915

Developing-Country Aquaculture and Harmful Algal Blooms*

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Abstract

Toxic algal blooms began to have significant impacts on developing-country aquaculture in the 1970s, including toxic shellfish and mass mortalities of fish and shrimp. Based on the experiences of developed countries, the potential exists in the waters of developing countries for a wide range of presently unrecorded toxins as well as other effects of algal blooms. The implications of these hazards are discussed. Their economic impact, which extends beyond the aquatic sector, is also discussed. Management measures used in both developed and developing countries at the industry as well as the government level are described and assessed. Finally, evidence of a relationship between red tides and aquaculture is discussed.

Introduction

Many scientists believe that visible algal blooms or red tides are environmental indicators in as much as there is a strong correlation between the number of red tides and the degree of coastal pollution or use of coastal waters for aquaculture (see e.g., Anderson 1989; Lam and Ho 1989; Okaichi 1989; Seliger 1989; Smayda 1989).

Potentially nuisance blooms in the sea have been around for a long time. Captain Cook observed *Trichodesmium* blooms in the Coral Sea in 1770 and along with his crew suffered ciguatera poisoning in the New Hebrides in 1774 (Hallegraeff 1990). The first paralytic shellfish poisoning report on the Pacific coast of north America was in 1793 during explorations by Vancouver in British Columbia (Conte 1984). The naturalist Poeppig was the first to record a red tide in Chile, in 1827. Darwin saw the next one there in 1835 (Unesco 1982).

In recent years, there appears to have been a rapid global increase in red tides which is reflected, according to Anderson (1989), in the increase in countries represented at the international meetings on toxic dinoflagellates, from three at the first such meeting in 1974, 17 at the second in 1978, 22 in the third (1985), to 27 in the fourth (1989).** New occurrences have been reported in a variety of locations at each meeting. A new directory of experts in the fields includes over 390 persons from 42 countries (White 1990a).

Whether the blooms are increasing or not, there has been a rapid growth of relevant literature. The 163 references in the present review, for example, while by no means

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**The topic of apparent increases in blooms has been pursued in recent reviews by Hallegraeff (1992, 1993) - Eds.

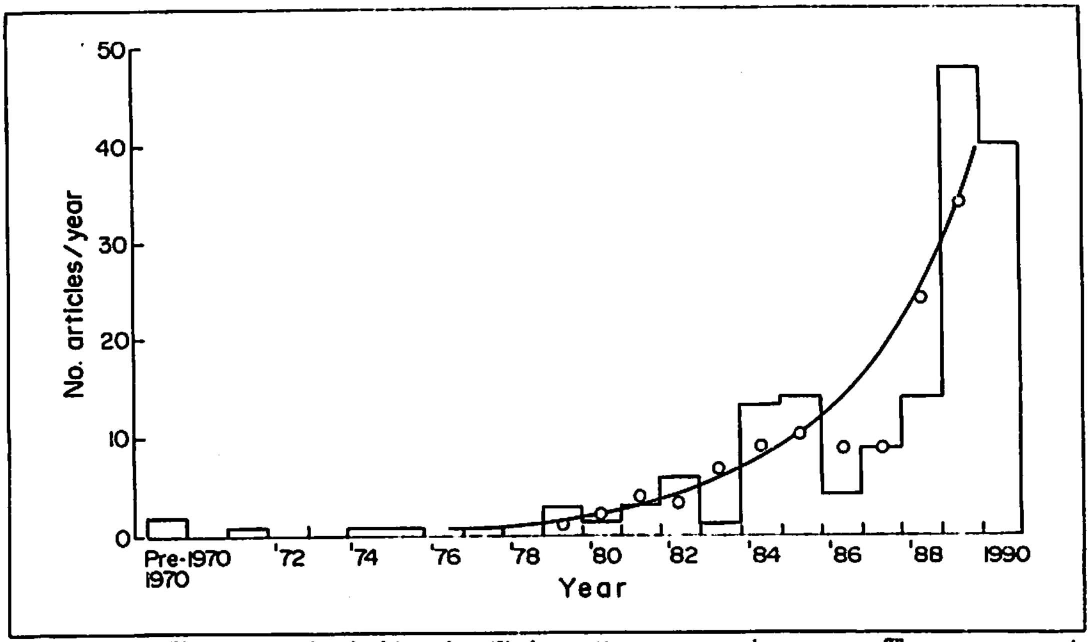

Fig. 1. Growth of literature as cited in this review. Circles are three-year running averages. The curve represents literature doubling every 2.25 years.

constituting a bibliography, exemplify the literature explosion. A plot of the publication dates (Fig. 1) shows that over 50% come from 1989 and 1990; the curve suggests a doubling of the annual literature output every 2-2.5 years over the past two decades. For comparison, aquaculture literature output doubled every five years from 1960 to 1980 (Maclean 1986).

One of the features of these algal blooms is a shift over the years from generally benign diatom blooms to flagellate blooms, possibly associated with decreased Si:P and N:P ratios, in part due to coastal enrichment from pollution and river-borne nutrients according to Smayda (1989). The flagellates include most of the toxic forms. Lam and Ho (1989) pointed out a clear shift from diatom- to dinoflagellate-dominated phytoplankton in Tolo Harbour, Hong Kong, as the waters became more polluted. Red tides there have increased dramatically also (Fig. 2). As Zou et al. (1985) pointed out, "A red tide can be looked upon both as a product and as a process in the eutrophication of estuarine and coastal ecosystems."

At the most recent international conference on toxic marine phytoplankton (Granéli et al. 1990), the influence of human activities was debated. In the conference overview, Taylor (1990) stated that "some blooms are plainly in response to eutrophication but others are equally plainly not." The following careful recommendation was approved and conveyed to the

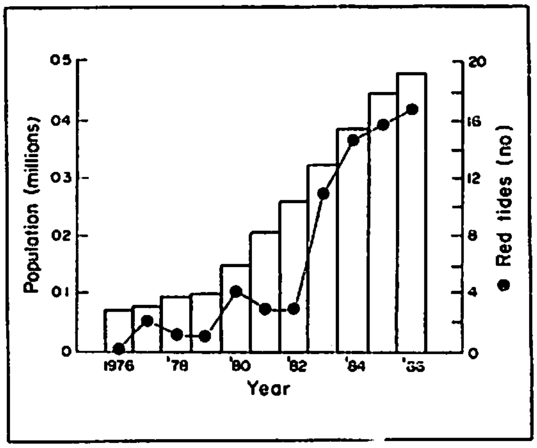

Fig. 2. Annual number of red-tides in Tolo Harbour, Hong Kong, and human population levels in Hong Kong from 1976 to 1986. From Lam and Ho (1989).

International Oceanographic Commission, Paris: "The conference participants reached a consensus that some human activities may be involved in increasing the intensity and global distribution of blooms and recommended that international research efforts be undertaken to evaluate the possibility of global expansion of algal blooms and man's involvement in this phenomenon" (Granéli et al. 1990, p. 517).

From the point of view of aquaculture, toxic algal blooms can be considered as part of the environment in which this sector is presently developing.

The coastal zone in many developing countries is becoming a major focus of attention to environmental managers in view of the largely uncontrolled developments taking place there, entailing massive destruction of natural habitats (Chua, this vol.) The Group of Experts on the Scientific Aspects of Marine Pollution (GESAMP) stated that "at the end of the 1980s, the major causes of immediate concern in the marine environment on a global basis are coastal development and the attendant destruction of habitats, eutrophication, microbial contamination of seafood and beaches..." (GESAMP 1990). Thus, it is the coastal environment of developing countries where we can expect to see dramatic increases in toxic algal blooms. The recent spreading of red tides in the Indo-Pacific (Maclean 1989a), for example, may be only a prelude to worsening problems ahead.

Meanwhile, that environment itself is in all probability undergoing changes such as atmospheric temperature increases and changing relative sea levels (Stewart et al. 1990), associated with global climatic changes which may mask, reinforce or negate some of the more local pollution/enrichment effects. We are dealing then with a growing problem in which there may be so many contributing factors - both primary and secondary - that it may be impossible or impractical to attempt to isolate any one cause or group of causes.

In the developing world, most of the interest in and workshops about harmful algal blooms (and, coincidentally over 80% of world aquaculture production) have been in Asia, and have happened fairly recently. A 1954 symposium on plankton in the Indo-Pacific made no mention of red tides or harmful algal species (FAO/Unesco 1954). Toxic blooms of *Pyrodinium* in Papua New Guinea in 1972, western Borneo in 1976 and 1980 and in the Philippines in 1983 (Maclean 1989b); fish kills in Hong Kong beginning in 1980 (Wong and Wu 1987); and an unprecedented red tide in Jinhae Bay, Korea (Park et al. 1989), raised enough concern for two regional meetings in 1984 (White et al. 1984; CSIRO 1985). The incidents in Hong Kong, Korea and the Philippines all involved aquaculture losses which continue to the present. The *Pyrodinium* situation was sufficiently alarming to warrant a special workshop in 1989 (Hallegraeff and Maclean 1989). In Latin America, a workshop on red tides on the Pacific coast was held in 1982 (Unesco 1982). There are no reports of similar activities in Africa. At the international level, two conferences in 1987 specifically addressed the impact of algal blooms on aquaculture (Dale et al. 1987; Jensen 1988).

In this review, I have attempted to consolidate information on the nature of the potential hazards, the extent of the algal bloom problem in developing-country aquaculture and, despite the uncertainty of future environments, some likely future scenarios.

