

A Review of the Effects of Algal Blooms on Shellfish and Aquaculture

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Abstract

Toxic algal blooms occur worldwide and in some areas they are a common and seasonal occurrence. Historically, attention has been focused on blooms of toxic dinoflagellates (e.g., *Protogonyaulax tamarensis*). More recently, attention has been turned to other species (e.g., *Dinophysis*, *Aureococcus*, *Gymnodinium*). These blooms often present problems with respect to optimal utilization of the shellfish resources, and the magnitude of economic losses can be catastrophic. Nevertheless, successful culture facilities and commercial harvests persist in areas prone to toxic algal blooms.

This paper reviews the literature available on occurrences of toxic algal blooms, discusses the means by which harvesters, managers, and industry cope with the problems associated with toxic algal blooms, and makes recommendations for the most efficient and successful utilization of resources in the face of environmental instability.

Occasionally, in response to favorable changes in their environment, certain algal species undergo a rapid population increase when their numbers may attain several million cells per litre and may form visible patches on the surface referred to as "red tides." Actual "bloom" concentrations vary among species, e.g., 10^5 cells/L constitutes a bloom of *Protogonyaulax* whereas 10^9 cells/L were present during blooms of *Aureococcus anophagefferans* (Casper et al. 1987). These blooms may or may not be visible and they may or may not be red. Some of the most devastating blooms of late were actually brown (Tracey 1988; Casper et al. 1987; 1988). Red water blooms are predominantly a surface phenomena, driven by winds and currents. There is evidence that nearly all, if not all, major blooms originate in the ocean and not within bays (Holligan 1985). These blooms may be either toxic or noxious, that is either producing specific toxins or causing anoxia through the decay process or, in some cases, simply clogging the gills of filter-feeding animals. In addition, blooms can appear and render shellfish toxic virtually overnight.

The presence of toxic algae and the potential for blooms have clear, negative effects on the development of aquaculture (Taylor

1989). Not only do these outbreaks pose a threat to public health (numerous deaths have been attributed to paralytic shellfish poisoning over the years), but they also are responsible for mass mortalities of shellfish and they can result in great economic hardship to the coastal fishing industries and aquaculture facilities. The problems associated with toxic algal blooms are no longer limited to the dinoflagellates and are becoming increasingly severe on a global scale (Table 1; Fig. 1). There have been a number of studies and symposia which have focused on the most predominant species of toxic dinoflagellates, e.g., LoCicero (1975), Taylor and Seliger (1979), Anderson et al. (1985), Okaichi et al. (1989), Dale et al. (1987), Parker and Tett (1987), Shumway (1988), and Granéli (1990).

Recent algal blooms attributed to a previously undescribed chrysophyte were responsible for the reduction in extent and biomass of eelgrass beds and caused starvation and recruitment failure of commercially important bay scallop populations in Long Island waters (Casper et al. 1987). These so-called "brown tides" were also responsible for the near elimination of mussel populations in certain areas of Narragansett Bay (Olsen 1986; Sieburth et al. 1986). Areas

TABLE 1. A summary of toxic and noxious algal blooms and their effects on shellfish. Taxonomic names appear as in the original publications.

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Dinophysis acuminata</i>	<i>Mytilus edulis</i>	highly toxic	Netherlands	Kat (1983; 1985; 1989)
<i>Dinophysis acuminata</i>	<i>Chlamys nipponensis</i>	toxic	Japan	Anraku (1984)
<i>Dinophysis fortii</i>	<i>Fulvia mutica</i> <i>Gomphina melan-aegis</i> <i>Mactra chinensis</i> <i>Mytilus coruscus</i> <i>Mytilus edulis</i> <i>Meretrix la-marchii</i> <i>Patinopecten yes-soensis</i> <i>Pecten albicans</i> <i>Ruditapes philippinarium</i>			
<i>Dinophysis acuminata</i>	<i>Mytilus edulis</i>	toxic	Netherlands	Kat (1983; 1985)
<i>Dinophysis acuta</i>	<i>Mytilus edulis</i>	DSP	Sweden	Edler and Hageltorn (1990)
<i>Dinophysis sacculus</i>	shellfish	ban on marketing	France	Lassus and Berthome (1988)
<i>Dinophysis</i> spp. including: <i>acuta</i> <i>acuminata</i> <i>norvegica</i>	<i>Mytilus edulis</i>	highly toxic; remained toxic for up to 7 months	Sweden, Norway, Denmark	Underdal et al. (1985); Yndestad and Underdal (1985); Krogh et al. (1985); ICES (1988)
<i>Dinophysis</i> spp.	<i>Mytilus edulis</i> , <i>Mercenaria mercenaria</i> , <i>Mya arenaria</i>	"probably source of DSP"	New York, USA	Freudenthal and Jijina (1988)
<i>Dinophysis sacculus</i>	<i>Mytilus</i> sp.	DSP; first report from area	Portugal	Alvito et al. (1990)
<i>Dinophysis acuta</i>	<i>Cerastoderma</i> sp.	toxic	Portugal	Sampayo et al. (1990)
<i>Dinophysis</i> spp.	<i>Crassostrea</i> sp.	DSP	Karnataka coast, India	Karunasagar et al. 1989
<i>Prorocentrum</i> sp.	<i>Katelysia</i> sp. <i>Meretrix</i> sp. <i>Paphia</i> sp.			
<i>Dinophysis</i> sp. <i>Prorocentrum</i> sp.	<i>Mytilus edulis</i>	DSP; first report from area	East Friesian Wadden Sea, Germany	Meixner and Luckas (1988)
<i>Prorocentrum micans</i>	<i>Mytilus edulis</i>	40–50% mortality; probably due to low oxygen	Northern Brittany	Lassus and Berthome (1988)
<i>Prorocentrum micans</i>	<i>Donax serra</i>	toxic	Cape Town, South Africa	Horstman (1981) (Personal communication)
<i>Prorocentrum micans</i>	<i>Cardium edule</i> <i>Mytilus edulis</i> <i>Tapes decussatus</i>	toxic; PSP	Portugal	Pinto and Silva (1956)

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Prorocentrum minimum</i>	<i>Tapes japonica</i>	highly toxic; human fatalities	Japan	Nakazima (1965a; 1965b; 1965c; 1968)
<i>Prorocentrum minimum</i>	oysters	mortalities in old animals	South Atlantic coast France	Lassus and Berthome (1988)
<i>Prorocentrum</i> sp.	<i>Mercenaria mercenaria</i>	reduced growth	Long Island Sound	Wikfors (personal communication)
<i>Exuviaella mariae</i> <i>Lebouriae</i>	<i>Venerupis</i> <i>mi-</i> <i>descussata</i> <i>Ostrea gigas</i>	over 100 human deaths; and 300 illnesses	Japan	Nakazima (1965a; 1965b; 1965c; 1968)
<i>Gonyaulax tamarensis</i>	<i>Cerastoderma edule</i> <i>Lucinoma borealis</i> <i>Macoma blathica</i> <i>Venus striatula</i> <i>Pecten maximus</i> <i>Mytilus edulis</i>	moribund or dead moribund or dead moribund or dead highly toxic; not adversely affected	Northumberland United Kingdom	Adams et al. (1968); Ayres and Cullum (1978)
<i>Gonyaulax tamarensis</i>	<i>Pecten maximus</i> <i>Chlamys opercularis</i> <i>Mytilus edulis</i>	toxic	Northumberland United Kingdom	Ingham et al. (1968)
<i>Gonyaulax tamarensis</i>	<i>Perna perna</i>	first record from Caribbean; 1 human fatality	Venezuela	Reyes-Vasquez et al. (1979)
<i>Gonyaulax tamarensis</i>	<i>Mytilus edulis</i>	toxic	Spain	Fraga et al. (1984); Blanco et al. (1985); Fraga and Sanchez (1985)
<i>Gonyaulax tamarensis</i> (<i>Protogonyaulax</i>)	<i>Artica islandica</i> <i>Mytilus edulis</i> <i>Mya arenaria</i> <i>Spisula solidissima</i> <i>Placoptecten magellanicus</i> <i>Modiolus modiolus</i> <i>Thais lapillus</i> <i>Polynices heros</i> <i>Buccinum undatum</i> <i>Colus stimpsoni</i> <i>Neptunea decemcostata</i>	highly toxic	Gulf of Maine and eastern Canada; Bay of Fundy and St. Lawrence regions	Hurst (1975); Hartwell (1975); Tufts (1979); Shumway et al. (1988); Prakash et al. (1971); Caddy and Chandler (1968); Prakash (1963); Medcof (1972)
<i>Protogonyaulax catenella</i>	<i>Crassostrea gigas</i>	16 persons developed numbness of mouth	Japan	Onoue et al. (1980, 1981a, 1981b)
<i>Protogonyaulax tamarensis</i> ¹	<i>Perna viridis</i>	63 cases of PSP; 1 human fatality	Pran Buri, Southern Thailand	Tamiyavanich et al. (1985); Maclean (1984)
<i>Protogonyaulax tamarensis</i>	<i>Soletellina diphos</i>	first recorded toxic bloom in area	Taiwan	Su et al. (1989)

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Protogonyaulax tamarensis</i>	<i>Crassostrea gigas</i> <i>Chlamys nipponensis</i> <i>Patinopecten yesoensis</i> <i>Mytilus edulis</i>	toxic	Japan	Oshima et al. (1982)
<i>Gonyaulax monilata</i>	<i>Crassostrea virginica</i> <i>Donax variabilis</i>	moribund	Gulf Coast, USA	Ray and Aldrich (1965); Wardle et al. (1975)
<i>Gonyaulax monilata</i>	<i>Brachidontes recurvus</i>	mortality high at 10 ⁶ cells/L	laboratory study	Sievers (1969)
<i>Gonyaulax monilata</i>	<i>Polynices duplicata</i> <i>Thais haemastoma</i> several species of bivalves	moribund	Galveston, Texas	Wardle et al. (1975)
<i>Gonyaulax acatenella</i>	<i>Clinocardium nuttalli</i> <i>Crassostrea gigas</i> <i>Mya arenaria</i> <i>Mytilus edulis</i> <i>Protothaca staminea</i> <i>Tresus capax</i> <i>Venerupis japonica</i>	several cases of PSP; one human fatality from eating cockles	British Columbia	Prakash and Taylor (1966)
<i>Gonyaulax catenella</i>	<i>Crassostrea gigas</i> <i>Mytilus edulis</i> <i>Mytilus californianus</i> gaper clams, cockles, Washington clams, horse-neck clams and littleneck clams <i>Hinnites multirugosus</i>	toxic 1 death from eating viscera	California	Sharpe (1981)
<i>Gonyaulax catenella</i>	<i>Crassostrea virginica</i> <i>Clinocardium nuttalli</i> <i>Chlamys hastata</i> <i>Hinnites multirugosus</i> <i>Mytilus edulis</i> <i>Mytilus californianus</i> <i>Ostrea lurida</i> <i>Ostrea edulis</i> <i>Pecten caurinus</i> <i>Pecten</i> sp. <i>Protothaca staminea</i>	toxic	Pacific coast states, USA	Nishitani and Chew (1988)

