

**HARMFUL
PHYTOPLANKTON
SURVEILLANCE IN
WESTERN AUSTRALIA**



Waterways Commission
Report No 43
1994



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CONTENTS

Acknowledgments	i
Summary	vi
1 Background	1
1.1 International scene	1
1.1.1 New Zealand	3
1.1.2 North America	3
1.1.3 Europe	3
1.1.4 Asia and Japan	4
1.1.5 South America	4
1.2 Threats to Australia	4
1.2.1 Paralytic Shellfish Poisoning (PSP)	5
1.2.2 Diarrhetic Shellfish Poisoning(DSP)	6
1.2.3 Amnesic Shellfish Poisoning (ASP)	6
1.2.4 Neurotoxic Shellfish Poisoning (NSP)	6
1.2.5 Hepatotoxicity	6
1.2.6 Ciguatera	7
2 Blue-green algal blooms in WA	7
2.1 <i>Nodularia</i> in Peel-Harvey estuary, Vasse-Wonnerup, Swan-Avon and Blackwood River.	7
2.1.1 Toxicity testing	8
2.1.2 Threats to human health	8
2.1.3 Threats to domestic animals and fish	11
2.1.4 Ecological threats	11
2.2 Future of blue-green blooms post-Dawesville Channel	11
2.2.1 Future of impacts in the estuarine system	12
2.2.2 Future of blue-green blooms in tidal rivers	14
2.2.3 Toxicity testing	14
2.3 Urban and rural wetlands and farm dams	14
2.3.1 Toxicity testing	15
2.3.2 Human health threats	15
2.3.3 Threats to domestic animals	16
2.3.4 Ecological threats	16
2.4 Other waterways	17
2.4.1 Swan-Canning River	17
2.4.2 Future threats to the Swan Estuary	18
2.4.3 Other waterways	19
2.4.4 Coastal waters	19



3	Dinoflagellates in Western Australia	20
3.1	Current estuarine occurrences	20
3.1.1	<i>Scrippsiella</i>	21
3.1.2	<i>Prorocentrum</i>	22
3.1.3	<i>Alexandrium</i>	25
3.1.4	<i>Dinophysis</i>	26
3.1.5	<i>Gymnodinium</i> , <i>Gyrodinium</i> , <i>Cochlodinium</i> and <i>Amphidinium</i>	28
3.1.6	<i>Ensiculifera</i>	28
3.1.7	<i>Ostreopsis</i>	29
3.1.8	Other dinoflagellate species	29
3.2	Other estuaries in WA	29
3.3	Near shore marine areas	29
3.3.1	Cockburn Sound to Geographe Bay	29
3.3.2	Perth metropolitan waters	29
3.3.3	Other marine areas	30
3.3.4	Ports and harbours	30
4	Toxic diatoms, Raphidophytes, Prymnesiophytes and other red tide organisms	32
4.1	Diatoms	32
4.1.1	<i>Pseudonitzschia</i>	32
4.1.2	<i>Chaetoceros</i>	32
4.1.3	<i>Rhizosolenia</i> cf. <i>chunii</i>	33
4.1.4	<i>Amphora coffeaeformis</i>	33
4.2	Silicoflagellates	34
4.3	Raphidophytes	34
4.4	Prymnesiophytes	35
4.5	Chlorophytes	36
4.6	Cryptophytes	36
4.7	Euglenophytes	36
4.8	Chrysophytes	37
4.9	Coccolithoporids	37
4.10	Cholera amoeba and other threats	37
5	Current mechanisms for surveillance in Western Australia	38
5.1	Surveillance	38
5.2	Toxicity testing	38
5.3	Surveillance of toxic blooms in WA	39



5.4 Gaps in current efforts	40
5.4.1 Surveillance	40
5.5 Solutions for Western Australia	41
5.6 Toxicity testing and risk assessment	41
5.6.1 Detection and quantification of algal toxins	41
5.6.2 Paralytic Shellfish Poisoning (PSP)	41
5.6.3 Diarrhetic Shellfish Poisoning (DSP)	42
5.6.4 Amnesic Shellfish Poisoning (ASP)	42
5.6.5 Neurotoxic Shellfish Poisoning (NSP)	43
6 Conclusions	45
7 References	46

APPENDICES

1	Potentially harmful phytoplankton blooms and red tides observed in Western Australian waterways excluding the Swan-Canning River	49
2	Potentially harmful phytoplankton blooms observed in the Swan-Canning and Avon rivers in Western Australia	51
3	Harmful algal blooms and red tides in the Mandurah area, excluding the Peel-Harvey estuary	56
4	Harmful algal blooms and red tides in the Peel-Harvey estuary (1983-85)	57
5	Harmful algal blooms and red tides in the Peel-Harvey estuary (1985-1992)	58
6	Relationships between W A stakeholders and issues of harmful phytoplankton surveillance	60
7	Relationships between W A stakeholders and areas of phytoplankton surveillance	61
8	A review of harmful algal blooms and their apparent global increase (Hallegraeff G M, 1993).	62

TABLES

1	Clinical symptoms of various types of shellfish poisoning	5
2	Acute mammalian toxicity values for <i>Nodularia</i> in Peel-Harvey estuary compared to common pesticides	10

FIGURES

1	Map showing some of the estuaries and rivers in south Western Australia.	2
2	Phytoplankton composition in the Harvey Estuary 1991-1992	8
3	Mean <i>Nodularia</i> cell densities for the Harvey Estuary from 1983 to 1992	10
4	The densities of <i>Synechococcus</i> in the Peel-Harvey estuary in 1990	12
5	<i>Anabaena</i> bloom in the Peel-Harvey estuary spring 1993	14
6	Mean dinoflagellate densities from the Peel-Harvey estuary 1993-94.	21
7	<i>Prorocentrum minimum</i> densities from sites in the Harvey Estuary in 1993.	23



PHOTOGRAPHS

1	Potentially harmful <i>Alexandrium minutum</i> from the lower Swan River estuary	6
2	<i>Nodularia spumigena</i> from the Peel-Harvey estuary and Avon River in Western Australia	9
3	Thick <i>Nodularia</i> bloom scum accumulations on the Harvey Estuary foreshore	11
4	<i>Anabaena</i> cf. <i>circinalis</i> filaments from the Serpentine River	13
5	<i>Oscillatoria</i> and <i>Anabaena</i> from the Canning River	15
6	<i>Microcystis</i> spp. from the Serpentine River at Mandurah	16
7	Warning signs erected adjacent to the Canning River at Kent Street Weir	17
8	<i>Anabaena</i> cf. <i>spiroides</i> from the Canning River	18
9	<i>Nodularia</i> with akinetes from the upper Canning River in 1994	18
10	<i>Aphanizomenon</i> from the Blackwood River	19
11	<i>Trichodesmium</i> (<i>Oscillatoria</i>) <i>erythraeum</i> from the Hardy Inlet	20
12	<i>Scrippsiella</i> spp. from the Swan River estuary	21
13	Dinoflagellate bloom scum adhering to the skin of swimmers from the Swan River estuary	22
14	<i>Prorocentrum minimum</i> from the upper Swan River estuary	23
15	<i>Prorocentrum dentatum</i> from the lower Swan River estuary	24
16	Other <i>Prorocentrum</i> species from Western Australia waters	25
17	<i>Alexandrium minutum</i> from the Swan River estuary	26
18	<i>Dinophysis</i> species from the Peel-Harvey estuary and <i>Dinophysis fortii</i> from Oyster Harbour, Albany	26
19	<i>Phalacrocoma</i> (<i>Dinophysis</i>) <i>rotundatum</i> species from the lower Swan River estuary	27
20	<i>Dinophysis caudata</i> from the Swan River estuary	27
21	<i>Ensiculifera</i> from the Swan River estuary and the Murray River	28
22	Benthic dinoflagellate <i>Ostreopsis</i> from Princess Royal Harbour, Albany	29
23	Ballast water discharge in the Bunbury Inner Harbour	30
24	Sampling for dinoflagellate cysts in harbour sediment at Bunbury	31
25	Dinoflagellate cysts from the Swan River estuary	31
26	<i>Pseudonitzschia pungens</i> from the Swan River Estuary	32
27	<i>Chaetoceros</i> diatom species with spines	33
28	<i>Distephanus speculum</i> , <i>Ebria tripartita</i> and the silicoflagellates <i>Dictyoch</i> and <i>fibula</i> from Oyster Harbour, Albany	34
29	Raphidophyte <i>Heterosigma</i> and <i>Fibrocapsa</i> species from the Swan River estuary	35
30	<i>Cricosphaera</i> scum from a canal in the Murray River at Mandurah	36
31	<i>Coccolithoporids</i> from the lower Swan River estuary	37
32	<i>Microtox</i> unit at the Chemistry Centre of WA	39
33	Remote sensing image of a <i>Eutroptiella</i> bloom from an upper Swan River estuarine red tide	39
34	High Performance Liquid Chromatography (HPLC) unit at the Chemistry Centre of WA	43



PHOTOGRAPHS

1	Potentially harmful <i>Alexandrium minutum</i> from the lower Swan River estuary	6
2	<i>Nodularia spumigena</i> from the Peel-Harvey estuary and Avon River in Western Australia	9
3	Thick <i>Nodularia</i> bloom scum accumulations on the Harvey Estuary foreshore	11
4	<i>Anabaena</i> cf. <i>circinalis</i> filaments from the Serpentine River	13
5	<i>Oscillatoria</i> and <i>Anabaena</i> from the Canning River	15
6	<i>Microcystis</i> spp. from the Serpentine River at Mandurah	16
7	Warning signs erected adjacent to the Canning River at Kent Street Weir	17
8	<i>Anabaena</i> cf. <i>spiroides</i> from the Canning River	18
9	<i>Nodularia</i> with akinetes from the upper Canning River in 1994	18
10	<i>Aphanizomenon</i> from the Blackwood River	19
11	<i>Trichodesmium</i> (<i>Oscillatoria</i>) <i>erythraeum</i> from the Hardy Inlet	20
12	<i>Scrippsiella</i> spp. from the Swan River estuary	21
13	Dinoflagellate bloom scum adhering to the skin of swimmers from the Swan River estuary	22
14	<i>Prorocentrum minimum</i> from the upper Swan River estuary	23
15	<i>Prorocentrum dentatum</i> from the lower Swan River estuary	24
16	Other <i>Prorocentrum</i> species from Western Australia waters	25
17	<i>Alexandrium minutum</i> from the Swan River estuary	26
18	<i>Dinophysis</i> species from the Peel-Harvey estuary and <i>Dinophysis fortii</i> from Oyster Harbour, Albany	26
19	<i>Phalacrocoma</i> (<i>Dinophysis</i>) <i>rotundatum</i> species from the lower Swan River estuary	27
20	<i>Dinophysis caudata</i> from the Swan River estuary	27
21	<i>Ensiculifera</i> from the Swan River estuary and the Murray River	28
22	Benthic dinoflagellate <i>Ostreopsis</i> from Princess Royal Harbour, Albany	29
23	Ballast water discharge in the Bunbury Inner Harbour	30
24	Sampling for dinoflagellate cysts in harbour sediment at Bunbury	31
25	Dinoflagellate cysts from the Swan River estuary	31
26	<i>Pseudonitzschia pungens</i> from the Swan River Estuary	32
27	<i>Chaetoceros</i> diatom species with spines	33
28	<i>Distephanus speculum</i> , <i>Ebria tripartita</i> and the silicoflagellates <i>Dictyoch</i> and <i>fibula</i> from Oyster Harbour, Albany	34
29	Raphidophyte <i>Heterosigma</i> and <i>Fibrocapsa</i> species from the Swan River estuary	35
30	<i>Cricosphaera</i> scum from a canal in the Murray River at Mandurah	36
31	<i>Coccolithoporids</i> from the lower Swan River estuary	37
32	<i>Microtox</i> unit at the Chemistry Centre of WA	39
33	Remote sensing image of a <i>Eutroptiella</i> bloom from an upper Swan River estuarine red tide	39
34	High Performance Liquid Chromatography (HPLC) unit at the Chemistry Centre of WA	43



1 BACKGROUND

Microscopic algae are important as oxygen producers, and food sources for filter-feeding shellfish and the larvae of commercially important crustacea and finfish. Proliferation of microscopic algae can be of great benefit to aquaculture and wild fisheries through increased productivity. In some circumstances however, severe economic losses to aquaculture, fisheries, native birdlife and tourism, and serious human health impacts can arise when unacceptable levels of particular phytoplankton species occur. Species with barbed spines can rupture delicate gill membranes in fish and shellfish, some produce gill-clogging mucus and other species can produce powerful neurotoxins, which accumulate in edible tissues. One species of dinoflagellate which has been responsible for the deaths of millions of fish, is stimulated to germinate from its benthic cyst by the presence of fish excreta, release its neurotoxins then feed on the fish (Huyghe, 1993). Even non-toxic phytoplankton species can cause anoxic water conditions when decay and respiration of dense blooms occur. Shell fisheries in Florida are regulated on the basis of cell counts of *Gymnodinium breve* as low as 5,000 cells per litre.

The presence of these species can result in reduced growth rates and vigour in aquaculturally grown animals, and the potential for extensive fish and marine life kills. Extensive fish kills have occurred in Western Australian waterways in the Murray, Capel and Swan rivers, the Vasse-Wonnerup estuary and the Yunderup Canals. Some of these have been explained by anoxia following the collapse of algal blooms. The Vasse-Wonnerup estuary contains a Prymnesiophyte species, not found elsewhere in Australia, which has been associated with mass fish mortalities overseas.

Human health threats and fatalities can occur after the consumption of shellfish that have been contaminated with algal neurotoxins. Even small fish such as *Sardinella* and *Decapterus* can be PSP toxic because the intestines contain *Pyrodinium* cells (Ming and Wong, 1989). These neurotoxins will accumulate in shellfish if their food source includes toxic species of microalgae/dinoflagellates. There are indications that during toxic bloom events molluscs other than bivalves can become toxic. These organisms include gastropods such as abalone and predatory and scavenging whelks. The alimentary canals of predatory crabs, crayfish and starfish may contain toxins in the digestive system, but rarely in the normally consumed muscle tissue (Marsden, 1993).

These effects can lead to reduction in the growth or vigour of aquacultural animals or their death, and possible human health threats.

1.1 International scene

The international scene with regard to the spread of harmful phytoplankton blooms has been thoroughly covered by Dr G Hallegraeff (1993) (Appendix 3). At present the paralytic shellfish poisoning must be considered as a global problem that requires both better professional as well as public awareness (Kao, 1993).

There has been a major expansion in the frequency, magnitude and geographic extent of toxic algal blooms and red tides over the past two decades globally (Hallegraeff, Appendix 3). This increased occurrence is attributed to:

- i) increased pollution and eutrophication in coastal waters,
- ii) increased awareness by scientists and better analytical capabilities,
- iii) dispersal of species by contaminated ballast water discharges,
- iv) dispersal of species by ocean currents,
- v) greater numbers of aquaculture establishments which have amplified the potential for sentinels for contamination.

Many countries have established comprehensive surveillance programs, to monitor for potentially toxic and harmful phytoplankton species in their waters in order to safeguard human health and to protect valuable aquaculture industries. The practice by India, of scrapping the hulls of old ships in their coastal waters, could be releasing potentially toxic dinoflagellate cysts, and other potentially harmful species, accumulated in the sediments of ballast tanks over 20-30 years of international travel.

