of individual algal species in blooms, the transfer and fate of toxins through the food chain, and perhaps most disturbingly, the influence of human activities on these processes. Also lacking are many of the tools needed for efficient management of potentially toxic fish or shellfish. In particular, sensitive, rapid alternative assay methods are needed for dockside or market-place testing.

The United States lags far behind many other countries in its approach to the management of problems caused by harmful algae and marine biotoxins. Canada, France, Norway, Sweden, China, and others have coordinated national research programs that include workshops or meetings to exchange results and search for solutions to common problems, sustained funding in directions identified as being of high priority, and continual re-evaluation of progress and plans for the future. The United States, in contrast, has had only small, fragmented research programs carried out by individual investigators, with small budgets that are rarely sustained through time. Thus, there is often insufficient communication between U.S. workers and no coordination of activities with respect to national priorities.

The shortfall in our ability to understand and manage these growing problems was reflected in fiscal years '91 and '92 priorities for Saltonstall-Kennedy funded research published in the Federal Register, many of which related to marine biotoxins. This emphasis on biotoxins is but one manifestation of the growing awareness that more of our national resources must be focused on this topic. During the past several years, other federal agencies have announced specific coastal research initiatives, some of which could, and should, include components on marine

biotoxins and harmful algae. It is disturbing to recognize that these initiatives are targeting a field that historically has been fragmented, uncoordinated, and poorly funded. Sound input is urgently needed from scientists, industry, and regulatory officials to keep these new research initiatives focused on high priority, productive endeavors. Thus, the primary goal of this workshop was to formulate a National Plan, consisting of a series of recommendations intended to address the major impediments to progress in the management of, and scientific research on, harmful marine algae and associated toxins.

The participants formulated the following overall goal and objectives for a *National Plan on Harmful Algae and Marine Biotoxins*:

GOAL: Effective management of fisheries, public health, and ecosystem problems related to marine biotoxins and harmful algae.

OBJECTIVES:

- To isolate toxins and their natural derivatives, and characterize their chemical structures and pharmacological action.
- To develop specific tests based on the unique chemistry and/or pharmacology of individual toxins.
- To develop forecasting capabilities for the occurrence and impacts of harmful marine algal blooms.
- To determine the source, fate, and consequences of algal toxins in marine foodwebs and fisheries.
- To develop management and mitigation strategies to minimize the impacts of marine biotoxins and harmful algae.
- To identify and improve access to databases for bloom incidence, toxin occurrence in shellfish, mass mortality events, and epidemiology.
- To develop communication programs that incorporate educational and public health materials, electronic communication and on-site training.
- To provide for rapid response to toxic and otherwise harmful marine algal outbreaks.

II. THE TOXINS

1. General Background

In the United States, the most significant economic and public health problems related to harmful algae are:

- Paralytic shellfish poisoning (PSP), which occurs in all coastal New England states as well as New York and along much of the west coast from Alaska to California. This problem has also extended to offshore areas in the northeast (causative species - the dinoflagellates *Alexandrium tamarense*, *A. fundyense*, and *A. catenella*; Anderson et al., 1982; Nishitani and Chew, 1988; Price and Kizer, 1990).
- Neurotoxic shellfish poisoning (NSP) and fish mortalities in the Gulf of Mexico and, more recently, extending northward to the coast of the Carolinas (causative species - the dinoflagellate *Gymnodinium breve*; Baden et al., 1984; Tester et al., 1991).
- Mortalities of farmed salmonids in the Pacific Northwest (causative species - the diatoms *Chaetoceros convolutus* and *C. concavicornis* and the raphidophyte *Heterosigma akashiwo*; Horner et al., 1990).
- Recurrent brown tides causing mass mortalities of mussel populations in Rhode Island, massive recruitment failure of scallops, and reduction of eelgrass beds around Long Island (causative species - the previously unknown chrysophyte, *Aureococcus anophagefferens*; Sieburth, et al., 1988).
- Ciguatera fish poisoning (CFP), a malady associated with dinoflagellate toxins accumulated in tropical fish flesh, occurring in virtually all sub-tropical to tropical U.S. waters (Florida, Hawaii, Guam, U.S. Virgin Islands, Puerto Rico, and many Pacific Territories; Ragelis, 1984; major causative species *Gambierdiscus toxicus*, *Prorocentrum* spp., *Ostreopsis* spp., *Coolia monotis*, *Thecadinum* sp., and *Amphidinium carterae*; Juranovic and Park, 1991).
- Amnesic shellfish poisoning (ASP) which occurred first in southeastern Canada in 1987, but has been a problem for the U.S. Pacific coast states over the past two years (causative species - the diatoms *Pseudonitzschia pungens* forma *multiseries* and *Pseudonitzschia australis*; Garrison et al., 1992; Buck et al., 1992; Fritz et al., 1992; Wood and Shapiro, 1992). This sometimes fatal illness is so named because one of its most severe symptoms is the permanent loss of short-term memory. The ASP toxin, domoic acid, has been detected in shellfish from both the West and East Coasts of the United States, and toxic *P. pungens* f. *multiseries* cells have been isolated from Gulf of Mexico waters, though no toxin has yet been detected in the field. Thus, the threat to U.S. shellfish consumers from this dangerous alga covers a broad geographic area. The name "ASP" understates the severity of this problem, as it is now known that domoic acid also accumulates in fish and in crab viscera along the west coast of the United States, where the impact of this toxin on non-molluscan fisheries may well exceed the loss to molluscan fisheries (e.g., razor clam).

Another serious threat is diarrhetic shellfish poisoning (DSP) which some consider the most serious and globally widespread phytoplankton-related seafood illness. The first confirmed incidence of DSP in North America occurred in 1990 when these toxins were detected in shellfish from the southern coast of Nova Scotia following numerous human illnesses (Quilliam et al., submitted ms.). Another DSP outbreak in Canada occurred in 1992 (Wright, pers. comm.). DSP-producing species of phytoplankton occur throughout all temperate coastal waters of the United States, and thus present a potential problem for the future, though no outbreaks of DSP have yet been confirmed.

2. Toxicology and Pharmacology

2.1 Background

Naturally-occurring toxins responsible for the intoxication syndromes associated with seafood are as diverse as are the algae that produce them (Table 1). Man is exposed principally

TABLE 1
TOXIN-DERIVED HUMAN TOXICOSES

TOXIN FAMILY (Number of Toxins)	SYNDROME¹	SOLUBILITY	ACTION ON
Brevetoxin (10)	NSP	Fat	Nerve, Muscle, Lung, Brain
Ciguatoxin /Maitotoxin (multiple)	CFP	Fat/Water	Nerve, Muscle, Heart, Brain
Domoic Acid (11)	ASP	Water	Brain
Okadaic Acid (3)	DSP	Fat	Enzymes
Saxitoxin (18)	PSP	Water	Nerve, Brain

¹ NSP= Neurotoxic Shellfish Poisoning, ASP = Amnesic shellfish Poisoning,
DSP = Diarrhetic Shellfish Poisoning, PSP = Paralytic Shellfish Poisoning,
CFP = Ciguatera Fish Poisoning

by consumption of contaminated seafood products, although one type of toxin (brevetoxin), because of aerosol formation due to wave action, also causes respiratory asthma-like symptoms during blooms of the toxigenic organism. In all cases, the diseases are caused by specific interaction of the toxins with tissues and organs responsible for carrying out vital cellular functions. By modifying these functions in deleterious ways, the toxins disrupt nerve electrical conduction, uncouple communication between nerve and muscle, and prevent critical physiological processes from occurring. Most of the toxins accomplish this by binding to specific receptors, or docking sites, on the tissue or organ leading to critical changes in intracellular concentration of ions such as sodium, calcium, and potassium. Some of the cellular changes lead to permanent effects in the exposed cells.

Seafood toxins bind with high affinity to specific receptor sites, often with binding constants in the 10^{-9} to 10^{-12} M range. Most binding is reversible, but dissociation times may be quite prolonged. Except for identification of the general category of toxin receptors in living organisms, virtually nothing is known about the chemical interaction of the toxins with their specific binding sites.

Many of the toxin classes are not single chemical entities, but instead represent families of compounds of similar chemical structure (Table 1). Each toxin derivative of the same parent compound is slightly altered in chemistry. This leads to wide-ranging variability in toxicity of the individual modified toxins.

Acute single-dose lethality of seafood toxins has been extensively studied in the laboratory (Shimizu, 1987). However, chronic and/or repeated exposure to marine seafood toxins, which is a more realistic phenomenon, has not been adequately examined. There is a serious lack of knowledge as to how the toxins are distributed throughout the body and eliminated. Other important questions include how long the toxins circulate before elimination, and how they are metabolized by living organisms. These knowledge gaps prevent researchers from devising antidotes or effective treatments which may alleviate or lessen the symptoms. Therapeutic intervention is primarily limited to symptomatic treatment and life support if necessary.

Similarly, statistical data collection on human exposure, intoxication duration, and number of incidences are limited and incomplete. Many cases of intoxication are not reported, or are reported inadequately based on hear-say evidence with little documentation.

Since 1978, illnesses in the U.S. due to natural algal toxins have included CFP, PSP, NSP and ASP. No incidents of DSP have yet been verified in this country. Although records are incomplete because reporting to the Centers for Disease Control (CDC) is voluntary, evidence indicates that ciguatera was responsible for about half of all seafood intoxications between 1978 and 1987 (Ahmed, 1991). A growing body of evidence indicates that incidents of ASP are on the increase (Buck et al., 1992; Garrison et al., 1992; Villac et al., in press; Horner and Postel, in press), and that DSP may shortly make its *début* in the United States. Certain of the toxicoses, like the toxigenic organisms, are focused geographically and result from consumption of particular species. However, with the increase in interstate and international transport of seafood, as well as international travel by seafood consumers, there are virtually no human populations that are free from risk.

ASP: Amnesic shellfish poisoning can be a life-threatening syndrome. It is characterized by both gastrointestinal and neurological disorders (Bates et al., 1989). Gastroenteritis usually develops within 24 hours of the consumption of toxic shellfish; symptoms include nausea, vomiting, abdominal cramps, and diarrhea. In severe cases, neurological symptoms also appear, usually within 48 hours of toxic shellfish consumption. These symptoms include dizziness, headache, seizures, disorientation, short-term memory loss, respiratory difficulty, and coma. In 1987, four victims died after consuming toxic mussels from Prince Edward Island, Canada. Since that time, Canadian authorities have monitored both the water column for the presence of the causative diatom, and shellfish for the presence of the toxin, domoic acid. Shellfish beds are closed to harvesting when the domoic acid concentration reaches 20 $\mu\text{g/g}$ shellfish meat. Fish

and crab viscera can also contain domoic acid, so the risk to human consumers and animals in the marine food chain is more significant than previously believed.

Ciguatera: *Ciguatera fish poisoning* produces gastrointestinal, neurological, and cardiovascular symptoms. Generally, diarrhea, vomiting, and abdominal pain occur initially, followed by neurological dysfunction including reversal of temperature sensation, muscular aches, dizziness, anxiety, sweating, and a numbness and tingling of the mouth and digits. Paralysis and death have been documented, but symptoms are usually less severe although debilitating (Miller, 1991). Recovery time is variable, and may take weeks, months, or years. Rapid treatment (within 24 hours) with manitol is reported to relieve some symptoms. There is no antidote, supportive therapy is the rule, and survivors recover. Absolute prevention of intoxication depends upon complete abstinence from eating any tropical reef fish, since there is currently no easy way to measure routinely ciguatoxin or maitotoxin in any seafood product prior to consumption.

DSP: *Diarrhetic shellfish poisoning* produces gastrointestinal symptoms, usually beginning within 30 min to a few hours after consumption of toxic shellfish (Yasumoto and Murato, 1990). The illness, which is not fatal, is characterized by incapacitating diarrhea, nausea, vomiting, abdominal cramps, and chills. Recovery occurs within three days, with or without medical treatment.