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Gonyaulax catenella</i>	<i>Saxidomus gigantus</i>			
	<i>Schizothaerus capax</i>			
	<i>Siliqua patula</i>			
	<i>Aulacomya ater</i> <i>Mytilus chilensis</i> <i>Chlamys patagonicus</i>	toxic	Chile	Avaria (1979); Guzman and Campodonico (1978)
<i>Gonyaulax excavata</i>	<i>Mytilus edulis</i> <i>Buccinum undatum</i>	toxic; shellfish mortalities	Faroe Islands	Mortenson (1985); Dale et al. (1987); Gaard and Poulson (1988)
<i>Gonyaulax excavata</i>	<i>Mytilus edulis</i>	first report from area	Newfoundland	White and White (1985)
<i>Gonyaulax excavata</i>	<i>Mytilus edulis</i>	toxic	Argentina	Carreto et al. (1985)
<i>Gonyaulax excavata</i>	<i>Mytilus edulis</i>	physically clogs gills	Los Angeles, California	Oguri et al. (1975)
<i>Gonyaulax polygramma</i>	oysters		Bay of Agu, Japan	Nishikawa (1901)
<i>Gonyaulax polygramma</i>	<i>Mytilus perna</i>	mass mortalities of fish and invertebrates result of low oxygen	Cape Town, South Africa	Grindley and Taylor (1962)
<i>Alexandrium minutum</i>	mussels	PSP; first recorded from area	France	Nezan et al. (1989)
<i>Alexandrium tamaransis</i>	mussels	toxic	Kamchatka, USSR	Konovalova (1989)
<i>Alexandrium acatenella</i>				
<i>Alexandrium</i> spp.	<i>Mytilus edulis</i>	significant reductions in growth	laboratory study	Nielsen and Stromgren (1989)
<i>Protogonyaulax tamaransis</i>	<i>Perna perna</i>	toxic	Venezuela	Ferraz-Reyes et al. (1985)
<i>Gonyaulax monilata</i>				
<i>Protogonyaulax catenella</i>				
<i>Gonyaulax grindleyi</i>	<i>Donax serra</i>	mass mortalities (virtually entire populations)	Cape Town, South Africa	Horstman (1981); Grindley and Nel (1970); Popkiss et al. (1979)
<i>Gonyaulax catenella</i>	<i>Choromytilus meridionalis</i>			
<i>Gonyaulax</i> sp.	<i>Mytilus edulis</i>	toxic	Uruguay	Davison and Yentsch (1985)
<i>Protogonyaulax tamaransis</i>	<i>Chlamys nipponensis</i>	toxic	Japan	Anraku (1984)
<i>Protogonyaulax catenella</i>	<i>Chlamys nobilis</i> <i>Crassostrea gigas</i> <i>Gomphina melan-aegis</i> <i>Mytilus corusicus</i> <i>Mytilus edulis</i>			

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Gymnodium breve</i>	<i>Patinopecten yesoensis</i>			
	<i>Ruditapes philippinarum</i>			
	<i>Crassostrea virginica</i>	all highly toxic; mass mortality of <i>Spisula</i>	Florida Gulf Coast	Cummins et al. (1971); Hemmert (1975); Joyce and Roberts (1975); Tiffany and Heyl (1978)
	<i>Donax variabilis</i>			
	<i>Macrocallista nimbosa</i>			
	<i>Mercenaria campechiensis</i>			
<i>Ptychodiscus brevis</i>	shellfish	toxic shellfish; scallop mortality; recruitment failure	North Carolina, USA	Tester et al. (1988); Barris (1988); Tester and Fowler (1989); Summerson and Peterson (1989)
	<i>Argopecten irradians</i>			
<i>Gymnodinium catenatum</i>	<i>Crassostrea gigas</i>	mussels more toxic than oysters; long line cultured mussels more toxic than those cultured intertidally	Tasmania	Hallegraeff and Summer (1986); Hallegraeff et al. (1989); Dale et al. (1987); Oshima et al. (1987a, 1987b)
	<i>Equichlamys bifrons</i>			
	<i>Mimachlamys asperimus</i>			
	<i>Mytilus edulis planulatus</i>			
	<i>Pecten fumata</i>			
<i>Gymnodinium catenatum</i>	<i>Crassostrea iridescens</i>	3 human deaths; 18 illnesses (mostly juveniles)	Mexico	Mee et al. (1986) Morey-Gaines (1982)
	<i>Donax</i>			
<i>Gymnodinium catenatum</i>	<i>Venus verrucosa</i> <i>Cytherea chione</i>	PSP toxins; first report from area	Mediterranean Sea	Bravo et al. (1990)
<i>Gymnodinium catenatum</i>	<i>Crassostrea gigas</i> <i>Mytilus edulis</i> <i>Pecten albicans</i> <i>Ruditapes philippinarium</i> <i>Saxidomus purpuratus</i> <i>Scapharca broughtonii</i>	first report of toxicity by this species in Japan	Japan	Ikeda et al. (1989)
<i>Gymnodinium catenatum</i>	<i>Mytilus edulis</i>	PSP outbreaks	northwest Spain	Campos et al. (1982)
<i>Gymnodinium catenatum</i>	<i>Cerastoderma</i> sp.	PSP outbreaks	Portugal	Franca and Almeida (1989)
	<i>Mytilus</i> sp.			
<i>Gymnodinium galatheanum</i>	<i>Ruditapes</i> sp.	significant reduction in growth killed oysters	laboratory study	Nielsen and Stromgren (1989)
	<i>Mytilus edulis</i>			
<i>Gymnodinium nagsakiense</i>	pearl oysters	killed oysters	Nagasaki, Japan	Fage (1953)
<i>Gymnodinium splendens</i>	<i>Crassostrea gigas</i> <i>Venerupis japonica</i>	acutely toxic to larval stages	Puget Sound	Cardwell et al. (1979)

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Gymnodinium splendens</i>	<i>Ostrea lurida</i>	mortalities in adult and juvenile oysters		Woelke (1961); Nightingale (1936)
<i>Gymnodinium veneficum</i>	<i>Buccinum undatum</i> <i>Lasaea rubra</i> <i>Mytilus edulis</i> <i>Pecten maximus</i>	shellfish mortalities; excitable tissues blocked	laboratory experiments	Abbott and Ballantine (1957)
<i>Gymnodinium</i> sp. <i>Gonyaulax</i> sp.	<i>Mytilus edulis</i>	considerable mortality of shellfish	south coast Ireland	O'Sullivan (1978)
<i>Gyrodinium aureolum</i>	<i>Mytilus edulis</i>	some mussel deaths	Norway	Tangen (1977)
<i>Gyrodinium aureolum</i>	<i>Pecten maximus</i>	post-larvae ceased feeding; mortalities in young scallops	France	Lassus and Berthome (1988)
<i>Gyrodinium aureolum</i>	<i>Pecten maximus</i>	numbers of larvae declined during bloom	Lough Hyne, Ireland	Minchin (1984)
<i>Gyrodinium aureolum</i>	<i>Mytilus edulis</i>	reduced clearance rates; marked cellular damage in gut	Plymouth, UK	Widdows et al. (1979)
<i>Gyrodinium aureolum</i>	<i>Crassostrea gigas</i>	larval survival reduced	Conwy, Wales	Helm et al. (1974)
<i>Gyrodinium aureolum</i>	<i>Mytilus edulis</i>	significant reduction in growth	laboratory study	Nielsen and Stromgren (1989)
<i>Gyrodinium spirale</i> <i>Gyrodinium aureolum</i>	shellfish; clams	shellfish mortalities	N. Brittany	Lassus and Berthome (1988)
<i>Gyrodinium</i> cf. <i>aureolum</i>	<i>Pecten maximus</i>	high mortality in post larvae and juveniles; reproduction and growth inhibited in adults	France	Erard and Dao (1990)
<i>Pyrodinium bahamense</i>	<i>Anadara maculosa</i> <i>Chama</i> spp. <i>Crassostrea echinata</i> <i>Crassostrea amasa</i> <i>Barbatia parvivillosa</i> <i>Modiolus auriculatus</i> <i>Ostrea trapezina</i> <i>Pinctada maxima</i> <i>Pinna</i> sp. <i>Pterocarpa</i> sp. <i>Pycnodonte hyotis</i>	toxic shellfish; some human fatalities	New Guinea	MacLean (1973, 1975); Worth et al. (1975)
<i>Pyrodinium bahamense</i>	<i>Anadara granosa</i> <i>Meretrix meretrix</i> <i>Perna viridis</i>	highly toxic	Brunei	Jaafar and Subramaniam (1984); Beales (1976)

TABLE 1. Continued.

Algal species	Shellfish species affected	Notes	Location	Reference
	<i>Saccostrea cucullata</i>			
<i>Pyrodinium bahamense</i>	<i>Amphichaena kindermanni</i>	26 human deaths; 185 illnesses	Guatemala	Rosales-Loessener et al. (1989)
<i>Pyrodinium bahamense</i>	<i>Perna viridis</i>	highly toxic	Philippines	Gacutan et al. (1985); Arafiles et al. (1984); Gonzales et al. (1989)
<i>Pyrodinium bahamense</i>	<i>Perna viridis</i> <i>Amusium pleuronectes</i>	several human fatalities; mostly juveniles	Philippines	Estudillo and Gonzales (1984)
	<i>Pinctada margaritifera</i>			
<i>Pyrodinium bahamense</i>	<i>Anadara</i> spp. <i>Atrina</i> sp. <i>Crassostrea belcheri</i> <i>Gafranium</i> sp. <i>Meretrix</i> spp. <i>Oliva</i> sp. <i>Saccostrea cucullata</i>	highly toxic	Sabah	Sang and Ming (1984); MacLean (1984, 1989)
<i>Pyrodinium bahamense</i>	<i>Barbatia</i> sp. <i>Lopha cristagalli</i> <i>Modiolus</i> sp. <i>Saxostrea mordax</i> <i>Spondylus butleri</i> <i>Tridacna crocea</i> <i>Tectus</i> sp.	toxic	Palau	Harada et al. (1982)
<i>Pyrodinium phoneus</i>	clams		Belgium	Koch (1939)
<i>Pyrodinium</i> (?)	<i>Crassostrea gigas</i>	toxin profile similar to <i>Pyrodinium</i> although no cells found	Solomon Islands	Oshima et al. (1987a, 1987b)
<i>Amphidoma</i> sp.	<i>Mytilus chilensis</i>	mildly toxic	Chile	Avaria (1979); Campodonico and Guzman (1974)
<i>Aureococcus anophagefferens</i>	<i>Argopecten irradians</i>	larval shell growth reduced and mortality increased	laboratory study	Gallager et al. (1988)
<i>Aureococcus anophagefferens</i>	<i>Mytilus edulis</i> <i>Argopecten irradians</i>	mass mortalities	Long Island embayments NY; Narragansett Bay, RI; Barnegat Bay NJ	Cosper et al. (1987); Tracey et al. (1988); Tracey (1985); Smayda and Fofonof (1989)
<i>Aureococcus anophagefferens</i>	<i>Mytilus edulis</i>	inhibition of ciliary activity	laboratory study	Draper et al. (1989)
<i>Aureococcus anophagefferens</i>	<i>Mytilus edulis</i>	reduced feeding; reproductive failure; mass mortalities	Narragansett Bay	Tracey (1988)

TABLE 1. Continued.