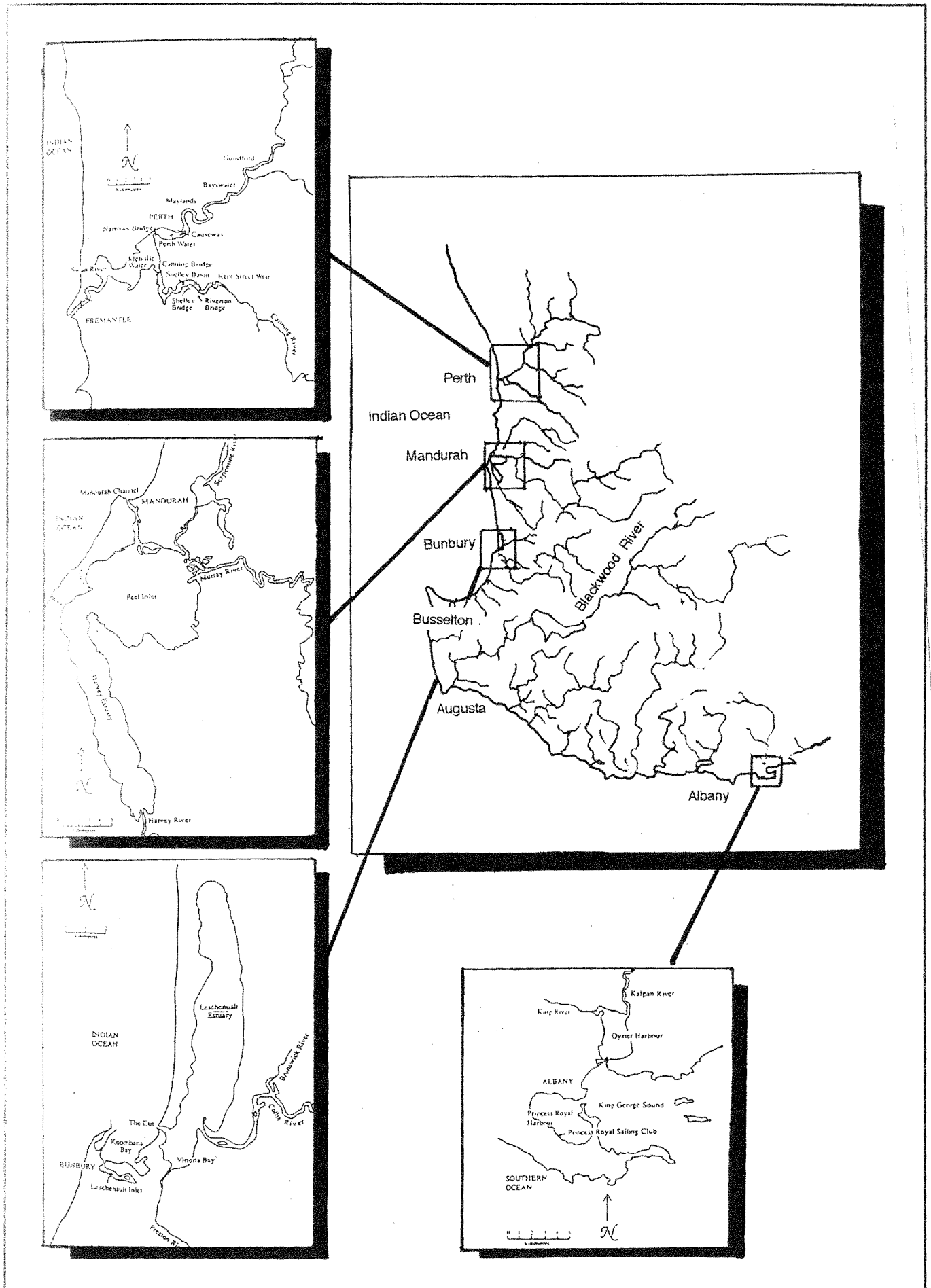


Figure 1. Map showing some of the estuaries and rivers in south Western Australia



1.1.1 New Zealand

Prior to January 1993, no NSP producing toxic phytoplankton species had been recorded from the coastal waters surrounding New Zealand. By March 1993 a dinoflagellate species *Gymnodinium cf. breve* through a relatively minor component of the total phytoplankton, was identified as the cause of 180 cases of shellfish poisoning through the consumption of affected shellfish (MacKenzie, 1993). A valuable export industry was quarantined, causing massive economic loss to shellfishermen (Smith et al., 1993). As a result testing is now required for Paralytic Shellfish Poisoning (PSP), Diarrhetic Shellfish Poisoning (DSP), Amnesic Shellfish Poisoning (ASP), and Neurotoxic Shellfish Poisoning (NSP) at an annual cost of \$3 million.

1.1.2 North America

In the United States of America, PSP, DSP, NSP, brown tides, ASP, ciguatera, fish mortality (diatoms and Raphidophytes) and noxious macroalgal problems occur in coastal waters and estuaries. The USA is creating a National Plan for Marine Biotoxins and Harmful Algae to direct and co-ordinate what were *ad hoc* responses to individual outbreaks. The Federal National Oceanic and Atmospheric Administration is to comprehensively examine harmful algal blooms through its Coastal Ocean Program and its National Marine Fisheries Service laboratories and grant programs. Rapid response teams will be organised for sudden bloom outbreaks (IOC UNESCO, 1992).

In Canada in 1987, an outbreak of domoic acid accumulation (ASP) in shellfish caused by diatom blooms resulted in the deaths of three people and the hospitalisation of hundreds of others. This toxin which causes Amnesic Shellfish Poisoning (ASP) was also detected in Australia for the first time in Victoria in 1993. Here in Western Australia we have the potential for a similar scenario to develop therefore an appropriate routine surveillance and management program should be established.

In Canada the Science Section of the Department of Fisheries and Oceans conducts a large national research program on harmful marine algae in an effort to protect consumers of seafood as well as the wild and aquaculture fishing industry (IOC UNESCO, 1992).

1.1.3 Europe

In French coastal waters, *Dinophysis* blooms which cause Diarrhetic Shellfish Poisoning (DSP) have been spreading and *Gyrodinium cf. aureolum* blooms have caused heavy losses of bivalves. The potential PSP producing *Alexandrium minutum* blooms have not so far caused problems in France. In 1988 to satisfy the demand for knowledge on the status of the marine coastal environment, the Ministries of the Environment, of the Sea and of Scientific Research encouraged fundamental research on conditions and mechanisms of harmful phytoplankton blooms in French waters (IOC UNESCO, 1992).

Though open waters around Greece are oligotrophic, some gulfs are undergoing discolouration by red tides and gel patches. Some research on phytoplankton, public health and seafood and oceanography is being carried out (IOC UNESCO, 1992).

Harmful algal blooms have not yet posed too serious an impact on German coasts. They expect that these are likely to occur at any time and have set up the Institute for Baltic Research which will establish a working group on aspects of harmful algal blooms (IOC UNESCO, 1992).

Aquaculture in Ireland is a very valuable industry. Red tides that kill fish and induce DSP toxicity in shellfish have ensured that a comprehensive monitoring program is maintained in Irish waters. The Fisheries Research Centre of the Department of the Marine is the designated laboratory for algal toxins in shellfish meats, cyst distribution, and routine and intensive phytoplankton monitoring in Ireland (IOC UNESCO, 1992).

In Italy, mucilage from benthic algae has had a severe effect on the fishery and tourism industry. Three *Alexandrium* species and *Gymnodinium catenatum* are present in coastal waters. The presence of bans on the taking of mussels due to cholera since 1973 is believed to have prevented human health effects by these species. There is no co-ordinated national project on harmful algal blooms in Italy (IOC UNESCO, 1992).

Following a major outbreak of PSP in 1976, Spain has an established monitoring program carried out by the Instituto Espanol de Oceanografia. Toxins are monitored by regional and national health authorities. PSP (*Alexandrium minutum*, *Gymnodinium catenatum*) and DSP (*Dinophysis* spp.) are the two prime concerns in Spanish waters (IOC UNESCO, 1992).

There is no co-ordinated national UK. research program aimed at harmful algal blooms. Funding is provided by the European Community, government department budgets and the National Environment Research Council. PSP, DSP and ichthyotoxic blooms are the main causes of problems in coastal and estuarine waters. The Ministry of Agriculture, Fisheries and Food carries out the routine monitoring for shellfish toxicity and toxic phytoplankton. The National Rivers Authority has a bathing waters monitoring program (IOC UNESCO, 1992).

Swedish Baltic waters are prone to blue-green (*Nodularia*) blooms (Sivonen et al., 1989) which affect children and particularly dogs (Elder et al., 1985), and also *Chrysochromulina* blooms which affect fish, invertebrates and algae. Mussels have also been affected



by *Dinophysis* species. A surveillance group has been established in Sweden with contacts with similar programs in Norway and Denmark. The Swedish National Marine Monitoring Program includes one regular pelagic station sampled twenty five times a year since 1976 (IOC UNESCO, 1992).

1.1.4 Asia and Japan

In China the first harmful algal bloom recorded was in 1933. Since then 70 species have been responsible for harmful algal blooms. The State Oceanic Administration and local coastal government agencies have announced measures to minimise the harmful effects of blooms in coastal waters. Losses of shrimp cultures resulted in the formation of a network of monitoring, investigation and research, management and information exchange (IOC UNESCO, 1992). In China investigation is being carried out on the association of high liver cancer incidence and the presence of blue-green algae in drinking water (Falconer, 1994).

Japan is experiencing serious harmful algal bloom problems with regard to aquaculture and public health. It has lost from the economy between 1972 and 1991 about 21.5 billion yen, due to fish mortalities associated with red tides.

Japan's problems arise from

- noxious algal blooms which cause mass mortality of marine organisms
- toxic species which cause human illness.

These areas are the subject of studies and research co-operatively by the Fisheries Agency and the Ministry of Education, Science and Culture. Ecology and oceanography, taxonomy and genetics, toxicology and toxin chemistry are some of the research projects operated by these agencies. The regular toxin monitoring program works very well and no case of poisoning has occurred since 1980 in shellfish sold in the market (Fukuyo, 1993).

1.1.5 South America

In Uruguay, four dinoflagellate species of phytoplankton are associated with periodic toxicity including *Alexandrium tamarense* in spring, and *A. fraterculus*, *Dinophysis acuminata* and *Gymnodinium catenatum* occurs from summer to autumn. The Instituto Nacional de Pesca is responsible for monitoring for harmful phytoplankton and arranging for toxicity testing (Mendez et. al., 1993).

In Chile, a massive marine fish kill occurred in 1988 as a result of a bloom of *Heterosigma akashiwo*. The Association of Chilean Salmon Farmers started a phytoplankton monitoring program which has been a useful tool for fish farming management decisions and planning (Clement and Lembeye, 1993).

1.2 Threats to Australia

Prior to 1985, toxic dinoflagellate blooms were virtually unknown in Australia. As a result, no monitoring programs or surveys for the detection of these toxins were conducted. During 1985-6, the first toxic dinoflagellate blooms in Australia were observed. There are now many reports of the impacts of harmful phytoplankton in Australia (Hallegraeff, 1993, Appendix 3). In the Port River (SA) *Alexandrium minutum* occurs, in Port Phillip Bay (Vic.) *Alexandrium catenella* is found and in Tasmania *Gymnodinium catenatum* blooms are regular events. The potential PSP producer species *Alexandrium tamarense* occurs in SA, Victoria and Tasmania. This species can affect birds, fish, mammals as well as humans (Hallegraeff, 1991).

In 1979, an outbreak of hepatitis-like symptoms occurred in 85 aboriginal children on a tropical island off the north eastern coast of Australia, after copper sulphate was added to the water supply reservoir to kill a blue-green bloom of predominantly *Cylindrospermopsis* which was found to contain a cytotoxic alkaloid (Falconer, 1994). In 1991 one of the biggest cyanobacterial (blue-green) blooms in Australia and perhaps the world, occurred along 1000 kilometres of the Darling/Barwon river system. Residents along the river were warned not to use the water for drinking, watering, washing or cooking purposes. In the summer of 1992-3 the first recorded bloom of *Nodularia spumigena* in Tasmania waters occurred. It was shown to produce the hepatotoxin, nodularin.

In the summer-autumn of 1993-94, significant new outbreaks of toxic cyanobacterial blooms were discovered in the rivers and wetlands of Western Australia. The deaths of fish are occurring with greater regularity in Western Australian waterways.

In Western Australia one case of suspected Paralytic Shellfish Poisoning (PSP) occurred in 1991. Many other cases may have gone unreported due to a lack of awareness of the causes and symptoms. Potential harmful impacts here are great because Australia has a high reliance on direct contact recreation, wild and cultured seafood consumption and export of mariculture produce.

The main health threats from phytoplankton-caused toxicity are summarised below.



Table 1 Clinical symptoms of various types of shellfish poisoning

PSP	DSP	ASP	NSP	CIGUATERA
<30 mins	>30 mins - 3 hrs >12 hrs	3-5 hrs	>3 hrs	12-24 hrs
Mild case				
Tingling and numbness around the lips, spreading to the face and neck; prickly sensation in fingertips and toes; headache dizziness, nausea and diarrhoea	Severe diarrhoea, nausea, vomiting, abdominal pain, chills	Nausea, vomiting, diarrhoea, abdominal cramps	Neurologic and gastrointestinal symptoms; chills, headache, diarrhoea, muscle weakness, stomach and joint pain; nausea, vomiting,	Gastrointestinal symptoms; diarrhoea, abdominal pain; vomiting, nausea
Extreme case				
Muscular paralysis, pronounced respiratory difficulty; choking sensation; death through respiratory paralysis may occur within 2 to 24 hrs of ingestion	Chronic exposure may promote tumour formation in the digestive system	Decreased reaction to deep pain; dizziness, hallucinations, confusion, short term memory loss; seizures	Paraesthesia, difficulty breathing, double vision, trouble talking and swallowing, dizziness	Neurological symptoms; numbness and tingling in the hands and feet; cold objects feel hot; difficulty in balance; low heart rate and blood pressure; rashes. Death through respiratory failure.
Treatment				
Stomach pumped; artificial respiration	Recovery after 3 days			No antitoxin or specific treatment. Calcium and mannitol may relieve symptoms. The patient may take many months to recover.

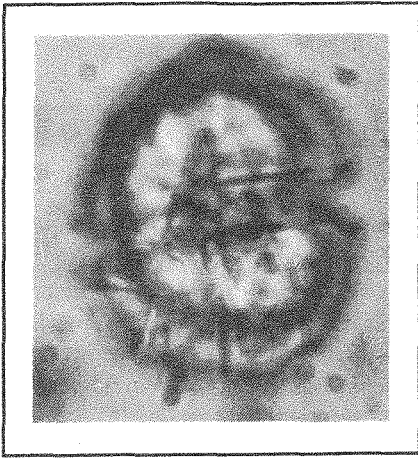
(Hallegraeff, 1993; Bates et. al., 1993)

1.2.1 Paralytic Shellfish Poisoning (PSP)

Paralytic Shellfish Poisoning can lead to temporary paralysis or death due to the presence of dinoflagellate saxitoxins in contaminated edible tissues. The toxins that cause PSP produce virtually identical symptoms to the chemically-different tetrodotoxin from puffer fish (Kao, 1993). The saxitoxins that are responsible for PSP are a family of at least 18 different compounds of widely differing potencies (Anderson, 1994).

The mortality rate of PSP varies considerably. There is a real increase in the PSP which has been spreading globally. Saxitoxins are water soluble compounds that bind to the outside of the sodium channels blocking propagation of the action potential of nerve cells. The family of PSP toxins includes 18 structurally-related chemical compounds (Kao, 1993). Intracellular bacteria from *Alexandrium tamarense* have been shown to produce saxitoxins (Anderson, 1994).

Shellfish which contain more than 80µg/100g of PSP are considered unfit for human consumption (Hallegraeff, 1993).



Photograph 1

Potentially PSP-producing dinoflagellate *Alexandrium minutum* from the lower Swan River estuary (a formalin preserved sample). (Size : 16-25 μm diameter.)

In WA in April 1993, a visiting Canadian scientist, Ms I Montagne, displayed typical PSP symptoms after eating seafood in Western Australia. Onset of her symptoms occurred at 2 am after eating 4 hours earlier. Symptoms included numbness around the lips, thumbs and face, tingling of the fingertips and toes and respiratory difficulties. She admitted herself to Royal Perth Hospital, but the doctor was unfamiliar with PSP symptoms. She suffered two further attacks and respiratory difficulties before recovery.

This case is indicative of possible misdiagnoses of serious conditions by local medical practitioners because of a lack of awareness of symptoms. This case was only identified because of the patient's international experience in this area. Some toxicity symptoms may be clinically similar to hyperventilation, shellfish 'allergy' or bacterial gastric infections according to the St John Ambulance Association.

1.2.2 Diarrhetic Shellfish Poisoning (DSP)

Diarrhetic Shellfish Poisoning can lead to temporary bouts of severe diarrhoea and even promote tumour formation in the alimentary canal because of the presence of dinoflagellate okadaic acid in contaminated edible tissues. Okadaic acid is a potent inhibitor of the serine/threonine protein phosphatases causing a build-up of phosphorylated proteins in the cells. This leads to continuous fluid secretion from the gut cells. DSP symptoms are similar to those of bacterial gastric symptoms (Jeffrey and Hallegraeff, 1990).

Either $2\mu\text{g}/\text{gram}$ of okadaic acid and/or $1.8\mu\text{g}/\text{gram}$ of dinophysistoxin-1 in shellfish hepatopancreas is considered to render the shellfish unfit for human consumption (Hallegraeff, 1993).

1.2.3 Amnesic Shellfish Poisoning (ASP)

Amnesic Shellfish Poisoning can lead to temporary or permanent loss of memory and even death due to the presence of diatom-based domoic acid in contaminated edible tissues. Domoic acid is a potent neurotoxin which is found in both diatoms and the red macroalgae species *Chondria armata* and *C. baileyana* (Pirquet, 1988). It stimulates the kainate-sensitive type of glutamate receptor on nerves, causing depolarisation, influx of calcium and eventually cell death. Memory loss apparently results from lesions in the hippocampus where Kainate receptors abound (Anderson, 1994).

The recent detection and verification of domoic acid in Victorian scallops indicates the potential threat to consumers and shellfish leases. Shellfish which contain more than $20\mu\text{g}/\text{gram}$ of tissue of domoic acid are considered unfit for human consumption (Hallegraeff, 1993).

1.2.4 Neurotoxic Shellfish Poisoning (NSP)

Neurotoxic Shellfish Poisoning can lead to neurologic and gastrointestinal symptoms because of the presence of dinoflagellate brevetoxins in contaminated edible tissues. These complex polycyclic polyether compounds have high lipid solubility. They depolarise nerves, causing increased transmitter release and loss of nerve conduction. They act on sodium channels by keeping them open, allowing greater sodium inflow. Brevetoxins are potent fish poisons causing muscle inco-ordination and paralysis and death by respiratory failure (Lavery, 1993).