NSP: *Neurotoxic shellfish poisoning* produces an intoxication syndrome nearly identical to that of ciguatera. In this case, gastrointestinal and neurological symptoms predominate. As noted above, formation of toxic aerosols by wave action can produce respiratory asthma-like symptoms. No deaths have been reported and the syndrome is less severe than ciguatera, but nevertheless debilitating. Unlike ciguatera, recovery is generally complete in a few days. Monitoring programs (based on *G. breve* cell counts) generally suffice for preventing human intoxication, except when officials are caught off-guard in previously unaffected areas.

PSP: *Paralytic shellfish poisoning*, like ASP, is a life threatening syndrome. Symptoms are purely neurological and their onset is rapid. Duration of effects is a few days in non-lethal cases. Symptoms include tingling, numbness, and burning of the perioral region, ataxia, giddiness, drowsiness, fever, rash, and staggering. The most severe cases result in respiratory arrest within 24 hours of consumption of the toxic shellfish. There is no antidote, supportive therapy is the rule and survivors recover. PSP is prevented by large-scale proactive monitoring programs (assessing toxin levels in mussels, oysters, scallops, clams) and rapid closures to harvest of suspect or demonstrated toxic areas.

2.2. Impediments and Recommendations

The *Toxins* working group identified three major impediments to progress in the area of toxin pharmacology and toxicology, and recommended solutions to these impediments. Priority order was not assigned to the impediments.

IMPEDIMENT: *Reference toxin is difficult to obtain, is not always reproducible, and is generally costly.* This impedes development of methods for detection, prevents detailed studies of physiology, and inhibits development of molecular pharmacology to explain toxin interaction at the receptor level.

RECOMMENDATION: Establish reference toxin supplies for the five major classes of toxins.

This can be accomplished by isolation and purification of toxin from fish and shellfish, from mass cultures of the toxic phytoplankton, or synthesis of less accessible toxins. Production of radiolabeled toxin first requires adequate supplies of purified standards. Three levels of standards are required: Pharmacological Standards, Analytical Standards, and Certified Standards.

Toxic marine dinoflagellates are some of the most difficult algae to grow in mass culture. Facilities required are extensive and sophisticated, and careful control of nutrient levels, pH, temperature, lighting, security, and cleanliness is necessary. Likewise, the facilities necessary to extract and purify multi-milligram quantities of these highly potent materials are extensive, and require special instrumentation for separation, isolation, detection and storage. At many such facilities, detailed safety plans are implemented. Although this is an objective which should be achieved as rapidly as possible, it must be recognized that a continuing financial commitment is necessary to implement the production of consistent, reliable standards. With the exception of ciguatera-related toxins, facilities expansion to produce the desired quantities of all levels of standards is a logistical possibility within 2-3 years. The ciguatera toxins will require extensive research prior to standards availability (see toxin standards section).

A major problem with available biotoxin supplies is their distribution to monitoring agencies and the research community. A well-defined distribution plan for biotoxin standards isolated or synthesized with the assistance of federal funds needs to be prepared. It is an overwhelming consensus that toxins should be made available at a minimal cost, for example, as is done by the National Hormone and Pituitary Program.

IMPEDIMENT: The annual incidence of seafood toxin poisoning is poorly documented.

Without knowledge of the actual magnitude of the problem, little can be done to evaluate remedial measures aimed at reducing incidence. Effects of episodic and chronic exposure have been totally neglected. It is also apparent that part of the reporting problem is due to lack of toxic syndrome recognition.

RECOMMENDATION: Develop a database in collaboration with the CDC for marine seafood intoxication. Explore development of better reporting tools, including mandatory reporting. Incorporate episodic or chronic exposures. Educate physicians, public health officials, and consumers in issues of seafood poisoning.

Incidences of seafood intoxication are thought to far exceed the actual reporting. Valuable information regarding seafood intoxication does not reach the proper reporting officials for many reasons. Initial symptoms often are gastrointestinal in nature and are misdiagnosed as the "flu." Only later, after intensification of symptoms or a significant number of individual events, are alternative diagnoses considered. Early identification of the toxicologic syndrome is necessary to permit effective therapeutic intervention. Once recognized, proper and prompt reporting alerts official agencies to implement regulatory directives. If the syndromes are recognized, and the reporting is mandatory, the data will be comprehensively recovered. This is a long-term objective, and has appended to it a continuing effort to collect and analyze data, to educate, and

to develop the program further. Initial funds (1-2 yrs) should be provided to trained epidemiologists and biostatisticians for examining the status of the situation and providing additional recommendations for full implementation of a program. Ideally, this should be undertaken by an epidemiologist or group of epidemiologists with interest and experience in marine toxin poisoning. Full implementation of this program would take 5-10 years.

IMPEDIMENT: Poorly-defined aspects of molecular pharmacology prevent development of therapeutic agents and appropriate receptor-based assays for marine toxins.

RECOMMENDATION: Identify primary tissues of toxicologic action in animals and man, and develop *in vitro* models that reflect the primary toxicologic action. Identify the molecular characteristics of specific receptors for saxitoxin/tetrodotoxin, brevetoxin/ciguatoxin, maitotoxin, okadaic acid, and domoic acid. Explore structural modification of toxins in relation to toxicity and binding affinity.

The molecular mechanism of intoxication is not known in detail for any marine toxin. This is in large part due to the fact that we do not know the primary targets for the biotoxin in the human (or animal) body. It is critical to define the primary relevant site of action for marine biotoxins, before funding substantial amounts of research on nonrelevant tissues.

Laboratory studies which employ radiolabeled toxins are capable of defining specific binding or recognition sites on a molecular level. Detailed knowledge of the toxin receptors is essential for understanding why "toxins" are toxic. Elucidation of the mechanism of action provides information essential for the development of receptor-based methods for detection, and practical methods for treatment of intoxication. Receptor-derived assays correlate well with toxicity, and may be enhanced using molecular recombinant technology, thereby reducing the need for animal-based toxicological assays. With our current capabilities and available molecular probes, brevetoxin research will continue (complete in 2-3 years), and pharmacologic probe synthesis and investigation initiated for saxitoxin, okadaic acid, and domoic acid (3-6 yrs). Maitotoxin and ciguatoxin(s) will require more structural information prior to direct analysis (> 7 yrs).

3. Analysis, Standards, Chemistry

3.1. Background

The chemical structures of ASP, DSP (okadaic acid family), NSP and PSP toxins are known (Baden, 1984; Shimizu, 1984; Yasumoto et al., 1984; Shimizu et al., 1986; Wright et al., 1989; Hall and Strichartz, 1990). Structures of the toxins responsible for ciguatera are not yet known, except for ciguatoxin in fish from the Tahitian region (Murata et al., 1990). In addition, the structure and role of maitotoxin (MTX) is only partially understood (Murata et al., 1992), as is its role in ciguatera fish poisoning. Determination of the structures of newly-recognized marine biotoxins requires the dedication of substantial fiscal resources, instrumentation, and personnel time. Currently, the only certified standard(s) available is for domoic acid (Institute for Marine Biosciences, National Research Council of Canada, Halifax, NS, Canada). Reference material is also available for domoic acid. Moreover, suites of standards for the different naturally-occurring toxins are necessary.

Biological tests using mice or rats are available for all listed toxins, but the bioassay for ASP toxins lacks sufficient sensitivity (Quilliam et al., 1989). Other biological tests that do not use animals have yet to be subjected to collaborative review. Accepted chemical analytical methods have been developed only for the ASP toxin, domoic acid (Quilliam, et al., 1991; Pocklington, et al., 1990). Chemical methods exist for the other toxins but have not gone through appropriate collaborative review (e.g., for PSP) or need further refinement (CTX, DSP, NSP).

In recent months, the detection of known toxins in unexpected vectors (e.g., PSP toxins in crab viscera, ASP toxins in fish and crab viscera) has accentuated deficiencies in assay procedures. The extension of certain assay procedures to "new" tissues can sometimes be done routinely when the organisms are related, but new procedures need to be developed when organisms not covered by the original method are investigated. Substitution of marine mammal liver for clam tissue in PSP testing, for example, can lead to erroneous conclusions.

3.2. Impediments and Recommendations

The *Toxins* working group identified three major impediments to progress associated with the development of standards and assays and provided recommended solutions to these non-prioritized impediments.

IMPEDIMENT: The chemical complexity of marine biotoxins impedes the development of a viable seafood safety program and successful fisheries resource management.

RECOMMENDATION: Determine the structure, chemical properties, and pharmacological behavior of marine toxins. Develop methods for extraction and purification of toxins from natural sources, their synthesis, and preparation of toxin derivatives.

The determination of the structure, properties, and behavior of marine toxins is an essential first step for the development of new and improved methods for the detection of toxins, for mitigating their presence and effects in seafoods, and for a successful fisheries resource management program. This expanded knowledge will augment the characterization of new seafood toxins as they arise. The structural characterization of marine toxins has challenged researchers for many years due to lack of appropriate expertise, resources, and specialized equipment and techniques not commonly available, such as nuclear magnetic resonance (NMR) spectrometers and "soft" ionization mass spectrometers.

Mass culture of toxic algae, chemical synthesis, chemical modification, and derivatization methods are necessary for the economical production of toxin standards and related compounds, and are vital for the preparation of easily-detected derivatives for use in monitoring programs and toxicological studies. Toxicological and pharmacological studies are necessary for the development of antidotes or other medical intervention techniques. These activities might require 3-5 years for known toxins, and 5-7 years for unknown or poorly understood toxins.

IMPEDIMENT: Every study related to marine biotoxins is compromised by a lack of toxin standards. It is impossible to develop methods for the detection and quantification of marine toxins without the availability of defined marine toxin standards.

RECOMMENDATION: Isolate or synthesize, and characterize sufficient quantities of purified toxin(s) for the development of appropriate standards. Establish a toxin standard development, maintenance, and distribution system, so that these toxins are equally accessible and available to all qualified research groups.

Biological, toxicological, and chemical studies require submilligram to milligram quantities of rigorously characterized marine toxins. This work will require the collaboration of biologists to produce adequate quantities of source materials, and chemists to isolate, purify, and characterize the toxins. Once prepared, either by biological or synthetic means, these toxin standards must be readily and reliably available to research and monitoring programs so that public health is adequately protected and fisheries resources are effectively managed. These standards should have international acceptance.

IMPEDIMENT: *Current assay methods for marine biotoxins are inadequate for most monitoring and research purposes. Most monitoring programs utilize whole animal assays which lack sensitivity and are becoming increasingly unacceptable.*

RECOMMENDATION: Develop rapid and cost-effective methods for detecting and quantifying marine toxins that are *internationally* acceptable.

The seafood industry is a global activity that requires coordination of safety regulations between trading nations. To avoid conflicts, the development of marine toxin action levels and methods must be evolved in close cooperation with other international agencies. A particularly important challenge is the development of rapid field methods, such as enzyme or immunoassay-based kits. Sensitivity of all methods must relate to human toxicity and to levels present in contaminated seafoods. The methods developed must be subjected to verification through formal, collaborative studies.

III. BLOOM BIOLOGY AND ECOLOGY

1. Bloom Dynamics

1.1. Background

The impacts from harmful or toxic blooms are necessarily linked to the population size and distribution of the causative algae. Efforts to manage fisheries resources affected by algal blooms or to assess the possible impacts of anthropogenic influences on harmful species requires an understanding of bloom biology and ecology.

The growth and accumulation of individual harmful algal species in a mixed planktonic assemblage are, however, exceedingly complex processes involving an array of chemical, physical, and biological interactions. Blooms can occur over wide geographic areas and may involve long-distance transport to affected resources. Harmful blooms can also occur on the ocean bottom, caused by either microscopic or macroscopic algal species. Macroalgal blooms need not produce toxins to be harmful. They can dominate planktonic or benthic communities, changing food web structure and altering habitats for many marine organisms.