Algal species	Shellfish species affected	Notes	Location	Reference
	<i>Mercenaria mercenaria</i>	reduced feeding		
<i>Aureococcus anophagefferens</i>	<i>Argopecten irradians</i>	76% reduction in adductor weights; recruitment failure of year class	Long Island, NY	Bricelj et al. (1987)
<i>Ceratium furca</i> var. <i>berghii</i> (?)	shellfish	outbreak of DSP first report from South Africa	South Africa	Horstman, (personal communication)
<i>Ceratium fusus</i>	<i>Crassostrea gigas</i>	acutely toxic to larval stages	Puget Sound	Cardwell et al. (1979)
<i>Ceratium fusus</i>	oysters	oyster farms completely destroyed; result of low oxygen	Korea	Cho (1979)
<i>Ceratium tripos</i>	<i>Placopecten magellanicus</i> <i>Arctica islandica</i> <i>Spisula solidissima</i> <i>Homarus americanus</i>	nontoxic; mass mortalities due to oxygen depletion	New York Bight	Mahoney and Steimle (1979)
<i>Chrysochromulina polylepis</i>	<i>Mytilus edulis</i>	some mortalities; mass mortalities of other invertebrates	Skagerrak, Kattegat	Rosenberg et al. (1988); Dundas et al. (1989)
<i>Chrysochromulina polylepis</i>	<i>Mytilus edulis</i>	fertilization of ova and successful development of embryos completely inhibited	Skagerrak	Granmo et al. (1988)
<i>Chrysochromulina polylepis</i>	<i>Mytilus edulis</i>	significant reduction in growth	laboratory study	Nielsen and Stromgren (1989)
<i>Cochlodinium heteroiobatum</i>	<i>Crassostrea virginica</i>	larval mortality; reduced calcium uptake	York River, Virginia USA	Ho and Zubkoff (1979)
<i>Cochlodinium</i> sp.	<i>Perna perna</i>	symptoms similar to PSP; several human fatalities; many illnesses	Venezuela	Reyes-Vasquez et al. (1979)
<i>Dictyocha speculum</i>	oysters	mortalities; probably due to anoxia	N. Brittany	Lassus and Berthome (1988)
<i>Hornellia</i> (= <i>Chatonella</i>) <i>marina</i>	shrimp	30–50% mortalities in ponds where blooms occurred; gill clogging	Johor Straits, Malaysia	Khoo (1985); Maclean (1989)
<i>Nannochloris</i> sp. <i>Stichoccus</i> sp.	<i>Crassostrea virginica</i>	recurrent blooms caused failure of oyster industry	New York	Ryther (1954)

TABLE 1. *Continued.*

Algal species	Shellfish species affected	Notes	Location	Reference
<i>Nitzschia pungens</i>	<i>Mytilus edulis</i>	highly toxic; over 106 illnesses and 3 human deaths	Prince Edward Island, Canada	Bates et al. (1988, 1989); Subba Rao et al. (1988); Addison and Stewart (1989); Smith et al. (1990)
<i>Olisthodiscus luteus</i>	oyster larvae	affects survival	laboratory studies	Anonymous (1982)
<i>Phaeocystis pouchetii</i>	<i>Mytilus edulis</i>	reproductive failure probably caused by feeding inhibition	Dutch Wadden Sea	Pieters et al. (1980)
<i>Prymnesium calathiferum</i>	shellfish	mortalities	New Zealand	Chang (1985)
<i>Rhizosolenia chunii</i>	<i>Mytilus planulatus</i> <i>Pecten alba</i> <i>Ostrea angasi</i>	bitter taste rendered shellfish unmarketable for 7 mos; digestive gland lesions and subsequent shellfish mortalities	Australia	Parry et al. (1989)
not specified	<i>Meretrix casta</i> <i>Crassostrea cucullata</i>	highly toxic	Mangalore, India	Karunasagar et al. (1984)
not specified; probably <i>Gonyaulax catenella</i>	<i>Ostrea gigas</i>	34 mild cases of PSP	Vancouver Island, B.C.	Davies et al. (1958); Anderson (1960)
not specified	<i>Soletellina diphos</i>	toxic shellfish	South Taiwan	Hwang et al. (1989)
not specified; probably <i>Protogonyaulax</i> sp.	<i>Mytilus edulis</i> <i>Mytilus corsucus</i> <i>Patinopectin yesoensis</i> <i>Chlamys farreri</i> <i>Perunidia venulosa</i> <i>Ruditapes philippinarum</i>	toxic	Korea	Jeon et al. (1988)

¹ In a later study (Kodama, ed. 1985) it was demonstrated that the strains of *P. tamarensis* in this area are nontoxic and that the toxicity exhibited by shellfish is due primarily to *P. cohorticula*.

of the Swedish west coast are currently being plagued by blooms of the prymnesiophyte, *Chrysochromulina polylepis*, previously unknown to the area.

The toxins associated with these various algae are potent, and molluscs feeding on them are prone to accumulation of toxins derived from these algae. The filter-feeding shellfish then become vectors in various forms of shellfish poisoning including paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning (DSP) and neurotoxic shellfish poisoning (NSP). Humans are poi-

soned by eating bivalve shellfish which have been exposed to toxic algae. Crustaceans (e.g., lobsters, crabs, shrimp) do not accumulate the toxins and are thus marketable even during intense blooms.

Shellfish toxicity and its association with exceptional blooms of plankton is not a new phenomena and has been around for centuries. It is particularly well-defined in the case of PSP, a recurrent phenomenon in some areas, which continues to present a serious health threat if proper control measures are not ensured. Slowly, progress is

being made towards understanding the nature and causes of toxic shellfish, but they still pose serious problems to harvesters, seafood processors, consumers and regulatory agencies.

The presence of toxic algae and/or the potential for blooms have clear, negative effects on the development of aquaculture. The problems are more acute in some areas than others. The Alaska butter clam industry was essentially destroyed over 40 years ago (Table 2). Alaska has 33,000 miles of coastline, over 100 species of clams and in 1917 the industry produced 5 million pounds of shellfish products (Neve and Reichardt 1984). Today the commercial clam fishery is virtually nonexistent and all Alaskan beaches are considered at risk at all times (Nishitani and Chew 1988). Approximately 70% of the British Columbian coastline is closed to commercial harvesting of shellfish because blooms of toxic dinoflagellates occur sporadically and unpredictably (Kitts et al. 1989; Chiang 1988). Mussel culture in Sweden and Norway was rapidly increasing up until 1984–1985. Blooms of the dinoflagellate *Dinophysis* spp. caused a major setback, and mussel culture was reduced to a minimum in both countries (Edler, personal communication). The industry was closed for up to a year with complete shut down of many farms. The highly successful mussel production in Spain (employing approximately 10,000) suffered severe setbacks due to both DSP and PSP (Table 1). During outbreaks, closure affects all secondary activities as well, including canning, marketing and transportation.

The most effective means of controlling quality during outbreaks of toxic algae is either by blanket closure during certain times of the year or by instituting a shellfish toxicity monitoring program. This has been done in many areas commonly plagued by such blooms. Under such conditions, the blooms are not quite so detrimental to affected shellfish-based industries. Culture of mussels in the northeastern United States and scallops in Japan have not been hampered by the presence of toxic dinoflagel-

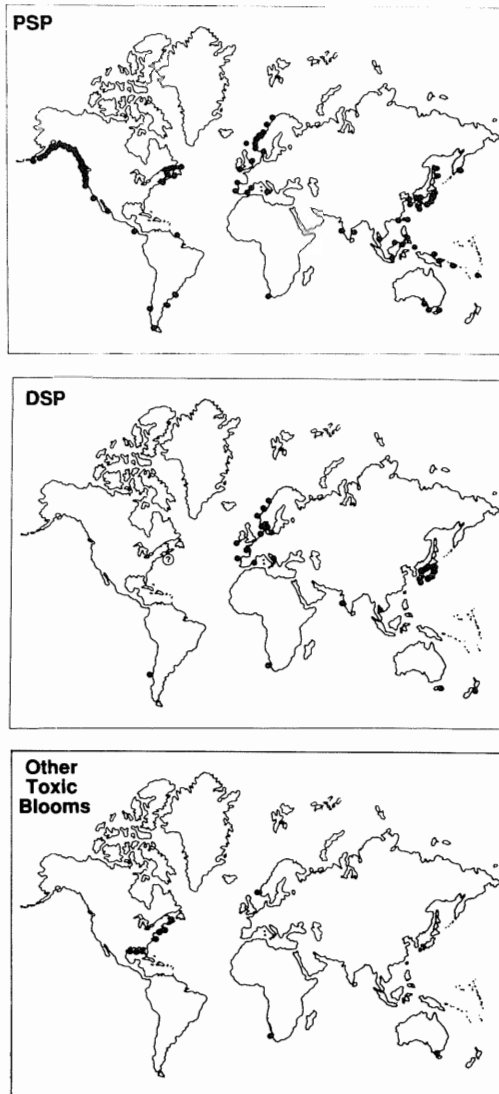


FIGURE 1. Distribution of (a) PSP, (b) DSP and (c) other toxic algal blooms. Data from Table 1.

lates due, primarily, to the presence of efficient monitoring programs which ensure public safety and at the same time minimize the disruption of harvesting. Other regions of the world have also established monitoring programs and more are currently being initiated.

Concern about toxic algal blooms has arisen in recent years because of their impact on public health and on the economics of shellfisheries and aquaculture. There is evidence that the incidence of blooms has

TABLE 2. Estimated losses to the shellfish industry as a result of toxic or noxious algal blooms.

Algal	Shellfish	Location	Loss (\$U.S. Millions)	Reference	
<i>Protogonyaulax tamarensis</i>	<i>Mytilus edulis</i>	Gulf of Maine 1972	>10	National Marine Fisheries Service	
	<i>Mya arenaria</i>		1- 9- 80		
<i>Protogonyaulax catenalla</i>	<i>Protothaca staminea</i>	British Columbia (annually)	2+	Lutz and Incze (1979)	
	<i>Saxidomus giganteus</i>				
	<i>Venerupis japonica</i>				
	<i>Panope generosa</i>				
	<i>Crassostrea gigas</i>	California, USA 1980	0.63		Conte (1984)
	<i>Crassostrea virginica</i>				
	<i>Ostrea edulis</i>	Washington, USA	no fishery since 1979		Nishitani and Chew (1988)
	<i>Saxicomus giganteus</i>	Alaska, USA	no fishery since 1946		McFarren et al. (1958) Nishitani and Chew (1988)
	<i>Clinocardium nuttalli</i>	Alaska, USA	no fishery since 1962		Nosho (1972) Nishitani and Chew (1988)
	<i>Ptychodiscus brevis</i>	<i>Mercenaria mercenaria</i>	North Carolina, USA		>24.7
<i>Crassostrea virginica</i>					
<i>Aureococcus anophagefferens</i>	<i>Mytilus edulis</i>	Narragansett Bay, USA 1987	0.1*	Fuchsberg (1985)	
	<i>Argopecten irradians</i>	Long Island, USA 1987	>2	Kahn and Rockel (1988)	
<i>Ceratium fusus</i>	oyster	Korea 1978	4.5	Cho (1979)	
<i>Nitzia pungens</i>	<i>Mytilus edulis</i>	P.E.I. Canada 1987	0.3*	Anonymous (1988)	
<i>Pyrodinium bahamense</i>	<i>Perna viridis</i>	Philippines	0.5	White et al. (1984)	
	<i>Spondylus butleri</i>	Philippines Western Samar	2.2	Gacutan et al. (1984)	
	<i>Tridacna corcea</i>				
	<i>Septifer bilocularis</i>				
	<i>Perna viridis</i>				
	<i>Septifer bilocularis</i>				
"Red tide" PSP	pearl oysters	Japan 1933	7	Fage (1953)	
	scallops	Japan 1978	10	Ito (pers. comm.)	
<i>Ceratium tripos</i>	shellfish	New Jersey, USA	60	Sindermann and Swanson (1980) Falkowski et al. (1980)	
	<i>Spisula solidissima</i>	New Jersey, USA	430†	Ropes et al. (1979)	
	<i>Arctica islandica</i>		120†	Figley et al. (1979)	
	<i>Placopecten magellanicus</i>		1.3†		
	<i>Homarus americanus</i>		7.1†		

been increasing in recent years (Maclean and White 1985; Smayda 1990). Some attribute the apparent increased incidence to an increased awareness and number of observers, but there seems little doubt that the increase in outbreaks is very real (Table 1). There is an increasing frequency, intensity and duration, and geographic spreading of outbreaks (Anderson et al. 1982).