1.2.5 Hepatotoxicity/Neurotoxicity from cyanobacterial blooms

Hepatotoxins damage the liver by deranging the cytoskeletal architecture of the hepatocytes (Falconer and Yeung, 1992). Nodularin is an hepatotoxic cyclic pentapeptide. Microcystins are hepatotoxic cyclic heptapeptides. These toxins cause hepatocyte degeneration and necrosis of the liver, gastroenteritis and can promote tumour growth.



The neurotoxins can cause neuronal depolarisation, inhibit cholinesterase or block neuronal ion channels (Resson et. al., 1993). Alkaloid neurotoxins are produced mainly by *Anabaena* and *Aphanizomenon*. They can produce neuro-muscular and respiratory disorders and even death in livestock. Stock affected by blue-green algal poisoning can show symptoms such as diarrhoea, paralysis, muscular weakness and difficulty in breathing, or symptoms that resemble strychnine poisoning (Aplin et. al., 1983).

Humans and animals contain injured cells of which some may spontaneously form cancers. Some of these dormant pre-cancerous cells will grow into a diversity of cancers when stimulated with blue-green tumor promoters. Potential liver and gut cancers will be stimulated into growth in the presence of microcystins in water supplies. *Lyngbya* (tropical seas), *Schizothrix* (temperate freshwater) and *Microcystis* are species which contain tumor-promoting substances (Falconer, 1994).

Allergic sensitivity to blue-green proteins and lipopolysaccharides can cause skin irritation, eye irritation and asthma when bathing or showering in water contaminated with blue-green algae and their extracts (Falconer, 1994).

1.2.6 Ciguatera from tropical coral reef dinoflagellate toxins

Ciguatera affects up to 50,000 people each year throughout the world. Many cases go unreported because the symptoms are similar to other illnesses or the disease is so common it goes unreported (Steidinger, 1993). Ciguatoxin is a heat stable highly oxygenated lipid soluble compound. It increases the permeability of excitable membranes to sodium ions. Maitotoxin is a water soluble compound and increases the permeability of excitable membranes to calcium ions. These toxins are produced by dinoflagellates associated with seaweeds and coral reefs, and are biomagnified up the food chain through small fish into larger fish. *Gambierdiscus toxicus*, *Ostreopsis siamensis*, *O. lenticularis*, *O. ovata*, *Prorocentrum lima*, *P. mexicanum*, *P. hoffmannianum*, *P. concavum* and *Coolia monotis* are the dinoflagellates suspected as the cause of ciguatera poisoning in tropical and sub-tropical regions, including northern Australia (Jeffrey and Hallegraef, 1990; Steidinger, 1993).

These species produce neurotoxic, hemolytic and/or hemagglutinating toxins which are lipid and water soluble. The toxins include ciguatoxin, maitotoxin, scaritoxin, gambiertoxin and others (Steidinger, 1993). Ciguatera causes gastrointestinal, cardiovascular, neurovascular, neurological, psychological and dermatological disorders. These symptoms can recur for years when people become sensitized to the toxins.

2 BLUE-GREEN ALGAL (CYANOBACTERIAL) BLOOMS IN WA

Blue-green algal blooms have been a regular occurrence in many Western Australian waterways for some years (Appendices 1 and 2). Recently the incidence and awareness of these potentially toxic blooms has increased. Affected waterways include urban wetlands, poorly flushed south west estuaries, river pools, farm dams and coastal marine areas. Research indicates that different toxic strains can be found within any bloom and that the variable toxicity of blooms could be due to these strains being sampled. Still and impounded waters tend to promote conditions where blooms of cyanobacteria develop. Even waters not considered to be eutrophic, may still contain small amounts of cyanobacterial scum accumulations (G Jones, pers. comm., 1994). Occasionally taste and odour problems occur in the Harding River Dam due to low numbers of cyanobacteria species. Usually cyanobacteria are not a problem in water supply dams (K Partridge, pers. comm., 1994).

From the Peel-Harvey estuarine system over thirty five species of cyanobacteria have been isolated (Huber, 1980). Genera reported to be toxic to livestock include, *Anabaena*, *Anacystis*, *Aphanizomenon*, *Gloeotrichia*, *Gomphosphaeria*, *Lyngbya*, *Nostoc* and *Nodularia* (Aplin et. al., 1983).

2.1 *Nodularia* in the Peel-Harvey estuary, Vasse-Wonnerup, Swan-Avon and Blackwood River.

In the Peel-Harvey estuary, almost annual blooms of *Nodularia* occur from September to January (annual *Nodularia* cell counts for the growth cycle are shown in Figure 1). The blooms in the Peel-Harvey were shown to be hepatotoxic as early as 1982 (Runnegar et. al., 1988). Outflowing tides of *Nodularia* from the Peel-Harvey estuary contaminate the waters of the Indian Ocean. *Nodularia* blooms occur in the Vasse-Wonnerup estuary, Blackwood River and the upper Swan River estuary, and have occurred in Glen Avon Pool (Waterways Commission unpublished data). Density details of the Swan River bloom were not available, but it was observed to be forming a small to moderate bloom with scumming visible at the Middle Swan Bridge.



Composition of phytoplankton from the Harvey Estuary from January 1991 to May 1992

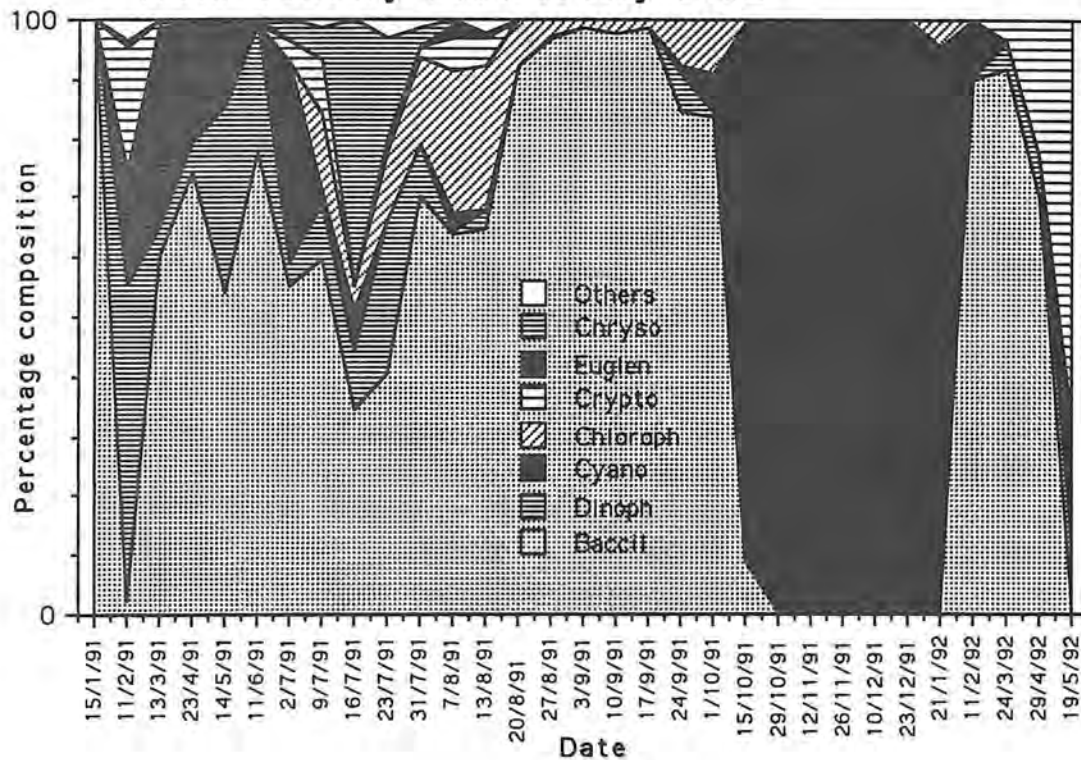


Figure 2 Phytoplankton composition in the Harvey estuary 1991-92.

2.1.1 Toxicity testing

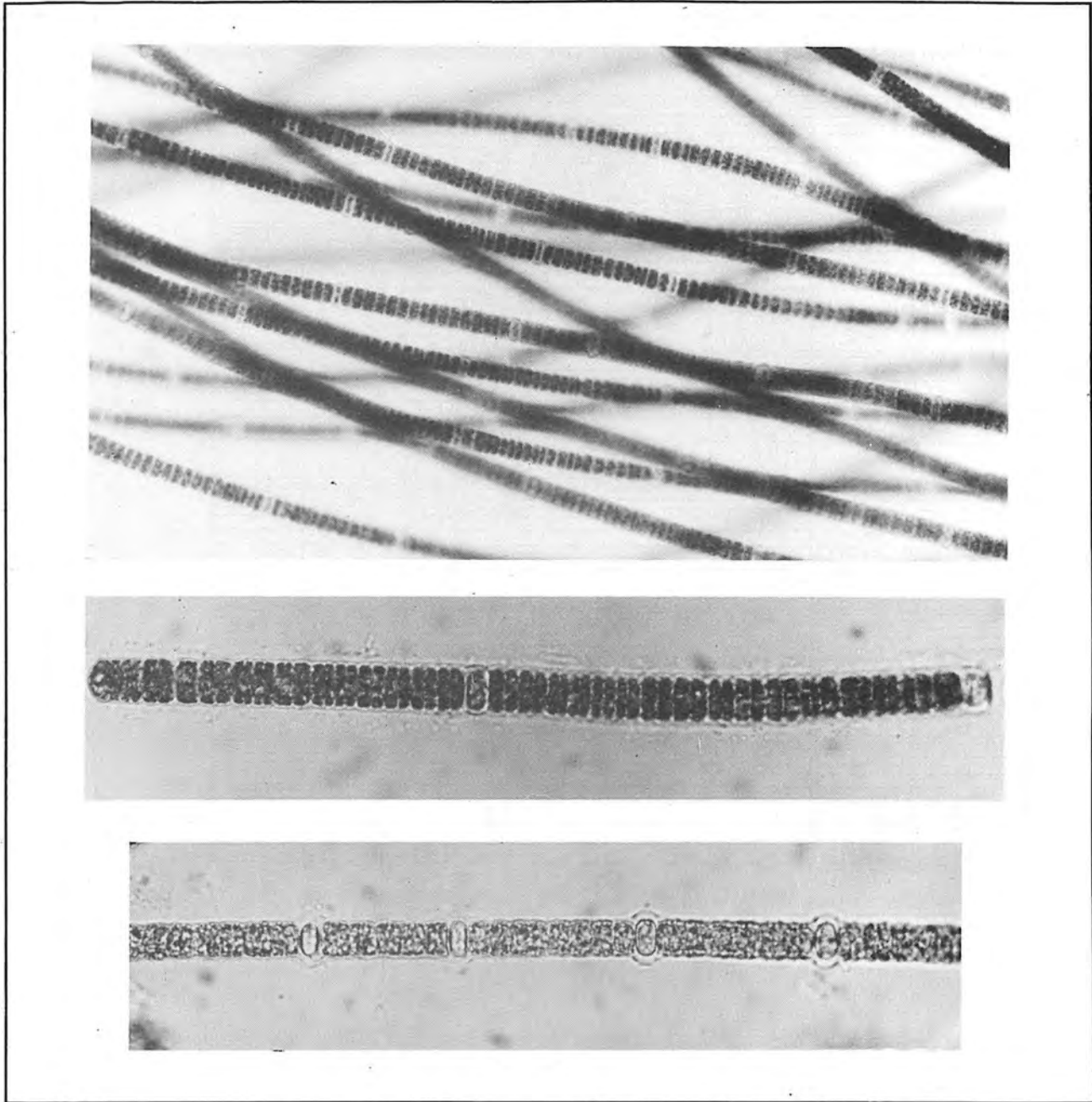
Nodularia was first associated with toxicity in Lake Alexandrina in South Australia by Francis in 1878. The toxicities of *Nodularia* cells and the edible mussels which accumulate the cells from the bloom-affected areas in the Peel-Harvey estuary, have been tested for the Waterways Commission by the University of New England (NSW) and by the Western Australian Department of Agriculture.

No toxicity testing has been carried out on the *Nodularia* blooms from the Blackwood River, Vasse-Wonnerup or the Swan River estuary to date. Results from mouse bioassays show LD_{50} values of 20-40mg/kg during the most toxic phase of the bloom. Table 2 compares these values with common pesticides. The lower the LD_{50} value the more toxic a substance.

2.1.2 Threats to human health

Nodularia poses a threat to humans by direct skin contact and consumption of filter-feeding shellfish. During bloom years *Nodularia* causes toxicity in mussels from the Peel Inlet from October to December (Falconer et al., 1992; J Allen, Agriculture Dept, unpublished data). These toxic effects require a warning on recreational activities and the taking of wild mussels *Mytilus edulis* from the area.

In 1992, thick buoyant *Nodularia* scum accumulations became trapped in a canal development adjoining the Mandurah entrance channel by prevailing south westerly breezes. The scum began to decay, giving off nauseating smells, which generated many complaints to the Peel Inlet Management Authority by affected residents. Aerial surveillance of the area showed the bright blue sunburnt crusty surface of the accumulation.



Photograph 2 *Nodularia* filaments from the Peel-Harvey estuary (upper photos) and the Avon River (lower photo).
(Size : cell diameter 9-12 μm)



Harvey Estuary mean (sites 1,31 & 58) integrated *Nodularia* cell counts for the period 1983-86, 1988-89 & 1991-93

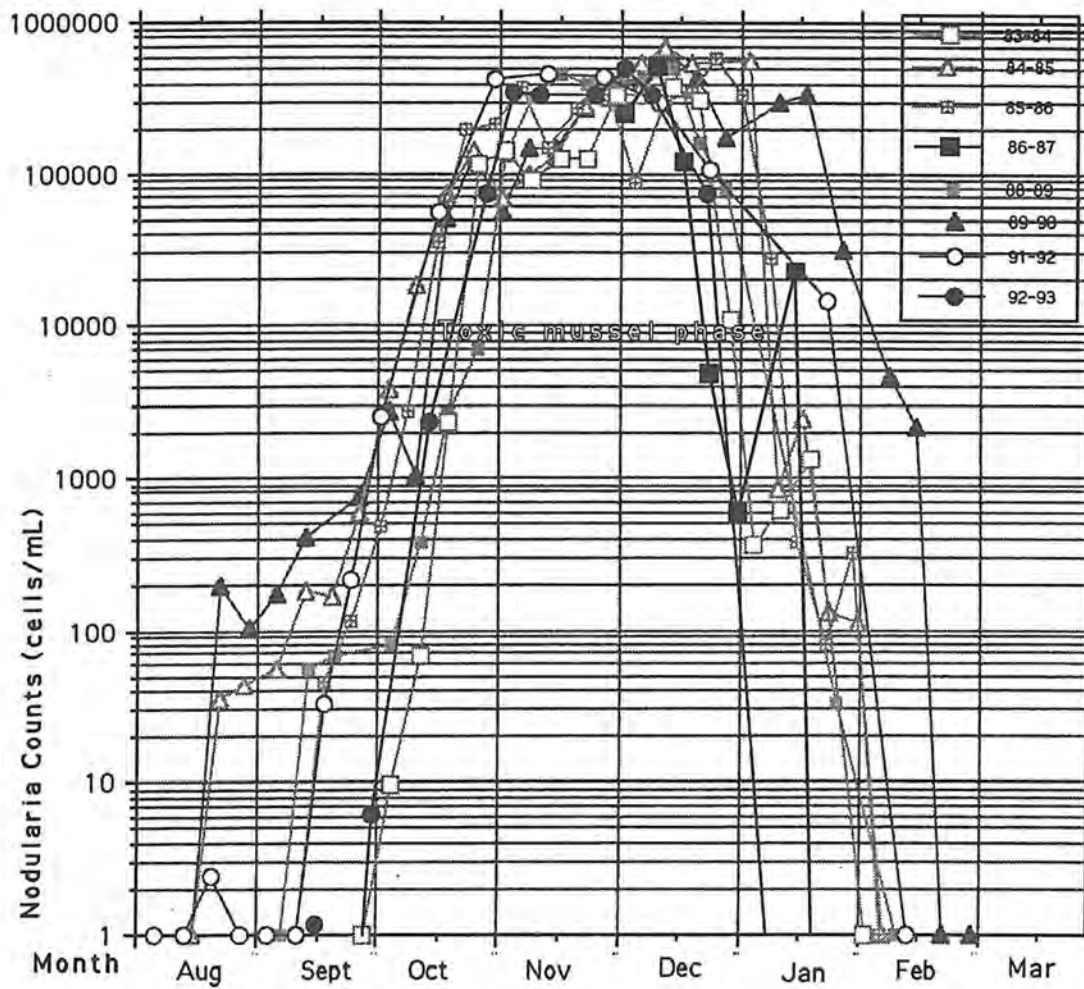


Figure 3 Mean *Nodularia* cell densities for the Harvey estuary from 1983 to 1992. (No bloom developed in 1987, 1990, only a minor bloom occurred briefly in mid-December 1993).

Table 2 Acute mammalian toxicity values for *Nodularia* in the Peel-Harvey estuary compared to common pesticides.