Causative Organisms

The algae under consideration come from the Cyanophyta, cyanobacteria; Chrysophyta, chrysophytes; Pyrrhophyta, dinoflagellates; Raphidophyta, chloromonads, Bacillariophyta, diatoms; and Prymnesiophyta, prymnesioids - a mixture of primitive eukaryotes and prokaryotes (Taylor 1985). Some authors use classes rather than phyla to describe these organisms (e.g., Hargraves et al. 1989; Fukuyo et al. 1990) and the taxonomy of individual species as well as of groups remains under revision (e.g., several papers in Granéli et al. 1990).

Larger dinoflagellates commended most attention at earlier workshops but in recent years the importance of other groups - the raphidophytes, particularly *Chattonella*; chrysophytes, especially the brown tide *Aureococcus*; the prymnesiophyte *Chrysochromulina*; and the cyanobacteria - has been recognized. The picoplankton (0.2-2.0 mm or bacteria-sized cells), have been found to be a source of blooms and toxins; they include small flagellates and cyanobacteria. These organisms bloom seasonally in coastal waters at densities orders of magnitude higher than larger forms, viz up to $10^9 \cdot l^{-1}$ vs. $10^6 \cdot l^{-1}$ (Hargraves et al. 1989). Often the picoplankton are the dominant primary producers in the sea (Sieburth and Johnson 1989).

There is no comprehensive list of causative species and their number is certainly still growing. Taylor (1990) gave a current "minimum list" of well-established harmful marine phytoplankton species (i.e., excluding those which have been associated with oxygen depletion or gill clogging) as follows: dinoflagellates 27; chloromonads 5; chrysophytes 1; prymnesioids 5; silicoflagellates (chrysophytes) 1; and diatoms 4. The most recent list from Japan includes 300 freshwater and marine algal species including "not only causative organisms of red tide but also toxic species and organisms which are associated with other dominant species in red tides along Japanese and Southeast Asian coastal waters" (Fukuyo et al. 1990). Shumway (1990) lists about 44 toxic algae which affect shellfish. Some 12 genera of Cyanophyta have been implicated in producing acute lethal toxins (Carmichael et al. 1990).

Toxic Products

The various toxins produced by these algae (and by a variety of other marine organisms) have in common the property of modifying the functions of ion channels across cell membranes. On this basis, toxins can be grouped into three types: activators, stabilizers and occluders (Strichartz and Castle 1990).

Toxins affecting or potentially affecting aquaculture operations include activators such as the toxins causing neurotoxic shellfish poisoning, diarrheic shellfish poisoning, ciguatera and domoic acid, and occluders such as the paralytic shellfish poisons (saxitoxins) tetrodotoxin and anatoxins (Dale et al. 1987; Carmichael et al. 1990; Strichartz and Castle 1990). A list of the better known toxins is given in Table 1.

Neurotoxic Shellfish Poisons

Eight polyether toxins, collectively called brevetoxins, are presently known to be produced by the dinoflagellate *Gymnodinium breve*, the Florida red tide organism (Shimizu 1989). The toxins are potent fish killers and in aerosol form cause human respiratory irritation. They also cause a mild form of poisoning, similar to ciguatera, and of similar chemical structure to the ciguatoxins in humans ingesting contaminated shellfish. A bloom in North Carolina at the end of 1987 caused closure of 150,000 ha of shellfish grounds and lowered the crop value by $2 million (Tester and Fowler 1990). There seem to be no reports of hazards from this alga in other waters.

Diarrheic Shellfish Poisons

Diarrheic shellfish poisoning (DSP) in humans is caused by ingesting shellfish which have accumulated toxins from dinoflagellates of the genus *Dinophysis* or the benthic dinoflagellate *Prorocentrum lima*. Elever polyether toxins have been identified from shellfish in three groups - okadaic acid and derivatives (dinophysistoxins); pectenotoxins; and yessotoxin (Yasumoto and Murata 1990).

Table 1. Properties of various algal toxins.

Generic name	Number known	Molecular weight	Effect	LD$_{50}$ mg/kg intraperitoneal mouse assay	Source
Brevetoxins	8	870-970	Potent ichthyotoxins NSP	60 (rats)	Shimizu (1989) Poli et al. (1990)
Saxitoxins	17	~300	PSP	10	Hall et al. (1990)
Anatoxins	2	165, 252	Neurotoxic poisoning of animals, fish	20-200	Carmichael et al. (1990)
Hepatotoxins	8-10	800-1,000	Hepatoenteritis	30-1,000	Carmichael et al. (1990)
Diarrheic shellfish toxins	11		Nonlethal intestinal disorders	100-770 (LD$_{99}$)	Yasumoto and Murata (1990)
Domoic acid	1	311	Gastrointestinal disorders, memory loss	-	Todd (1990)
Ciguatoxins	2	1,100	Gastrointestinal, cardiovascular and neurological disorders	0.45	Frelin et al. (1990)

Symptoms in humans are gastrointestinal and not fatal. DSP was only recognized in the mid-1970s and was probably previously confused with bacterial spoilage. However, recent reports (e.g., Dale et al. 1987; Shumway 1990) show that DSP is a significant problem in Europe and Japan, with incidences also in Australia, Chile, India and New Zealand. *Dinophysis* spp. are cosmopolitan phytoplankters, so the potential for problems elsewhere is high, and closures of shellfish farms may be longlasting. For instance, mussels in Sweden remained toxic almost continuously from October 1984 until summer 1986 (White 1988).

Ciguatoxins

Ciguatoxins are a group of disparate polyether toxins which cause the well-known illness in humans, ciguatera, from eating various tropical reef fish. The two major toxins are ciguatoxin (of which there are two "species") and maitotoxin. A third compound, scaritoxin, may be a form of ciguatoxin, while new "minor toxins" have recently been discovered (Legrand et al. 1990). Another different polyether toxin, palytoxin, has been found responsible for poisonings by triggerfish and xanthid crabs (Yasumoto and Murata 1990). The poisons are detected via mouse bioassay.

The benthic marine dinoflagellate *Gambierdiscus toxicus*, the major alga responsible for ciguatera, has now also been found to form toxic blooms on its macroalgal substrate. Population explosions of the alga in Tahiti after some 10 years of relative dormancy were closely associated with toxicity in the grazing surgeonfish *Ctenochaetus striatus* (Bagnis et al. 1990). *Gambierdiscus* showed clear seasonal population trends in Queensland, Australia

(Gillespie et al. 1985), but no such trend in Tahiti (Bagnis et al. 1985).

There are no records of ciguatera from farmed fish but the toxins could become important if ranching of tropical reef fish becomes practical and even for farming tropical carnivorous fish in marine enclosures, where they have access to small prey entering the enclosures.

Paralytic Shellfish Poisons

These poisons are produced by a number of marine dinoflagellates as well as by the predominantly freshwater cyanobacteria *Aphanizomenon flos-aquae*. *Aphanizomenon* produces aphantotoxins which have been found to be identical to saxitoxins (Shimizu et al. 1990). The best known dinoflagellate toxin producers are: *Gonyaulax* spp. now called *Alexandrium* spp. (Balech 1985), which have exhibited toxic blooms almost all around the world in tropical and temperate waters (e.g., Taylor and Seliger 1979; Anderson et al. 1985; Okaichi et al. 1989a); *Gymnodinium catenatum* which causes PSP in Australia, Europe, Japan and Venezuela (Hall et al. 1990); and *Pyrodinium bahamense* around Southeast Asia, the South Pacific (Maclean 1989d) and recently several countries along the Pacific coast of central America (F. Rosales-Loessener 1989, pers. comm.).

Eighteen closely-related "saxitoxins" have been discovered and a further six may possibly be found in future, based on the molecular structure of the group. The known "saxitoxins" include saxitoxin *per se*, neosaxitoxin and several gonyautoxins and decarbamoyl saxitoxins (Yasumoto et al. 1984; Hall et al. 1990). Several of these compounds in different proportions are found in the various algae and their consumers; each algal species exhibits a unique toxin "profile".

Saxitoxins accumulate mainly in filter-feeding bivalves, which in general are not lethally affected by them, and pass along the foodchain. The consumers include some gastropods, which prey on bivalves and become toxic, and also humans who are poisoned, sometimes fatally from paralytic shellfish poisoning (PSP). There are, of course, other pathways through zooplankton to fish and marine mammals, as well as to birds. Humans have contracted PSP by eating planktivorous fish (see below). The symptoms in humans are neurological, gastrointestinal and respiratory disorders (e.g., Pastor et al. 1989). Timely respiratory support prevents death. There is no proven antidote although the Philippine folk remedy of drinking coconut milk and brown sugar was reported to be effective in reducing toxicity of crude toxins (Gacutan 1986).

Amnesic Shellfish Poison

This form of intoxication in humans from eating bivalves was first reported in late 1987 in eastern Canada, when consumers of cultured mussels, *Mytilus edulis*, developed gastrointestinal disorders accompanied by short-term memory loss. There were some deaths. Unlike in other forms of algal poisoning, the neurological damage is permanent (Addison and Stewart 1989; Smith et al. 1990; Todd 1990).

The toxin was found to be domoic acid, a neurotoxic amino acid, detected by mouse bioassay and high-performance liquid chromatography (HPLC). The source was apparently a bloom of the diatom *Nitzschia pungens*. Agricultural run-off was suspected as the cause by Smith et al. (1990), whereas Addison and Stewart (1989) did not discount the possibility that the intensive aquaculture in the area may have been a cause of the bloom.

So far this toxin has not been found in developing countries. *N. pungens*, however, is a ubiquitous coastal species (Fukuyo et al. 1990).