A number of factors are thought to enhance blooms including: nutrient enrichment (eutrophication (Holligan 1985)); decreased grazing pressure (Lindahl 1983; Lindahl and Hernroth 1983, 1988); large scale hydrometeorological changes (Holligan 1985); upwelling of nutrient rich bottom water (Tangen 1977, 1983) and heavy precipitation and fresh water run off (Edler et al. 1982; Cembella et al. 1988a, 1988b) and even the presence of previous blooms of other phytoplankton species (Silva 1985). It has also been firmly established that there is a direct correlation between the number of red tides and the extent of coastal pollution (particularly from sewage and some forms of industrial wastes) (Jingzhong et al. 1985; Richardson 1989; Anderson 1989; Wong 1989). The potential hazards to the shellfish industry are staggering and shellfish monitoring programs designed to protect the general public have become a necessity in previously unaffected areas, especially southeast Asia and other Pacific areas (Maclean 1984).

Finally, there is increasing evidence that toxic species are being transported to new areas via ships' ballast or through infected shellfish (Maclean 1989; Hallegraeff et al. 1988, 1989). When shellfish are transported from a toxic area to clean waters, they will begin to self-depurate and there is a real risk of infecting the "clean" area with cysts and/or motile cells, thus, making it possible to seed a future bloom. The Netherlands has established regulations whereby it is prohibited to place mussels from potential "PSP risk" areas to other areas in an effort to control spreading of blooms. The increase

in blooms worldwide is disturbing for several reasons. Developing countries often lack the "expertise and managerial infrastructure to deal with sudden PSP outbreaks" (White 1987) and the toll on human health as well as the industry can be enormous, e.g., the first experience with a PSP event in the Philippines in 1983 left 21 people dead, nearly 300 ill, and the harvest and sale of all shellfish banned for 8 months (Table 1).

Toxic Blooms and their Effects

The sources of these toxic blooms have been most commonly associated with dinoflagellates, particularly those of the genera *Protogonyaulax*, *Gymnodinium*, and *Pyrodinium* (vectors of PSP), *Dinophysis* (vectors of DSP), and *Ptychodiscus* (vectors of NSP).

PSP. The chemical and biochemical nature of PSP toxins has been the subject of several recent reviews (Hall and Reichardt 1984; Shimizu 1978, 1988; Sullivan 1988; Kodama and Ogata 1988). The "suite" of toxins produced by dinoflagellates is comprised of 12 sulfocarbamoyl and carbamate toxins and is water soluble. In addition, the decarbamoyl toxin derivatives can be present in shellfish due to enzymatic action on sulfocarbamoyl and carbamate toxins (Sullivan et al. 1983; Shimizu and Yoshioka 1981). Thus there can be up to 18 different toxins present in shellfish, depending on which toxins are produced by the dinoflagellate prevalent in the local area, the presence of selective uptake and storage of the various toxins in the shellfish, and any subsequent metabolism of the toxins in the shellfish tissue (Sullivan 1988). To date, the most widely utilized technique for determination of paralytic shellfish toxins is the mouse bioassay (Horowitz 1984) and is based on the original assay of Sommer and Meyer (1937) and Medcof et al. (1947). This method has several drawbacks (Sullivan 1988) and several authors have proposed chemical assay techniques including the ox-

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* Losses reported by one farm only.

† Represents estimated potential losses including dockside value, marketers and processors.

idation/fluorescence method originated by Bates and Rapoport (1975). In addition, a number of immunological based assay techniques have been reported (Sullivan 1988). Other methods have been proposed using the housefly, chicken embryo and microbial bioassays. High performance liquid chromatography (HPLC) has recently been utilized in many investigations concerning PSP toxins (Sullivan and Iwaoka 1983; Sullivan and Wekell 1984; Sullivan et al. 1985). Most recently, efforts have been focusing on the potential for development of an antiserum (Kitts et al. 1989) and the production of a "Saxitoxin Kit" based on an Elisa system and now available commercially (N.C.T. Inc., Quebec, Canada).

The toxic marine dinoflagellate *Protogonyaulax tamarensis* (Lebour) Taylor (also known as *Gonyaulax tamarensis*, *G. excavata*, *G. tamarensis* var. *excavata*, *Gessnerium tamarensis*, *Alexandrium tamarense* or *A. fundyense* by various authors (Cembella et al. 1988a, 1988b)) is the organism responsible for PSP in many parts of the world (Table 1; Fig. 1a) and, because of the serious damage it can cause to the shellfish industry, is probably the best documented of any of the toxic species. Other species of *Gonyaulax* are also known to cause toxicity in shellfish, especially in the Pacific northwest and Europe. Further, the resting cysts of these species are also highly toxic (Yentsch and Incze 1982; Anderson 1984).

Pyrodinium bahamense var. *compressa* also produces paralytic shellfish toxins and has been associated with severe outbreaks of shellfish poisoning in southeast Asia. It was first observed in Papua, New Guinea, in 1969. One of the worst episodes to date took place during 1983 in the central Philippines where many mussel farms exist. The red tide persisted from June to September and resulted in 21 deaths and 278 reported illnesses (White 1987). While this species is most common in the East Indies and Philippines, it also occurs in Thailand and India. A recent outbreak in Guatemala resulted in 26 deaths and 185 serious illnesses

(Anonymous 1987). Villagers harvested clams (*Amphichaena kindermanni*) for a local feast and death ensued soon after. The toxin was so virulent that "mice died in the hands of the lab technicians as they injected them with a diluted concentration of the toxin." There was no prior history of PSP in the area (Rosales-Loessener et al. 1989).

A third, highly toxic group of dinoflagellates belongs to the genus *Gymnodinium* and are also responsible for outbreaks of PSP. Outbreaks have been reported from Tasmania, Mexico, Japan, Portugal, Spain, Argentina, Ireland and the northwest coast of North America (Table 1). This species was responsible for the closure of extensive mussel farms in northwest Spain and Tasmania in 1985–1986 and for the death of three children along the west coast of Mexico in 1979 (White 1987). In addition to producing highly potent toxins, these species have been responsible for shellfish mortalities which include larval, juvenile and adult oysters, and mussels among others.

NSP. Ptychodiscus brevis (formerly *Gymnodinium breve*) causes NSP with symptoms similar to, but milder than, PSP. The brevetoxins are lipid soluble and no deaths have been reported due to this species. Toxins are detected by a standardized mouse bioassay (Delaney 1985). Outbreaks of *Ptychodiscus* are annual events, primarily limited to the coasts of Florida and Texas but seen as far north as North Carolina (Pietrafesa et al. 1988), and the State of Florida monitors routinely for presence of the dinoflagellate.

DSP. Diarrhetic shellfish poisoning is easily confused with gastroenteritis and general stomach upsets associated with eating shellfish or contaminated shellfish. Consequently, DSP has only been recognized as a disease for the past ten years (Yasumoto et al. 1978) and no mortalities have been recorded (Lee et al. 1988). Toxins associated with DSP include okadaic acid, dinophysistoxin-1 and dinophysistoxin-3 (Lee et al. 1989) which are lipid-soluble. The most seriously affected areas are Europe and Ja-

pan, although there is increasing evidence that the problem may be more widespread than previously thought.

Dinophysis acuminata is considered to be the main source of DSP in European episodes although there is no direct proof thus far (Krogh et al. 1985). *Dinophysis acuta* and *D. norvegica* are the most likely organisms responsible for DSP in Norway (Dahl and Yndestad 1985). *Dinophysis fortii* is believed to be one of the causative organisms in Japanese waters along with *Prorocentrum* spp. (Yasumoto et al. 1985; Yasumoto et al. 1984). *Prorocentrum lima*, a benthic dinoflagellate, has been implicated in Spain and harvesting is limited or stopped for a few weeks every summer and fall in anticipation of its presence. A bloom is not necessarily needed to induce toxicity as it has been shown that concentrations as low as 100 cells/L may produce high levels of DSP in mussels (Dahl and Yndestad 1985).

Methods of detection for DSP include the suckling mouse assay (Hamano et al. 1985), a mouse assay (Yasumoto et al. 1978) and a test based on cytotoxicity (Underdal et al. 1985). The majority of work on DSP has been carried out in Japan by Yasumoto and co-workers (Sullivan 1988) and the HPLC method appears to be the most promising analytical procedure for routine shellfish toxicity monitoring. Detection of the toxins in European monitoring programs is generally by rat assay but the method lacks sensitivity, specificity and accuracy. Okadaic acid was considered to be the major component of toxic shellfish from Spain, France, Netherlands and Sweden (Kumagai et al. 1986). Research on the toxins has been hampered by the fact that *Dinophysis acuminata* and *D. fortii*, two species suggested to produce okadaic acid and dinophysistoxins, have never been raised in pure culture. The Japanese have developed a chemical assay which is very sensitive and allows identification of toxins in trace amount of sample such as a plankton sample (Lee et al. 1987; UBE Industries 1988). They have been able to demonstrate complex toxin

profiles from Japanese shellfish which include up to ten individual toxins, the relative ratios of which fluctuate seasonally, yearly and regionally (Yasumoto 1987). Lee et al. (1988) have recently identified multiple toxin profiles in mussels from Norway as well, adding dinophysistoxin-1 and yessotoxin to the list, thus providing the first evidence for multiple toxin profiles from Europe. Further research is obviously needed to assess the toxin profiles associated with DSP from various geographic regions. In addition, efforts should continue to culture *Dinophysis* spp. if specific outbreaks of the dinoflagellates are to be associated with episodes of DSP.

Like some PSP toxins which persist long after the blooms have disappeared, *Dinophysis acuta* and *D. norvegica* toxins may remain in mussels, *Mytilus edulis*, for up to five months after accumulation in Swedish waters (Underdal et al. 1985). In other areas, toxins have been known to persist for many months and the economic damage to the shellfish industries due to prolonged closures can be devastating.