Item	Mammalian	LD ₅₀ mg/kg
<i>Nodularia</i> paste	20-40	
Heptachlor	100-162	
Chlorpyrifos		2,000
Malathion		4,100

(Toxicity Data: Professor I Falconer, University of New England, Dr J Allen, Department of Agriculture, Pesticide data from The Pesticide Manual 1979).



2.1.3 Threats to domestic animals and fish

There is evidence that the deaths of many dogs in Sweden were caused by the animals licking their fur after swimming in *Nodularia*-affected waters. Stock have died after drinking bloom-affected farm dam waters in WA, but there has been little documentation of these occurrences, and no confirmed cases in WA to date.



Photograph 3. Thick accumulation of *Nodularia* along the Harvey estuary foreshore

2.1.4 Ecological threats

The ecological threat of *Nodularia* is that fish and crabs avoid the area affected by the dense blooms. Laboratory trials jointly carried out by the Fish Health section of the Department of Agriculture and the Waterways Commission using Black Bream showed that the fish were not affected in *Nodularia* blooms where the densities exceeded 10^6 cells/mL. The trials were abandoned after one week because ammonia concentrations were becoming too high. No pathological harmful effect was observed in the fish (J Langdon, T Thorne and W Hosja, unpublished data). *Nodularia* increases the nitrogen content of estuaries in which it blooms, because of its ability to fix atmospheric nitrogen in its heterocysts. Increased nitrogen in contaminated waters may stimulate blooms of other potentially toxic species. Decomposition of collapsing blooms causes water anoxia and toxic hydrogen sulphide production. The disappearance of *Cladophora* macroalgal infestations from the Peel-Harvey estuary is believed to be caused by the light-shading effect of dense overlying blooms of *Nodularia*.

The deaths of hundreds of crabs in a canal on the Mandurah entrance channel in 1992 were attributed to the decomposition of *Nodularia* bloom material. Tests carried out by the Waterways Commission showed that bottom water dissolved oxygen concentrations were depleted to levels that could not sustain marine organisms (anoxia) as bacterial decay action of the bloom took place.

2.2 Future of blue-green blooms post-Dawesville Channel

It is anticipated that blooms of *Nodularia* are likely to be dramatically reduced in the main body of the estuary through unfavourably higher salinities. Blooms of *Nodularia* are likely to persist in the Serpentine River however, where the expected benefits of flushing will be reduced. There was a bloom of the freshwater blue-green *Anabaena* sp. (90-250,000,000 cells/L), in the Harvey Estuary during October 1983 after a very wet winter which suppressed salinities for longer than usual. Frequently, dense blooms of the filamentous benthic blue-green *Oscillatoria* occur at the water surface following the collapse of *Nodularia* blooms in the Peel-Harvey estuary. It is possible that these two species will still occasionally form bloom of shorter duration once the Dawesville Channel is opened to the sea in 1994. Dense surface accumulations of *Anabaena circinalis* (8,000,000,000/L) were recorded in the Serpentine river in the 1992-3 bloom season.



2.2.1 Future of impacts in the estuarine system

With the anticipated reduction in *Nodularia* blooms after the Dawesville Channel opening, it is not known if *Synechococcus*, *Trichodesmium* or another blue-green species will become a threat. A bloom of the blue-green picoplanktonic species of phytoplankton *Synechococcus* of untested toxicity occurred in 1990 with maximum densities exceeding 36,000,000,000 cells per litre (Waterways Commission, unpublished data). Some strains of *Synechococcus* are toxic (Skulberg et al., 1993). It is possible that relatively rare harmful or toxic phytoflagellate species may become more dominant in the absence of *Nodularia* blooms.

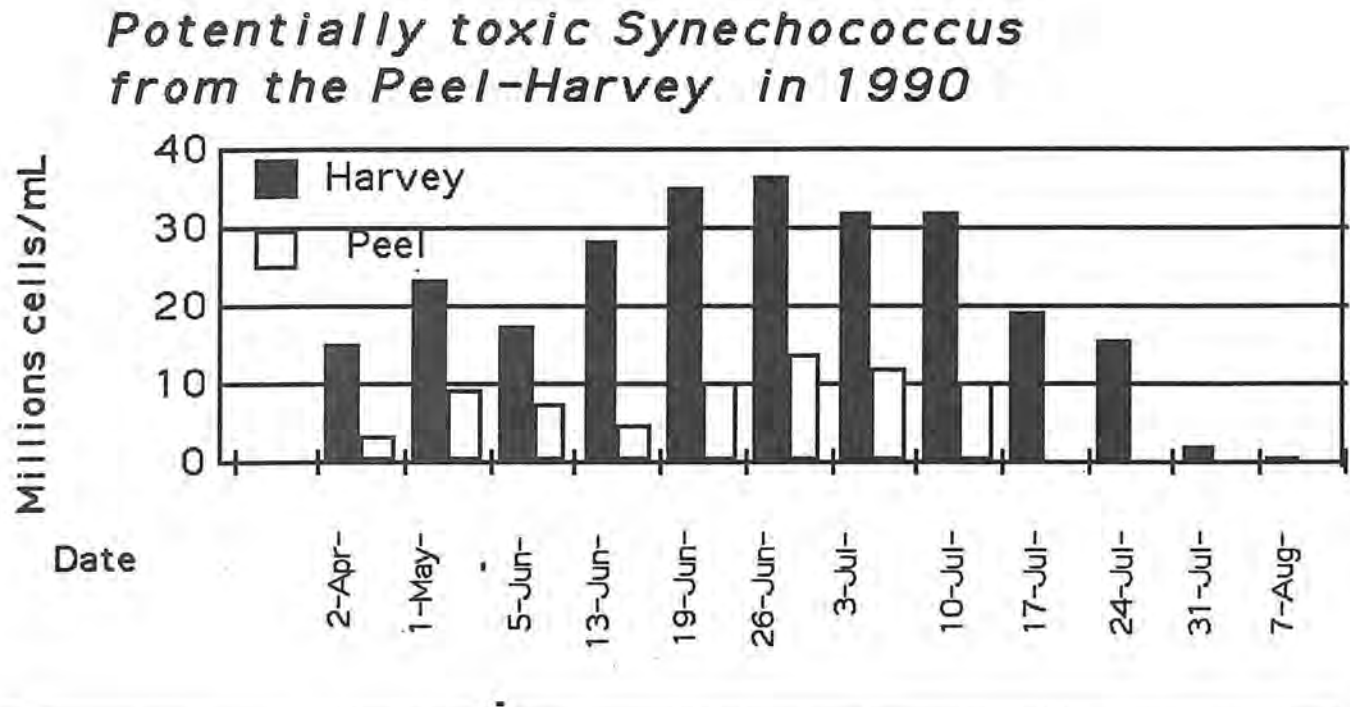
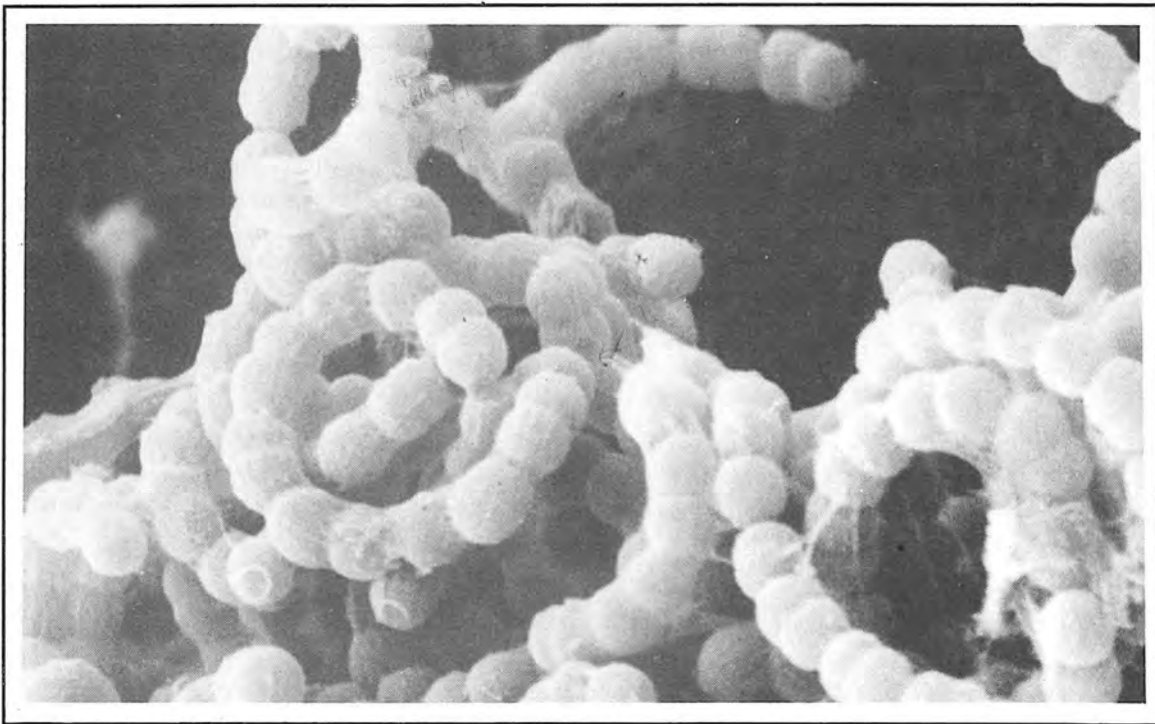


Figure 4 The densities of *Synechococcus* in the Peel-Harvey estuary in 1990.

In the winter-spring of 1983 which was very wet, the first recorded bloom of the freshwater blue-green algal species *Anabaena* and *Microcystis* developed in the Harvey estuary. The opening of the Dawesville Channel might not prevent such events occurring during years of very heavy rainfall.

With the expected longer duration of saline water phases occurring in the Peel-Harvey estuary after the opening of the Dawesville Channel, it is anticipated that there will be a change to the present established pattern of phytoplankton composition and density. Blue-green algal species such as the potentially toxic *Synechococcus* could bloom more regularly, especially in the Harvey estuary in the saline waters. If shown to be toxic this could have health implications on wild shellfish consumption.



Photograph 4 *Anabaena cf. circinalis* from the Serpentine River. Upper - live cells. Lower - Scanning Electron Micrograph courtesy of G M Hallegraeff. (Size : cell diameter 9-11 μm)

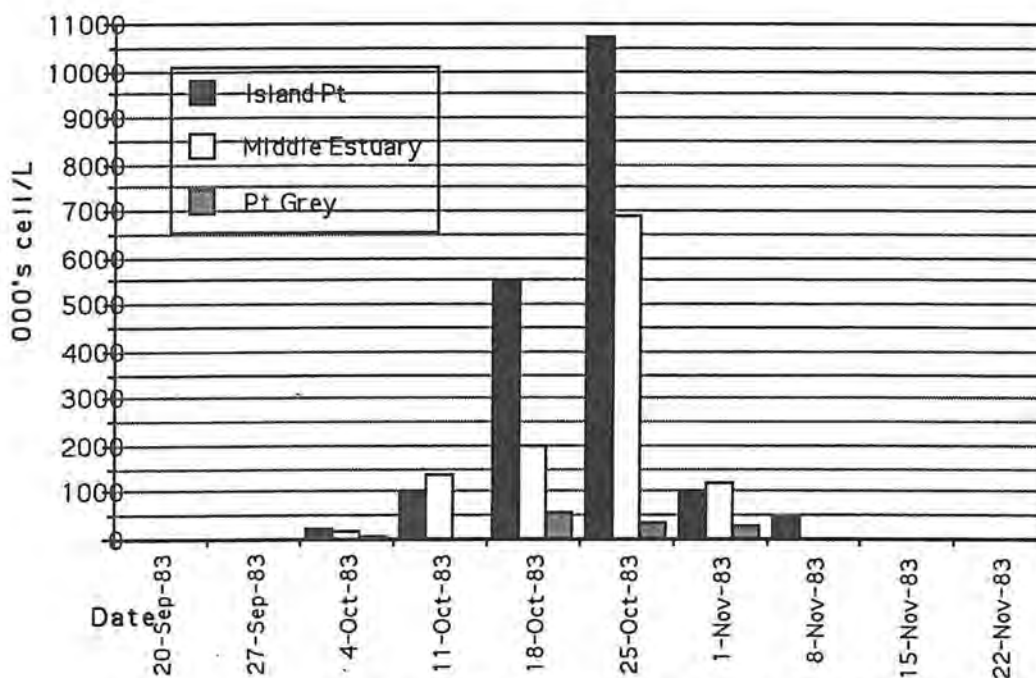


Figure 5 *Anabaena* bloom in the Peel-Harvey estuary spring 1983

2.2.2 Future of blue-green blooms which can tolerate the higher salinities in the estuary and tidal rivers

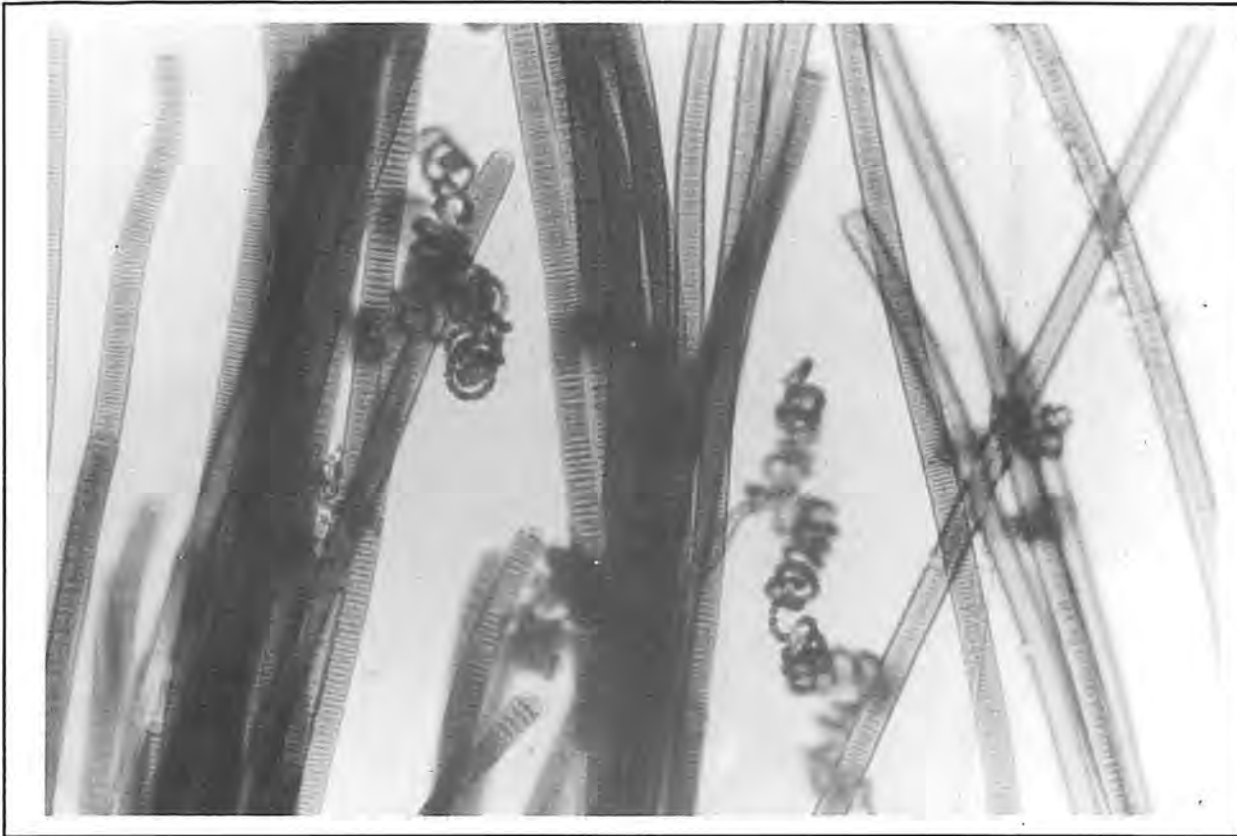
In January 1994, a bloom of *Nodularia* and *Anabaena* occurred in the Serpentine River when no significant bloom occurred in the Peel-Harvey estuarine system. Blue-green blooms are likely to continue to occur in the Serpentine River where the cleansing effects of the Dawesville Channel will be attenuated and catchment nutrient exports remain high. It is likely that these areas will continue to require monitoring and health warnings in the future. An *Anabaena* species, able to tolerate brackish water (14 ppt), has been recorded in the Avon River in 1991 (Avon River Management Authority, unpublished data).

2.2.3 Toxicity testing

Little toxicity testing has been carried out on either *Oscillatoria* or *Anabaena* in these areas. The Waterways Commission and the Department of Agriculture have recently arranged to carry out testing on WA species of *Oscillatoria* which can accumulate as scums along shorelines. These species are suspected of producing a powerful neuro-muscular blocking agent anatoxin a and microcystin (hepatotoxins).

2.3 Urban and rural wetlands and farm dams

Blooms of *Nodularia*, *Microcystis* and *Anabaena* occur in many of the urban lakes (Forrestdale, Mary Carol, North, Bibra, Blue Gum, Booragoon, Monger, Tranby, Hazelmere Lakes etc.) and wetlands and in rural farm dams in Western Australia. Blooms are observed in lakes in spring and summer. They are observed in eutrophic lakes which are subject to nutrient inputs from agricultural fertilisers, domestic runoff and leachates from rubbish tips and intensive horticulture. Wetlands having low levels of urban or rural development do not generally show phytoplankton bloom activity. Wetlands that are highly coloured tend to have fewer blooms due to light inhibition.