Our level of knowledge about each of the many harmful algal species varies significantly, and even the best-studied remain poorly characterized with respect to bloom or population dynamics. Resolution of various rate processes integral to the population dynamics (e.g., input and losses due to growth, grazing, encystment, excystment, and physical advection) has not been accomplished, but is fundamental to the long-term management of fisheries resources or marine habitats affected by harmful algae. Many of the processes are difficult to quantify in the field because harmful species are often only a small fraction of the biomass in natural samples. **The end result is that there are no predictive models of population development, transport, and toxin accumulation for any of the major harmful algal species in the United States.**

Within the past two decades, the incidence of toxic blooms caused by formerly undetected taxa (the so-called "hidden flora") has increased (Anderson, 1989; Smayda, 1990). The basic biology and environmental triggers for toxic activity of these cryptic species have not been characterized. U.S. coastal waters are generally becoming nutrient enriched, often because of human influences. *The impact of increased nutrients on harmful algal bloom events remains uncertain, however, and the relative importance of natural variance vs anthropogenic influences on blooms is not known (Smayda, 1990).* To further confuse the issue, global changes and trends in several physical and chemical parameters, such as temperature and UV radiation, as well as nutrient enrichment, may also affect harmful algal blooms.

The long-anticipated potential of remote sensing is becoming a reality in the study of harmful bloom dynamics. Near real-time sea surface temperatures have been used successfully to identify oceanic features and water masses associated with blooms of two harmful species in two different hydrographic regimes (Keafer & Anderson, in press; Tester et al., 1991). This approach needs further refinement and should be extended to other species and regions of the United States.

There is a serious deficiency in our understanding of the physiology and genetics of toxin production. Potentially harmful algae exhibit genetic variability to the extent that toxic and non-toxic strains occur within individual species, and toxic species exhibit a range of inherent potencies. These differences in toxin composition and content are genetically and environmentally regulated, and increase the difficulty in identifying and evaluating the harmful effects of these algae. Development of molecular probes and other techniques for genetic characterization would aid in the identification and separation of harmful algae present in mixed natural populations.

1.2. Impediments and Recommendations

The *Bloom Biology and Ecology* working group identified five major impediments to progress in the area of algal population dynamics, biology, and ecology, and one impediment in the area of phytoplankton monitoring. The group recommended solutions to these impediments.

IMPEDIMENT: Adequate documentation of harmful algal events is difficult because of the lack of rapid, species-specific methods for counting and separating cells from natural samples.

RECOMMENDATION: Support cooperative development of molecular probes (nucleic acid and antibody-based) and other techniques for genetic characterization. Provide access to appropriate facilities and equipment; disseminate technology and probes.

Harmful and benign organisms are currently difficult to distinguish in a timely fashion. This restricts identification and separation of harmful algae for their rapid quantification and analysis within multi-species planktonic assemblages. Nucleic acid and antibody probes, which target different cellular components, offer high flexibility and specificity in their design and application. Some laboratories which have the appropriate skills, expertise, and equipment can provide training and support for others that have the need for probes but lack these resources. Equipment should be made available for the analysis of field and laboratory samples, perhaps through a dedicated facility with a flow cytometer and equipment for nucleic acid and protein analysis. Probes are in an early stage of development for several harmful species, but for most species, there is no sequence information or other biochemical characterization that can be used to design specific probes. Knowledge about one species can frequently be applied to closely related species, greatly accelerating the rate at which unstudied species can be characterized. With immediate support, species-specific probes for several harmful algae could be available within one to two years. A battery of probes against many harmful species could follow within 5 years. Once the genes involved in toxin production are identified and characterized, probes can be developed to identify only toxic species. The use of such probes in quantifying target cells requires additional studies of the physiological variability of their molecular targets under different environmental conditions.

IMPEDIMENT: Population dynamics, including the rate processes required in predictive models of harmful blooms, cannot be adequately described or predicted, although this information is of fundamental importance to effective resource management.

RECOMMENDATION: Determine biological rate processes and initiate studies of coastal hydrography and water circulation for development of physically/biologically coupled models at temporal and spatial scales appropriate to harmful algal blooms.

Despite the fundamental importance of predictive models for harmful algal blooms in different regions, no such models exist for U.S. problem species. Knowledge of the rate processes that determine the net accumulation of cells and physical models of the regional hydrographic features that influence the initiation, distribution and maintenance of blooms are both indispensable to such models.

Information on bloom dynamics can be gained through laboratory and field studies that define nutrient uptake kinetics, growth rates, loss terms, and life cycle dynamics. While field conditions such as circulation, meteorology, and water chemistry have long been recognized as critical elements in blooms of some toxic species, neither the initial boundary conditions, nor the hydrographic regimes within which harmful blooms occur are clearly understood. Regional multi-disciplinary field efforts, adequate to characterize the physical circulation models, are needed. Ideally, these would be 3-5 year programs. The ultimate goal is to couple population dynamics with physical circulation models for a given hydrographic regime, and to refine the physically/biologically coupled models using field bloom observations and toxicity patterns. Laboratory studies could be accomplished within about 2 years per species. Field studies can be greatly facilitated by timely accessibility to archived and *in situ* environmental information.

IMPEDIMENT: Competitive outcomes in species selection and succession cannot be predicted, nor can the relative effects of natural vs. anthropogenic factors be resolved.

RECOMMENDATION: Undertake experimental studies on factors regulating selection and succession, emphasizing grazing, nutrients and related anthropogenic variables, and allelopathic effects of toxins.

Prediction of harmful species occurrences and evaluation of potential stimulation by anthropogenic influences are essential for effective resource management. The few available long-term data sets strongly suggest a link between nutrient enrichment and increasing occurrences of known harmful species as well as formerly undetected taxa ("hidden flora").

Prediction of the outcomes of competitive interactions between harmful algae and other food web components depends upon understanding the processes regulating growth, toxicity, and encystment of individual harmful species. Laboratory experiments (2-3 years) can be used to examine growth across gradients of nutrients (i.e., absolute concentrations and variable supply ratios), temperature, salinity, light, mixing, and grazing by appropriate predators. Given this knowledge, experiments can be expanded to include natural communities (e.g., in mesocosms, field enclosures; 5 years) in order to examine competition, grazing, allelochemical effects, and other influences on selection and succession of harmful algae. These field data can be used to estimate rate constants for accumulation and loss terms which, in turn, would enable construction of mathematical models needed to assess mitigation strategies under variable environmental conditions.

Understanding the influence of anthropogenic effects will require analysis of data bases for phytoplankton communities and tractable anthropogenic variables such as inputs of nutrients and other pollutants. Initiation or expansion of long-term monitoring programs of at least 10-years duration must include both episodic events and nutrient time-series studies. Short-term and long-term correlations between pollutant inputs and abundances of harmful algal species, together with information from the autecological studies, will provide a basis for mesocosm-scale experiments. These experiments (3-5 years duration) are needed to test potential mitigation strategies and strengthen interpretations about the influences of anthropogenic variables on bloom species selection and succession.

IMPEDIMENT: There is insufficient knowledge of the physiology of algal growth and toxin production in response to environmental variables.

RECOMMENDATION: Conduct experimental studies of organism physiology, emphasizing environmental tolerances and factors which influence growth and toxin production. Expand culture collections to include broad geographical representation of all potentially harmful species; include multiple clones from single populations.

Tolerance ranges and optima for growth and toxin production in response to environmental variables such as salinity, temperature, and light must be determined for multiple toxic and non-toxic (if available) clones of each species in batch culture. In addition, classical steady-state analyses of nutrient requirements and uptake rates and toxin physiology (including content and composition) of each species are necessary. This work will depend upon a supply of appropriate isolates, our ability to manipulate them in culture, and the availability of sensitive and reliable methods of toxin analysis. Physiological experiments can be carried out in tandem with studies of tolerance ranges and optima once the basic individual growth requirements are determined, and will take 3 yrs to complete for each species.

Individual clones of a single species exhibit marked variation in numerous characteristics, including growth and toxin production (Maranda et al., 1985; Bomber, et al., 1989; Cembella et al., 1987; Hayhome et al., 1989), and thus may not be representative of local or regional populations. A few laboratories in the United States have initiated "syndrome-based" culture collections of harmful marine microalgae. Presently, isolates housed in these collections do not adequately represent the full range of variants characteristic of each harmful species. It is essential that new clones be established from throughout the geographical range of each harmful species. Establishment of clones should be accompanied by basic screening programs in order to select ideal clones for physiological and toxicological studies.

Production of toxins in quantities sufficient for their purification and characterization requires identification and culture of "high performance" clones and knowledge of their growth requirements. Such collections could be established within a 3-year period. Completion of basic screenings may extend each project into a fourth year.

There are anecdotal and circumstantial accounts of bacterial involvement in toxin production by harmful algal species, but only one set of published data demonstrates bacterial synthesis of PSP toxins (Kodama, 1990). The existence of toxigenic bacteria and their association with harmful algal species must be investigated. This work will rely on the isolation of bacteria and the

development of techniques that optimize our ability to detect toxin production. Verification of toxigenic bacteria will take about one year for each species of harmful algae once methods are accepted for unequivocally demonstrating the presence or absence of the bacteria.

2. Phytoplankton Monitoring

2.1. Background

Testing shellfish and other seafood for possible toxins is expensive and time-consuming. Further, current seafood monitoring efforts are often limited by cost and geographic area covered and may not even test the food product most affected by a particular toxin. An easier and possibly more effective approach involves regular, routine sampling and analysis of phytoplankton samples, especially in areas where aquaculture and/or recreational harvesting are common. If potentially toxic phytoplankton species are found, then more expensive seafood testing must be done.

Routine phytoplankton monitoring would provide long-term data on the occurrence of harmful algal species and foster the development of testable hypotheses and insights into the status and trends in harmful algal bloom events. Retrospective analyses of the few existing historical data sets and initiation of time-series will allow assessment of the role of improved monitoring programs and strategies. This information will also promote development of badly needed mitigation methods.

2.2. Impediments and Recommendations

IMPEDIMENT: *Coastal environmental programs are inadequate for bloom detection, monitoring and mitigation of bloom effects.*

RECOMMENDATION: Identify regional expertise and facilities. Establish species-specific monitoring programs on a regional scale, using shipboard techniques and remote sensing where appropriate. Identify sentinel species appropriate for each specific toxin and habitat type. Organize regional response teams and logistical support for unexpected events, and reporting centers to accommodate rapid response. Develop practical response protocols for protecting aquaculture sites on a regional and/or species-specific basis. Coordinate and develop national and regional training programs (e.g., sampling and identification methods). Develop and disseminate adequate reference materials.

Adequate phytoplankton monitoring programs can serve as early warning systems to moderate the effects of blooms on public health, aquaculture, and fisheries. Response teams, organized by region using existing expertise and maintained as part of a national program, should augment species-specific monitoring programs in areas of recurring bloom events. In-water monitoring and remote sensing provide the early warning systems needed by the aquaculture industry and government officials. Further, long-term data sets are needed for trend analyses. These recommendations are a high priority and must be implemented through federal/state/academic/private industry partnerships.

A network of sentinel sites might be composed of local residents and user groups who are often the first to recognize a bloom event and notify local government agencies. Other sentinel sites

could be located at coastal aquaculture facilities. Government and/or industry personnel must be able to sample and quickly identify the causative organism and determine whether it is a known or potentially toxic species. Samples must be sent to taxonomic experts for verification. Technical training will provide the expertise needed for early warning systems and local response. Training should be structured at several levels.

These recommendations should be implemented immediately and continue indefinitely, although possibly on a somewhat reduced level after 5 years, depending on trend analysis and local needs. The Canadian domoic acid experience has shown that phytoplankton monitoring can be an effective component in a program to protect seafood consumers from marine biotoxins.

IMPEDIMENT: The causes and effects of harmful blooms of benthic and planktonic macroalgal species are poorly understood.

RECOMMENDATION: Evaluate the manner in which macroalgal species composition can be influenced by nutrient enrichment, coastal erosion, and other human activities. Determine the effects on habitats and food-chain structure that are associated with macroalgal blooms.

Much of the focus in this program is on microscopic algal species which bloom in surface waters, but harmful blooms of macroalgae also occur. These can cause harm by altering benthic habitats through the displacement of indigenous species, and by changing food-chain structure and dynamics. One manifestation of coastal nutrient enrichment is the enhancement of benthic (and, on occasion, planktonic) macroalgal abundance, with certain opportunistic species often dominating. Not only will studies of benthic algal species succession and dominance be necessary for effective management of coastal resources, but the changing distribution and abundance of these species through time and space may provide strong evidence of the extent of human impacts on algal populations in general.