Tetrodotoxin

Tetrodotoxin (TTX), best known for its dramatic effects on consumers of *fugu* puffer fish, is also found in other fish, frogs, newts, octopus, gastropods, starfish, crabs, flatworms, zooplankton, algae and bacteria (*Vibrio* spp. and several other genera) (Jeon et al. 1986; Narita et al. 1987; Tamplin 1990). As with saxitoxins, it is possible that bacteria are the primary producers of tetrodotoxin, which then accumulates up the foodchain (Narita et al. 1987; Tamplin 1990). Tetrodotoxin is not known to be associated with any dinoflagellate blooms, but is usually discussed in the same fora and has implications for aquaculture.

There are four tetrodotoxin toxins - tetrodotoxin itself being the best known. They are detectable by mouse bioassay and several more sophisticated biological as well as chemical techniques (Onoue et al. 1984; Tamplin 1990).

Pufferfish are farmed in Japan (and may be potential export crops in Asian developing countries). Saito et al. (1984) found that pufferfish from farms in several localities in Japan were all nontoxic. Toxicity was found only in wild-caught specimens. Not all pufferfish species are toxic. Those that are have very high resistance to interperitoneal administration of the toxins (minimum lethal dose of 300-750 mouse units (MU)/20-g body weight), compared to 1-20 MU for nontoxic pufferfish species (Saito et al. 1985b). The toxin seems to be a biological defense agent for the pufferfish (Saito et al. 1985c). Human poisonings are through ingestion of *fugu* or by being bitten, in the case of the Australian blue-ringed octopus.

Hepatotoxins

Anabaena and some other predominantly freshwater cyanobacteria - *Microcystis aeruginosa, M. viridis, Nodularia spumigena* and *Oscillatoria agardhii* - produce hepatotoxins, short peptide toxins which affect the liver and are also acutely lethal to animals (Carmichael et al. 1990). Six chemically related hepatotoxins are known which have about one-fifth the toxicity of saxitoxins and account for most of the cases of animal poisonings from freshwater cyanobacteria (Gorham and Carmichael 1988).

To date there are no confirmed instances of human death from these sources, but allergic and gastrointestinal problems are known and pets, livestock and wildlife, including fish, have been killed.

Toxicity problems from freshwater cyanobacteria have been recorded in Australia, Bangladesh, China, (12 countries of) Europe, India, Israel, Japan, Latin America, North America, South Africa, Thailand and the USSR (Carmichael et al. 1990). These countries are a fair cross-section of the globe and it is likely that in others, especially developing countries, the problem has not been fully recognized. Some of the species, e.g., *Anabaena flos-aquae, Microcystis aeruginosa* and *M. viridis*, are ubiquitous (Fukuyo et al. 1990). *Microcystis* is reported to dominate continuously warmer, shallow, eutrophic waters such as Indian temple ponds and Lake George in Uganda (Stirling and Dey 1990).

Anatoxins

Anatoxins are a group of neurotoxins produced by the freshwater cyanobacteria *Anabaena flos-aquae*. Six compounds have been isolated which have from one-twentieth to one-half the potency of saxitoxins (Carmichael et al. 1990). They can be detected by mouse bioassay and HPLC. The toxins are lethal. No antidotes are available (Gorham and Carmichael 1988).

Cytotoxins

Carmichael et al. (1990) noted that some freshwater cyanobacteria produce a variety of bioactive compounds of much lower toxicity to humans, collectively called cytotoxins, which cause dermatitis or irritation on contact. They can be lethal to mice, when administered interperitoneally. These authors pointed out that the various neurotoxic, hepatotoxic and dermatotoxic compounds produced by cyanobacteria are a direct and growing threat to animal and human water supplies.

Other Harmful Effects

Oxygen Depletion and Gill Clogging

Blooms of many algal species are not toxic in themselves but cause mortalities through oxygen depletion when blooms collapse. In theory this situation could apply to almost all algal bloom species. Some examples of these problems are given in Table 2.

Oxygen depletion in eutrophic ponds at night is a well known consequence of "excess" algal biomass, but similar problems are emerging in open marine waters. For example, in the southern Kattegat between Denmark and southern Sweden, "Oxygen deficits resulting from decomposition of algal matter, with the ensuing death of fish and other animals, consitute a regular environmental problem there" (Dahl et al. 1989).

Gill clogging due to mucus secretion was thought to be a major factor in fish kills caused by several flagellates (White 1988). However, species mentioned by White (1988) have been found to act through toxin production. One, the dinoflagellate *Gyrodinium aureolum* was found to kill seawater acclimatized rainbow trout (*Oncorhynchus mykiss*) by causing degeneration of gill tissue. Involvement of toxin(s) was presumed (Roberts et al. 1983). There is recent evidence of toxin production by the chloromonad *Heterosigma akashiwo* (R.J. Gowen, pers. comm.), also noted by White (1988) as causing only mucus clogging of gills.

Some larger dinoflagellates with spinous skeletons, such as *Chaetoceros convolutus, C. concavicornum* and *C. danicus*, can cause physical damage to gills, such that fish die of asphyxiation; this may become a serious problem for fish farmers (R.J. Gowen, pers. comm.).

The massive mortalities of farmed yellowtail (*Seriola quinqueradiata*) in the Seto Inland Sea of Japan by the chloromonad *Chattonella* are due to gill damage and production by the algae of highly unsaturated fatty acids which decrease the pH of the fish's blood, making gas exchange difficult (White 1988).

Nutrient Stripping

Uno and Sasaki (1989) report that diatom blooms affect nori (*Porphyra tenera*)

Table 2. Examples of algal species/situations causing anoxic/hypoxic conditions in aquaculture facilities in developing countries.

Species	Country	Commentary	Source
Cyanobacteria			
Trichodesmium erythraeum	Thailand (Gulf)	Extensive fish kills in farms	Suvapepun (1989)
Microcystis sp.	Philippines (Laguna de Bay)	Fish kills in fishpens	Ronquillo (1987)
Dinoflagellates			
Noctiluca sp.	Philippines (Manila Bay)	Deaths of fish in cages, mussels oysters, shrimp and crabs	Ronquillo (1988)
Noctiluca and other genera	Hong Kong	Fish kills in farms	Wong and Wu (1987); Lam and Yip (1990)

seaweed culture in Japan. Seasonal blooms of *Eucampia, Chaetoceros* and *Nitzschia* strip the seawater of nutrients, resulting in fading of dark brown color of the seaweed and lowering its commercial value. An index of photosynthetic activity per unit biomass has been developed which gives a 2-4 week warning to growers (Yamamoto and Fujisaki 1989).

Discoloration

A bloom of the ubiquitous ciliate *Mesodinium rubrum* was reported to have caused red discoloration of oysters (*Ostrea edulis*) in the Netherlands (Kat 1984). The author noted previous incidents of orange discoloration in oysters in Italy, pink oysters in Texas caused by a bacteria, blue-green oysters in France caused by a diatom and green oysters in Greece caused by coccolithophorid.

Tainting

Tainting of fish from algal blooms is another factor to be considered. Earthy tainting of freshwater fish flesh with geosmin was noted by Stirling and Dey (1990) as an effect of decomposing blooms. From personal experience, tainting is a seasonal problem in tilapias in Laguna de Bay, Philippines, attributed by some researchers to blooms of *Microcystis aeruginosa* but by others to actinomycetes (de Guzman 1990). A shipment of pond-grown penaeid shrimp from Ecuador was unmarketable as a result of intense musty flavor from geosmin, caused by cyanobacteria (Lovell and Broce 1985). A bloom of the diatom *Rhizosolenia chunii* in southeastern Australia in 1987 caused a strong bitter flavor in cultured and wild bivalve molluscs. Over a seven-month period, some 500 tonnes of mussels worth about $1 million were discarded (Parry et al. 1989).

In summary, many toxins are produced by algae which in blooms can render bivalve molluscs toxic to humans. Some toxins can be fatal to humans - the paralytic shellfish poisons (PSP) and amnesic shellfish poison - while others, diarrheic and neurotoxic shellfish poisoning, are milder but severe enough to close fish farms and fisheries for long periods. Human intoxication from fish containing PSP toxins has been known as well as from fish containing the more common ciguatera toxins. Fish and other organisms can be killed or even if they survive have their growth and fitness reduced by micro-algae via neurotoxins (anatoxins, brevetoxins, hepatotoxins and PSP toxins) as well as gill-clogging mechanisms and bloom-induced hypoxic or anoxic conditions. Finally, some algal bloom species can cause off-flavors and discoloration in various aquatic organisms.

Economic Impact

Economists seem rarely to have turned their attention to red tides. Yet the losses caused by blooms can be large, especially by developing-country standards. A list of estimated losses from blooms to mainly north American and Japanese shellfish industries given by Shumway (1990) shows figures ranging from $0.1 million to an astounding $430 million. These include fisheries as well as farms. Maclean (1989c) divided economic impact into three facets: occasional acute episodes; chronic situations; and permanent closures.

Acute Bloom Situations

In occasional acute situations, the public is taken by surprise and large numbers of people are affected by contaminated organisms. The number of persons affected ranges usually from 50 to 300, as in the first outbreaks of *Pyrodinium* in Sabah, Malaysia (Ting and Wong 1989), the Philippines (Gonzales 1989a), and Guatemala (Rosales-Loessener 1989); of domoic acid poisoning in eastern Canada (Todd 1990); and of neurotoxic shellfish poisoning in New Jersey, USA (Tester and Fowler 1990). In the Philippines, a second acute *Pyrodinium* episode occurred in 1987 (four years after the first) and resulted in over 200 cases of illness (Maclean 1989b). Such illness affects the resource rent from the fishery or aquaculture sector to some extent. The full impacts of blooms appear rarely to have been determined but are probably much larger than published figures imply.