Photograph 5 *Oscillatoria* and *Anabaena* from the Canning River

2.3.1 Toxicity testing

Very little toxicity testing has been carried out on these wetland blue-green blooms. The Department of Agriculture may be able to provide a relatively inexpensive rapid, routine service for testing of urban wetland blue-green blooms (J Allen, pers. comm., 1994).

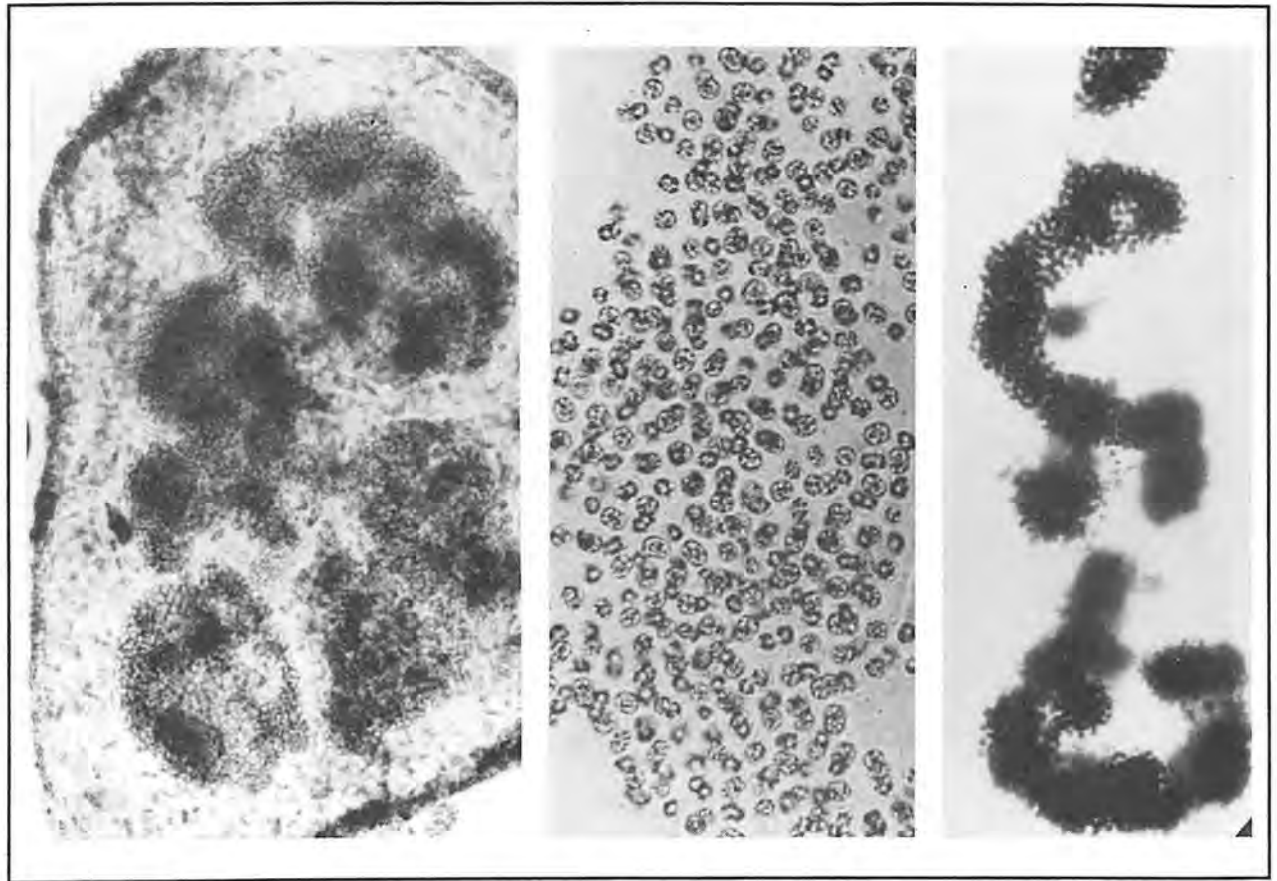
It is imperative that Western Australia develops its own monitoring and testing facilities for rapid identification and assessment of threat. In the past, Western Australia was heavily reliant on assistance from overseas and interstate specialists for testing for toxicity. Though complete monitoring and testing facilities are not currently available locally the resources have greatly improved recently. The Chemistry Centre of Western Australia will be capable of providing HPLC analysis for Anatoxin-a and microcystin in blue-green algae or water samples with some method development (S Jones, Chemistry Centre of WA, pers. comm., 1993).

Toxicity testing of blue-green algal samples is relevant to the particular sample and it is safer to assume that large accumulations of *Anabaena* and *Microcystis* may be toxic at any stage and therefore should be treated with respect (Working Party on Blue-green Algae in Water Supplies, 1990).

2.3.2 Human health threats

Some lakes are used as source waters for sprinkler irrigation. At times these can spray measurably large densities of potentially toxic cyanobacteria at the sprinkler heads (up to 18,500,000 cell/L and 200,000,000 cell/L *Anabaena* sp. in municipal sprinklers, Jan 1994 and November 1994, respectively). These cells are being sprayed in irrigation waters over areas that the public use for active recreation. Some species of neurotoxic *Anabaena* could cause asthma-like symptoms in people if inhaled in the fine mist spray (Falconer, pers. comm., 1994).

Blue-green species blooming in urban and rural wetlands are capable of producing toxins. Tests on *Microcystis* scum in a small lake adjacent to the Swan River foreshore produced a LD_{50} of 100 mg/kg toxicity (J Allen, unpublished data, 1994). The human health threat is reduced because of low levels of direct contact recreation or consumption of wetland animal species. However children swimming in contaminated wetlands may be at risk. In Sweden children have been adversely affected by contact with blue-green algal blooms.



Photograph 6 Two estuarine (left) and one freshwater species (right) of *Microcystis* (*Anacystis*) found in the Serpentine River at Mandurah, and the South Perth foreshore. (Size : cell diameter 4-7 μm)

2.3.3 Threats to domestic animals

These toxic species are a threat to stock and domestic animals that may drink or swim in affected waters. There have been numerous reports of stock losses from contaminated farm dams in WA, but these have not been well documented. Many popular recreational areas adjacent to lakes which have blue-green blooms are used for the exercising of domestic dogs. These are at risk of neurotoxic or hepatotoxic poisoning if they drink from the water's edge or swim in bloom-affected waters. Intraruminal and intraperitoneal (IP) doses of *Anabaena circinalis* (IP toxic at LD_{50} 17 mg/kg in mice) were compared. It was calculated that lethality was not observed in sheep intraruminally until a dose of 1710 mg/kg was given. This was the equivalent of 8.5 litres of thick algal bloom. This suggested that the IP mouse test is an unreliable method for judging potential oral toxicity in livestock (Runnegar et al., 1988).

Dogs have been poisoned after eating benthic *Oscillatoria* species off rocks in Scotland (Codd, pers. comm, 1994).

2.3.4 Ecological threats

The impacts of toxic phytoplankton blooms are not well understood. Considerable work has been done on changes to invertebrate fauna following nutrient enrichment (Davis and Rolls, 1992). Native birds have died from blue-green algae-affected lakes. The majority of these birds (up to 70%) do not recover from the effects of blue-green algal bloom toxicity (H Barnes, pers. comm., 1994).

It is well established that wetlands experiencing regular phytoplankton blooms have a reduced invertebrate species diversity with the complete absence of several key groups. Increased numbers of nuisance-causing chironomid midges are associated with contaminated wetlands. Waterbird deaths have been reported from botulism associated with contaminated wetlands. The role of phytoplankton in these ecological changes in the wetlands is poorly understood.



2.4 Other waterways

2.4.1 Swan-Canning River

In January 1994, blooms of the cyanobacteria *Anabaena cf. spiroides*, *A. circinalis* (neurotoxic) and *Microcystis* (hepatotoxic), occurred in the freshwater section of the Canning River, upstream of the Kent Street Weir. This was the first record of an *Anabaena* bloom in this part of the river. This area of the river is frequently used for recreational swimming by people and for the exercising of dogs. The river water is used for irrigation and stock watering under licence to the Water Authority of WA. Maximum surface cell densities of 480,000,000 cells/litre were measured by the Swan River Trust during the 1994 *Anabaena spiroides* bloom.



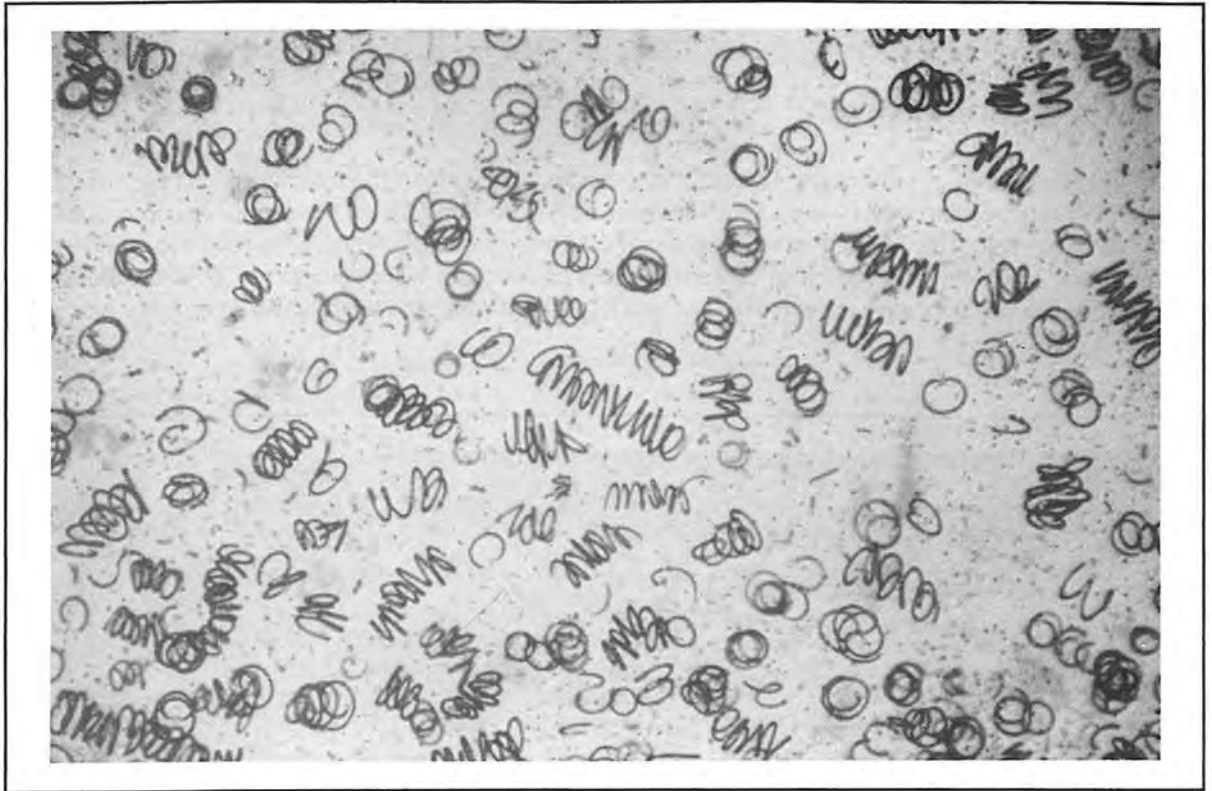
Photograph 7 Warning sign erected adjacent to the Canning River at Kent Street Weir in 1994.

The Western Australian Health Department and the Canning and Gosnells City Councils issued health risk notices and signposted swimming areas with warnings (Photograph 7). Intraperitoneal tests carried out by the Department of Agriculture showed LD₅₀ toxicity at less than 320mg/kg (*A. spiroides*) in January 1994 and 50mg/Kg in February (*A. circinalis*) (J Allen, unpublished data). The second Canning River bloom in February 1994 also contained appreciable numbers of *Microcystis* colonies, a potentially hepatotoxic blue-green species. Potentially toxic *Oscillatoria* scum was found along the River edges in March.

Filaments of *Nodularia* (green coloured due to the absence of gas vesicles) were recorded along the edges of the Canning River in the vicinity of the Nicholson Road Bridge area in February 1994 (Photograph 9).

The cyanobacterium *Microcystis littoralis* occurs in the estuarine parts of the Canning, upper Swan and Serpentine rivers. This species tested from the lower Canning in 1993 was shown to have low toxicity with an LD₅₀ of 400 mg/kg (J Allen unpublished data).

Short-lived *Nodularia* blooms have previously been recorded in the upper Swan and the Avon rivers without toxicity testing (unpublished data). Appendix 2 shows the summarised history of observed phytoplankton blooms in the Swan River estuary.

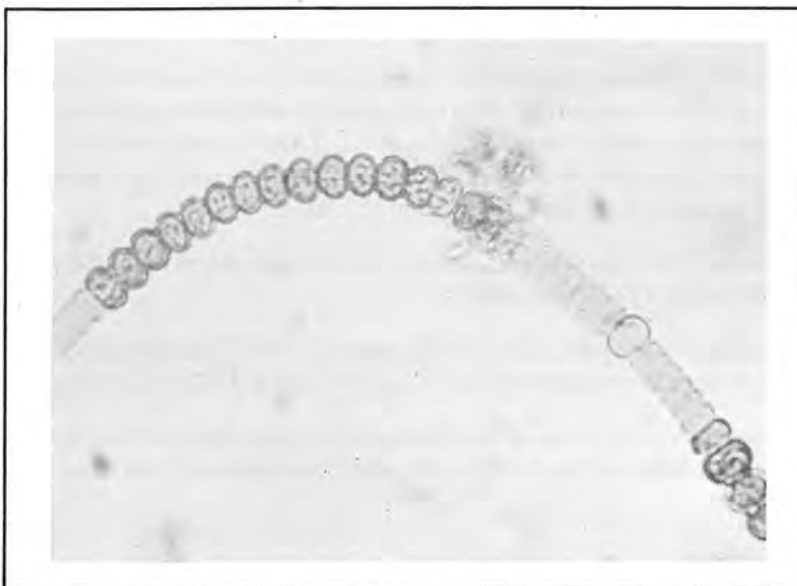


Photograph 8 *Anabaena* cf. *spiroides* from the Canning River.

2.4.2 Future threats to the Swan Estuary

A recent water quality monitoring program has found other species of blue-green algae in samples from the upper Swan River estuary. Surface runoff from heavy rains into contaminated wetlands frequently flushes thick blue-green algal scums to the Swan River estuary during both summer and winter.

Ellen Brook has a low Nitrogen:Phosphorus ratio of around 6:1. Ratios of nitrogen and phosphorus less than 15:1 (the Redfield ratio) favour the growth of blue-green species which are able to 'fix' their own atmospheric nitrogen and out-compete other less toxic species which have a high requirement for nitrogen and are not able to 'fix' atmospheric nitrogen. *Microcystis* blooms have been observed in the lower Ellen Brook (Swan River Trust unpublished data).



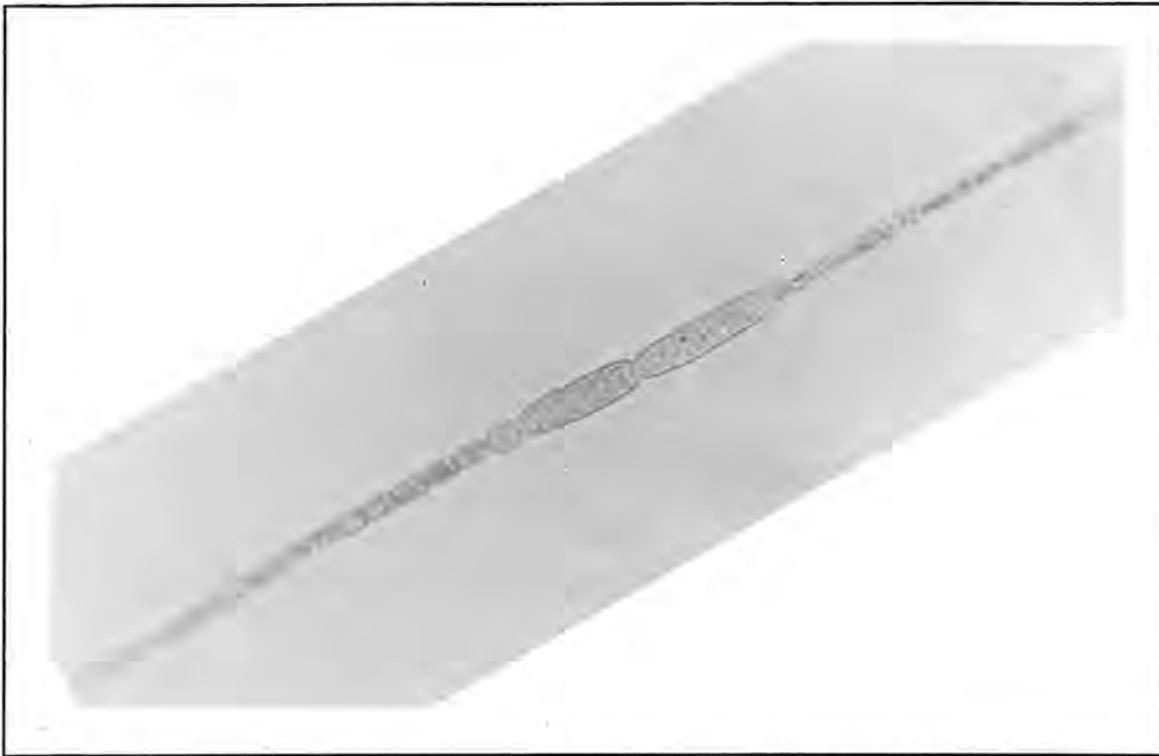
Photograph 9 Freshwater *Nodularia* with akinetes and heterocysts from the upper Canning River in 1994.