IV. FISHERIES AND FOOD WEBS

1. General Background

As biotoxins move up through marine food webs, they can have a broad spectrum of effects on marine animals in inshore, offshore, pelagic, and benthic habitats (Table 2). The scope of these effects, resulting from both chronic and acute exposure to the toxins, has become more evident in recent years (Anderson and White, 1989; White, 1980, 1988; White, in press; White et al., 1989). A wide variety of animals can accumulate biotoxins and act as intermediate vectors to consumers at higher trophic levels. Certain groups of animals, as direct consumers of microalgae, have received primary attention with regard to specific biotoxins. The best-known examples are filter-feeding bivalve molluscs as vectors for PSP, NSP, DSP, and ASP (Shumway, 1990). Phycotoxins are, however, increasingly being detected in a wide range of marine animals, such as gastropod molluscs, zooplankton, planktivorous fish, benthic crustaceans, sea birds and marine mammals (Quayle, 1969; Halstead, 1978; White, 1981b; Smayda, 1992).

Marine fish and shellfish kills caused by harmful algae may have significant economic impacts on coastal communities through lost recreational and commercial fishing revenues and adverse aesthetic effects on tourism (e.g., fish kills in Florida and the southeastern United States) and decimation of bay scallop stocks and reduction of eelgrass nursery habitat by brown tides in New York; Cosper et al., 1987). Harmful algae also may have direct (non-food chain) and catastrophic economic effects on finfish aquaculture. There is no uniform recording or reporting of fish kills, but the frequency of these events may be increasing.

Coastal waters in the United States harbor a number of harmful phytoplankton species that could cause, or already may have caused, massive fish and shellfish mortality, judging from recent events in other parts of the world. For example, *Chatonella antiqua*, various silico-flagellates, and *Prymnesium* spp. are present here but have not been documented to cause the fish and shellfish kills and other mortality events seen elsewhere in the world. Other toxic species remain to be identified, such as an unusual dinoflagellate species responsible for a number of fish kills in North Carolina (Burkholder et al., 1992). It is possible, even likely, that this dinoflagellate has caused fish kills in all of the mid-Atlantic states for decades or more.

It is known that biotoxin conversions (e.g., saxitoxin in butter clams) and magnification (e.g., ciguatera) during food-chain transfers can occur and may be important in understanding the fate of phycotoxins in the marine environment, although these processes are poorly understood (Shimizu, 1987). Shellfish differ markedly in their physiological responses, and in their ability to accumulate, metabolize, and eliminate various biotoxins (Shumway, 1990; Shumway and Cucci, 1987). Therefore, information obtained for one species is not necessarily applicable to others.

TABLE 2

ALGAL SPECIES WHICH POSE A THREAT TO FINFISH, SHELLFISH AND WILDLIFE IN NORTH AMERICA

Harmful Algal Species	Geographic Area	Affected Organisms *
<i>Alexandrium</i> spp. (PSP)	Northern Atlantic and Pacific Coast of North America	Mussels, surfclams, softshell clams, sea scallops, butterclams, ocean quahogs, oysters, gastropods, lobsters, crabs Herring, salmon, menhaden, sandlance, mackerel and possible other fish species. Whales, sea lions*, sea otters*, sea birds Squid, zooplankton, and other benthic invertebrates
<i>Alexandrium monilata</i>	Gulf of Mexico	Oysters, coquinas, mussels, gastropods, fish
<i>Pseudonitzschia pungens</i> f. <i>multiseries</i> (ASP)	Gulf of Maine; eastern Canada, Puget Sound, WA	Mussels
<i>P. pseudodelicatissima</i> (ASP)	New Brunswick, Canada	Mussels
<i>P. australis</i> (ASP)	California	Anchovies, sea birds
Probably <i>P. australis</i> (ASP)	Washington, Oregon	Razorclams*, Dungeness crabs*
Unidentified (ASP)	Massachusetts	Bay scallops*
	Maine	Sea scallops*
<i>Dinophysis</i> spp. (DSP)	Nova Scotia, Gulf of St. Lawrence, Canada	Mussels*
<i>Prorocentrum lima</i> (DSP)	Nova Scotia, Canada	Mussels*
<i>Prorocentrum</i> spp.	Long Island Sound	Northern quahogs, bay scallops
<i>Gyrodinium aureolum</i>	Northern New England (Maine)	Mussels, softshell clams*
<i>Aureococcus anophagefferens</i>	New York, Rhode Island, New Jersey	Bay scallops, mussels <i>Anchoa</i> sp., cladocerans
<i>Gymnodinium breve</i> (NSP)	Gulf of Mexico, South Atlantic Bight	Bay scallops, surfclams, oysters, southern quahogs, coquinas. Tunicates Many commercial and recreational species of fish. Sea birds*, sea turtles, manatees*, dolphins*
<i>Chaetoceros</i> spp.	Pacific northwest	Salmon aquaculture, possibly other species
<i>Heterosigma akashiwo</i>	Pacific northwest Narragansett Bay	Salmon aquaculture zooplankton
Unnamed gonyaulacoid	Mid-Atlantic region	Striped bass, flounder, croaker, mullet, menhaden, pinfish, sea trout, blue crabs, bay scallops
<i>Gambierdiscus toxicus</i> <i>Prorocentrum lima</i> * <i>P. concavum</i> * <i>P. hoffmannianum</i> * <i>Ostreopsis lenticularis</i> * <i>O. siamensis</i> *	South Florida, Florida Keys Puerto Rico, U.S. Virgin Islands Hawaii, Guam	Grouper, snapper, mackerel, jack, barracuda, parrot fish, tang, goat fish, and other finfish Gastropods

* Found to contain algal toxins, or be adversely affected by marine algae

* Causative algae implicated, not confirmed.

Marine mammals and wildlife including endangered species are also threatened by toxic algae. Over the past few years, PSP toxins transferred through mackerel have been implicated in the mass mortality of humpback whales in the Northeast (Geraci et al., 1989); domoic acid transferred through anchovies has been implicated in the death of brown pelicans and cormorants in the Southwest (Work et al., in press; Fritz et al., 1992) and brevetoxins possibly transferred through menhaden were implicated in the mass mortality of bottlenose dolphin in the southeast (Anderson and White, 1989). The transmission of dinoflagellate toxins through marine food chains can also have ecologically significant sub-lethal effects. PSP toxins sequestered by butter clams function as an effective chemical defense against important seabird, sea otter and fish predators, and may influence the distributions of these species (Kvitek and Beitler, 1991; Kvitek, in press). The ecological impact of dinoflagellate toxins in the marine food chain may therefore have profound consequences for conservation biology and our attempts to preserve and protect endangered species. The scope of this overall problem is unknown.

Algal blooms may have harmful effects not related to production of toxins, such as oxygen depletion of the water column (Ropes et al., 1979), fish suffocation from stimulation of gill mucus production, or mechanical interference with filter-feeding structures (Horner et al., 1990).

The *Fisheries and Food Webs* working group identified seven major impediments to progress in the biology and ecology of toxic shellfish; three impediments in the areas of fish kills and aquaculture of finfish; two in the area of the effects of toxins on the marine food web, and five in shellfish monitoring programs. The group recommended solutions to these impediments.

1.1. Shellfish: Impediments and Recommendations

IMPEDIMENT: *Available information on toxin kinetics (toxin uptake and detoxification/depuration) and anatomical distribution of toxins in shellfish is restricted and limited to a few bivalve species.*

RECOMMENDATION: Determine factors controlling accumulation and loss of toxins in commercially important inshore and offshore shellfish, including environmental factors, characteristics of the phytoplankton assemblage (e.g., relative abundance and toxicity of implicated algal species), and prior history of exposure to toxins.

Field studies relating bloom dynamics to shellfish toxicity patterns at the appropriate spatial and temporal scales are extremely rare. This information is necessary to identify potential aquaculture species which are less susceptible to accumulation and long-term retention of toxins, select suitable indicator species, and evaluate the potential for species-specific closures of shellfish harvesting grounds. These data will also allow optimization and streamlining of costly monitoring efforts (e.g., determination of optimum sampling frequency) and development of mitigation strategies. Field studies correlating phytoplankton and shellfish toxicities in combination with experimental toxification studies will allow unequivocal cause-effect linkage between shellfish toxicity episodes and their source.

Emphasis should be placed on:

- **Understanding reaction products and kinetics of metabolic transformations of toxins in shellfish tissues.**

Two- to three-year studies in which the history of toxication is well characterized are best suited to meet this objective. Results obtained in the laboratory should be compared with those documented in field populations where the source of toxin may be unknown or poorly characterized. Toxin conversions in shellfish tissues may increase public health risk. For example, low potency PSP toxins in algal cells are converted to more potent metabolic end products in some bivalve tissues.

- **Anatomical/cytochemical localization and transfer of toxins among tissues, especially when only some tissues are marketed, (e.g., in scallops, surf clams and razor clams).**
- **Characterization of magnitude and causes of variability in toxin accumulation within a population (e.g., in relation to body size, reproductive condition, or feeding zone), and among different species.**
- **Development of methods to enhance the rate of toxin depuration (detoxification), especially in species of high economic value that are characterized by prolonged toxin retention (e.g., surf clams, butter clams and sea scallops).**

Treatment of toxic shellfish should involve relatively short time scales compatible with industry needs. Treatment methods include manipulation of natural environmental variables (e.g., temperature), development of food processing technology, or artificial methods such as treatment with ozone.

- **Determine the relationship between algal population dynamics and seasonal and spatial patterns of toxicity in shellfish populations (e.g., how do vertical distribution of algal cells and benthic re-suspension affect toxin transfer?).**

This requires high-frequency sampling of shellfish stocks, phytoplankton populations and hydrographic features at selected field sites that are readily accessible and where toxic/noxious blooms are known to be a recurrent problem. Inter-annual variability should be determined.

- **Develop predictive models of toxin kinetics (uptake and depuration by shellfish) based on integration of field and laboratory studies.**

Simple bioenergetics-based models have been used previously to describe and predict the accumulation of anthropogenic contaminants in aquatic systems. Modeling efforts should be especially useful in identifying the likely source and history of shellfish intoxication in areas where extensive phytoplankton monitoring is unavailable or impractical. Predicting the risk of contamination in areas as yet unaffected by shellfish toxicity episodes, but where the presence

of toxic algae has already been documented, may also be possible. Models will likely aid in the design of an optimum sampling schedule for the monitoring of a particular shellfish resource.

IMPEDIMENT: *Information is lacking on the relative sensitivities of different life history stages to harmful algae, and on the long-term effects of algal toxins/metabolites on growth, reproductive success and recruitment of shellfish populations. Past work has largely focused on adult stages of a few species and on short-term effects on individuals.*

RECOMMENDATION: **Assess the short- and especially long-term concentration-dependent chronic effects of harmful algae on various life history stages of shellfish (e.g., larvae, juveniles, adults). Determine the mode of action and effects of toxins on these developmental stages at both organismal and population levels.**

Blooms of harmful algae may exert sublethal effects on shellfish populations, and thus affect long-term persistence as well as harvestable yields of the resource. For example, toxic *Alexandrium* cells (Shumway and Cucci, 1987; Bricelj et al., 1991) and direct contact with *Aureococcus anophagefferens* cells (Tracey, 1988) can significantly inhibit feeding activity in some bivalve species. Field and laboratory studies should assess the relevance of such transient physiological effects on population fitness traits (e.g., growth rates and reproductive performance), and identify the most critical developmental stages affected. Studies involving natural populations will depend on bloom incidence in the field. Mitigation strategies such as transplanting of stocks to unaffected areas during a harmful bloom, or modification of culturing practices and schedules (e.g., early or delayed planting of seed), and stock rehabilitation efforts following a toxic episode could thus be designed to minimize adverse effects. The time frame for such studies will depend on the lifespan and growth rate of the species or developmental stage under consideration, but useful data could be provided within 2-3 years.