While the majority of DSP outbreaks are described from Japan and Europe, it is probable that the occurrences are not limited to those areas. Because the symptoms of DSP are so closely aligned with common gastroenteritis, cases of DSP may have previously gone unnoticed or unreported. The causative organisms described from other geographic areas are commonly present in other areas. Freudenthal and Jijina (1988) reported 12 species of *Dinophysis* in Long Island waters with *D. acuminata*, *D. norvegica* and *D. acuta* being the most prevalent. *Dinophysis fortii*, *D. acuminata* and *Prorocentrum micans* are common members of the phytoplankton community in many areas including British Columbia and the northeast coast of the United States. McAlice (1975) reported the presence of ten species of *Dinophysis* including *D. acuminata*, *D. acuta*, and *D. norvegica* in the Gulf of Maine. Shumway et al. (1987) reported the presence of *D. acuminata* and *Dino-*

physis spp. in the digestive tracts of scallops (*Placopecten magellanicus*) and *D. acuminata*, *D. acuta*, *D. norvegica*, *D. rotundata* and *Dinophysis* sp. from mussel (*Mytilus edulis*) guts from Maine waters (Newell et al. 1989).

Maranda and Shimizu (1987) conducted a two year survey for DSP in Narragansett Bay. They noted the presence of several species of *Dinophysis* but were not able to link the presence of these dinoflagellates to toxic shellfish. Stamman et al. (1987) concluded that, "while the potential for DSP certainly exists in the US, no definitive evidence that any DSP cases have occurred as a result of shellfish consumption in this country." Despite this lack of evidence for the presence of DSP, there is every reason to believe that it is a potential hazard.

DSP has emerged as a potential toxin problem in Maine. In the fall and winter of 1988, shipments of European oysters (*Ostrea edulis*) originating in Casco Bay, Marine, were tested by the Netherlands Institute for Fishery Investigations (RVV) and found to be positive for DSP. The shipments were subsequently refused as have been others since that time (Hurst, personal communication). These samples were tested using the rat bioassay and scored "+-". Subsequent analyses of oysters from the same area by the Department of Fisheries and Oceans, Canada, using the mouse test for fluid accumulation (Ministry of Health and Welfare Japan 1981) indicated that these oysters were negative for DSP. A further analysis using the suckling mouse assay (Hamano et al. 1985) also indicated that the samples were negative for DSP. This positive test, coupled with the inability to test for and certify that oysters from Maine are free of DSP toxins has resulted in an economic loss to Maine oyster farmers and shellfish dealers of approximately \$500,000 (Hurst, personal communication).

Even though the tests for DSP were, in this instance, inconclusive, there appears to be every reason to believe that the potential for DSP exists in waters off the northeast United States. The dinoflagellate species

commonly associated with DSP are routinely present, and the lack of documented cases may simply be due to the fact that DSP is so easily confused with other maladies. It seems premature to establish a major sampling or monitoring program for DSP in these waters; however, with the increasing demand for mussels in the US, growers, marketers and managers should be aware of the potential problem.

ASP. A prime example of a sudden and unexpected outbreak of toxic shellfish occurred in the Cardigan River region of Prince Edward Island, Canada, in 1987 when cultured mussels (*Mytilus edulis*) were implicated in 129 cases of poisoning and 2 deaths (Bates et al. 1988). The causative agent was identified as domoic acid (Wright et al. 1989), a naturally occurring compound previously unknown as a source of shellfish poison. Domoic acid can be regarded as "a conformationally restricted form of glutamic acid that disrupts normal neurochemical transmission in the brain by binding to certain glutamate receptors of neuronal cells. This results in increased firing of the neurons and eventual rupture of the cell" (Bird and Wright 1989). This represents the first known occurrence of human poisoning from this neurotoxin and the establishment of a new illness, amnesic shellfish poisoning (ASP). The symptoms include abdominal cramps, neurologic responses involving memory loss and disorientation and, in some instances, mortality. It has been suggested that the toxin is derived from a diatom, *Nitzschia pungens* (Rao et al. 1988; Smith et al. 1989), a common member of the phytoplankton community not previously known to produce toxins. Thus far, only the forma *multiseries* from eastern Prince Edward Island has been shown to produce the toxin (Bird and Wright 1989).

The primary method of assay is HPLC. The standard mouse assay, however, gives a distinct reaction which is easily distinguished from the reaction induced by paralytic shellfish toxins. The injected mouse first begins to scratch behind the ears with the hind legs, the tail becomes rigid and the

animal then goes into severe convulsions. The reaction takes longer than the standard mouse bioassay and is qualitative rather than quantitative in nature. The HPLC method is more exact and is currently employed in both Canada and the United States.

The Canadian outbreak prompted Maine officials to monitor several areas for the presence of this toxin. Sea scallop (*Placopecten magellanicus*) digestive glands were analyzed for domoic acid by the Food and Drug Administration, Winchester, Massachusetts (Hurst, personal communication). One area, Broad Cove, Eastport, produced scallops with domoic levels of 568–595 $\mu\text{g/g}$ tissue. Several other low levels of this toxin were noted at Blue Hill Bay, Jonesport, Kittery, Scarborough, Harpswell, Machias Bay, Northeast Harbor and Swans Island (Range 0.1–10.2 $\mu\text{g/g}$ tissue) (Hurst, personal communication). Domoic acid is a highly potent neurotoxin and its presence in the phytoplankton has caused great concern amongst the shellfish industry and managers alike.

Other effects of blooms. *Gyrodinium aureolum* (which may or may not be synonymous with *Gymnodinium nagasakiense* found in Japanese waters), although not implicated in any outbreaks of PSP [Tangen 1977; Pybus 1980; (Thain and Watts 1987 found no PSP-type toxins in water samples collected throughout a bloom of this species)], has been shown to cause mortalities in a number of species (Boalch 1979; Griffiths and Dennis 1979) (Table 1) and should be considered a major threat to aquaculture activities in specific geographic regions, most notably northwestern Europe. In some instances, the kills can be attributed to deoxygenation of the water when the bloom begins to decay. In others, however, there is clear evidence that biotoxins are in effect. Only recently has a fat-soluble cytotoxin been identified in *G. cf. nagasakiense* (Partensky et al. 1989).

Ottway et al. (1979) reported that the infaunal species, *Mya* sp., *Ensis* sp., *Cerastoderma edule* and *Tapes decussata*, all surfaced on the beaches during a bloom of *Gyrodinium* in Youghal, Ireland. Cross and

Southgate (1980) reported mortalities of shore animals associated with a bloom of *Gyrodinium aureolum* with some mussels being affected. Widdows et al. (1979) reported that during a large bloom of *Gyrodinium*, *Mytilus* exhibited decreased clearance rates and that the *Gyrodinium* cells caused marked cellular damage to the gut, although the animals were capable of rapid recovery once the cell concentration was reduced. Larval survival of *Crassostrea gigas* was reduced in the presence of *G. aureolum* cells; however, mussels tested for toxicity during the same period presented no health hazard (Helm et al. 1974). Thain and Watts (1987) have proposed the use of oyster (*Crassostrea gigas*) embryonic development as a bioassay to indicate variations in water quality before, during and after blooms. Their method shows real promise as a monitoring tool for mariculture.

A massive shellfish kill was reported in late September, 1988, at Maquoit Bay, Maine, USA, one of the most productive shellfish bays in the state. Unfortunately, water samples were only available after the tragedy and it will be difficult, if not impossible, to determine with certainty the cause of the kill. Subsequent analyses of these water samples did indicate that a "red-tide" had occurred and that the causative organism was *Gyrodinium aureolum* (Haugen and Selvin, personal communication). The cell counts were as high as 1.8×10^6 cells/L after the "tide" had passed. Several individuals and agencies are currently trying to analyze environmental conditions surrounding the bloom and examining possible causes of such a massive shellfish kill (Campbell 1989; Heinig 1989).

The problems associated with toxic algal blooms are no longer limited to the dinoflagellates and are becoming increasingly severe on a global scale (Fig. 1; Table 1). Relative newcomers to the scene are the recently described chrysophyte, *Aureococcus anophagefferens*, responsible for the reduction of eelgrass beds and the collapse of the bay scallop industry on Long Island and the near elimination of mussel populations in Nar-

ragansett Bay (Bricelj et al. 1987; Cosper et al. 1987; Tracey 1988), and the haptophycean alga, *Chrysochromulina polylepis*, previously unknown to Swedish waters but currently causing problems in that area. Blooms of *C. polylepis* primarily endanger the salmon aquaculture industry; however, Granmo et al. (1988) have demonstrated that *C. polylepis* is acutely toxic to eggs and larvae of *Mytilus edulis*, with fertilization of ova and successful development of embryos completely inhibited. This is the first report of such toxic effects for this species and the implications for potential damage to culture facilities are obvious. *C. polylepis*, originally described from the English Channel (Manton and Parke 1962), is found in other areas, e.g., British Columbia and the Baltic Sea, and appears to be cosmopolitan in distribution (Estep and MacIntyre 1989), and there is no certainty that it will not create problems in other regions.

Prorocentrum minimum var. *mariae lebouriae* was responsible for a disastrous case of shellfish poisoning in Japan in 1942 when 324 cases of shellfish poisoning and 114 deaths were attributed to eating of the short-neck clams, *Venerupis semidecussata* (Nakazima, 1965a, 1965b, 1965c). *P. minimum* is a common organism in Portugal and occasionally has been associated with shellfish toxicity. It has spread to Norway, Sweden and Denmark although it has not yet attributed to any shellfish toxicity in these areas. The effects of several other species of algae on shellfish are summarized in Table 1. Another red tide phenomenon known to play an important role in controlling the settlement of green mussels (*Perna viridis*) is *Noctiluca* blooms.

Another example of new and unexpected blooms occurred on the west coast of South Africa. This was the first occurrence of DSP in that area and the causative organism was tentatively identified as *Ceratium furca* var. *berghii* (Horstman, personal communication). *C. furca* has not previously been associated with outbreaks of DSP; however, during an episode of DSP in Sweden (Krogh

et al. 1985) *Ceratium furca* was the dominant organism during the bloom. *D. acuminata* was still considered to be the main source of DSP by the authors who noted that "no report on toxin production by *Ceratium* species is available, and accordingly we do not suspect them to be the causative organisms in this DSP outbreak, but rather *Dinophysis* species, such as *D. acuminata*, although we have not yet addressed this point specifically."

It has often been reported in the literature that the toxic dinoflagellates have little or no effect on the shellfish themselves. In addition to massive kills reported above that may result from the toxic blooms directly or indirectly, a recent series of studies demonstrated a number of direct effects on the shellfish (Cucci et al. 1985; Shumway et al. 1985; Shumway and Cucci 1987; Shumway et al. 1987; Gainey and Shumway 1988a, 1988b). These responses are species specific, geographically specific and often dramatic. Of particular interest to culturists are feeding responses, byssus production and mortality, all of which are adversely affected by the presence of *Protogonyaulax tamarensis*.

Inasmuch as no geographic area seems to be immune from outbreaks of algal blooms, culturists and managers must be aware of the potential problems that these blooms can incur.