2.4.3 Other waterways

Various other species of blue-green algae have been recorded in other waterways. *Microcystis* has been found in the Vasse River (LD_{50} 50 mg/kg), *Anabaena* in the Sleeman River, *Oscillatoria* in the Avon River, Yunderup Canals and the Walpole and Nornalup estuaries. *Microcystis* and *Anabaena* have been recorded in the Dodson Wetlands (LD_{50} 200 mg/Kg). Some species of *Oscillatoria* can produce hepatotoxicity. *Oscillatoria* is common in many Western Australian rivers, estuaries and canal developments as benthic mats which float to the water surface and form bands of scum along the foreshores. These blue-green species may produce toxins but there has been little research work on their toxicity. Continued excessive nutrient inputs and the accumulation of blue-green akinetes stores in sediments mean that persistence of these blooms is highly likely.

In January 1993 and again in 1994, extremely dense blooms of the cyanobacteria *Aphanizomenon* (max 4,800,000,000 cells/L) along with *Anabaena* and *Nodularia* occurred in stretches of the Blackwood River. In the literature one species of *Aphanizomenon* is reported to be toxic. The affected areas were closed to recreational use, domestic and stock watering by the local health departments, after dogs were reported to be ill after drinking from the river.



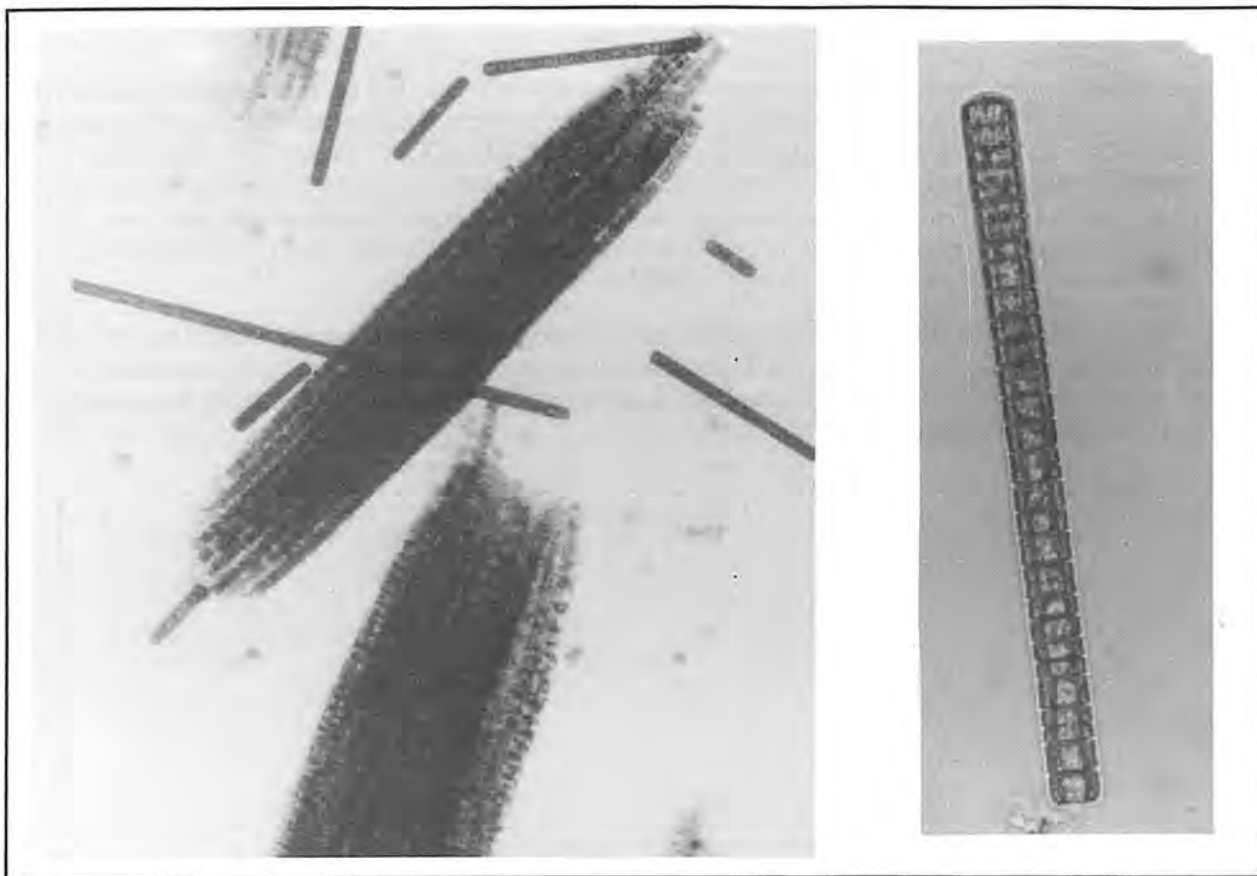
Photograph 10 *Aphanizomenon* from the Blackwood River. (Size : cell diameter 5-6 μ m)

There are reports of successful treatment and control of freshwater blue-green algal blooms in Britain using barley straw at the addition rate of 10 grams per cubic metre of water (Bird, 1991). To date in Australia (1993/94), scientifically based trials have failed to come up with the same successful results of preventing or stopping blue-green blooms, even at higher dosage rates.

2.4.4 Coastal Waters

Trichodesmium erythraeum blooms occur from the North West coastal region from November-March, and off the metropolitan beaches from April-August (Creagh, 1985). Blooms have been recorded as far as Esperance in the south. Precautionary beach closures were made by a local environmental health officer on the south coast of Western Australia during a bloom when a swimmer was taken to hospital after developing a severe skin rash in 1990. A related tropical species *T. thiebautii* has been shown to be toxic (Hawser and Codd, 1992). Little identification and no toxicity testing of *Trichodesmium* blooms in the North West coastal waters has been carried out.

A bloom of *T. erythraeum* was associated with the deaths of crayfish at Fremantle in 1994. Tests on this *Trichodesmium erythraeum* from the bloom in metropolitan coastal Western Australian in 1994 were negative for toxicity indicating that gill clogging and oxygen depletion might have been responsible for these deaths (J Allen unpublished data). The appearance of these blooms is not believed to be associated with eutrophic water conditions. Scums form in calm conditions and are blown ashore by onshore winds and blooms disintegrate releasing pigments. Senescing *Trichodesmium* blooms have entered the Peel Inlet with the incoming tide, where they have released red stains into the water. Some thick accumulations of this species have caused the deaths of marine organisms by lowered oxygen and the clogging of respiratory structures (Creagh, 1985). A bloom accumulation of *T. erythraeum* was observed in the Hardy Inlet in January 1994 by Dr E P Hodgkin.



Photograph 11 *Trichodesmium (Oscillatoria) erythraeum* bundles and a single filament from the Hardy Inlet bloom. (Size : Cell diameter 7-12 μm)

3 DINOFLAGELLATES IN WESTERN AUSTRALIA

About 20 species of dinoflagellates produce a variety of toxins that can be accumulated by filter-feeding shell fish.

Toxic blooms can exist in narrow bands some metres below the sea surface (MacKenzie, 1993). Dinoflagellates have well established vertical migration patterns, rising in the surface mid-morning and sinking mid-afternoon (Maclean, 1989).

Screening of waters for harmful species should be through vertical net towers. Cell enumeration should not be estimated from net hauls (Anderson, 1989).

The threat of dinoflagellate toxins in shellfish is of international concern and there needs to be an appropriate surveillance program in place (Hallegraeff, 1993, Appendix 3; MacKenzie, 1993; Kao, 1993). Two species of potentially toxic (Paralytic Shellfish Poisoning-PSP) *Alexandrium* dinoflagellate have been recorded in Western Australian waters thus far (Hallegraeff and Hosja, 1993). The identification of these dinoflagellate species requires specialised techniques and equipment. As a minimum, a phase contrast attachment on the compound microscope is required to observe the plate sutures of specially stained cells. The ideal method used by scientific personnel working with harmful dinoflagellates is a combination of epifluorescence after pre-staining with *Calcofluor* and/or Scanning Electron Microscopy (SEM).

Many *Prorocentrum* and *Dinophysis* blooms have occurred in local estuaries. *Dinophysis fortii* has been found in Oyster Harbour at Albany. Collapsing dinoflagellate blooms have contributed to fish and invertebrate deaths due to anoxia in Western Australian waters on more than one occasion.

3.1 Current estuarine occurrences

Dinoflagellate red tides are regularly observed in the Swan-Canning estuary, Peel-Harvey and Leschenault Estuary/Collie River areas, in the Wilson Inlet and in some coastal lagoons (Appendices 1 & 2). Potentially toxic dinoflagellate *Alexandrium*, *Prorocentrum* and *Dinophysis* species exist in Western Australian estuarine and coastal waters.

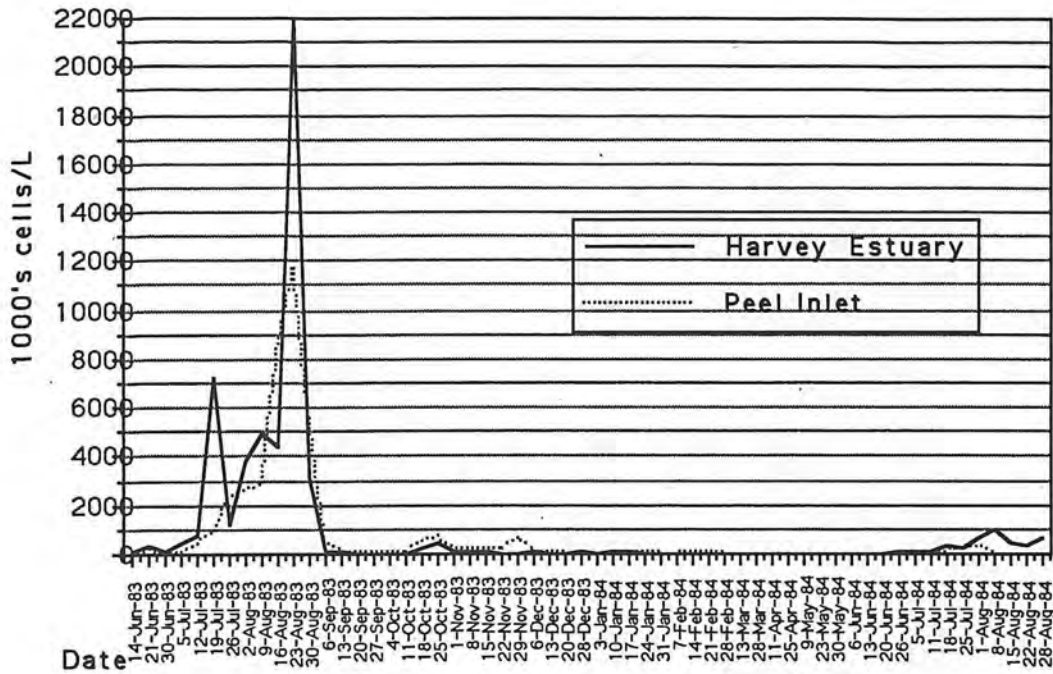
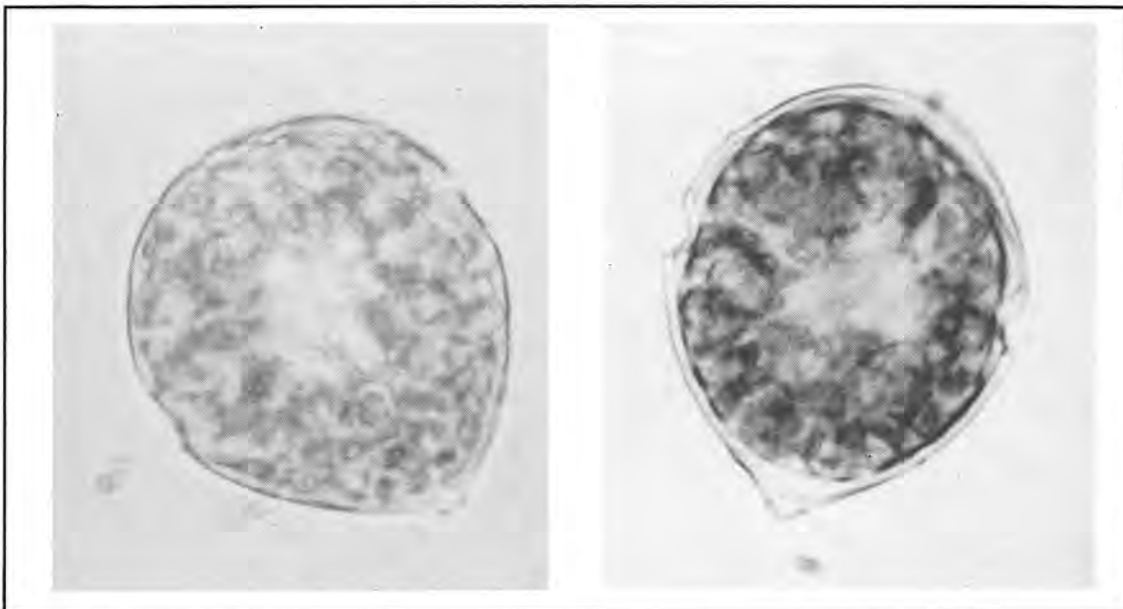


Figure 6 Mean dinoflagellate densities in the Peel - Harvey estuary 1983-84

3.1.1 *Scrippsiella*

Scrippsiella has previously caused fish kills in Australia (Hallegraeff, 1990). Blooms of non-toxic *Scrippsiella* and a *Scrippsiella*-like species are regular occurrences in the Swan River estuary. A maximum red tide density of up to 500,000,000 cells/L occurred in October 1993. Samples of estuarine mussels taken from within the bloom area were submitted for PSP toxin testing by the Chemistry Centre (WA) in 1994. No PSP toxicity was identified. In coastal waters a *Scrippsiella* red tide caused concern when it occurred in the sheltered waters of Mindarie Keys in February 1992 (WWC unpublished data).

A bloom of an unidentified, mucus-producing *Scrippsiella*-like species, caused a loss of amenity for river users in January 1990 when it adhered to swimmers' skin, hair and bathers at Ascot along the Swan River estuary.



Photograph 12 *Scrippsiella* species (left) and *Scrippsiella*-like species (right) from the Swan River estuary. (Size : cell length 18-30 μ m)



Photograph 13 Dinoflagellate bloom scum adhering to the skin of swimmers from the Swan River estuary (Photograph reproduced with the kind permission of Mr R Irving, Community Newspapers).

3.1.2 *Prorocentrum*

Prorocentrum species have been associated with Diarrhetic Shellfish Poisoning (DSP) and Venerupin poisoning (Tangen, 1983). The latter symptoms can take 36–48 hours to manifest themselves and lead to symptoms similar to bacterial food poisoning. Hence many cases of this type of poisoning may currently go unrecognised and unreported. Scanning electron microscopy reveals details of *Prorocentrum* surface morphology not apparent in previous light microscopy, which is useful in identification of the species (Faust, 1990).

Extremely dense blooms of *Prorocentrum minimum* have occurred in the Peel-Harvey/Yunderup and Waterside Mandurah Canals and in the Swan-Canning (up to 200,000,000 cells/L). A single test carried out in Japan on the Waterside Mandurah mussels (*Mytilus edulis*) collected during a bloom peak fortunately produced negative results for DSP (Oshima, unpublished data). This is not to be interpreted as indicating a lack of toxicity in WA strains because toxicities in phytoplankton vary with seasonal and other environmental factors. *Nodularia* in the Peel-Harvey is only toxic for part of the bloom cycle. The role of bacteria in toxicity production by microalgae is not fully understood.