IMPEDIMENT: *The identity, mode of action, and species-specific impacts of toxins/metabolites associated with previously unimplicated harmful algae (e.g., *Aureococcus anophagefferens*, and *Gyrodinium aureolum*) have not been clearly established.*

RECOMMENDATION: **Identify the potentially noxious bioactive compounds associated with these algae, determine their mode of action on shellfish, and develop sensitive bioassays for their rapid detection.**

Blooms of *Aureococcus anophagefferens* have recurred in New York waters since 1985, and were also documented in RI and NJ waters in 1985 (Cosper et al., 1989). Blooms of a related picoplanktonic alga recently occurred in Texas (Stockwell et al., in press). *Aureococcus* caused weight loss of adult bay scallops and mortality of adult mussels and recruitment failure of bay scallops (Tracey, 1988; Bricelj and Kuenstner, 1989), but only anecdotal information is available for the effects on other commercially important bivalves, such as the American oyster and hard clam. Less susceptible species might provide a viable aquaculture alternative during brown tide episodes.

Gyrodinium aureolum, a species with ichthyotoxic properties, has been shown to cause mortalities in a number of bivalves (scallops, oysters and mussels) in Europe (Tangen, 1977; Partensky and Sournia, 1986) and was recently considered responsible for mass shellfish mortalities in Maquoit Bay, ME (Heinig and Campbell, 1992). Preliminary studies have shown deleterious effects of this alga on feeding of nine juvenile species, and mortality in some species (Shumway, unpublished data).

Medium-term (3 years) laboratory studies are required to determine the effects of these algae on a broad range of species. Such studies should verify the existence of a concentration threshold below which no adverse effects are observed. Understanding of concentration-dependent effects will help to develop mitigation strategies (e.g., site selection for aquaculture ventures). Bioassays could be developed over the short-term (2 years), before chemical characterization of bioactive compounds has been achieved, and should determine if these active compounds are extracellular and/or intracellular.

IMPEDIMENT: *An extensive historical database on accumulation and depuration of PSP toxins has been collected by state and federal monitoring agencies, but this "grey" literature is scattered and not readily accessible to potential users.*

RECOMMENDATION: Compile, integrate and interpret existing data in order to further elucidate general patterns of toxification/detoxification in commercially important shellfish on a regional and national basis.

IMPEDIMENT: *Availability of isolates of toxic/noxious algae is limited. These are essential for physiological studies on effects and mode of action of toxins.*

RECOMMENDATION: Establish additional cultures of algal isolates/clones, and develop culture techniques where these are not available (e.g., *Dinophysis* spp.).

For some species of harmful algae that affect the United States, only single cultures are available (e.g., *Aureococcus anophagefferens*). This severely biases laboratory studies towards isolates that may not be representative of the natural populations. Efforts to culture the DSP-producing *Dinophysis* spp. have so far been unsuccessful. Continued research on *Dinophysis* species is highly desirable, but does not warrant a major investment of funds until DSP is shown to be a real rather than a perceived problem to U.S. shellfish.

IMPEDIMENT: *Rapid, reliable methods for field-testing of shellfish are lacking. Standards for quantification of toxins are limited and often unavailable. Analytical methods for detection and quantification of toxins in animal tissue (e.g., PSP and DSP toxins) need improvement. Lack of radiolabeled toxic compounds limits the scope of laboratory studies on toxin transfer in shellfish.*

RECOMMENDATION: Provide low cost, certified toxin standards; develop and test rapid methods for *in situ* detection of toxins; improve analytical methods and provide radiolabeled toxins for quantification of toxins in shellfish tissues.

1.2. Finfish: Impediments and Recommendations

IMPEDIMENT: *The physiological responses of fish exposed to toxic and other marine harmful algae are poorly known.*

RECOMMENDATION: Complete the identification of known or suspected fish-killing algal species and their toxins or harmful metabolites. Establish the mechanisms underlying algal-caused fish kills, the routes of toxin delivery, and the varying effects on different life history stages of fish. Develop standardized laboratory bioassay techniques for key fish species. Examine bioaccumulation of toxins and possible physiological feedback mechanisms between fish and phytoplankton.

Although fish exposed to harmful algae are known to die of respiratory failure in some cases, the underlying causes and physiological mechanisms may vary among species or are unknown.

Severe economic damage is caused by several species of harmful algae, although the routes of toxin delivery and physiological reactions of fish are poorly understood. Modes of toxin production and ichthyotoxic action should be studied because human consumers of fish may be at risk if toxin accumulates in an unpredictable fashion in fish muscle tissue. This limits the ability to mitigate the problem for aquaculture and to understand and predict the consequences for wild fish stocks. There may be multiple causes of fish death due to harmful phytoplankton, and their relative contribution is difficult to detect or predict. For example, the raphidophyte microflagellate *Heterosigma akashiwo* (found on both coasts of the United States) and related species (*Chatonella marina* in Japan) may suffocate fish due to massive mucus production by the gills, by neurotoxin suppression of respiration, or by destruction of blood components (Onoue, 1990; Chang et al., 1990; Black et al., 1991; MacKenzie, 1991; Rensel Associates and PTI Environmental Services, 1991). Tools and methods of investigation are generally available and the above recommendations could be achieved in 5 to 7 years.

IMPEDIMENT: *Harmful phytoplankton blooms are a major impediment to the operation and development of marine finfish aquaculture.*

RECOMMENDATION: Develop more effective methods of mitigating the effects of harmful algal blooms on finfish aquaculture.

Catastrophic losses of aquaculture fish have occurred in the United States in recent years due to species of harmful phytoplankton previously not recognized as toxic or present (Horner et al., 1990). Monitoring by fish farmers in coastal waters provides a valuable link and permanent sampling platforms for assessing the frequency and trends of harmful blooms. Mitigation based on physical movement of water into net-pens or oxygenation is feasible and already practiced in some cases, but once the underlying causes of fish mortality are known, other types of mitigation

will be possible. Such strategies are necessary for harmful algal species that often occur throughout the water column or are difficult to detect in aerial or boat surveys. The time lines involved in developing effective mitigation techniques are short-term (2-4 years), depending on the species involved.

IMPEDIMENT: *Investigators of fish-kills involving algal blooms often lack training, specialized equipment, and communication networks needed to detect, investigate, and determine causes.*

RECOMMENDATION: Promote and expand communication among aquatic user groups, resource agencies, and specialists. Develop field sampling protocols and streamlined physiological or pathological assays to determine which algal toxins or species were responsible for fish losses. Establish systematic reporting and data base management of marine fish kill data.

Marine user group and resource agency personnel are generally not experienced or trained regarding harmful marine phytoplankton blooms. Fish kills often occur quickly, before agency or research personnel are able to react. As a result, little knowledge about the causes of algal-related fish kills has been gained. Although fish kills may be increasing in frequency, determination of actual trends is impossible at present. Many resource agencies will investigate fish kills first for anoxic/hypoxic water conditions, or will suspect diseases, chemical spills or pollution before searching for other causes. Phytoplankton sampling and/or testing for toxin content is often a low priority or is not attempted. Development of handbooks similar to those used to investigate fish kills in freshwater (e.g., Meyer and Barclay, 1990) will allow agency staff or volunteers to collect fish tissues in a proper manner for analyses. Fostering communication between university or federal experts and aquatic user groups (fishermen, aquaculturists) will expedite reporting and accurate assessment of the causes and extent of fish kills. This is a short-term effort that will require periodic review.

1.3. Food Web Effects: Impediments and Recommendations

IMPEDIMENT: *Investigative responses to kills of marine animals are often ineffective.*

RECOMMENDATION: Establish rapid response research capability and associated geographic information system (GIS) data base.

Present studies of sudden toxic bloom events and resulting contamination and kills of marine biota are not pre-planned, are poorly coordinated, and often take place too long after the event to be useful. Thus, determination of the causative organism(s), the routes of toxin transfer, the nature of the effects on marine animals, and the risks to public health is difficult or impossible. As such, these responses are usually reactive, not proactive. By the time stricken animals arrive at analytical facilities, toxins may no longer be present at detectable levels. Infrastructure and funding should be provided to enable the investigation of toxic and noxious blooms at their peaks in terms of toxin assays of plankton, intermediate vectors, fish, and other wildlife. Selected

species should be assayed over time to determine duration of toxin retention and changes in toxin profiles. Professionals in the fields of wildlife veterinary medicine and biology, and toxin analysis, should be trained in sampling protocols aimed specifically at marine biotoxins. Regional rapid response teams should be coordinated with the simultaneous efforts of similar teams investigating blooms and the origin and fate of the toxins (see below). All results could be stored as overlays in a GIS data base. This type of archive and retrieval system offers the most effective means of linking spatially-related data and testing hypotheses about the importance of oceanographic processes, land-use practices, and environmental factors to harmful algal bloom dynamics and food web effects.

IMPEDIMENT: Risks of biotoxins to marine animals or to public health resulting from the movement of toxins through the marine food web chain currently cannot be addressed.

RECOMMENDATION: Develop quantitative models of the fate and consequences of biotoxins in the marine food web.

Important pathways of toxin transmission have not been identified, nor has toxin accumulation potential and sensitivity been determined for many key marine species (i.e., species that are either commercially important, endangered, or serve as major food web links). The information necessary for development of these models could be obtained primarily by multi-disciplinary, rapid response groups investigating bloom dynamics and toxin transmission in the food web, coordinated with other supporting studies (e.g., laboratory and field feeding and observational studies).

2. Shellfish Monitoring Programs

2.1. Background

In the United States, the monitoring of marine biotoxins and/or associated phytoplankton in seafood and the environment has been primarily the responsibility of state-sponsored programs. The relative success of these programs can be measured by the lack of overt public health morbidity or mortality from consuming contaminated seafood (Ahmed, 1991; Bean et al., 1990). However, the sampling programs of individual states vary in magnitude (Table 3), dependent upon the degree of seafood production or import, commitment of resources, and political will.

The responsibility of the state programs can extend to products from international waters and interstate imports. These programs can implement the closure of a fishery based upon existing action limits for only two marine biotoxins, paralytic shellfish toxin and amnesic shellfish toxin, or they can exercise other policy considerations such as the lack of sufficient information about a fishery. Presently, closure limits based on the presence of other marine biotoxins have not been established. When few monitoring stations are employed to cover large coastal areas and the frequency of sampling is relatively low, program officials close large fishery areas as a conservative measure to protect the consumer and consumer confidence in the seafood industry. This is the case especially when health officials attempt to deal with toxic effects from an

TABLE 3

1989 STATE PSP MONITORING STATIONS

STATE	NUMBER OF SAMPLES (NO. STATIONS)
Maine	3500 (200)
Washington	1900 (50)
California	1100 (not fixed)
Oregon	780 (16)
Massachusetts	700 (98)
Alaska	654 (40)
Connecticut	51 (5)
Rhode Island	40 (8)
New Hampshire	34 (1)

unidentified toxic substance. Monitoring is not only important in the closure process, but is critical for establishing re-openings.

International imports are subject to routine inspection by United States Customs and can be embargoed if found to be contaminated. On the other hand, international exportation of seafood products from the United States are subject to the seafood importation standards at the point of destination.

The collection of a sample can be accomplished in many ways. Sub-samples can either be taken by program personnel, delivered from commercial harvests at the point of landing, or before delivery to market. State programs support "sentinel" species programs whereby specific or mixed species of molluscs (e.g., mussels for PSP) are strategically placed along coastal areas that are subject to harvesting activity. These sites are sampled regularly to provide an early warning of the presence of toxins. The relative effectiveness of a sentinel program can be measured by the number of sampling sites per area covered and the frequency of sampling. In addition, with few exceptions, sentinel sites typically are positioned near-shore and do not provide information about offshore toxin distribution. In practically all cases, the analyses for marine biotoxins are carried out by state or federal laboratories, the exception being the availability of uncertified commercial laboratories that conduct analyses for domoic acid.