Intoxification and Detoxification of Shellfish

Rates of intoxication and detoxification are species-specific and are, in most cases, directly related to the number of cells available to the animals (Sribhibhadh 1963; Gillfillan et al. 1976; Prakash et al. 1971; Saunders et al. 1982). Rate of loss is also known to vary with season (Prakash et al. 1971; Hwang et al. 1987) and low water temperatures which apparently retard toxin loss and size (Aalvik and Framstad 1981); however, the degree to which temperature affects the uptake and release of toxins is not clearly understood (Madenwald 1985). Differences in rates of toxins of toxin accu-

mulation have recently been demonstrated between wild and cultured *Mytilus* (Desbiens et al. 1989). Further, the rate of detoxification is highly dependent on the site of toxin storage within the animal, i.e., toxins in the gastrointestinal tract (e.g., *Mytilus*) are eliminated much more readily than toxins bound in tissues (e.g., *Placopecten*, *Spisula*, *Saxidimus*).

Table 3 summarizes the existing data on toxin retention for a number of bivalve species. Mussels (*Mytilus* spp., *Modiolus* spp.) are known to accumulate PSP toxins faster than most other species of shellfish and also to eliminate the poison quickly. While oysters do not accumulate the toxic species as readily as mussels, they may take considerably longer to detoxify (Neal 1967; Hurst, personal communication; Shumway et al. 1990; Table 3). In contrast, some species (e.g., *Saxidimus giganteus*, *Spisula solidissima*) may remain toxic for extended periods, e.g., in excess of two years (Quayle 1965; Blogoslawski and Stewart 1978; Chambers and Magnusson 1950).

Some species are known to avoid toxic dinoflagellates (Shumway and Cucci 1987). One species of particular interest is *Mercenaria mercenaria*. During the outbreak of a bloom of *Gonyaulax* (= *Protogonyaulax*) *tamarensis* in 1972, the entire coastline of Massachusetts came under interdict (Bicknell and Collins 1973). Monitoring of the coast indicated that some 2,800 acres of shellfish harvesting areas were contaminated. Bioassays of shellfish samples showed toxin in the range of 3,000–5,000 $\mu\text{g}/100\text{ g}$ tissue with the most heavily contaminated shellfish being the mussel (*Mytilus edulis*), soft-shelled clam (*Mya arenaria*) and bay scallops (*Argopecten irradians*). It was specifically noted that no quahogs (*M. mercenaria*) or oysters were affected. *Mercenaria mercenaria* was reported to be toxic in the Bay of Fundy and St. Lawrence regions by Bond and LaChance (1959). Studies in this laboratory have shown that in the presence of bloom concentrations of *P. tamarensis* the quahog first retracts its siphons and then

completely isolates itself from the external environment by means of shell valve closure. The animals did not reopen their shell valves until after the addition of clean sea water. Efforts to induce toxicity by feeding *P. tamarensis* for extended periods were unsuccessful.

It is also possible that *Mercenaria* responds to the presence of other dinoflagellates in the same manner. Castagna (personal communication) has observed that quahogs exposed to red-tide blooms (nontoxic) in Virginia bury themselves very deep in the experimental trays. He further noted that "wild" populations were found at depths of up to 14 inches below the sediment surface during these blooms as opposed to their usual 6 inches.

Data are presented in Table 4 which summarizes toxicity data on samples of mussels and quahogs collected from the same localities in Maine during 1979–1986. At no time were quahogs found to be toxic, although mussels from the same areas showed toxin levels as high as 2,600 $\mu\text{g}/100\text{ g}$ tissue. Bricelj et al. (1989) have demonstrated that, although *M. mercenaria* are unlikely to become toxic when fed on pure cultures of *Protogonyaulax tamarensis*, they did become toxic when fed low concentrations of toxic dinoflagellates in combination with a known good algal food source. Such differences in toxin accumulation and retention between species should be taken into account before choosing a species to be reared in areas prone to toxic algal blooms.

Various attempts have been made at detoxifying shellfish contaminated with paralytic shellfish toxins in an effort to reduce the duration of "off market" times. The most obvious method is to transplant shellfish to waters free of the toxic organisms and allow them to self-depurate. While this is a satisfactory method for many species of shellfish, rates of detoxification vary considerably between species (Table 3), and some species remain toxic for extended periods of time. Detoxification using temperature or salinity stress has been tried with mar-

TABLE 3. Approximate times of toxin retention for various species of bivalve molluscs (represents time taken for toxin levels to fall below either quarantine or detection levels).

Species	Toxin source	Retention time	Reference
<i>Anadara maculosa</i>	<i>Pyrodinium bahamense</i>	6 weeks	Worth et al. (1975)
<i>Arctica islandica</i>	<i>Protogonyaulax tamarensis</i>	2 months <i>in vivo</i>	Shumway, unpublished
<i>Choromytilus meridionalis</i>	<i>Gonyaulax catenella</i>	3 months	Popkiss et al. (1979)
<i>Clinocardium nuttalli</i>	<i>Gonyaulax acatenella</i>	9 weeks	Quayle (1965)
<i>Crassostrea cucullata</i>	not specified, probably <i>Pyrodinium bahamense</i>	2 months	Karunasagar et al. (1984)
<i>Crassostrea echinata</i>	<i>Pyrodinium bahamense</i>	3 weeks in closed system; longer periods <i>in vivo</i>	Maclean (1975)
<i>Crassostrea gigas</i>	<i>Gonyaulax acatenella</i>	4 months 1-9 weeks	Worth et al. (1975) Quayle (1965; 1969); Sharpe (1981)
<i>Crassostrea iridescens</i>	<i>Gymnodinium catenatum</i>	1 month > 1 month	Sribhibhadh (1963) Mee et al. (1986)
<i>Crassostrea virginica</i>	<i>Gymnodinium breve</i>	2-6 weeks	Morton and Burklew (1969)
<i>Meretrix casta</i>	not specified, probably <i>Pyrodinium bahamense</i>	1 month	Karunasagar et al. (1984)
<i>Modiolus auriculatus</i>	<i>Pyrodinium bahamense</i>	6 weeks	Worth et al. (1975)
<i>Modiolus modiolus</i>	<i>Gonyaulax tamarensis</i>	up to 60 days ¹	Gilfillan et al. (1976)
<i>Mya arenaria</i>	<i>Gonyaulax acatenella</i> <i>Gonyaulax tamarensis</i>	5 weeks 4-6 weeks	Quayle (1965) Prakash et al. (1971); Bicknell and Collins (1973)
<i>Mytilus californianus</i>	<i>Gonyaulax catenella</i>	up to 45 days ¹ < 1 month	Gilfillan et al. (1976) Sommer and Myer (1937); Sharpe (1981)
<i>Mytilus edulis</i>	<i>Protogonyaulax tamarensis</i> <i>Gonyaulax acatenella</i> <i>Gonyaulax excavata</i>	10 days-7 weeks up to 50 days 11 weeks 4 weeks 2-3 weeks	Oshima et al. (1982); Gilfillan et al. (1976); Prakash et al. (1971) Quayle (1965) Sharpe (1981) Gaard and Poulsen (1988)
<i>Patinopecten yessoensis</i>	<i>Dinophysis</i> spp. <i>Protogonyaulax tamarensis</i>	1 week 6 weeks-5 months	Haamer et al. (1989) Oshima et al. (1982); Iioka et al. (1964)
<i>Placopecten magellanicus</i>	<i>Protogonyaulax tamarensis</i>	6 months in closed system; can be toxic year round <i>in vivo</i>	Bourne (1965); Shum- way et al. (1988)
<i>Protothaca staminea</i>	<i>Protogonyaulax acatenella</i>	5 weeks	Quayle (1965)
<i>Saxidomus giganteus</i>		2 years +	Quayle (1965); Anony- mous (1974)
<i>Saxidomus solidissima</i>	<i>Gonyaulax catenella</i>	< 1 month	Sommer and Myer (1937)
<i>Spisula solidissima</i>	<i>Protogonyaulax tamarensis</i>	up to one year	Medcof et al. (1947); Blogaslawski and Stewart (1978)
<i>Spondylus</i> sp.	<i>Pyrodinium bahamense</i>	still highly toxic after months	Worth et al. (1975)
<i>Tresus capax</i>	<i>Gonyaulax acatenella</i>	11 weeks	Quayle (1965)
<i>Venerupis japonica</i>	<i>Gonyaulax acatenella</i>	5 weeks	Quayle (1965)

¹ Dependent on initial level of toxicity.

ginal success (Gilfillan et al. 1976; Blogoslawski and Neve 1979). Chlorination has been used in France; however, this process alters the flavor of the shellfish and thus decreases marketability.

Ozonation appears to be the most promising method although its capabilities are limited. Several authors have reported effective inactivation by ozone of PSP toxins in shellfish exposed to *Gonyaulax tamar-ensis*, *G. catenella* and *G. breve* blooms (Thurberg 1975; Blogoslawski et al. 1975, 1979; Dawson et al. 1976; Blogoslawski and Stewart 1978). A subsequent study by White et al. (1985) gave results totally contradictory to previous studies in that no detoxification occurred in *Mya arenaria* exposed to ozone treatments. More recently, preliminary studies by Gacutan et al. (1985) demonstrated that both ozone gas and PVP-iodide-iodine may effectively inactivate PSP toxins from *Perna viridis* contaminated by *Pyrodinium bahamense*.

In a recent review (Blogoslawski 1988), it was concluded that ozonized seawater can be of value in detoxification of shellfish contaminated recently by the vegetative cell phase of toxic dinoflagellates. In a study during a red tide outbreak, it was shown that ozone treatment of the seawater does prevent shellfish (*Mytilus edulis*, *Mya arenaria* and *Guekensia demissus*) from accumulating paralytic shellfish toxins. Blogoslawski concluded that inactivation could be achieved in bivalves exposed to and contaminated by motile dinoflagellate cells bearing PSP without measurably altering the physical state of the treated bivalves and that this inactivation could be achieved in a marketable species such as *Mya* within an economically feasible time frame (Blogoslawski et al. 1979). Ozone is useless in detoxifying cysts or in bivalves that have ingested cysts or have the toxins bound in their tissue over long periods of time. Further, detoxification over long periods of time is not economically feasible. In general, the effectiveness of ozonation in the detoxification of shellfish remains highly questionable (Blogoslawski 1977).

TABLE 4. Toxicity levels (μg toxin/100 g tissue) of mussels and quahogs collected at various locations in Maine over a period of 7 years.

	Mussels	Quahogs
1979		
Ben Island	non-toxic	non-toxic
Birch Point	83	non-toxic
Cape Poroise	536	non-toxic
Wildwood Park	64	non-toxic
1980		
Basin Point	non-toxic	non-toxic
Cliff Island	non-toxic	non-toxic
Birch Point	non-toxic	non-toxic
Gurnet	non-toxic	non-toxic
Lumbos Hole	non-toxic	non-toxic
Dyer Cove	84	non-toxic
1981		
Birch Point	540	non-toxic
Lab Wharf	2,604	non-toxic
Spinney Creek	not tested	non-toxic
Thurlows Pine Point	not tested	non-toxic
1982		
Barnes Point	non-toxic	non-toxic
1984		
Spinney Creek	161	non-toxic
Flying Point	non-toxic	non-toxic
Mere Point	70	non-toxic
1986		
Spinney Creek	1,340	non-toxic
	835	non-toxic
	245	non-toxic
	95	non-toxic
New Meadows	not tested	non-toxic
Bethel Point	230	non-toxic

At present the economic feasibility of efficiently detoxifying shellfish on a large scale in artificial systems is not promising. In areas prone to regular outbreaks of toxic algal species, culturists and commercial fishermen alike must still depend on reliable monitoring systems to warn of toxic shellfish and plan their activities accordingly. Economic losses can be kept to a minimum through the combined efforts of an intensive monitoring program and culture of "rapid release" species (e.g., *Mytilus edulis*), species known to avoid toxic dinoflagellates (e.g., *Mercenaria*, most oysters) or scallops (adductor muscles rarely if ever toxic) (Shumway et al. 1988).