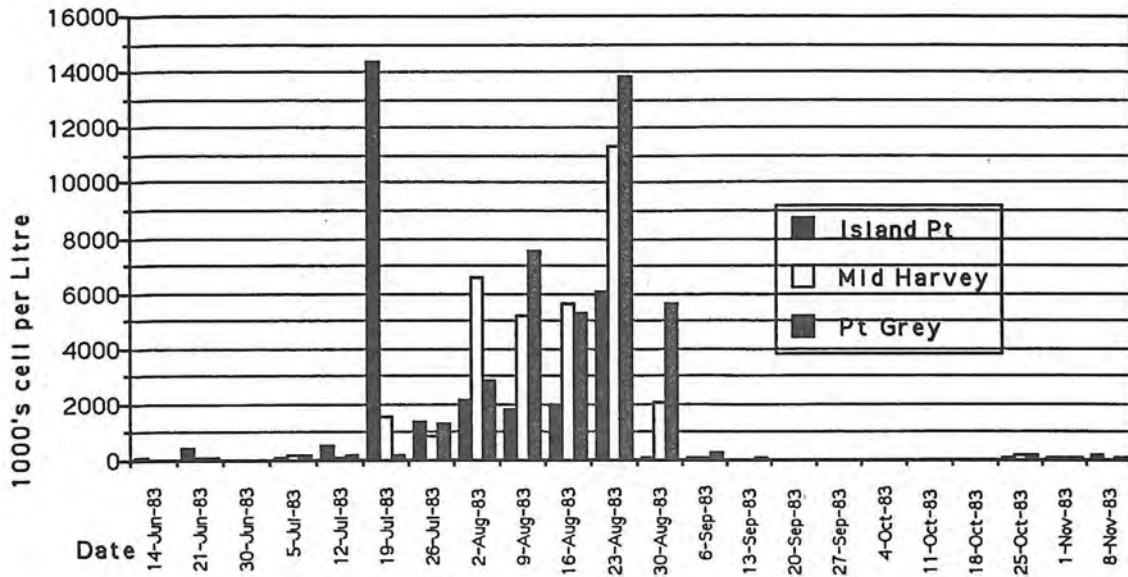
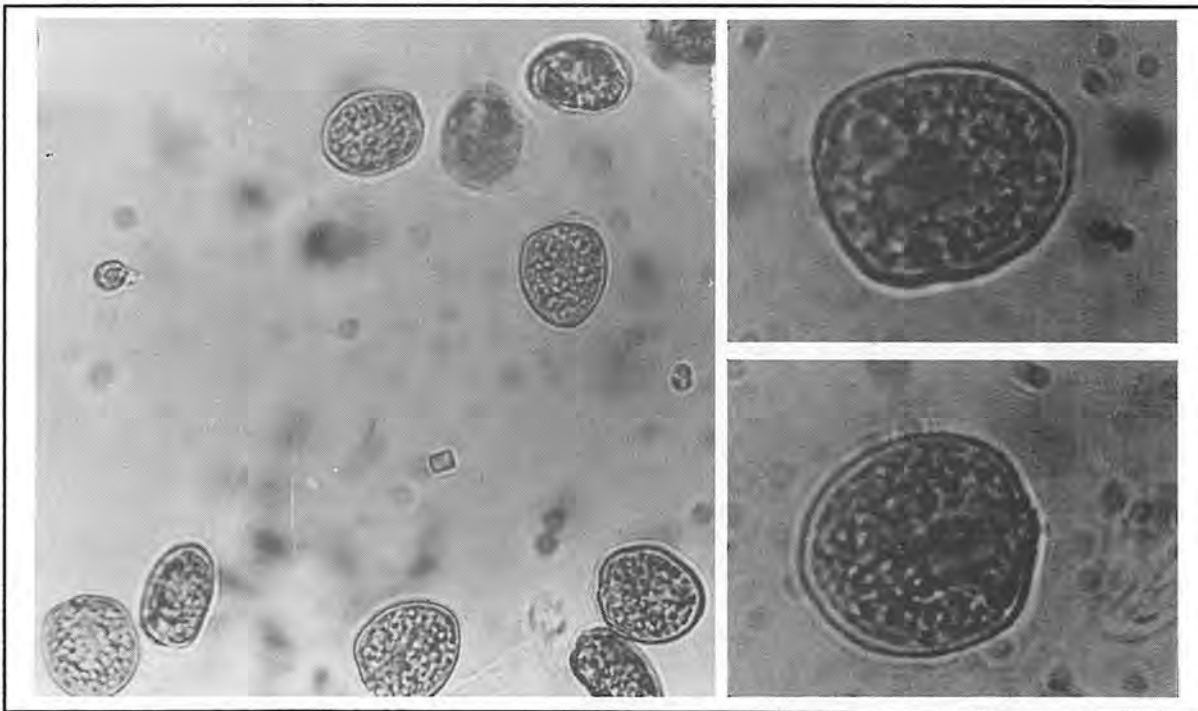


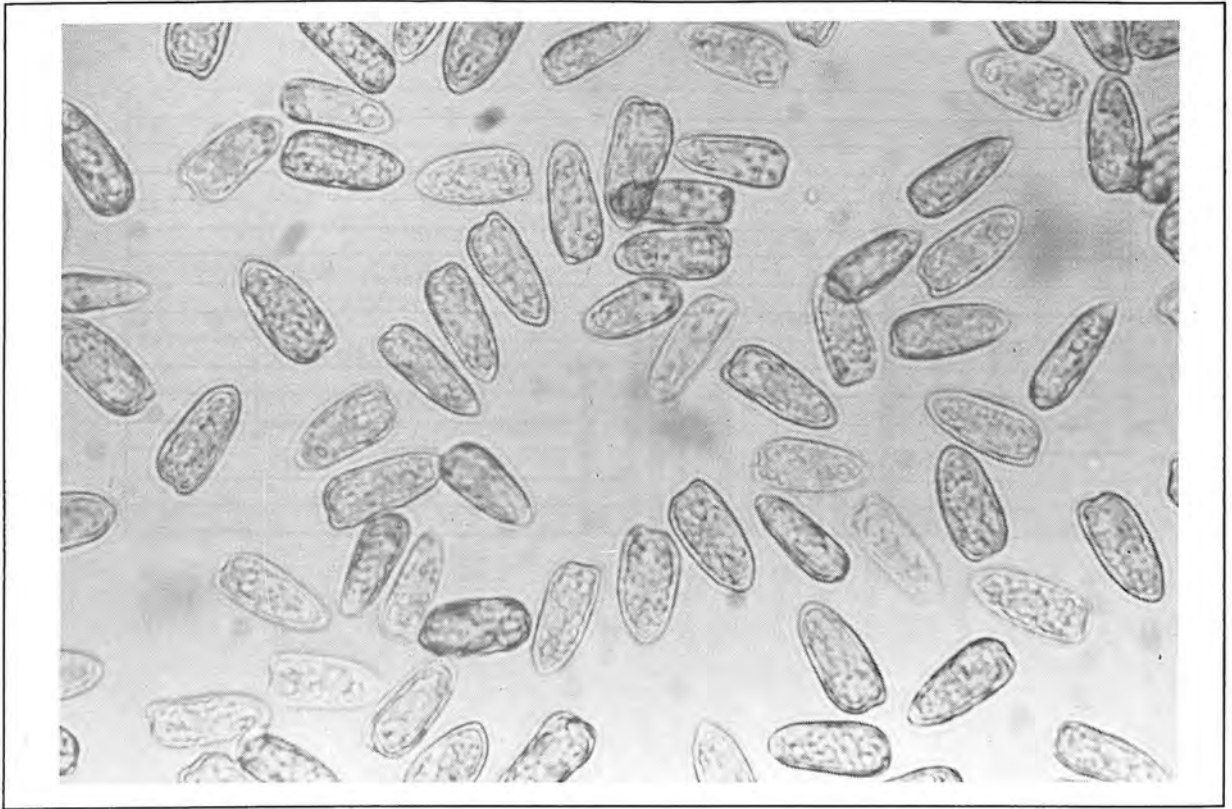
Figure 7 *Prorocentrum minimum* densities from sites in the Harvey estuary from June 1983 to November 1983

Fish, mollusc and crustacean kills in the Swan River and Yunderup Canals were attributed to the collapse of dense *Prorocentrum minimum* blooms which resulted in extremely low water dissolved oxygen concentrations, with toxic H_2S and NH_4-N release from sediments. *Prorocentrum (Exuviella)* bloom respiration at night and the oxidation of dead cells has been observed to cause dangerous oxygen diminution, reaching the threshold for lethal limits for invertebrates and fish (Bodeanu and Usurelu, 1979).



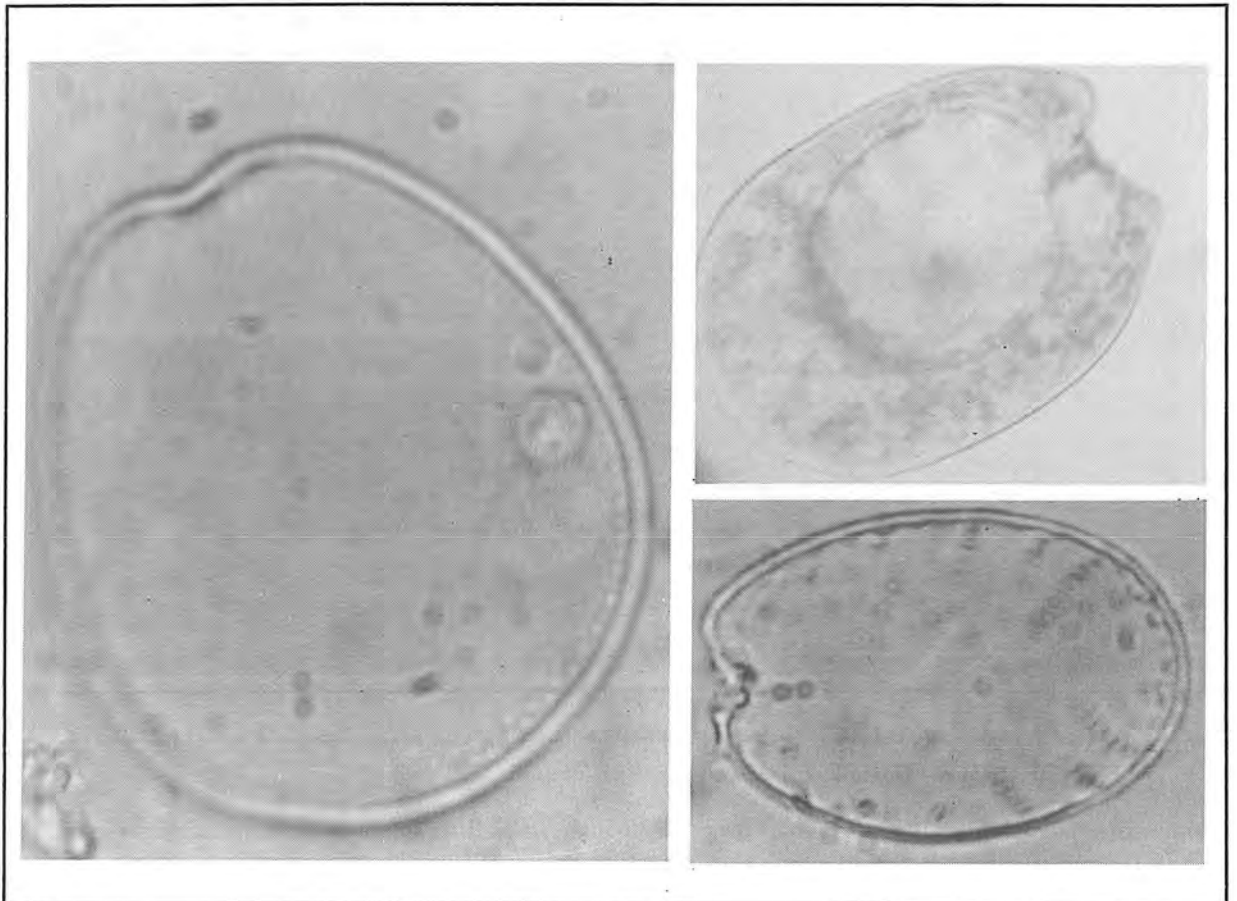
Photograph 14 The cosmopolitan potentially toxic dinoflagellate *Prorocentrum minimum* from a bloom in the upper Swan River estuary in 1992. (Size : cell length 16-20 μm)

Prorocentrum dentatum formed its first recorded major bloom in the Swan River in May 1993 at a maximum recorded density of 1,500,000,000 cells/L in Perth Water two weeks after heavy autumn rains followed hot warm weather conditions. Some of the cells formed chain-like colonies.



Photograph 15 *Prorocentrum dentatum* from the lower Swan River estuary. (Size : cell length 18-20 μm)

A benthic dinoflagellate species, *Prorocentrum* cf. *lima* cultured by the CSIRO from Wilson Inlet proved negative for the DSP toxin (Okadaic Acid). A number of similar as yet to be identified species are present in the Swan River, Peel Inlet, Wilson Inlet, Oyster Harbour, Leschenault Estuary, Parker Point at Rottnest, and in Cockburn Sound. *Prorocentrum lima*, *P. lima marinum* form and *P. mexicanum* are all similar. The toxicity profile of these species is not well understood. *Prorocentrum lima* and *P. mexicanum* both appear to be present in Princess Royal Harbour. A strain of *Prorocentrum mexicanum* has been shown to be toxic (Faust, 1990).





Photograph 16 Four benthic dinoflagellate *Prorocentrum mexicanum* species (bottom right page 24) from Western Australian waters.

3.1.3 *Alexandrium*

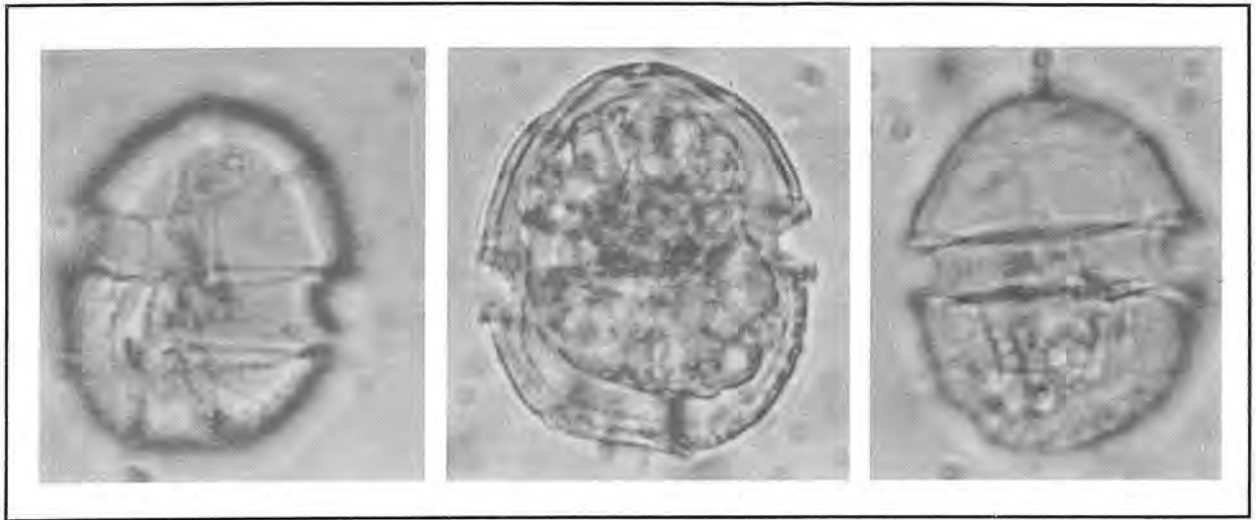
Alexandrium spp. can be PSP producers. *Alexandrium minutum* has been present in samples from the Swan River estuary since at least 1983. A sample of *Alexandrium minutum* collected from the Swan in January 1993 is being cultured at Murdoch University. Toxicity tests will be carried out by the Chemistry Centre of WA when a sufficient quantity of the cultured phytoplankton material is available for HPLC analysis. *Alexandrium minutum* was found in the Canning River estuary at Riverton Bridge in February 1994.

At present *Alexandrium* occurs in the saline phase of estuarine waters during summer and autumn. No complete monitoring of the density of *Alexandrium* in Western Australian waters has been carried out. The species can bloom well below the water surface (MacKenzie, 1993) therefore special sampling techniques need to be employed at various depths to determine its true distribution and density, and thus its true risk potential to human health.

Alexandrium minutum is widespread in the estuaries and in south western coastal waters of Western Australia. It is likely that *Alexandrium* is being translocated to the south west coast by the Leeuwin Current. Recently, *Alexandrium* was found in samples of water collected from the Hardy Inlet area (E.P. Hodgkin, *peis. comm.*). The ecotoxicology of this species is poorly understood.

No PSP testing of oysters in Oyster Harbour at Albany leases is currently being undertaken. It is likely that *Alexandrium* species are being transported to southern waters by the Leeuwin Current in a southward dispersal of marine animals (Hutchins, 1991). This could explain the presence of the species in south western coastal water samples taken by Hallegraeff and Hosja in 1993. The Albany Waterways Management Authority has begun collecting phytoplankton by net sampling of the waters in Oyster Harbour for potentially toxic phytoplankton species.

PSP toxicity of wild mussels occurs in the Port River in South Australia and is caused by *Alexandrium minutum* red tides (Hallegraeff et al., 1988). The Port River is subjected to sewage discharges which promote massive blooms. Blooms of 100,000 cells/L are sufficient to cause PSP in shellfish. In Western Australia, there is a projected increase in nutrient rich wastewater discharges to the Indian Ocean from 200 million to over 350 million litres per day (Cary and Simpson 1991). There are enormous loads of nitrogen and phosphorus entering the Indian Ocean from the Swan River. This will only serve to increase the risk that the densities of potentially toxic *Alexandrium* in coastal areas could in turn affect farmed and wild shellfish.