Within state programs, however, the responsibility of seafood monitoring can be decentralized, with different programs charged with the management of different seafood industries (e.g.,

shellfish industry, finfish industry, crab industry, etc.). The specific mandates of each of these sub-programs may present a narrow focus to a particular biotoxin problem, depending upon the region and indigenous biotoxins present. While focused programs may provide adequate protection to the consumer, these efforts can be inflexible when attempting to respond to the presence of newly-recognized biotoxin threats. Communications between state programs and interagency entities can at times be tenuous, even during crises.

Ideally, these efforts are designed to lead to a proactive response. That is, the toxins are detected within the seafood prior to commercial sale of product. In practice, this may be viewed as reactive, since a toxic sentinel site or product is already contaminated and the monitoring effort is not 100% for all seafood (nor should it be). The State of Florida exercises a "preemptive" monitoring program, which combines seafood and phytoplankton monitoring with aspects of remote sensing to recognize algal blooms prior to their impact on fisheries.

2.2. Impediments and Recommendations

IMPEDIMENT: *Current monitoring efforts are too limited and inflexible to measure the full impact of marine biotoxins. The extent of the distribution of marine biotoxins in consumed seafood products is not known.*

RECOMMENDATION: In those instances where newly-recognized toxins impact a fishery, state and federal resources should be made available to aid monitoring programs. Various seafood products should be surveyed on a region-by-region basis for the most important biotoxins and the products affected.

Existing routine monitoring programs are, by design, limited to managing specific resources. On those occasions where marine biotoxin crises arise, routine state and federal funding mechanisms are inadequate to address the immediate concerns. This places a huge burden on state and federal agencies to redirect resources from predetermined programs to deal with the crisis. Contingency funds and a mechanism for rapid deployment are needed to help state and federal programs respond to crises such as toxic plankton blooms, fish kills, and marine mammal kills. Time is of the essence if these efforts are to be effective.

All coastal areas contain various types of marine life and biotoxins. The utilization of existing biotoxin monitoring elements to address all biotoxin concerns may not be appropriate. Therefore, the search for affected seafood products and additional biotoxins will be an evolving process. The ultimate goal of this effort will be to create more efficient and cost-effective programs.

IMPEDIMENT: *The public health community and seafood industry require early warning of toxic/harmful phytoplankton blooms to protect seafood consumers and producers.*

RECOMMENDATION: Identify the best indicator species within specific regions. Identify the best sites and sampling strategies for these indicator species.

Presently, bivalve molluscs (e.g., mussels, oysters or clams) are employed as the primary sentinel organisms. These species may or may not reflect accurately the true nature of a toxic algal bloom. For example, mussels have been employed as sentinel organisms for the detection of domoic acid (Haya et al., 1991). They may not, however, always be the best predictor of the presence of domoic acid, as was shown on the West Coast when razor clams were toxic but mussels were not (Wood and Shapiro, 1992). A primary sentinel species may, in fact, be a non-consumed species with characteristics of toxin uptake, distribution, and retention that provide useful data for particular toxins. Further studies on a regional basis are required to determine which species best accommodate specific biotoxins and analyses. These studies should be conducted locally to account for regional variations and should identify species with both long- and short-term retention times. These studies would take 2-3 years for completion.

Monitoring programs are only as good as the analytical support they receive. Present laboratory support is limited by the number of facilities and methods currently available. In addition, in those instances where newly-recognized toxins are found, a full understanding and characterization of the toxin must be obtained before an appropriate sentinel organism can be identified.

In order to identify sites and sampling strategies, models that describe temporal and spatial distributions of toxins must be developed. The presence of biotoxins in marine life is subject to the effects of regional hydrographic conditions. In order to develop the model and account for the environmental conditions, the distribution of toxins among sites and individual organisms must be evaluated statistically and include both nearshore and offshore locations.

IMPEDIMENT: *The collection, preservation, and handling of naturally-occurring toxic seafood and algal samples for use by researchers and public health officials is hampered by a lack of standardized procedures.*

RECOMMENDATION: **Develop internationally accepted and appropriate collection, preservation, and shipping protocols.**

With the lack of standardized handling and shipping methods, the implementation of public health measures is hampered. Furthermore, the development and refinement of methods for biotoxin detection are also impeded. In particular, it is necessary to understand the fate and stability of biotoxins in natural seafood matrices. Currently, the methods used for the collection and handling of contaminated seafood are not standardized. It is not known how these conditions affect toxin stability. If sample integrity is compromised, analytical results are questionable. This could be easily tested by conducting spiking studies or splitting contaminated samples prior to shipment. The initial method development and subsequent collaborative studies could be completed within 2-3 years.

Due to increasing interest and awareness of marine toxins and algal blooms on the part of the general public, scientists, and public health officials, we can assume that in the future, new toxins or the extension of known toxins to new seafood items will be documented. As the public and

health professionals inevitably begin to associate illness with the consumption of a seafood product, there will be increasing demands to "explain the cause of the sickness". There is a need for the development of standardized, generic laboratory procedures, techniques, protocols, and surveys that could be compiled and be "ready-to-use" by local and state public health agencies to meet and deal with these inquiries.

IMPEDIMENT: *Slightly different policies for dealing with fishery closures due to a marine toxin outbreak can lead to industry problems in contiguous states sharing the coastline and the toxin problem.*

RECOMMENDATION: Encourage the states to form regional communication networks and harmonize risk management policies.

If one state closes a fishery while the others do not, processors and fishermen move from the affected state to the state where the fishery is still open. This places stress on the open industry and appears to place the closed state at a competitive disadvantage. In addition, if the toxin outbreak does impact several states, regulatory tracking of risk species becomes virtually impossible due to lengths of coastline, mobility of fishermen, and multi-state licensing of fishing boats. It is better if states sharing coastline and toxin risks develop a harmonized closure action plan.

IMPEDIMENT: *Authority for dealing with both known and new marine toxins occurring in seafood is sometime fragmented within state management agencies, leading to inter- and intra-agency jurisdictional overlap and confusion.*

RECOMMENDATION: Encourage the states to streamline programs dealing with marine toxin risks by placing monitoring and management control in a single regulatory agency.

In many states there is a fragmentation of authority between state agencies for action on a fishery due to the presence of a toxin. For example, in some states, authority is divided among the Departments of Agriculture (for crustacea and finfish), Health (molluscan shellfish), and Fisheries (resource closures). There may be a variety of good reasons for this fragmentation; nonetheless, when a toxin such as PSP is found in a "non-traditional" species such as Dungeness crab, it becomes difficult for the state to formulate and take appropriate action. States should be encouraged to streamline their programs in dealing with marine toxin risks, i.e., place monitoring and control in one agency.

V. SUMMARY

The preceding text lists numerous impediments to progress in the area of marine biotoxins and harmful algae and addresses each with a series of recommendations. The length of these lists defines the major challenge before us if the goal of the National Plan is to be realized. Some might conclude from the many impediments that past research has made little progress, but this is certainly not the case. The scientific and policy disciplines involved are healthy and maturing, though underfunded relative to the expanding problem.

The rate and extent of progress from here will depend in large part on how effectively the recommendations in this National Plan are implemented. Our hope is that numerous state and federal agencies will use this document to identify topics that relate to their particular responsibilities or purviews, and that scientists and private industry will use these ideas to guide their activities as well. No single agency can address all of the identified impediments, but most can be covered by the combined efforts of several organizations. Overlap and omissions are likely however, unless further coordination is attempted at the agency level. The network has been established to make this possible, but concerted efforts will be necessary to keep the lines of communication and coordination open.

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VII. REFERENCES

- Ahmed, F. E. (Ed.). 1991. *Seafood Safety*. National Academy Press, Washington, DC, 432 pp.
- Anderson, D. M. 1989. Toxic algal blooms and red tides: a global perspective. In: *Red Tides: Biology Environmental Science and Toxicology*, T. Okaichi, D. M. Anderson and T. Nemoto (Eds.), Elsevier, NY, pp. 11-16.
- Anderson, D. M. and A. W. White. 1989. *Toxic Dinoflagellates and Marine Mammal Mortalities*. Woods Hole Oceanographic Inst. Tech. Rept. WHOI-89-36 (CRC-89-6).
- Anderson, D. M., D. M. Kulis, J. A. Orphanos, and A. R. Ceurvels. 1982. Distribution of the toxic red tide dinoflagellate *Gonyaulax tamarensis* in the southern New England region. *Est. Coast. Shelf Sci.* **14**: 447-458.
- Baden, D. G., T. J. Mende, M. A. Poli, and R. E. Block. 1984. Toxins from Florida's red tide dinoflagellate, *Ptychodiscus brevis*. In: *Seafood Toxins*, E. Ragelis (Ed.), Amer. Chem. Soc. Symposium Series, Washington, D.C. pp. 359-367.
- Bates, S. S., C. J. Bird, A. S. W. de Freitas, R. Foxall, M. Gilgan, L. A. Hanic, G. R. Johnson, A. W. McCulloch, P. Odense, R. Pocklington, M. A. Quilliam, P. G. Sim, J. C. Smith, D. V. Subba Rao, E. C. D. Todd, J. A. Walter, and J. L. C. Wright. 1989. Pennate diatom *Nitzschia pungens* as the primary source of domoic acid, a toxin in shellfish from eastern Prince Edward Island, Canada. *Can. J. Fish. Aquat. Sci.* **46**: 1203-1215.
- Bean, N. H., P. M. Griffin, J. S. Goulding, and C. B. Ively. 1990. Foodborne disease outbreaks, 5-year summary, 1983-1987. *MMWR* **39** SS-1, 15-57.
- Black, E. A., J. N. C. Whyte, J. W. Bagshaw and N. G. Ginther. 1991. The effects of *Heterosigma akashiwo* on juvenile *Oncorhynchus tshawytscha* and its implications for fish culture. *J. Appl. Ichthyol.* **7**: 168-175.
- Bomber, J. W., D. R. Tindall, and D. M. Miller. 1989. Genetic variability in toxin production among seventeen clones of *Gambierdiscus toxicus* (Dinophyceae). *J. Phycol.* **25**: 617-625.
- Bricelj, V. M., J. H. Lee and A. D. Cembella. 1991. Influence of dinoflagellate cell toxicity on uptake and loss of paralytic shellfish toxins in the northern quahog *Mercenaria mercenaria*. *Mar. Ecol. Prog. Ser.* **74**: 33-46.
- Bricelj, V. M. and S. Kuenstner. 1989. Effects of the "brown tide" on the feeding, physiology and growth of juvenile and adult bay scallops and mussels. In: *Novel Phytoplankton Blooms: Causes and Impacts of Recurrent Brown Tides and Other Unusual Blooms*, E. M. Cosper, V. M. Bricelj, and E. J. Carpenter (Eds.), Springer-Verlag, Berlin, pp. 491-509.

Buck, K. R., L. Uttal-Cooke, C. H. Pilskaln, D. L. Roelke, M. C. Villac, G. A. Fryxell, L. Cifuentes and F. P. Chavez. 1992. Autoecology of *Pseudonitzschia australis* Frenguelli, a suspected domoic acid producer, from Monterey Bay, California. *Mar. Ecol. Prog. Ser.* **84**: 293-302.

Burkholder, J. M., E. J. Noga, C. H. Hobbs, and H. B. Glasgow Jr. 1992. New "phantom" dinoflagellate is the causative agent of major estuarine fish kills. *Nature* **358**: 407-410.

Cembella, A. D., J. J. Sullivan, G. L. Boyer, F. J. R. Taylor, and R. J. Anderson. 1987. Variation in paralytic shellfish toxin composition within the *Protogonyaulax tamarensis/catenalla* species complex: red tide dinoflagellates. *Biochem. System. Ecol.* **15**: 171-186.

Chang, F. H., C. Anderson and N. C. Boustead. 1990. First record of a *Heterosigma* (Raphidophyceae) bloom and associated mortality of cage-reared salmon in Big Glory Bay, New Zealand. *N.Z. J. Mar. Freshw. Res.* **24**: 461-469.

Cosper, E. M., V. M. Bricelj, and E. J. Carpenter, (eds.). 1989. *Novel Phytoplankton Blooms: Causes and Effects of Recurrent Brown Tides and Other Unusual Blooms*. Springer-Verlag, Berlin, 799 pp.