A good example in the developing-country aquaculture context is the first outbreak of mussel (*Perna viridis*) poisoning in Manila Bay, Philippines, in 1988:

> In August and September 1988, the first outbreak in Manila Bay occurred. Thanks to the media, the whole seafood industry nearly ground to a halt, while mussel growers even tried to implicate freshwater products in an effort to offset the swing by consumers to tilapias and other freshwater organisms! All fish markets in Manila were depressed for over three months, similar to the case in San Francisco in 1980. Manila's seafood market handles 35% of the nation's landings. Thus, the losses were large, up to $300,000/day at the height of the scare. Japan and Singapore banned shrimp imports from the Philippines for an unknown period (although they were clean), which would have meant losses of $500,000/day if the produce was not subsequently sold. Losses by mussel growers for a three-month period were more modest, about $950,000 in all" (Maclean 1989c).

Even the vinegar industry was affected, because mussels are usually eaten with a vinegar-based sauce. Quite probably, the prices of unaffected commodities such as poultry rose in response to the general scare on seafoods.

This "halo effect" (Shumway 1990) of red tide outbreaks into other sectors of the economy is not confined to developing countries; examples from developed countries are given by Maclean (1989c) and Shumway (1990). Misinformation in the media is a common cause.

As far as losses to aquaculturists themselves is concerned, there are few data from developing countries. Thai fish farmers lost some $1.16 million worth of fish in 1983 when a huge *Trichodesmium* bloom in the Gulf of Thailand collapsed, causing anoxic conditions along the coast (Suvapepun 1989). Mussel farmers in Manila Bay lost about $1 million worth of (condemned) produce during the 1988 *Pyrodinium* blooms there (Maclean 1989c). The single mussel farm in Brunei lost most of its crop which fell from the farm's longlines during a long ban on marketing in 1988 due to high PSP levels in shellfish. This involved total losses, including expenses in growing the crop, of about $0.1 million (Jaafar et al. 1989). A pond shrimp kill by an unknown dinoflagellate in Hupei and Shandong provinces of China in 1989 caused a financial loss of 300 million yuan (US$1=5.3 yuan) (Ger Guo Chang, pers. comm.).

Chronic Bloom Situations

In chronic red tide situations, large losses may still occur, a good example being the 71 billion yen loss of cultured yellowtail in the Seto Inland Sea of Japan in 1972 and previous and ongoing lesser but significant annual losses there (Fig. 3) (Okaichi 1989). However, in chronic situations, the public eventually recognizes that only particular aquatic products are at risk, thus reducing the "halo effect". Costs of regular monitoring become part of the annual loss of resource

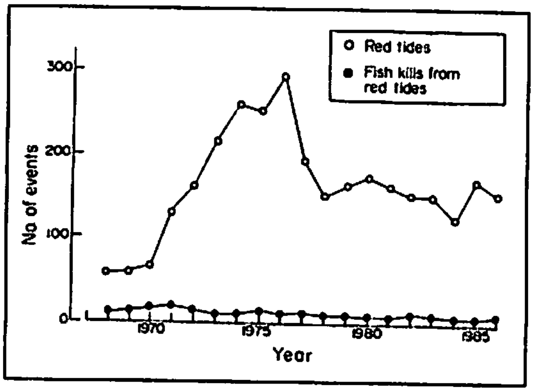

Fig. 3. Red tides in the Seto Inland Sea, 1968-1986. After Okaichi (1989).

rent. These costs may be $10,000-50,359 per year per affected area based on some recent examples (Maclean 1989c). A Canada-wide program of collection, testing, enforcement, management and information dissemination for the shellfish industry costs over $1 million per year (Pirquet 1988 cited in Shumway 1990).

Where red tides have been recognized as becoming chronic, interested parties in both government and the private sector can act more decisively. For example, in one of the earlier salmon farming fish kills in Norway, Tangen (1982) reported losses of 4 million Kroner from two farms due to *Gyrodinium aureolum* in 1982. In 1988, Norwegian farmers averted losses of $200 million by towing 120 farms out of threatened areas (Anon. 1988). Such strategies are further discussed below.

Permanent Closures

Permanent closures mean an indefinite loss of potentially high resource rents as well as losses of protein. There are examples of permanent and "near-continuous" closures in Canada (Taylor and Seliger 1979; White 1982). In developing countries, such economic sacrifices may appear to be untenable but could become necessary to avoid loss of life. The west coast of Sabah, Malaysia, is an area where there have been several deaths from PSP in most years since 1976, although red tides have only been found on a few occasions (Ting and Wong 1989). Not only does the prospect of developing a mollusc culture industry there appear bleak but the Sabah government recommends that people eat only small quantities of shellfish and that children should not eat them at all, even during "safe" periods (Wong and Ting 1989). Similar situations are likely to develop in other *Pyrodinium*-affected areas, which include many countries on both sides of the tropical Pacific (see Table 6, p. 266).

A summary of the main actual and potential "costs" associated with the above conditions - acute, chronic and closed areas - is given in Table 3. Evaluating many of them and even prioritizing them in terms of severity or importance is a difficult task but one which governments must face in making management decisions. Only a few of the cost factors lie within the aquaculture sector, but most have a bearing on its development.

Table 3. Actual and potential costs and losses associated with *Pyrodinium* red tides. Numbers represent relative severity of each factor from least (1) to most (5) important. (Source: Maclean and Ross 1989)

5	Loss of life
5	Cost of resettlement in a chronic red tide situation (transmigration)
4	Reduced price of uncontaminated seafoods
4	Cost of maintaining public awareness
3	Reduced price of suspect seafood
3	Loss of confidence by consumers
3	Loss of income by fishers, fishfarmers
3	Cost of monitoring and research (personnel, equipment, supplies)
2	Loss of condemned seafood (by fishers, fishmongers or agents)
2	Cost of publicizing and enforcing bans
2	Potential loss of business opportunities (aquaculture, fisheries)
2	Potential loss of resource use
1	Loss of wages of hired labor (fishers, fishmongers, drivers, etc.)
1	Loss of income by victims, in medicine, time off work, hospitalization (socioeconomic costs)
1	Loss of tourism income (coral reef resources)
1	Loss of income by seafood restaurants
1	Loss of foreign exchange earnings (private and government) through need to import and loss of export

Implications for Marine and Brackishwater Aquaculture

Estuarine and coastal environments contain noxious algal species which have affected virtually every type of aquaculture operation. Bivalves react to the presence of toxic dinoflagellates in a variety of ways as summarized by Gainey and Shumway (1988) and Shumway et al. (1990). These include shell valve closure; reduced filtration rate; food selection; inhibition of byssus production; change in oxygen consumption; change in heart rate; neurophysiological effects; and mortality. Presence and extent of the responses depend on the algal and bivalve species as well as the presence/absence of prior exposure of the bivalves to the dinoflagellates (Gainey and Shumway 1988). Some recent examples: the brown tide chrysophyte *Aureococcus anophageffrens* apparently caused starvation of scallops and reduced feeding in mussels (various papers in Cosper et al. 1989.); the diatom *Rhizosolenia chunii* caused high mortality of shellfish in Port Phillip Bay, Australia (Parry et al. 1989).

The implication of these data is that bivalves are affected during algal blooms to a varying extent that may include starvation and death. Kodama (1990) has taken the further step of suggesting that bivalves may become toxic by ingesting toxin-producing bacteria directly in the absence of flagellate blooms. Effects on the bivalves in this case, if any, have yet to be determined.

Intramuscular injections of tetrodotoxin and paralytic shellfish toxins into a variety of marine and freshwater clams showed the clams to be highly resistant with LD_{100}/20-g weight of over 300 MU for both types of poison (Hwang et al. 1990), indicating that these molluscs can accumulate high levels of the toxins.

Toxic dinoflagellates which cause PSP also affect fish. Fish larvae died rapidly on a diet of *Gonyaulax excavata* (*Alexandrium tamarense*) or of copepods which had been eating the dinoflagellates (White et al. 1989). The authors calculated that a first-feeding fish larva (in this case red seabream, *Pagrus major*) would need to eat only 6-11 *G. excavata* cells for a lethal dose. Zooplankton are probably much more resistant; for example, the dose of *A. tamarense* to kill brine shrimp (*Artemia salina*) is 10 times that required to kill mice on a weight-for-weight basis (Betz and Blogoslawski 1982).

Toxicity of PSP to adult marine and freshwater fish was investigated experimentally by Saito et al. (1985a). For most fish, lethal doses, administered interperitoneally, were 1-10 times that for the mouse [a 20-g mouse is the standard bioassay animal (Horwitz 1990)]. However, by oral administration, with a PSP solution absorbed onto a commercial feed, the lethal dose was much higher than by the interperitoneal route. Three aquaculture species were tested using both routes (Table 4).

Marine fish sampled had similar responses to these freshwater species. The authors concluded that fish were very resistant to ingestion of PSP. However, the toxin given in feed would probably have eluted rapidly, such that the oral tests above might not be valid (S. Hall, pers. comm.).