Prediction and Monitoring

With all of these effects on the industry, the question is then asked, what can be done? The most obvious answer is: Find a way of predicting the onset of blooms. The advantages of being able to predict the occurrence of potentially detrimental algal blooms are obvious, and any means of predicting a bloom would be a great advantage. Early detection would allow officials to warn people of the impending blooms, and a forewarning to culturists could save them from economic disaster. Unfortunately, no practical way of predicting the development of blooms exists at present, although attempts are underway by many investigators.

There is increasing evidence that nearly all, if not all, blooms originate in the ocean, not in bays (Holligan 1985), and it is possible that key meteorological/oceanographic parameters might indicate a high or low probability of a bloom (Steidinger and Haddad 1981). Oceanographers can identify areas where there is a high probability that a bloom may occur; however, prediction is not a possibility at this time. Ouchi (1982) has proposed a model based on the discriminant analysis of temperature, salinity, total dissolved phosphates, dissolved inorganic nitrogen, dissolved organic nitrogen and particulate organic nitrogen. Fraga et al. (1988) have proposed the development of a bloom prediction capability for some dinoflagellate species based on an upwelling index indicative of the movement of offshore surface waters into rias. They combined meteorological, hydrographic and biological conditions to predict possible bloom conditions within the rias of northwest Spain, a major shellfish producing region, and suggest that the blooms in this region may be predictable in the future. Paerl (1988) has recently reviewed the commonalities of combinations of environmental factors most likely to elicit nuisance blooms. He also presented criteria for deeming a water body "bloom sensitive." Undoubtedly, as more studies are undertaken, correlations be-

tween bloom events and environmental parameters will provide a more precise predictive capability.

Since most blooms originate offshore, satellite imagery or satellite-tracked monitoring buoys can assist in early detection of blooms. Yentsch (1987) has proposed the use of remote sensing (the quantitative assessment of the numbers of algae and the dimensions of the patch without directly sampling the water mass) facilities to monitor the formation of algal blooms. This requires observations from considerable heights, and he proposes that vehicles such as satellites, aircraft or balloons could serve as a part of an early warning system (Figs. 2, 3, 4). These vehicles must be equipped with sensors designed to monitor specific environmental parameters previously associated with algal blooms. Instrumentation for satellites and aircraft have been developed which utilize the light absorption and/or light emitted as fluorescence from algae. Unfortunately there is no clear cut means of distinguishing toxic and nontoxic blooms.

One major system being established is in Norway: MARINET. The system is currently being tested and is expected to be in full operation soon. This is a warning and forecast service that will include noxious algae and algal toxins. The system is composed of various types of remote sensing, a network of rapporteurs and laboratories where algae and toxins will be identified. The ultimate goal is for the establishment of an international contact network (Tangen 1987). ICES has established National Coordinating Centers for Exchange of Information on Exceptional Algal Blooms (ICES 1988, 1989). Anyone planning a sea farming business or needing information about blooms would do well to contact the National Coordinating Center in his country.

While a successful method of prediction would be hailed by the shellfish industry, the above systems are expensive and not readily available at present. In the absence of capabilities for prediction, monitoring is

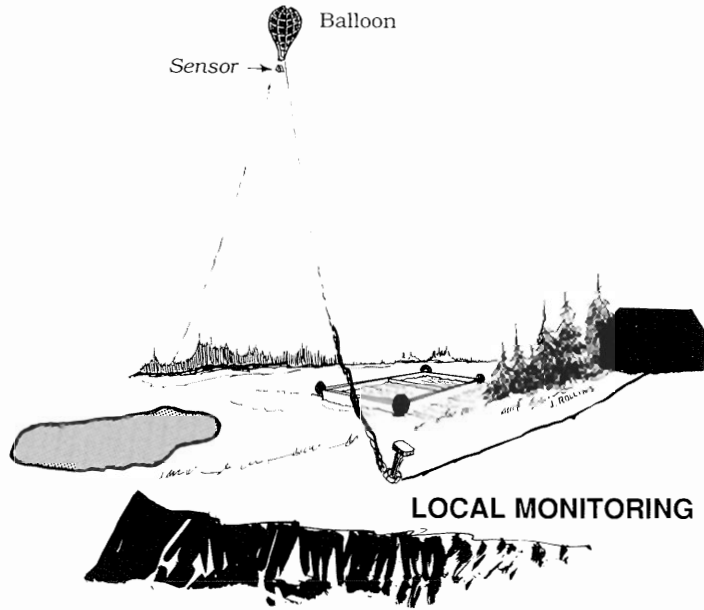


FIGURE 2. Local monitoring of water characteristics using a balloon arrangement. From Yentsch (1987) with permission.

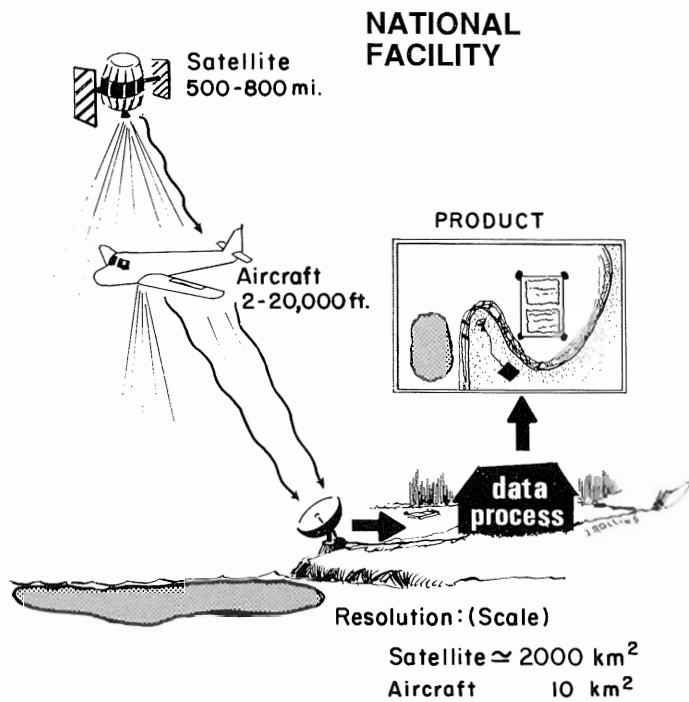


FIGURE 3. National Remote Sensing Facility from C. S. Yentsch (1987) with permission.

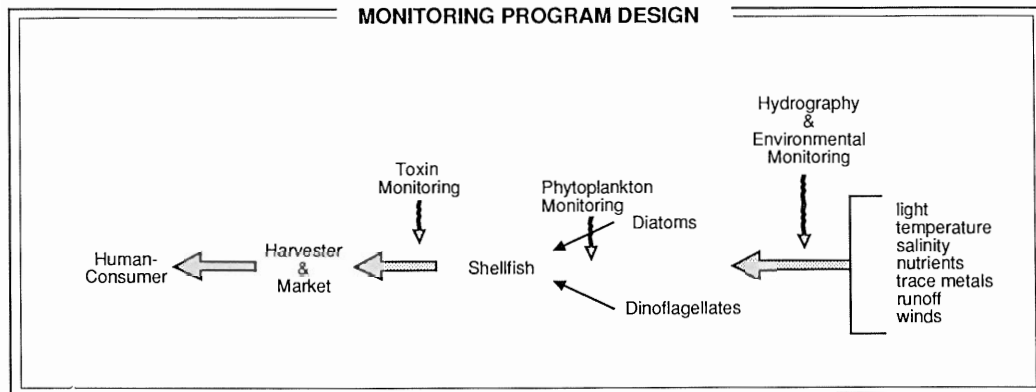


FIGURE 4. Monitoring program where early warning is provided by environmental monitoring. From C. S. Yentsch (1987) with permission.

still the most powerful tool available to management. Monitoring of phytoplankton can provide forewarning of potentially harmful conditions and also an early detection of accidentally introduced species that may pose potential hazards, e.g., the recent introduction of *Gymnodinium* to Tasmania via ships ballast (Hallegraeff et al. 1988). This type of monitoring is a routine aspect of mariculture in Japan where the cost of phytoplankton-caused damage can exceed millions of dollars annually. Early monitoring and warning paid off in Ireland in 1984 when a phytoplankton monitoring program showed the presence of *Protogonyaulax*. Nearby mussel farms were warned of the potential danger and their product tested positive for PSP. The potential harvest of harmful mussels was avoided (Doyle, personal communication). In another instance, Anderson et al. (1982) and Schrey et al. (1984) reported the presence of toxic cysts and motile cells of *P. tamarensis* in areas historically free from outbreaks of shellfish toxicity, a finding which should alert potential culturists to the necessity for monitoring local waters.

Many countries have established comprehensive monitoring programs, usually in response to a massive outbreak of toxic algae. Blooms resulting in fatalities seem to get the most immediate attention. Japan (Anraku 1984), USA (Hurst 1982; Shumway et al. 1988; Lutz and Incze 1979;

Yentsch and Incze 1980; Neve and Reichardt 1984) and Canada (Bruce and Delaney 1972) lead the field. Europe is currently developing programs (Fufari and Hunt 1981) and an extensive monitoring program was recently established in Tasmania. Certainly more are needed, especially in developing countries where primitive culture facilities are common and technical assistance may be lacking. Large scale programs such as those established in Japan, USA and Canada provide an early warning system and minimize the losses due to blooms by implementing both area- and species-specific closures.

While regular water sampling and satellite monitoring will help to locate red tides in their early stages of development, the methods are by no means failsafe, making it difficult for farms and aquaculture facilities to plan harvests. Even in the event of an early warning, it is impossible to prevent most species of bivalves from becoming toxic. An early warning can, however, prevent the selling and consumption of toxic shellfish and/or allow growers to harvest early or plan their harvests around the blooms to avoid unnecessary economic losses.

Economic Threat

Toxic algal blooms present not only a public health hazard, but a major economic threat as well. Blooms may affect the fish-

eries and culture efforts by rendering products toxic and thus unmarketable, by directly killing the shellfish, or by what is known as the halo (see below). It is difficult to assess the cost of a shellfish closure, and accurate economic analyses are generally not available (Conte 1984; Kahn and Rockel 1988). Table 2 is a summary of available data on economic losses associated with toxic algal blooms. This table is not intended to be an all inclusive analysis of economic losses, but rather an indication of the extent of losses which have been incurred.

Probably more devastating than the blooms themselves are the subsequent publicity, dissemination of misinformation and public uneasiness. The impact of these factors goes far beyond the dramatic decline in demand for the products. Bans on shellfish result in loss of jobs, unemployment for fishermen and the secondary industries such as processing, middlemen, suppliers, and the bans also pose problems for international trade and discourage the expansion of the industry into aquaculture.