Cosper, E. M., W. C. Dennison, E. G. Carpenter, V. M. Bricelji, Y. G. Mitchell and S. H. Kuenstner. 1987. Recurrent and persistent brown tide blooms perturb coastal marine ecosystems. *Estuaries* **10**: 284-290.

Fritz, L., M. A. Quilliam, J. L. C. Wright, A. M. Beale, and T. M. Work. 1992. An outbreak of domoic acid poisoning attributed to the pennate diatom *Pseudonitzschia australis*. *J. Phycol.* **28**: 439-442.

Garrison, D. L., S. M. Conrad, P. P. Eilers and E. M. Waldron. 1992. Confirmation of domoic acid production by *Pseudonitzschia australis* (Bacillariophyceae) cultures. *J. Phycol.* October.

Geraci, J. A., D. M. Anderson, R. J. Timperi, D. J. St. Aubin, G. A. Early, J. A. Prescott, and C. A. Mayo. 1989. Humpback whales (*Megaptera novaeangliae*) fatally poisoned by dinoflagellate toxin. *Can. J. Fish. Aquat. Sci.* **46**: 1895-1898.

Hall, S. and G. Strichartz (eds). 1990. *Marine Toxins: Origin, Structure, and Molecular Pharmacology*. American Chem. Soc. Symposium Series, Washington, DC, 377 pp.

Halstead, B. W. 1978. *Poisonous and Venomous Marine Animals of the World*. Darwin Press, Inc., Princeton, NY.

Haya, K., J. L. Martin, L. E. Burrige, B. A. Waiwood and D. J. Wildish. 1981. Domoic acid in shellfish and plankton from the Bay of Fundy, New Brunswick, Canada. *J. Shellfish Res.* **10**: 113-118.

- Hayhome, B. A., D. M. Anderson, D. M. Kulis, and D. J. Whitten. 1989. Variation among congeneric dinoflagellates from the northeastern United States and Canada. I. Enzyme electrophoresis. *Mar. Biol.* **101**: 427-435.
- Heinig, C. S. and D. E. Campbell. 1992. The environmental context of a *Gyrodinium aureolum* bloom and shellfish kill in Maquoit Bay, Maine. September 1988. *J. Shell. Res.* **11**: 111-122.
- Horner, R. A. and J. R. Postel. Toxic diatoms in western Washington waters. In: H. Van Dam (Ed.), *Proceedings of the 12th International Diatom Symposium*. In press.
- Horner, R. A., J. R. Postel, and J. E. Rensel. 1990. Noxious phytoplankton blooms in western Washington waters: a review. In: *Toxic Marine Phytoplankton*, E. Graneli, B. Sundstrom, L. Edler, and D. M. Anderson (Eds.), Elsevier, New York, pp. 171-176.
- Juranovic, L. R. and D. L. Park. 1991. Foodborne toxins of marine origin: ciguatera. *Rev. Environ. Toxicol.* **117**: 51-94.
- Keafer, B.A. and Anderson, D.M. In press. Use of remotely-sensed sea surface temperatures in studies of *Alexandrium tamarens* bloom dynamics. In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds), Proc. 5th Internat. Conf., Elsevier, Amsterdam.
- Kodama, M. 1990. Possible links between bacteria and toxin production in algal blooms. In: *Toxic Marine Phytoplankton*, Graneli, E., Sundstrom, B., Edler, L. and Anderson, D.M. (Eds.), Elsevier, New York, p. 52-61.
- Kvitek, R. G. In press. Paralytic shellfish toxins as a chemical defense in the butter clam (*Saxidomus giganteus*). In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds), Proc. 5th Internat. Conf., Elsevier, Amsterdam.
- Kvitek, R. G. and Beitler, M. K. 1991. Relative insensitivity of butter clam neurons to saxitoxin: a pre-adaptation for sequestering paralytic shellfish poisoning toxins as a chemical defense. *Mar. Ecol. Prog. Ser.* **69**: 47-54.
- MacKenzie, L. 1991. Toxic and noxious phytoplankton in Big Glory Bay, Stewart Island, New Zealand. *J. Appl. Phycol.* **3**: 19-34.
- Mahoney, Y. B., P. Olsen and M. Cohn. 1990. Blooms of a dinoflagellate *Gyrodinium* cf. *aureolum* in New Jersey coastal waters and their occurrence and effects worldwide. *J. Coast. Res.* **6**: 121-135.
- Meyer, F. P. and L. A. Barclay. 1990. *Field Manual for the Investigation of Fish Kills*. U.S. Fish and Wildlife Service Resource Publication 177. 120 pp.

- Murata, M., A. M. Legrand, Y. Ishibashi and T. Yasumoto. 1989. Structures of ciguatoxin and its congener. *J. Am. Chem. Soc.* **111**: 8929-8931.
- Nishitani, L. and K. K. Chew. 1988. PSP toxins in the Pacific Coast states: Monitoring programs and effects on bivalve industries. *J. Shell. Res.* **7**: 653-669.
- Onoue, Y., M. S. Haq and K. Nozawa. 1990. Separation of neurotoxins from *Chattonella marina*. *Nippon Suisan Gakkaishi* **56**: 695.
- Partensky, F. and A. Sournia. 1986. Le dinoflagellé *Gyrodinium cf. aureolum* dans le plancton de l'Atlantique nord: identification, écologie, toxicité. *Crypt. Algal.* **7**: 251-275.
- Pocklington, R., J. E. Milley, S. S. Bates, C. J. Bird, A. S. W. de Freitas and M. A. Quilliam. 1990. Trace determination of domoic acid in sea water and phytoplankton by high performance liquid chromatography of the fluorenylmethoxycarbonyl (Fmoc) derivative. *Intern. J. Environ. Anal. Chem.* **38**: 351-368.
- Price, D. W. and K. W. Kizer. 1990. California's paralytic shellfish poisoning prevention program, 1927-89. Calif. Dept. Health Serv. 36 p. (Unpublished Report)
- Quayle, D. B. 1969. Paralytic shellfish poisoning in British Columbia. *Fish. Res. Bd. Canada, Bull.* 168
- Quilliam, M. A., M. W. Gilgan, S. Pleasance, A. S. W. deFreitas, D. Douglas, L. Fritz, T. Hu, J. C. Marr, C. Smyth, and J. L. C. Wright. Submitted ms. Confirmation of an incident of Diarrhetic Shellfish Poisoning in eastern Canada.
- Quilliam, M. A., M. Xie and W. R. Hardstaff. 1991. A rapid extraction and clean-up procedure for the determination of domoic acid in tissue samples. *Inst. Mar. Biosci. Tech. Rpt.* 64 (NRCC No. 33001), 52 pp.
- Ragelis, E. P. 1984. Ciguatera seafood poisoning: Overview. In: *Seafood Toxins*, E. P. Ragelis (Ed.), Amer. Chem. Soc. Symp. Ser. No. 262, Washington, D.C., pp. 25-36.
- Rensel Associates and PTI Environmental Services. 1991. *Nutrients and Phytoplankton in Puget Sound*. Prepared for U.S. EPA, Region 10, Office of Coastal Waters. Report 910/9-91-002, Seattle, 130 pp.
- Rensel, J. E. In press. Severe blood hypoxia of Atlantic salmon exposed to the marine diatom *Chaetoceros concavicornis*. In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds), Proc. 5th Internat. Conf., Elsevier, Amsterdam.

- Roper, J. W., A. S. Merrill, S. A. Hurawski, S. Chang and C. L. MacKenzie, Jr. 1979. Impact on clams and scallops. In: *Oxygen Depletion and Associated Benthic Mortalities in New York Bight, 1976*. NOAA Prof. Paper 11, Rockville, MD, pp. 263-275.
- Shimizu, Y. 1984. Paralytic shellfish poisons. In: *Progress in the Chemistry of Organic Natural Products*, Vol. 45, W. Herz, H. Grisebach and G. W. Kirby (Eds.), Springer-Verlag, Wien, pp. 231-264.
- Shimizu, Y. 1987. Dinoflagellate toxins. In: *Biology of Dinoflagellates*, F. J. R. Taylor (Ed.), pp. 282-315.
- Shimizu, Y., H. N. Chou, and H. Bando. 1986. Structure of brevetoxin A (GB-1 toxin), the most potent toxin in the Florida red tide organism *Gymnodinium breve* (*Ptychodiscus brevis*). *J. Am. Chem. Soc.* **108**: 524-525.
- Shumway, S. E. 1990. A review of the effects of algal blooms on shellfish and aquaculture. *J. World Aquacult. Soc.* **21**: 65-104.
- Shumway, S. E. and T. L. Cucci. 1987. The effects of the toxic dinoflagellate *Protogonyaulax tamarensis* on the feeding and behavior of bivalve molluscs. *Aquat. Toxicol.* **10**: 9-24.
- Shumway, S. E. and A. D. Cembella. The impact of toxic algae on scallop culture and fisheries. *Reviews in Fisheries Science*. In Press.
- Sieburth, J. McN., P. W. Johnson, and P. E. Hargraves. 1988. Ultrastructure and ecology of *Aureococcus anophagefferens* gen. et sp. nov. (Chrysophyceae): the dominant picoplankton during a bloom in Narragansett Bay. *J. Phycol.* **24**: 416-425.
- Smayda, T. 1990. Novel and nuisance phytoplankton blooms in the sea: Evidence for a global epidemic. In: *Toxic Marine Phytoplankton*, Graneli, E., Sundstrom, B., Edler, L. and Anderson, D.M. (Eds.), Elsevier, New York. p 29-40.
- Smayda, T. 1992. Global epidemic of noxious phytoplankton blooms and food chain consequences in large ecosystems. In: *Food Chains, Yields, Models, and Management of Large ecosystems*, K. L. M. A. Sherman and B. D. Gold (Eds.), Westview Press, Boulder, CO, pp. 275-307.
- Stockwell, D. A., E. J. Buskey, and T. E. Whitedge. In press. Studies on conditions conducive to the development and maintenance of a persistent "brown tide" in the Laguna Madra, Texas. In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds), Proc. 5th Internat. Conf., Elsevier, Amsterdam.
- Tester, P. A., R. P. Stumpf, F. M. Vukovich, P. K. Fowler, and J. T. Turner. 1991. An expatriate red tide bloom: transport, distribution, and persistence. *Limnol. Oceanogr.* **36**: 1953-1961.

Tracey, G. A. 1988. Feeding reduction, reproductive failure and mass mortality of mussels (*Mytilus edulis*) during the 1985 'brown tide' in Narragansett Bay, RI. *Mar. Ecol. Progr. Ser.* **50**: 73-81.

Villac, M. C., D. L. Roelke, T. A. Villareal and G. A. Fryxell. Comparison of two domoic acid producing diatoms. H. Van Dam (Ed.), *Proceedings of the 12th International Diatom Symposium*. In press.

White, A. W. 1980. Recurrence of kills of Atlantic herring (*Clupea harengus harengus*) caused by dinoflagellate toxins transferred through herbivorous zooplankton. *Can. J. Fish. Aquat. Sci.* **37**: 2262-2265.

White, A. W. 1981a. Sensitivity of marine fishes to toxins from the red-tide dinoflagellate *Gonyaulax excavata* and implications for fish kills. *Mar. Biol.* **65**: 255-260.

White, A. W. 1981b. Marine zooplankton can accumulate and retain dinoflagellate toxins and cause fish kills. *Limnol. Oceanogr.* **26**: 103-109.

White, A. W. 1984. Paralytic shellfish toxins and finfish. In: *Seafood Toxins*, E. P. Ragelis (Ed.), American Chemical Symposium Series 262, ACS, Washington, DC, pp. 171-180.

White, A. W. 1988. Blooms of toxic algae worldwide: Their effects on fish farming and shellfish resources. In: *Proceedings of the International Conference on the Impact of Toxic Algae on Mariculture*, Aqua-Nor '87 Exhibition, Trondheim, Norway, 13-18 August 1987, pp. 9-14.