Table 4. Mimimum lethal doses (LD_{100}) of PSP in three aquaculture finfish species. Data from Saito et al. (1985a)

Species	LD_{100} MU/20-g body weight Interperitoneal	Oral
Cyprinus carpio	2-5	120
Salmo gairdneri (*Oncorhynchus mykiss*)	1-8	320-340
Oreochromis niloticus	2-5	>400

Hwang et al. (1990) subjected a large number of fish, crustaceans and molluscs to interperitoneal injections of tetrodotoxin and saxitoxins. Minimum lethal doses (LD_{100}/20-g weight) for fish were similar for the two groups of toxins and to values found by Saito et al. (1985a). The various crabs and shrimps tested were in many cases even more susceptible with LD_{100} values of 0.5-5.0 MU; shrimp were the most susceptible (0.5 MU).

In situ, adult fish may be killed by paralytic shellfish toxins during blooms. Estimated doses of toxins in various fish kills in Europe and north America (White 1984) were of the same order of magnitude as the experimental values in Table 4. White (1984) also found similar interperitoneal lethal doses to those of Saito et al. (1985a) and Hwang et al. (1990). He concluded that fish are unable to tolerate even small amounts of toxins in their bodies and hence are unable to accumulate them.

Nevertheless, there have been cases of PSP from eating planktivorous fish in Sabah, Malaysia (Ting and Wong 1989); Papua New Guinea (Maclean 1979) and the Philippines (Estudillo and Gonzales 1984). Samples tested shortly after capture in Brunei Darussalam showed that the toxin was confined to the digestive tract; the intestines were full of *Pyrodinium* cells (De Silva et al. 1989). It may be of interest that when Oshima (1989) re-examined the material after 18 months of storage, the intestines showed low toxicity, while the dorsal musculature was quite toxic.

Fish kills by other toxic algae, nearly all in fish farms, are widespread. Apart from *Chattonella*, the raphidophyte *Heterosigma akashiwo* has caused major losses of caged fish in Japan and also in Europe since the early 1970s (White 1988) and most recently (1989) a NZ$ 12 million loss of salmon in New Zealand (Hallegraeff 1990). Nontoxic algae cause similar damage by creating anoxic conditions (Table 2). *Pyrodinium* has caused extensive mortality of marine life during a decomposing bloom in Sabah, Malaysia (Maclean 1989a). This Sabah incident was around sheltered reefs while reefs exposed to currents were undamaged (E. Wood, unpubl. data).

A summary of recorded effects on existing aquaculture facilities in developing countries is given in Table 5. Although the list is not long, it shows that there must have been serious economic problems for farmers - major losses of shrimp in China and on a smaller scale in Malaysia; fish kills in Hong Kong, Singapore and Thailand; and public health problems - *Pyrodinium* has caused hundreds of illnesses and usually several deaths during each outbreak.

It is worthwhile drawing attention to the shrimp farming industry which despite boom and bust cycles due to disease and market idiosyncrasies, is a priority export-oriented industry for many developing-country governments in Asia (Bangladesh, China, India, Indonesia, Malaysia, the Philippines, Thailand, Vietnam) and Latin America (Brazil, Ecuador, Mexico, Panama, Peru). Cultured shrimp rose from 2% to 22% of total shrimp production between 1981 and 1988 (Liao 1990). As Table 5 shows, there has already been a large loss in shrimp farms from algal blooms in China and perhaps moderate losses in Malaysia. The Philippines has experienced shrimp export losses due to the "halo effect" as mentioned above, while development of shrimp farming in Brunei Darussalam and Malaysia (Sabah) is also threatened by the "halo effect" (Maclean 1989c).

A list of potential hazards in developing countries can be made by including (i) algal species in areas where they have exhibited toxicity to marine organisms which are not cultured but which suggest similar toxicity to or in potential aquaculture crops; and (ii) the "sleepers", planktonic algal species known to be present in a locality but not yet causing toxicity problems. Absence of problems may be due to absence of blooms, absence of aquaculture, absence of phytoplankton surveys, absence of toxin

Table 5. Incidence of toxic algal problems in developing-country aquaculture.

Country (site)	Algal species	Effect	Status	Relationship to aquaculture	Source
Brunei Darussalam	*P. bahamense* var. *compressum*	PSP	Chronic since 1976	Losses to mussel farmer	Jaafar et al. (1989)
China (Hupei and Shandong Prov.)	Unknown	Shrimp kill	First record, 1989	80% shrimp in 2,000 ha of ponds killed	Ger Guo-Chang (pers. comm.)
India (Tamil Nadu and Karnataka)	Unknown	PSP	First record, 1988	PSP, DSP found in shellfish harvesting beds, Karnataka	Baht (1981); Karunasagar et al. (1984, 1989)
Rep. of Korea (mainly Jinhae Bay)	26 species	Various	Chronic, increasing	"Severe damage" to farms	Park et al. (1989)
Malaysia (peninsular)	*Chattonella marina*	Shrimp kill	First report, 1985	Heavy losses in shrimp farms	Khoo (1985)
Malaysia (Sabah)	*P. bahamense* var. *compressum*	PSP	Chronic since 1976	Experimental cultured oysters toxic	Ting and Wong (1989)
Papua New Guinea	*P. bahamense* var. *compressum*	PSP	Maybe cyclic	Experimental cultured oysters toxic	Maclean (1989d)
Philippines	*P. bahamense* var. *compressum*	PSP	Increasing since first record, 1983	Caused mainly by eating cultured mussels	Gonzales (1989a)
Singapore	*Cochlodinium* *Chattonella* *Heterosigma*	Fish kills	Occasional	Mortality of groupers in cages	Lim (1989)
Taiwan (Tungkang)	Probably *Alexandrium tamarense*	PSP	First record, 1986	Caused by eating cultured clams (*Soletellina*)	Hwang et al. (1989); Su et al. (1989)
Thailand (Gulf)	Various	Fish kills	Chronic, increasing		Suvapepun (1989)
Venezuela	*Gymnodinium catenatum*	PSP		Stifled developing mussel culture industry	S. Hall (pers. comm.)

bioassays, or lack of recognition of a problem. The list given in Table 6 is no doubt incomplete.

Implications for Freshwater Aquacuture

The potential impact of toxic freshwater algae on aquaculture in developing countries, particularly in the tropics, depends to a large extent on resolving the paradox that: (a) lethal cyanobacterial blooms are common in temperate freshwaters; (b) resulting losses of fish crops and human illnesses are rare; and (c) the same algal species do not appear to be toxic in the tropics. It is worthwhile reviewing cyanobacteria toxicity before assessing its possible impact on aquaculture.

The prevalence of toxic cyanobacterial blooms can be gauged by a recent survey in Finland (Sivonen et al. 1990) in which 44% of 188 water bloom samples in fresh and brackishwater during 1985 and 1986 were found to be lethally toxic, containing either hepato- or neurotoxins. The authors noted that blooms were more common in the south, which was expected in view of the higher

Table 6. Potential hazards from various toxic algae to farmed aquatic organisms in some developing countries.

Algal species	Country (Locality)	Hazard	Source/Comments
Aphanizomenon spp.	Many countries	PSP in freshwater clams	
Chattonella spp.	Many countries	Fish kills	e.g., Singapore (Lim 1989); Philippines (Manila Bay) (Y. Fukuyo, pers. comm); India (Malabar coast) (Subrahmanyan 1954)
Dinophysis spp.	Many countries	DSP in shellfish	e.g., several *Dinophysis* species in Papua New Guinea (Maclean 1989a); Indonesia (Adnan, unpubl. data); Côte d'Ivoire (Dandonneau 1971); Mediterranean Egypt (Dowidar 1974); Puerto Rico (Margalef 1957); Thailand (Suvapepun 1989)
Gonyaulax monilata	Ecuador Pakistan (Arabian Sea)	Fish kills	Unesco (1982); Rabbani et al. (1990)
Microcystis aeruginosa	Many countries	Hepatoenteritis in humans; fish kills	Inadequate data to determine risk
Protogonyaulax sp.	Mexico (Gulf)	PSP	Described from Mexico; toxic isolates
Pyrodinium bahamense var. *compressum*	Costa Rica (Pacific) El Salvador Guatemala (Pacific) Mexico (Pacific)	PSP in cultured shellfish	Extensive *Pyrodinium* red tides causing PSP illnesses on the Pacific coast of Central America, November-December 1989. Previous Nicaragua (Pacific) episodes; also in Guatemala (see text)
	Fiji	PSP in shellfish	Causative organism unknown; most probably *Pyrodinium*. Five cases between 1976 and 1983 (Raj 1983)
	Indonesia	PSP in fish and shellfish	*Pyrodinium* not confirmed, but presumed by Adnan (unpubl. data); deaths from eating planktivorous fish, *Sardinella* sp. and *Selaroides* sp. (Adnan 1984)
	Palau	PSP in *Pyrodinium*	Cultured by Harada et al. (1982)
Unknown sp.	India	PSP in shellfish	In coastal shellfish (Sagar et al. 1988)
	Singapore	PSP in shellfish	PSP occurred in mussels (Tan and Lee 1986)
	Solomon Islands	PSP, ciguatera	PSP from oysters (*Crassostrea* spp.) affects 2 per 1,359 population; ciguatera "infrequent and unpredictable" (Eason and Harding 1987)
	Thailand (Gulf)	PSP	(Kodama et al. 1988)

population, industry, agriculture and forestry there. Carmichael et al. (1990) similarly stated that as waterbodies become more eutrophic they support higher production of cyanobacteria. Cyanobacterial bloom samples from 15 British freshwaters over recent years have all been lethal although mortalities to animals drinking water were recorded on only two occasions (Codd and Poon 1988).