In addition, the impact of a toxic algal bloom is not always restricted to the immediate area. During the 1972 outbreak of "red tide" in New England, shellfish (including mussels, clams, quahogs and oysters) were all removed from the market in Maine and Massachusetts. The scallop, *Argopecten irradians* was briefly included in the ban but later restored to the market as only the adductor muscle of this species is eaten. New York and Connecticut followed suit with a safety measure by stopping the importation and sale of shellfish from the affected states. Although the ban included only the species mentioned above, consumers responded by avoiding other species (including fish, lobsters, *Homarus americanus*, sea scallops, *Placopecten magellanicus*, and northern shrimp, *Pandalus borealis*). Losses to the fishermen were estimated by NMFS to be more than \$1 million due to the adverse publicity (Jensen 1975).

Conte (1984) reported that during a PSP

outbreak in California in 1980 (San Francisco Bay), the purchasing and consumption of oysters virtually ceased. This was primarily due to news media coverage, and the problem was intensified by the distribution of misleading information and failure to distinguish between affected and nonaffected areas. As often happens, there was a lack of positive news coverage after the ban was lifted. In this particular instance, the most severe impact of the bloom was disruption of the industry's cash flow, so that revenue necessary for reseeded operations by the commercial growers was not available (Conte 1984).

White et al. (1984) described the nature of reports during a toxic bloom in the Philippines where 41 press releases over a period of six months gave conflicting accounts. Further, in some areas where literacy was low, information passed by word of mouth added to confusion and public wariness (White et al. 1984).

A recent outbreak of red-tide in Manila Bay caused extensive economic damage (Maclean 1989). Prices of all seafood dropped to 40% of normal prices and landings were officially assessed. In yet another example of the "halo effect," Japan and Singapore "were said to have banned shrimp imports from the Philippines" (Maclean 1989). In this instance, losses to the vinegar industry and fuel oil revenue were also widespread.

Most recently, the occurrence of domoic acid in mussels from Canadian waters resulted in considerable economic losses not only to the Canadian mussel growers, but to mussel producers throughout New England. In addition, the general public was leery of most shellfish for an extended period of time after the "bloom," and restaurant owners also suffered.

Clearly, misinformation can have disastrous effects on sales of shellfish (both affected and unaffected species) (Fig. 5). Once consumer confidence is lost, it is a long and arduous process to reestablish it. Many agencies have attempted to rectify the sit-

uation by attempts to avoid the publication of misleading information. The Shellfish Institute of North America (SINA) has distributed special issues of informative newsletters and has met with Federal officials to exchange accurate information. SINA reported (Jensen 1975) that news releases issued by the U.S. Food and Drug Administration (FDA) were not always properly quoted by the news media and cited as an example the fact that species unaffected by red tide were not included in published news articles although they were named in the original release. In California, closure announcements and other information regarding PSP are issued to the news media by only one agency, the Department of Health Services, to avoid multiple or confusing reports (Nishitani and Chew 1988). In Canada, the Department of Fisheries and Oceans oversees a program including collection, testing, enforcement, management and dissemination of public information at an annual cost over \$1 million (Pirquet 1988).

The efficiency of various surveillance and monitoring programs has alleviated some of the problems associated with misinformation and adverse publicity, although headlines such as those depicted in Fig. 6 continue to appear, often with little in the way of clear and informative explanations of the extent and consequences of the blooms.

In areas prone to toxic algal blooms, consumers must be constantly reassured of the quality of seafood products being marketed. In addition to reports of the onset of blooms, news media must be encouraged to give equal coverage when the bans on shellfish are lifted. Further, information should be given which specifically identifies the shellfish species involved while at the same time identifying those species which remain unaffected. One of the best campaigns aimed at reversing the adverse effects of toxic algal blooms currently is being run by the Canadian government in the aftermath of the outbreak of ASP in Prince Edward Island and is designed to reestablish shellfish as a

top quality product. The campaign has already received \$2.2 million (Pirquet 1988) and should serve as an example to other areas prone to major economic losses associated with toxic algal blooms. Only through increased public awareness associated with accurate and positive publicity can shellfish harvesters hope to combat the detrimental and often disastrous results of publicity and misreporting of the effects of toxic algal blooms.

Considerations for Aquaculture

In light of the fact that one toxic algal bloom could represent financial disaster to an investor virtually overnight, considerable attention should be paid to site selection and habitat assessment. Criteria have been suggested by Parker (1987) for finfish farmers which are, for the most part, just as applicable to shellfish farmers. Dale et al. (1987) also considered the role of monitoring for toxic dinoflagellates in assessing the suitability of sites for starting aquaculture projects as part of a workshop convened to address the problems of toxic dinoflagellate blooms in aquaculture. The following activities, synthesized from the above two studies, should be considered in assessing site risk:

- 1) A thorough hydrographic survey including rates of water exchange and presence of offshore frontal or upwelling systems. High rates of exchange reduce the risks of *in situ* blooms but may allow transport inshore of offshore blooms, and frontal or upwelling systems may be seed areas for offshore blooms. Ideally, the site waters will be fully mixed and in contact with fully mixed sea areas.
- 2) An assessment of the nutrient status of the local waters and the accumulation of soft sediments (usually an indication of poor circulation).
- 3) A quick survey of the phytoplankton community and an investigation of previously recorded species, paying special attention to the presence of known nox-

ious species and outbreaks of PSP, DSP, ASP, etc.

- 4) A survey for the presence of cysts in local sediments, often an indication that a bloom is possible.

If at all possible, a pilot study should be undertaken prior to any major investment of time or money. This study should include a detailed analysis of commercial, financial and administrative aspects of the proposed venture. A small number of shellfish should be installed as test organisms with continuous monitoring of the environment as well as of the animals.

In addition to the considerations listed above, careful attention to species selection and method of grow-out will provide the shellfish farmers with some security. For instance, mussels are known to accumulate toxins much faster than other species and their culture in locations prone to outbreaks of toxic algae requires special attention to possible PSP, DSP and ASP hazards. The European oyster, *Ostrea edulis*, is known to feed selectively on dinoflagellates (Shumway and Cucci 1987) and in Maine waters is known to become toxic even before mussels (Shumway et al. 1988). On the other hand, in Maine waters the time of peak PSP danger coincides with the season when the quality of both oysters and mussels is lowest. Further, there is a conservation closure on wild *O. edulis* from June 15–September 15. Oysters (*Crassostrea* spp.), in general, tend to be less toxic than other species and also tend to release accumulated toxins at a rapid rate. *Crassostrea virginica* is rarely toxic (Hurst, personal communication; Shumway, in preparation) nor is the quahog, *Mercenaria mercenaria*, one of the most profitable species for culture in the United States (Castagna, personal communication). Scallops are the safest bet as long as the adductor muscle is the only product sold. To date, toxin levels above quarantine levels have not been reported from adductor muscles (Hurst, personal communication; Cembella and Shumway, unpublished). Further,

Shimizu and Yoshioka (1981) have demonstrated that some toxin inactivation takes place in the adductor muscle. Other scallop tissues, including the gonad, mantle and digestive glands, have been shown to be highly toxic throughout the year, even in the absence of any toxic algae (Shumway et al. 1988; Bourne 1965; Jamieson and Chandler 1983). Efforts are currently underway in Alaska (Anonymous 1989) to establish a commercial fishery based on pink or spiny scallops which are small and eaten whole. Particular attention should be paid to possible accumulation of PSP toxins before such efforts are expanded. Other species which retain toxin for extended periods, e.g., butter clams and surf clams, should be avoided.

Attention should also be paid to the method of culture. It has been demonstrated in some areas that species grown in rope or raft culture tend to toxify more quickly than bottom grown animals, e.g., scallops in Japan (Shimizu 1982) and mussels in Tasmania (Table 1). Knowledge of species-specific toxification and detoxification rates will allow estimates to be made of times of no harvest or "time off the market."

Finally, culturists should ascertain means for monitoring their shellfish or for having them tested regularly for the presence of toxins. Ensuring a supply of non-toxic shellfish to consumers is the responsibility of harvesters, processors and the regulatory agencies. More and more, the onus is on the grower to guarantee a clean product as indicated by the regulations set forth by the Maine Department of Marine Resources with regard to toxic shellfish:

"Chapter 15—General Shellfish Sanitation and Depuration Provisions

15.06. *Shellfish Contamination Standards—Paralytic Shellfish Poisoning*

- A. It shall be unlawful to buy, receive, sell, possess, ship, transport, shuck or otherwise process shellfish in any form, regardless of origin, at or prior to a retail sale to a consumer or at or prior to the

wholesale level in preparation for sale to another wholesale or retail establishment, where the shellfish contain more than 80 micrograms toxin per 100 grams of shellfish meat.

- B. *Embargo of Shellfish.* Where samples taken from shellfish indicate that those shellfish contain more than 80 micrograms toxin per 100 grams of shellfish meat, the Commissioner may embargo the contaminated shellfish, as well as any other shellfish which are likely to be contaminated in the same vehicle or facility, in accordance with the embargo powers granted to the commissioner in 12 M.R.S.A. § 6856(6).
- C. *Sampling.* The Department shall collect samples of shucked shellfish and shell stock from each shellfish certificate holder periodically in order to ensure that all shellfish meet contamination standards, described above. The Department shall also collect samples from shellfish shipped or transported into this state by shellfish dealers from other states or counties in order to ensure such shellfish comply with contamination standards, described above."

What practical measures can be taken to reduce the impact of a toxic algal bloom? Management options in the event of a bloom are few. Rafts can be moved, although this is probably impractical under most conditions. Shellfish can be marketed early, prior to intoxication, if the bloom is detected early enough. In the event of annual outbreaks, growers and harvesters can simply plan their market time around possible periods of closure. Unfortunately, there are no practical solutions to reduce the impact of a bloom. Careful site selection, species selection and an early warning system can certainly soften the impact.

Are toxic algal blooms and aquaculture mutually exclusive? The answer is, *absolutely not.* Toxic dinoflagellates represent only a small part of the phytoplankton community and blooms represent only a part of the total risk to aquaculturists. It is en-

couraging to note from the recent review by Nishitani and Chew (1988) that despite the apparent increases in occurrences of high PSP toxin concentrations in shellfish, aquaculture and the harvest of wild bivalves have increased in Alaska, Washington, Oregon and California during the last decade. This increase has been in both the number of species being harvested commercially and in the areas involved. These increases have been made possible at least in part by the expanded monitoring programs of each of these states. It must also be recognized that the impact of a toxic algal bloom goes far beyond the simple inability to harvest and sell the product. As more emphasis is placed on shellfish culture, so must an emphasis be placed on the establishment of reliable and accessible monitoring programs. Further research is needed to develop simple, reliable field tests and other laboratory tests (Hurst et al. 1985) for rapid determination of toxicity levels. Toxic algal blooms have assumed a global perspective; as more emphasis is placed on shellfish culture, so must emphasis be placed in ensuring that monitoring programs are established.

An interdisciplinary approach is important. Research and management are intimately linked in the formulation of regulations. Administrators cannot hope to develop and/or impose suitable and functional regulations without the input of knowledgeable researchers. Policies regarding public health and economic considerations must be based on sound scientific data if they are to be efficient, functional and accepted by the general populace. Finally, an emphasis must be placed on increased public awareness and education of the public sector through structured news releases and well informed medial personnel.

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¹ Original papers or copies of all references have been consulted.