White, A. W., O. Fukuhara and M. Anraku. 1989. Mortality of marine fish larvae from eating toxic dinoflagellates or zooplankton containing dinoflagellate toxins. In: *Red Tides: Biology, Environmental Science, and Toxicology*, T. Okaichi, D. M. Anderson and T. Nemoto (Eds.), Elsevier, New York, pp. 395-398.

White, A. W. In press. Recent occurrence of paralytic shellfish toxins in offshore shellfish in the northeastern United States. In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds.), Proc. 5th Internat. Conf., Elsevier, Amsterdam.

Wood, A. M. and L. Shapiro (Eds.). 1992. Amnesic shellfish poisoning: a workshop report. Oregon Institute of Marine Biology, Newport OR. 52 pp.

Work, T. M., A. M. Beale, K. Fritz, M. A. Quilliam, M. Silver, K. Buck and J. L. C. Wright. In press. Domoic acid intoxication of brown pelicans (*Pelecanus occidentalis*) in California. In: *Toxic Marine Phytoplankton*, T. J. Smayda and Y. Shimizu (Eds.), Proc. 5th Internat. Conf., Elsevier, Amsterdam.

Wright, J. L. C., R. K. Boyd, A. S. W. De Freitas, M. Falk, R. A. Foxall, W. D. Jamieson, M. V. Laycock, A. W. McCulloch, A. G. McInness, P. Odense, V. P. Pathak, M. A. Quilliam, M. A. Ragan, P. G. Sim, P. Thibault, J. A. Walter, M. Gilgan, D. J. A. Richard and D. Dewar. 1989. Identification of domoic acid, a neuroexcitatory amino acid in toxic mussels from eastern Prince Edward Island. *Can. J. Chem.* **67**: 481-490.

Yasumoto, T., M. Murata, Y. Oshima, G. K. Matsumoto and J. Clardy. 1984. Diarrhetic shellfish poisoning. In: *Seafood Toxins*, E. P. Ragelis (Ed.), Amer. Chem. Soc. Symp. Ser. No. 262, Washington, DC, pp. 207-214.

VIII. WORKSHOP AGENDA

Tuesday, April 21, 1992

- 8:30 Registration; coffee
- 9:30 Announcements - Dr. Sylvia Galloway
- 9:15 Welcome - Dr. Robert Kifer
- 9:30 Workshop Overview - Dr. Donald Anderson
- 9:45 International Perspective - Dr. Donald Anderson
- 9:55 NMFS Perspective - Dr. Sylvia Galloway
- 10:05 Coastal Oceans Program Perspective - Dr. Leon Cammen
- 10:15 FDA Perspective - Dr. Sherwood Hall
- 10:25 NIEHS Perspective - Dr. Daniel Baden
- 10:35 Break
- 10:50 Define National Program; Develop objective statement for the workshop
- 12:00 Lunch
- 1:15 Assignment to Working Groups; Charge by Co-Chair
- 1:30 Working Group Discussion; Goal to generate consensus document in assigned topic
- 5:00 Social gathering at picnic area, Marshlands House
- 6:00 Dinner - "FROGMORE STEW"

Wednesday, April 22, 1992

- 8:30 Coffee; Working Group discussion continued
- 12:00 Lunch
- 2:00 Plenary Presentations; Pharmacology/Epidemiology/Toxicology - Dr. Daniel Baden
- 2:30 Toxin Analysis/Assays/Chemistry Standards - Dr. Jack Wekell
- 3:00 Shellfish Monitoring - Dr. Richard Danielson
- 3:30 Plankton Monitoring - Dr. Rita Horner
- 4:00 Break
- 4:15 Foodweb Effects - Dr. Alan White
- 4:45 Shellfish Depuration/Physiology - Dr. Sandra Shumway
- 6:00 Dinner on your own

Thursday, April 23, 1992

- 8:30 Coffee; Plenary Presentations; Fish Mortalities - Mr. Jack Rensel
- 9:00 Bloom Biology/Ecology - Dr. Donald Anderson
- 9:30 Remote Sensing - Dr. Patricia Tester
- 10:00 Nutrient/Pollution Effects - Dr. Theodore Smayda
- 10:30 Break
- 10:45 Taxonomy/Genetics/Population Biology - Dr. Karen Steidinger
- 11:15 Hydrography/Physical Oceanography - Dr. Donald Anderson
- 11:45 Discussion: Report structure
- 12:00 Lunch
- 1:30 Working Groups meet to finalize reports
- 4:00 Discussion: Management (structure), networking, funding sources, collaborations
- 6:00 Dinner on your own; Evening Working Group writing (if necessary)

Friday, April 24, 1992

- 8:30 Coffee; Reports by three Working Groups - Strawman priority lists, Executive Summary
- 9:00 National Program Priorities Set, Discussion -
Dr. Donald Anderson/Dr. Sylvia Galloway
- 10:30 Break
- 12:00 Workshop Adjourned
- 1:30 Informal Lab Tour - John Babinchak/Fran Van Dolah

IX. PARTICIPANTS

Dr. Donald Anderson (Co-chair)
Biology Department
Woods Hole Oceanographic Institute
Woods Hole, MA 02543
(508) 457-2000, Ext. 2351
FAX: (508) 457-2169

Dr. Daniel G. Baden
Marine and Freshwater Biomedical Sciences
Center
University of Miami
4600 Rickenbacher Causeway
Miami, FL 33149-1098
(305) 361-4738; FAX: (305) 361-4711

Dr. Monica Bricelj
Marine Sciences Research Center
SUNY at Stony Brook
Stony Brook, NY 11794-5000
(516) 632-8663; FAX: (516) 632-8820

Dr. JoAnn Burkholder
N.C. State University
Department of Botany, Box 7612
Raleigh, NC 27695
(919) 515-2726; FAX: (919) 515-3436

Dr. Richard Danielson
Department of Health Services
Sanitation and Radiation Laboratory
2151 Berkeley Way, Room 465
Berkeley, CA 94704
(510) 540-2201; FAX: (510) 540-2053

Dr. Sylvia B. Galloway (Co-chair)
Southeast Fisheries Science Center
National Marine Fisheries Service, NOAA
P.O. Box 12607, 217 Fort Johnson Road
Charleston, SC 29422
(803) 762-1200; FAX: (803) 762-1998

Dr. Sherwood Hall
U.S. Food and Drug Administration
200 C Street, SW (HFF-521)
Washington, DC 20204
(202) 205-4818 OR (202) 254-3888
FAX: (202) 254-3982

Dr. Rita Homer
School of Oceanography, WB-10
University of Washington
Seattle, WA 98195
(206) 543-8599; FAX: (206) 543-0275

Mr. John W. Hurst
Fisheries and Health Science Division
Department of Marine Resources
West Boothbay Harbor, ME 04575
(207) 633-5572; FAX: (207) 633-7109

Mr. Raffael Jovine
Department of Biological Sciences
University of California at Santa Barbara
Santa Barbara, CA 93106
(805) 893-4319; FAX: (805) 893-4724

Dr. Rikk G. Kvitek
Moss Landing Marine Laboratories
P.O. Box 450
Moss Landing, CA 95039
(408) 633-5606; FAX: (408) 633-5642

Dr. Roy E. Martin
National Fisheries Institute
1525 Wilson Boulevard, Suite 500
Arlington, VA 22209
(703) 524-8883; FAX: (703) 524-4619

Dr. John S. Ramsdell
Division of Marine Biomedical and
Environmental Sciences
Medical University of South Carolina
Charleston, SC 29425
(803) 793-7988; FAX: (803) 792-0664

Mr. Jack Rensel
School of Fisheries, HF-15
University of Washington
Seattle, WA 98195
(206) 524-6331; FAX: (206) 524-6331

Dr. Yuzuru Shimizu (not present, but
contributed)
Department of Pharmacognosy
College of Pharmacy
University of Rhode Island
Kingston, RI 02881-0809
(401) 792-2751; FAX: (401) 792-2181

Dr. Sandra E. Shumway
Bigelow Laboratory for Ocean Sciences
West Boothbay Harbor, ME 04575
(207) 633-2173; FAX: (207) 633-6584

Dr. Theodore J. Smayda
Graduate School of Oceanography
University of Rhode Island
Kingston, RI 02881
(401) 792-6171; FAX: (401) 792-6682

Dr. Karen Steidinger
Florida Marine Research Institute
Florida Department of Natural Resources
100 Eighth Ave., SE
St. Petersburg, FL 33701
(813) 896-8626; FAX: (813) 823-0166

Dr. Patricia A. Tester
Southeast Fisheries Science Center
National Marine Fisheries Service, NOAA
Beaufort Laboratory
Beaufort, NC 28516
(919) 728-8792; FAX: (919) 728-8784

Dr. Donald Tindall
College of Science
Department of Botany and Plant Biology
Southern Illinois University
Carbondale, IL 62901
(618) 536-2331; FAX: (618) 453-3441

Dr. Jack Wekell
Northwest and Alaska Fisheries Science Center
National Marine Fisheries Service, NOAA
Seattle Laboratory
2725 Montlake Blvd., E.
Seattle, WA 98112
(206) 442-7746; FAX: (206) 553-4304

Dr. Marleen M. Wekell
U.S. Food and Drug Administration
P.O. Box 3012
Bothell, WA 98041-3012
(206) 483-4902; FAX: (206) 483-4996

Dr. Alan W. White
Northeast Fisheries Science Center
National Marine Fisheries Service, NOAA
Woods Hole, MA 02543
(508) 548-5123; FAX: (508) 548-5124

Dr. Jeffrey L.C. Wright
National Research Council of Canada
Institute for Marine Biosciences
1411 Oxford Street
Halifax, Nova Scotia B3H 3Z1
CANADA
(902) 426-8275; FAX: (902) 426-9413

**Representing the NMFS Southeast
Fisheries Science Center:**

Mr. John A. Babinchak
Dr. Gregory J. Doucette
Ms. Jeanne D. Joseph
Dr. Robert Kifer
Dr. Peter Moeller
Dr. Frances M. Van Dolah

Southeast Fisheries Science Center
National Marine Fisheries Service, NOAA
P.O. Box 12607, 217 Fort Johnson Road
Charleston, SC 29422
(803) 762-1200; FAX: (803) 762-1998

Representing NOAA Coastal Oceans Program:

Dr. Leon Cammen
National Sea Grant College Program, NOAA
R/OR1
1335 East-West Highway
Silver Spring, MD 20910
(301) 713-2435; FAX: (301) 713-0799



Biotoxin Workshop Participants (left to right): Sylvia Galloway, Rafael Jovine, Peter Moeller, Fran VanDolah, Jack Rensell, Alan White, Monica Bricelj, Sherwood Hall, John Hurst, Pat Tester, John Ramsdell, Rita Horner, Jack Wekell, Rikk Kvittek, Marleen Wekell, Rick Danielson, Jeff Wright, Ted Smayda, Greg Doucette, Dan Baden, Sandy Shumway, Jeanne Joseph, Don Tindall, Bernie Lanoue, Karen Steidinger, JoAnn Burkholder, Don Anderson (Leon Cammen and Roy Martin, not shown - early departure)

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16. Abstract (Limit: 200 words) Marine biotoxins and harmful algae represent a significant and expanding threat to human health and fisheries resources throughout the U.S. This problem takes many forms, ranging from massive "red tides" or blooms of cells that discolor the water to dilute, inconspicuous concentrations of cells noticed only because of the harm caused by the highly potent toxins those cells contain. Impacts include mass mortalities of wild and farmed fish, human intoxications and death from contaminated shellfish or fish, alterations of marine trophic structure, and death of marine mammals, seabirds, and other animals. The nature of the problem has changed considerably over the last two decades in the U.S. Where formerly a few regions were affected, now virtually every coastal state is threatened, in many cases over large geographic areas and by more than one harmful species. The U.S. research, monitoring, and regulatory infrastructure is not adequately prepared to meet this expanding threat. In an effort to surmount these problems, a workshop was convened to formulate a National Plan for the prediction, control, and mitigation of the effects of harmful algal blooms on the U.S. marine biota. This report summarizes the status of U.S. research knowledge and capabilities, and identifies areas where research funds should be directed for maximum benefit.		14.	
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