The mode of ingestion of these toxins is by drinking the contaminated water. However, saxitoxins have been found in freshwater clams (Y. Fukuyo, pers. comm.). Thus, paralytic shellfish poisoning from cultured freshwater bivalves is a distinct possibility.

Toxicity levels per unit biomass of cyanobacteria are known to vary widely from week to week and even within an individual bloom on a single occasion (Codd and Poon 1988).

Another confounding feature is that isolates of a single species may or may not contain toxins (Codd and Poon 1988), a feature shared with some dinoflagellates. There is mounting evidence that, for saxitoxins at least, the toxin source may be bacterial symbionts rather than endogenous (Kodama 1990; Tamplin 1990).

What triggers toxin production? Production of toxins in a marine cyanobacteria (*Synechococcus* sp.) varied with salinity, temperature, light intensity and most interestingly with growth phase; no toxins were produced in the exponential growth phase but appeared suddenly during the stationary phase (Mitsui et al. 1989). For several dinoflagellate species, toxin production in cultures during the exponential growth phase was found to increase at lower temperatures as a result of lower growth rate (Hall 1982; Ogata et al. 1989).

In warmer latitudes, cyanobacteria tend to dominate in summer and when waterbodies become more eutrophic; *Microcystis aeruginosa* is frequently dominant in wastefed fishponds (Colman and Edwards 1987). Yet there are no reports of fish mortality from cyanobacterial poisons in tropical waters such as Lake George (Uganda) and tropical wastefed fishponds (Colman and Edwards 1987).

There is limited evidence that higher temperatures may suppress toxin production. For *M. aeruginosa* the optimum temperature for toxin production is about 25°C. The effects of light intensity are ambiguous (Codd and Poon 1988). The strains used in experiments appear to be from temperate sources. Would tropical strains behave differently? The tropical blooming cyanobacteria *Cylindrospermopsis (Anabaenopsis) raciborskii* was recently found to be severely hepatotoxic, apparently causing a large outbreak of hepatoenteritis from a tropical Australian reservoir (Hawkins et al. 1985). The authors noted that hepatoenteritis is common in many tropical countries and that *C. raciborskii* is one possible cause. The species blooms in water warmer than 25°C, although the toxicity tests of Hawkins et al. (1985) were only at 25°C.

Testing at higher temperatures of tropical freshwater blooms for toxicity and effects on fish is needed to resolve these issues. New reliable assay methods are needed also (Codd and Poon 1988).

As far as growth of the cyanobacteria themselves is concerned, the optimum temperatures may be higher than those noted above. Overall maximum rates of photosynthesis and growth are 25°C or greater; 27.5-32°C for *M. aeruginosa* (Roberts and Zohary 1987).

Toxicity to Fish

Phillips et al. (1985b) exposed rainbow trout (*Oncorhynchus mykiss*) to a bacteria-free culture of *M. aeruginosa*. The algae proved to be harmless in this form, presumably because the trout would not ingest them, although the algal toxicity was proven by intraperitoneal injections of ultrasonically broken cells from the culture, which were lethal to the fish. The authors cited two 1948 reports of an instance where decomposing *Aphanizomenon flos-aquae* proved toxic to a range of tank-held fish species in a river in Wisconsin, USA; while Tabthipwon et al. (1988) suspected that toxins from *M. aeruginosa* retarded growth of experimental tilapia (see below).

Colman and Edwards (1987) cited experiments in which carp, probably *Cyprinus carpio*, exhibited erratic behavior and mortality when fed a mixture of live *Aphanizomenon* and *Microcystis*; and mortality of golden shiner *Notemigonus crysoleucas*, when toxin from *A. flos-aquae* was added to the water. As with the study by Phillips et al. (1985b), interperitoneal injections were toxic in both experiments.

Phillips et al. (1985a) summarized the adverse effects of algae on farmed (and wild) fish populations as related not only to the toxins but also to increased turbidity, diurnal fluctuations of dissolved oxygen and increased concentrations of harmful metabolites during bloom die-offs.

Other than cyanobacteria, the freshwater dinoflagellate *Peridinium polonium*, one of several *Peridinium* species which bloom in many artificial lakes in Japan, produces an ichthyotoxin comparable in strength to that of the Florida red tide organism *Ptychodiscus brevis (Gymnodinium breve)*, and causes occasional fish kills (Oshima et al. 1989).

Much future aquaculture development in freshwater in developing countries will take place in relatively stagnant waters, such as flooded ricefields and ponds, and in cages in lakes and reservoirs.

The riceland available for rice-fish farming is enormous: there are 77 million hectares of irrigated riceland, for example (IRRI 1988). It must be remembered that cyanobacteria, through their nitrogen-fixing ability, play a vital role in building soil fertility; in addition they also produce substances that promote the growth of rice plants (Roger and Kulasooriya 1980). Thus, farmers encourage blooms. If fish production further encouraged cyanobacterial blooms through increased eutrophication, one would expect that there would be potential for poisoning not only the fish but also livestock and perhaps even humans downstream. However, this has not proven to be the case: where rice-fish culture is practiced, there have been no reports of illnesses.

In fishponds, cyanobacteria may play a major role in nitrogen fixation (Lin et al. 1988) and as a direct feed for phytoplanktivorous fish like some of the tilapias, including the most promising species, *Oreochromis niloticus*. Recent work has shown that 90% of fish yield in organically manured ponds come from food webs originating with algae (Schroeder et al. 1990) Yet their potential toxicity has rarely been taken into account. *O. niloticus*, in fact, seemed to be retarded in its growth when *Microcystis aeruginosa* was a significant part of the diet and, indeed, liver deterioration of the fish was found at higher levels of *Microcystis* in the feed (Tabthipwon et al. 1988). Recall that *M. aeruginosa* produces lethally toxic hepatotoxins (Carmichael et al. 1990). Codd and Poon (1988) noted that the algal genera which are typically dominant are those with toxin-producing species.

The area of freshwater lakes and reservoirs in Asia alone is some 13-14 million ha of which reservoirs currently comprise 5.5 million ha (De Silva 1988). Asian reservoir area may increase to 20 million ha by the year 2000 (Costa-Pierce and Soemarwoto 1987). The potential for aquaculture development in these waterbodies is large.

Algal blooms occur in such waterbodies and can be cropped by fish. For example, in the Saguling Reservoir, Indonesia, there are constant blooms of *M. aeruginosa* and the introduction of phytoplanktivorous fish for culture and capture fisheries was suggested (Costa-Pierce and Soemarwoto 1990; Munro et al. 1990). Again, there appears to be potential for poisoning of fish or mammals drinking the water.

Beveridge (1984) provided a model to calculate the aquaculture carrying capacity of enclosed waterbodies based on phosphorus values as a measure of the maximum algal biomass permissible, that maximum depending on the other purposes for which the water is used. Stirling and Dey (1990) observed that such models, being based on annual nutrient loads, overlook the possibility of short-term deleterious blooms. However, they recommend an upper limit of fish production in shallow well-mixed lakes of 3-4 $t \cdot ha^{-1}$.

An elegant method of determining whether aquaculture activities contribute significantly to eutrophication in tropical enclosed waterbodies was provided by Costa-Pierce (1990). Through multiple regression analysis of water quality at several sites in an Indonesian reservoir, he determined that aquaculture, in this case cage culture of carp (*Cyprinus carpio*), had negligible impact on plankton compared to the effect of other endogenous and exogenous nutrients.

Freshwater algae themselves are now being grown for human food. The cyanobacteria *Spirulina* is the best known genus, used as a food supplement. But other genera such as *Anabaena*, which includes toxic strains, are coming on the market, posing potential health hazards (Gorham and Carmichael 1988).

Tropical countries would seem to have advantages for growing algal foods: strong sunlight and extensive ponds and paddies from which the algae might be harvested. Research is needed to identify and assess any hazards posed before this form of aquaculture develops. Monitoring toxicity of cultured blooms may become a routine expense.

Management Measures

Management at the Industry Level

Strategies for aquaculturists to offset the impact of toxic blooms include a variety of physical and chemical techniques.

MECHANICAL/PHYSICAL MEASURES

In Singapore, where affected fish cages and toxic plankton blooms are in the upper two meters, suggested steps to minimize contact between algae and fish include: a PVC "skirt" at least 2 m deep to surround individual cages or the whole farm; transfer of fish to deeper nets, 4 m deep; an airlift or water pump to draw up deeper water to disperse the algae; thinning out of the stocking density of sensitive fish; and towing of cage assemblages to safe areas (Lim 1989).

A management method for mussel beds in Samar, Philippines, was hinted at by Ronquillo (1987) in his observation that the poles on which the mussels grew were too close together and led to pollution, which provided nutrients for *Pyrodinium* to bloom. Moving the poles apart might encourage water movement and less waste buildup.

Adjusting depths of bivalves in cages or on longlines is unlikely to be a safe option. Maclean (1975) found toxic bivalves at 10 m depth in Port Moresby, Papua New Guinea, below depths of *Pyrodinium* blooms. Scallops on longlines in Funka Bay, Japan, were found to contain little toxin in the uppermost and lowest parts of the longlines but were highly toxic in the middle sections (S. Hall, reporting a presentation by V. Nishihama, pers. comm.). Recent experiments in eastern Canada showed that mussel growers cannot avoid toxicity in cultured mussels by altering the depth of holding structures due to the changing distribution of PSP toxin (from *Alexandrium excavatum*) in the mussels with depth over time (Desbiens et al. 1990).

Jones and Gowen (1985) modeled flushing rates and phytoplankton growth in Scottish sea-lochs and suggested that considerations of exchange and source water hydrography be included in aquaculture site selection there.

Dale et al. (1987) went further. They considered that a hydrographic survey should be undertaken as part of an aquaculture site selection process, followed by a pilot phase in which environmental parameters continue to be monitored; finally an ecological model should be set up to predict the occurrence of blooms. They also recommended that the literature be scanned to identify potentially harmful algae in the area; that sediments be examined for dinoflagellate cysts; and that available remote sensing data be obtained. However, it is difficult to imagine such guidelines being implemented in most developing countries.

CHEMICAL DISPERSANTS

Hallegraeff (1987) cited an earlier report on spraying copper sulfate from aeroplanes, which killed *Gymnodinium breve* blooms but caused anoxia when cells decomposed. White (1988) said that application of ammonium sulfate to brackishwater fishponds kills incipient blooms of *Prymnesium parvum.*

Chemical removal of a bloom of *Chattonella antiqua* in Shido Bay, Japan, was accomplished using 50 ppm of sodium percarbonate (Okaichi et al. 1989b). The bloom disappeared within two hours of application; the algae apparently sank and encysted. The area was suffering mortality of cultured yellowtail due to the *Chattonella* bloom at the time.

There does not seem to have been any further progress on using such chemicals but they may have a future role as for dispersants used in certain oil spill situations.

OZONATION

Ozone treatment was earlier reported to be effective against PSP toxins and brevetoxin (reviewed in Rosenthal 1981). Dupree (1981), summarizing previous literature, felt that 1-5 minute contact time at 0.56-1.0 mg $O_3 \cdot l^{-1}$ was sufficient to kill most pathogens but that ozone had high toxicity in itself, especially to oyster larvae. Blogoslawski (1988) concluded that ozonized seawater could not detoxify clams that had ingested cysts or had toxin bound in their tissues over long periods of time, but could destroy PSP in motile dinoflagellate cells.

There has been one experiment on the effect of ozone on *Pyrodinium*-toxic mussels (*Perna viridis*), in the Philippines (Gacutan et al. 1984), which reported that ozone was effective in lowering toxin levels, although rather slowly under the experimental conditions. The authors reported that PVP-iodide was also effective.*

DEPURATION

Experiments using clean seawater for toxin reduction in mussels (*Mytilus edulis*) in Korea showed that filtered seawater in an open system caused toxicity to drop by 40-92% over five days (Chang et al. 1988). White (1982) cited earlier experiments on clams and mussels with similar results. For *Pyrodinium*, toxic oysters (*Crassostrea echinata*) took three weeks to lose toxicity in a filtered open seawater system (Maclean 1975).

In vivo, however, depuration takes considerably longer. Shumway (1990) provided an extensive list of retention times of various bivalves exposed to different toxic dinoflagellate sources. From the list, an average figure would be 4-8 weeks for toxicity to drop below quarantine or detection levels, depending on mollusc and algal species. Thermal shock is also reported to speed up depuration (Blogoslawski and Neve 1979).

HEATING/CANNING

PSP toxins are largely unaffected by ordinary cooking methods and their activity is potentiated by the use of acid (vinegar) in preparations common in countries such as the Philippines.

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*New results on rapid destruction of *Gymnodinium breve* toxins by Schneider and Rodrick (in press) by both direct and indirect (pre-ozonated water) ozone treatment hold promise of safe, effective toxin removal. Eds.

Detoxification of PSP toxins through canning is a little explored area, although a standard canning process can reduce toxin levels in scallops from 400 $MU \cdot g^{1}$ to less than 4 $MU \cdot g^{1}$. Most of the toxin reduction is due to the process while the remainder takes place over 30 days storage, according to Noguchi et al. (1980). An earlier report by Schantz (1973, cited by Arafiles et al. 1984) indicated that canned and processed shellfish may still contain up to 50% of the toxin even if the product has been heated to 115°C.

There does not seem to have been much progress or interest in this field since the above papers. However, White (1982) suggested canning as a viable option for Bay of Fundy clams in the face of increasingly toxic *Gonyaulax* blooms.

There is no chemical explanation for toxin breakdown or deactivation at the temperatures involved and the phenomenon may be simply a redistribution of toxin between meat and liquid parts of the canned shellfish (S. Hall, pers. comm.). Oshima's (1989) observation, mentioned above, that PSP toxins became distributed throughout fish tissues after 18 months frozen storage is pertinent here.

Microcystis toxin (hepatotoxin) is heat labile, such that proper cooking of fish raised in *Microcystis*-dominated ponds should render any such toxin in the fish harmless to humans [B. Hepher, pers. comm. to Colman and Edwards (1987)].

For tetrodotoxin, a combination of cooking and washing was effective in reducing highly toxic pufferfish liver to safe levels (Tsubone et al. 1986).

Management at the National Level

Red tide action plans have been produced in some Southeast Asian countries. All involve monitoring programs and public awareness. Measures related to aquaculture are: in Brunei Darussalam, a ban on harvesting and/or marketing of suspect fish and shellfish (De Silva et al. 1989); in Sabah, Malaysia, to cease picking, selling or eating all types of shellfish and snails, and even in the safe season not to consume a large amount of shellfish in one meal - children are urged not to eat shellfish at all (Wong and Ting 1989); in the Philippines a ban on harvesting, marketing and transporting of all kinds of marine shellfish from contaminated waters and provision of emergency loans to affected fish farmers (Gonzales 1989b).

In Hong Kong, where the major impact of red tides is fish kills in fish farms, simple sampling kits are issued to aquaculture representatives and government staff. On-site tests are performed by a "mobile squad" and fish farmers in the area are alerted and advised of means to minimize losses (Wong and Wu 1987).

In El Salvador, food was distributed by the government to clam harvesters who were obliged to withdraw their product from sale during the second red tide episode along the western Central American coast in April 1990 (S. Hall, pers. comm.).

Virtually every report dealing with management aspects of algal blooms calls for continual monitoring of plankton and suspect organisms, usually shellfish. As mentioned, this can be a costly exercise. In the Philippines, good progress towards a blowfly bioassay, rather than the more expensive mouse bioassay, has been made (A. Mendigo, pers. comm.).

No consideration seems to have been given by governments in developing countries to zoning as a means of setting aside for aquaculture, sites where pollution levels can be kept low and red tide incidence is unlikely. Indeed, in Southeast Asia at least, red tide management plans are somewhat fatalistic, assuming permanent problems and neglecting (probably for economic reasons) the rezoning of aquaculture operations, reduction of pollution or engineering works to alter current patterns (e.g., Corrales and Gomez 1990).

Remedial management has been attempted elsewhere. For example, the number of red tides in Japan's Seto Inland Sea dropped markedly, beginning four years after the passing of the 1972 Seto Inland Sea Environment Conservation Law (see Fig. 3) (Okaichi 1989). The law reduced land reclamation and required treatment facilities to reduce the chemical oxygen demand (COD). Reduction of pollution lessened the number of red tides. Yet, the number of fish kill events has not been decreasing and the value of lost fish harvests has been increasing (Okaichi 1989).

For algae which are not clearly associated with eutrophication, the physical habitat itself would need to be altered to be less favorable for the occasional dominance of toxic species. In fact, the decimation of mangrove forests around Southeast Asia may be one reason why toxic blooms of *Pyrodinium* are not more common in the region. A bioluminescent lagoon in Puerto Rico, caused by a "resident" population of *Pyrodinium*, lost its glow when a new channel to the sea was created; the channel increased the flushing rate of the lagoon and the *Pyrodinium* could no longer maintain their presence (H.H. Seliger, pers. comm.).

Coastal engineering may be a solution to reduce eutrophication and associated blooms and anoxic conditions. Dredging operations, however, invoke the danger of seeding benthic dinoflagellate cysts into the water column (Hallegraeff 1987).

Table 7 presents some of the present measures used by governments and suggests some alternatives. Moving an affected industry to a "clean" location is an option that could be less expensive than extensive monitoring and attempts at pollution abatement; harvesting red tide organisms is not a cost-effective measure at present and would depend on profitable use being found for chemicals from the organisms.

Similarly, coastal engineering options have to be considered against the value of the threatened or potential aquaculture (or fisheries) harvests in the area, as well as other environmental impact aspects. As aquaculture expands, the economic and public health incentives to change the shape of a bay or move a river mouth, for example, may become comparable to those that lead to massive dam construction for agriculture, flood control or drinking water. So far the whole field of algal blooms has been dominated by biologists and toxicologists.

Table 7. Present management concepts and alternative approaches to dealing with algal blooms in aquaculture.

Present concept	Alternative approach
Routine monitoring	"Once only" movement of aquaculture industry
Move cages out of red tide event by towing	Remove red tide events by coastal engineering
Attempts to lower pollution levels	Rezone aquaculture away from pollution
Use chemical dispersants in aquaculture crises	Harvest red tides
Insure, make grants/loans to affected farmers	Abandon (legislate against) aquaculture in the affected area

Imaginative thinking on long-term and large-scale solutions is lacking.

Management at the International Level

Some form of regional or international cooperative activity is usually recommended at the various international fora on red tides, but until recently little had been done. Dale et al. (1987) recommended a "red tide brigade" of international experts. Something of the kind was subsequently formed by the Intergovernmental Oceanographic Commission's Ocean Science and Living Resources Group which recently included red tide as a major thrust with the formation of the (OSLR/IOC) Group of Experts on Harmful Algal Blooms. The Western Pacific Group (WESTPAC) of the IOC also has a red tide section sponsoring research and workshops, while the United Nations Environment Programme is setting up regional management projects of an institution-building nature. The International Council for the Exploration of the Sea (ICES) investigates algal blooms through a Working Group on Phytoplankton and the Management of their Effects.

An International Red Tide Information and Assistance Service (IRTIAS) is being formed at Woods Hole Oceanographic Institution to assist developing countries especially and which will include advice on preventive measures for aquaculturists (White 1990b).

Aquaculture and Red Tides: An Uneasy Association

Some authors feel that aquaculture contributes to conditions favoring red tides. Smayda (1989) stated that "shellfish and finfish aquacultural activities are often followed by blooms of both benign and toxic algal species." He only gave one example but noted that "aquacultural activities augment and modify the local grazing structure as do benthic and pelagic fishery operations" and that this may be a cause of blooms. Hallegraeff (1990) also said that "the effluent from fish farms stimulates algal blooms".

The evidence is circumstantial but sometimes persuasive. Anderson (1989) pointed out that PSP in one Philippine site (Balete Bay, Davao Oriental) was first recognized just one year after mussel (*Perna viridis*) culture was introduced there. Corrales and Gomez (1990) noted that red tide sites in the Philippines are mostly sites of mussel farming. Port Moresby in Papua New Guinea, where the first recorded outbreak of *Pyrodinium* in the Indo-Pacific took place, was also the site of a large pearl oyster farm at the time. However, the larger picture does not support an association between *Pyrodinium* and aquaculture (or fishery) operations. Neither activity was present in other outbreak sites in Papua New Guinea or elsewhere.

A much closer relationship exists between *Pyrodinium* and mangroves. The only bloom of *Pyrodinium* where mangroves have not been in the general vicinity is western Manila Bay, Philippines, where, however, there used to be mangroves (Maclean 1989d). Margalef (1957) had earlier placed the nontoxic variety of *Pyrodinium* (*P. bahamense* var. *bahamense*) as a member of the mangrove community. Mangrove areas are generally sheltered and cheap to lease for which reason most coastal aquaculture in the Indo-Pacific region takes place in these areas - the habitat of *Pyrodinium*.

Another interesting feature of *Pyrodinium* is that the fossil record of cyst distribution is much broader latitudinally than the present distribution of the species (Matsuoka 1989).

For other harmful microalgae, particularly those enhanced by forms of pollution (see below), it is difficult to reject the hypothesis that the increased nutrients around fish, shrimp or mollusc farms might stimulate algal blooms.

Dispersal of red tide organisms via coastal or ocean currents is a known, and perhaps the chief mechanism, a recent interesting example being that of a bloom of the Florida red tide organism *Gymnodinium breve* which was carried several hundred kilometers northward to North Carolina in the Gulf Stream (Tester et al. 1989). Seliger (1989) lists a number of areas where toxic blooms of *Gymnodinium* and *Alexandrium* first appeared in 1971/1972, as a result of water movements probably attributable to El Niño during that period. Further, there is the possibility of long-distance dispersal of red tide organisms in ballast water (Maclean 1977; Hallegraeff et al. 1990) which poses dangers for aquaculture. Such organisms have been proven capable of surviving long voyages in ships' holds and may be carried into important aquaculture areas; regulation of ballast water discharge is called for (Hallegraef et al. 1990). There is also the possibility that the organisms may be spread through spread of cultured aquatic animals, particularly bivalves, to new growing areas, as in the Philippines, for example.

Two Types of Bloom Organisms

There seem to be two major types of marine blooms - those in which nutrient additions to coastal systems are obviously implicated and those in which coastal enrichment is not an obvious factor. Organisms such as *Gymnodinium breve*, some *Alexandrium* (*Gonyaulax*) species and *Pyrodinium* fall into the second category according to these authors. Perhaps *Gambierdiscus* belongs here also. One environmental factor that triggers major blooms may be El Niño. As noted above, Seliger (1989) felt that this was so for *Gymnodinium* and *Alexandrium*. The case is even stronger for *Pyrodinium*, for which all major outbreaks in the western Pacific since 1972 correlate with El Niño-Southern Oscillation (ENSO) events (Fig. 4).

Coastal enrichment, attributable to human activities, is almost certainly involved for other species and situations, as in the Seto Inland Sea (*Chattonella* spp.) (Okaichi 1989), in Jinhae Bay, Korea (Park et al. 1989), and Hong Kong (Wong and Wu 1987). The last two areas host a cocktail of bloom species. In Hong Kong, the evidence of human impact is most compelling (see Fig. 2) (Lam and Ho 1989).

Where does this leave the relationship between aquaculture and algal blooms? If the two types of blooms - either obviously associated with coastal enrichment or not - are treated separately, some future scenarios can be drawn.

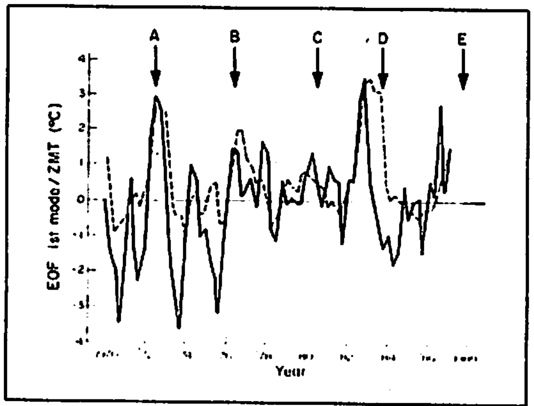

Fig. 4. Major toxic red tides in the western Pacific and ENSO events, 1972-1987. Graph shows empirical orthogonal functions (EOF, solid lines) of zonal wind anomalies, and zonal mean surface temperature anomalies (ZMT, broken line) over the near-equatorial eastern Indian and western Pacific Oceans. Strong positive anomalies are indicative of ENSO events and are seemingly correlated with *Pyrodinium* red tide events. Arrows show time of onset of red tides: A: Papua New Guinea, 1972; B: Borneo, 1976: C: Borneo, 1979-80; D: Philippines, 1983; E: Philippines, 1987. From Maclean (1989c).

Some Scenarios

What does the environmental future of developing-country aquaculture hold with respect to algal blooms? Obviously, either enrichment levels will be contained, increase or decrease.

In the unlikely event of decreasing levels of coastal enrichment, dominant algal species may revert to a mixture of more benign diatoms and of dinoflagellates that bloom with little (if any) enrichment, such as *Pyrodinium* in tropical waters. In this case, the affected countries can look forward to either regular or in the longer term cyclical toxic events. The short-term (but how short?) outlook is for chronic seasonal blooms that require monitoring and seasonal closures. The long-term perspective is for occasional acute events that may initially cause extensive losses of lives and investment (because monitoring activities will usually have lapsed between cycles).

If present levels of coastal enrichment can be contained, the outlook is for an ongoing uncertainty and lack of predictability about both the species and the severity of future red tides. Monitoring and insurance costs, occasional losses, public health concerns and uncertain investment returns may combine to make surviving coastal aquaculture products much more expensive.

Increased enrichment, if recent history is any guide, can only lead to a broader spectrum of toxic species or more or less continuous presence of a few dominant toxic species. Nuisance algal species which respond to coastal enrichment will increase in number and frequency of blooms as the level of enrichment increases. Most of these species cause fish kills through toxin production or by causing anoxic conditions. Aquaculture of finfish in such circumstances will become increasingly incompatible. Aquaculture operations themselves may contribute significantly to eutrophication, exacerbating the situation. If aquaculture continues, mollusc culture would be more suitable in these areas, since there is no additional nutrient input from feeding.

With regard to blooms that appear to be independent of coastal enrichment, the situation for aquaculture is unpredictable because the species appear to be spreading in geographic distribution. Most of them produce PSP toxins. Generally speaking finfish and crustacean culture would be more successful in such areas, because these organisms are neither susceptible to nor accumulate saxitoxins. Note that aquaculture activities in these areas may lead to eutrophication and perhaps a "succession" of bloom organisms towards those that obviously respond to enrichment.

K. Matsuoka (pers. comm.) suggested that a succession may be occurring in Manila Bay and nearby Zambales where *Pyrodinium* blooms have occurred in recent years. Noxious algal species such as *Chattonella* and *Alexandrium* were found in significant numbers there in a July 1990 survey in which he took part.

In enclosed freshwater waterbodies, given their multiple use, especially in tropical developing countries, cyanobacteria blooms are almost a certainty. Will these tropical blooms suddenly become toxic, as have other (marine) algal species? If the hypothesis is correct that bacterial symbionts (or parasites?) are responsible for toxicity in algae, then the "on" switch, i.e., an algal population assimilating toxic bacteria, may be easier than an "off" switch, i.e., an algal population losing its symbionts/parasites.

Finally, the prospect of abandoning aquaculture, e.g., through legislation against it in view of public health hazards that cannot be overcome reliably, is an alternative, which I found on sharing this manuscript, to have few supporters. For example, J.W. Hurst, Jr. (pers. comm.) wrote "I do not regard 'red tides' as an insurmountable barrier to the development of aquaculture. It must be understood that while the present methods for determining the safety of shellfish are expensive they are a necessary part of determining their safety."

My view is perhaps a jaded one in the light of the uncontrolled coastal developments taking place in most countries. There are so many sectors competing for use of inshore waters that the future of coastal aquaculture may be limited by them as much as by algal blooms. Red tides may be the "straw that breaks the camel's back" in this respect. However, I take heart from Dr. Hurst's (pers. comm.) conclusion that "Persons who are Third World nationals deserve high quality safe seafood and should be regarded as the most important customers of the aquaculture industry of that nation. This attitude will assure a high quality and safe product."

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