Photograph 17 *Alexandrium minutum* from the Swan River estuary. (Size : cell diameter 16-25 μm)

Alexandrium produces resting cysts which are capable of being transported in ballast water. Viable cysts of *Alexandrium catenella* have been germinated from the ballast sediments of a cargo vessel in Port Hedland (Jones, 1991). Cysts of *Alexandrium minutum* as yet have not been identified in sediments from the Swan River, the Port of Fremantle, Cockburn Sound, Leschenault Estuary, Geographe Bay or the Inner and Outer Harbours at Bunbury, even though viable organisms have been found in water samples (Hallegraeff and Hosja, 1993).

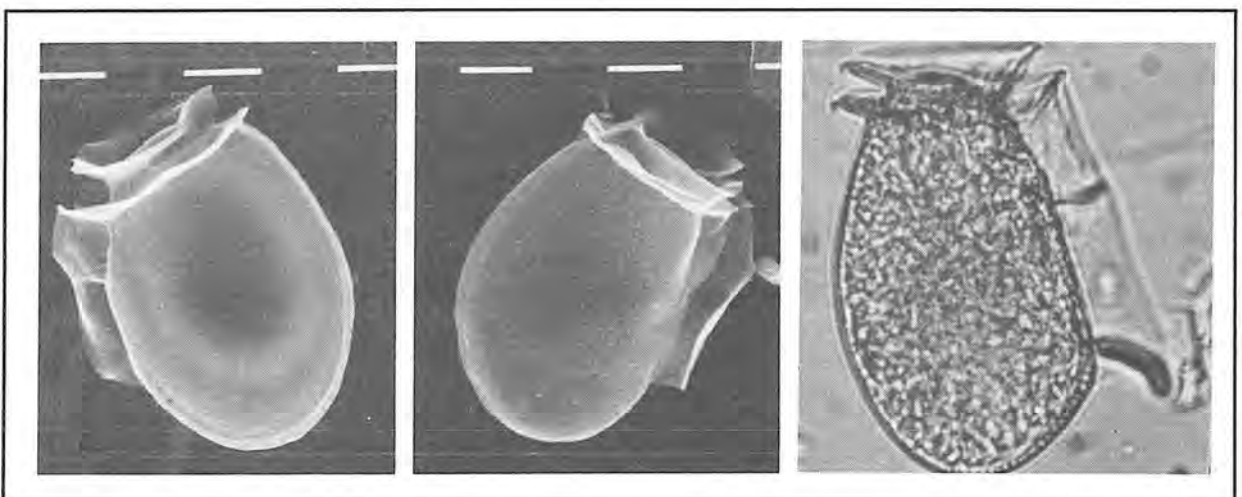
There are plans to open the port of Esperance to iron ore export which will increase the risk of cyst introductions from the ballast tanks of incoming carriers if the current voluntary guidelines are adhered to. Cyst surveillance of sediments is difficult and resource intensive but is a important task which must be carried out in all ports in Australia in order to assess the potential severity of this problem and to prevent the incorrect apportioning of blame to foreign sources. Some harmful *Alexandrium* species may already be present in Western Australia, but remain sub-visible through inadequate monitoring in the area.

Even if toxic species were discovered, it would not be a simple exercise to ascertain the introduction date of the species. In New Guinea the deaths of people from PSP toxins were first recorded in 1972. Research through medical records suggests that PSP may have been present for over fifty years (Maclean, 1991).

3.1.4 *Dinophysis*

The genus *Dinophysis* contains more than 200 species.

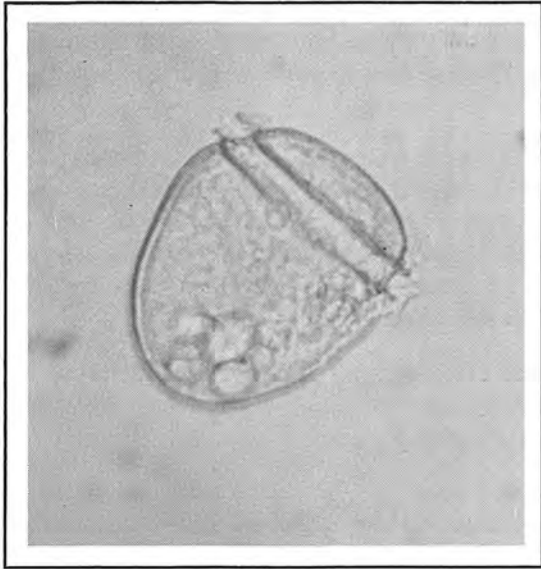
Several *Dinophysis* species have been responsible for DSP in the mussel and seafood industry. At present further testing is required to confirm whether *Dinophysis acuminata* cf. *ovum* and *D. cf. fortii* have been present in the Peel-Harvey. No mouse or HPLC testing of mussels for DSP has been undertaken during these blooms. *Dinophysis fortii* at densities as low as 200 cells per litre may lead to DSP outbreaks (Larsen and Moestrup, 1992). *D. fortii*, has been confirmed in samples from Oyster Harbour at low densities. (WWC, unpublished data.)



Photograph 18 Micrographs of *Dinophysis* from the Peel-Harvey estuary (GM Hallegraeff) and *Dinophysis fortii* from Oyster Harbour. (Size : cell length 45-60 μm)

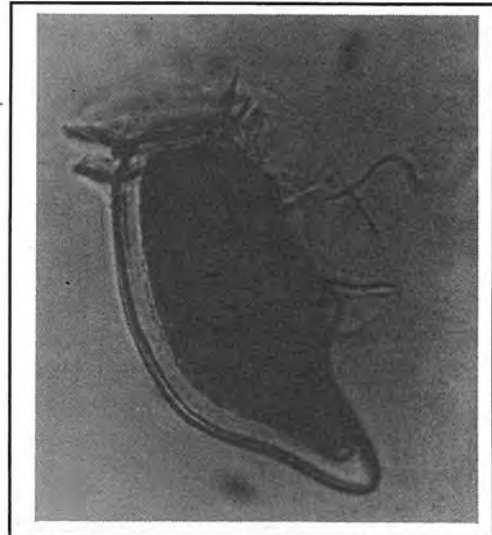


Phalacroma (Dinophysis) rotundatum is a species which is associated with toxicity (dinophysin-toxin-1) in Japan (Hallegraeff, 1991). This species occurs in low numbers in the lower Swan River estuary during spring-summer-autumn. No quantification of its cell numbers nor DSP toxicity tests have been undertaken.



Photograph 19 *Phalacroma (Dinophysis) rotundatum* from the lower Swan River estuary. (Size: cell length 35-50 μm)

Dinophysis caudata red tides were associated with mass mortalities of fishes (Fukuyo, 1990). The toxicity potential of *D. caudata* needs to be studied further (Larson and Moestrup, 1992). In Western Australian waters the same species is present in moderate numbers.



Photograph 20

Dinophysis caudata highly magnified showing large nucleus (left) and specimen preserved in Lugol's iodine showing flagella (above). (Size : cell length 80-90 μm).

3.1.5 *Gymnodinium*, *Gyrodinium*, *Cochlodinium* and *Amphidinium*

Gymnodinium cf. *breve* caused NSP in New Zealand in 1993 which resulted in the crippling of an export industry worth over \$2 million per week. The New Zealand Communicable Diseases Centre reported 162 cases of toxic shellfish poisoning. These 'naked' species (no hard covering) and can be difficult to identify in the preserved state and can therefore be overlooked during routine screening programs. Naked species are best identified in the fresh state or under the electron microscope following suitable preservation techniques.

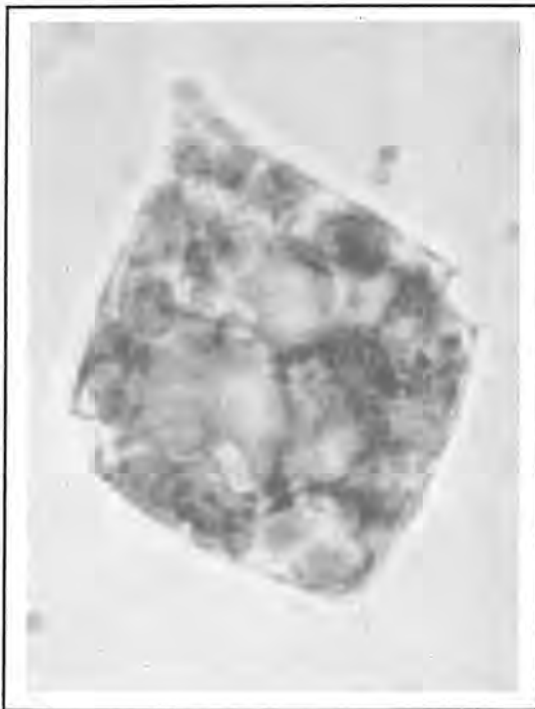
A dinoflagellate similar to *Gyrodinium aureolum* (*G. nagasakiense*), which is a fish-killing species, has formed dense blooms in the Peel-Harvey estuary. *Gyrodinium spirale*, which is associated with shellfish mortality in other parts of the world, also occurs in the Peel Inlet. Many other *Gymnodinium* and *Gyrodinium* species are yet to be positively identified. Blooms of *Gymnodinium simplex* have been recorded from the Collie and Swan rivers. A red tide bloom of *G. simplex* and *Gymnodinium* spp. between Ascot and West Midland in the Swan River was associated with a fish odour and anoxic black surface waters in March 1994. This resulted in further recreational contact warnings by the Swan River Trust and local health department authorities.

In Tasmania *Gymnodinium catenatum* causes PSP in shellfish farms. It is believed that cysts of these were introduced into Tasmanian waters in ballast tank waters. Domestic ballast water transfer is a threat within Australian coastal waters though water temperatures may restrict certain dinoflagellate species from becoming established in some ports.

Cochlodinium species are present in the Swan, Peel-Harvey and Leschenault estuaries. These species can produce ichthyotoxins. *Amphidinium carterae* which is associated with sediments in the Swan River estuary has caused fish and shellfish mortality elsewhere. No toxicity testing has been carried out during blooms of similar organisms in Western Australia. *Gymnodinium sanguineum* which is common to many WA estuaries has been implicated in losses of oysters in British Columbia. (Hallegraeff, 1991.)

3.1.6 *Ensiculifera*

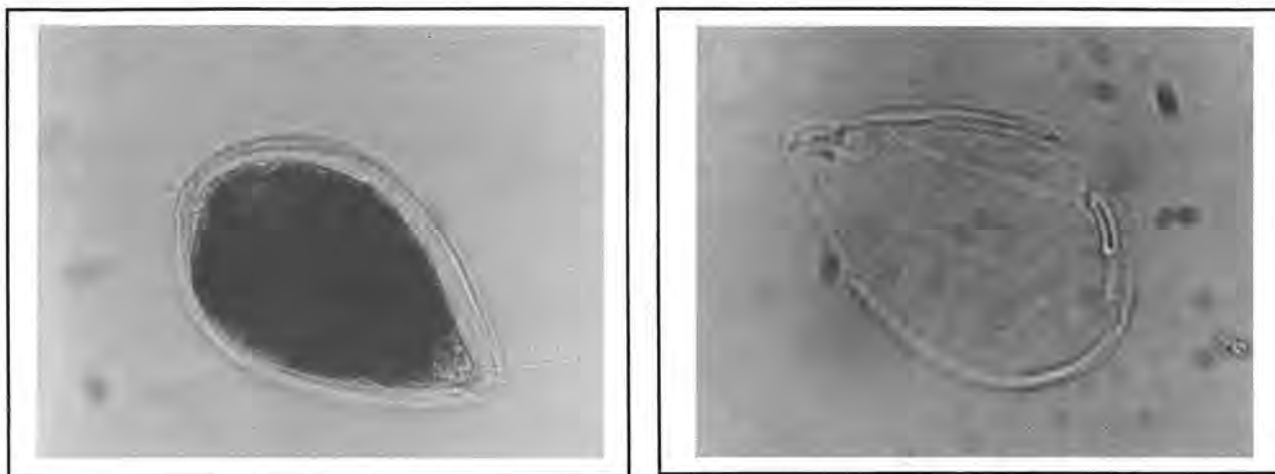
The dinoflagellate *Ensiculifera* is common to most estuaries and rivers in the south west of Western Australia (Waterways Commission unpublished data). As with *Prymnesium parvum* in Australia, *Ensiculifera* is uniquely isolated to Western Australia. The species forms calcareous resting dinocysts (Matsuoka et al., 1990). Calcareous cysts from *Ensiculifera* species have been found in the sediments of the Swan River and in the Bunbury area (Hallegraeff et al, 1993).



Photograph 21 *Ensiculifera* from the Swan River estuary and the Murray River (SEM micrograph, C J Bolch and G M Hallegraeff). (Size : cell length 40-50 μ m)

3.1.7 *Ostreopsis*

This dinoflagellate is associated with seagrass beds. Cells of *Ostreopsis* cf. *siamensis/lenticularis* were found in surface water samples from the Princess Royal Harbour at Albany in Western Australia. The species *Ostreopsis siamensis* is associated with ciguatera fish poisoning (Jeffrey and Hallegraeff, 1990). The phytoplankton in the Princess Royal Harbour and Oyster Harbour needs to be examined in detail to assess the presence and risk of harmful species.



Photograph 22 Benthic dinoflagellate *Ostreopsis* from Princess Royal Harbour, Albany. (Size : cell length 60-80 μ m)

3.1.8 Other dinoflagellate species

A bloom of *Alexandrium pseudogonyaulax* occurred in the Yunderup Canals in January 1994 at a density of 1,450,000 cells per litre. This taxa has been associated with weak ichthyotoxicity (Hallegraeff, pers. comm.). Around the time that the bloom occurred, a thick white proteinaceous surface foaming was observed in the canals (G Nelson, pers. comm., 1994). No samples of the foam were submitted for microscopic examination and the cause was not established.

Ceratium species are well established in Western Australian coastal waters and estuaries. *Ceratium furca* is one of the more densely blooming species in Cockburn Sound and the lower Swan River estuary.

3.2 Other estuaries in WA

Western Australia has an extensive coastline with a concentration of its population in coastal areas utilising these for recreational pursuits. Widespread agricultural production in most WA regions has seen losses of nutrients and sediment to increasingly more coastal waterways. Many of these waterways are only sampled occasionally on an *ad hoc* basis.

3.3 Near shore marine areas

3.3.1 Cockburn Sound to Geographe Bay

There are at least two *Alexandrium* species in Cockburn Sound and *Alexandrium minutum* has been recorded in coastal waters south to Geographe Bay. No toxicity has been found in surface mussels from Cockburn Sound but testing has not been comprehensive.

The Fisheries Dept of WA is currently conducting routine monitoring of surface phytoplankton and surface mussels. It has been found elsewhere that toxic species and mussel toxin accumulations may be restricted to deeper waters and not be observed in surface waters.

3.3.2 Perth metropolitan waters

More than 120 species of dinoflagellates (some yet to be identified) have been recorded in metropolitan coastal waters (J John, pers. comm.). A severe red tide of *Scrippsiella* occurred in Mindarie Keys in February 1992. On two occasions in 1993, unidentified plankton species have formed red tides in the Mindarie Keys marina (P Collins, pers. comm.). The species were neither identified nor tested for toxicity.

3.3.3 Other marine areas

Ciguatera is found in fish from tropical waters. Hallegraeff (1991) reported that cases of suspected poisoning have been recorded in Western Australia from North West Shelf waters. No surveillance of ciguatera causing dinoflagellates is currently carried out in Western Australian tropical waters.

3.3.4 Ports and harbours

Annually, about 155 million tonnes of ballast water is discharged into Australian ports of which 78% is from overseas ports and the remainder is from Australian ports (AQIS, 1993). Ballast water is recognised as a possible vector for the transfer of viable dinoflagellate cysts (Hallegraeff and Bolch, 1992). In Western Australia a ballast water study at Port Hedland found viable cysts of toxic dinoflagellates being discharged into the marine environment (Jones, 1991). Guidelines relating to ballast water discharges from international vessels coming into Australian ports require voluntary compliance.

The world-wide increase in toxic phytoplankton and the lack of rigorous surveillance programs mean that a significant threat remains unchecked. In Western Australia there are ports at Broome, Wyndam, Port Hedland, Dampier, Geraldton, Fremantle, Bunbury, Albany and Esperance. The Swan River Trust has taken a base-line sediment sample from the Port of Fremantle. The Western Australian Ports Authority Association (WAPAA) is keen to have all of these ports sediments surveyed for toxic dinoflagellate cysts.



Photograph 23 Ballast water discharge in the Bunbury Inner Harbour.

Some ballast water and harbour sediment monitoring surveys have been carried out in Western Australia. These have been far from extensive or complete. There are both international and national threats of ballast water transferring harmful species of phytoplankton and other organisms. Dredging operations in ports and harbours can mobilise dinoflagellate cysts into areas where high levels of nutrients and favourable water quality conditions for the species to germinate are found. No deepening dredging operations should be carried out in harbours until the sediments have been tested for the presence of harmful cyst. If the cysts are found to be present a protocol for dredging operations should be in place to prevent the transfer of these to other areas.

These could include:

- the disposal of spoil to inshore areas away from the water
- no ships to take on ballast during port dredging operations
- dredging to occur only during outgoing tides
- the treatment of dredge spoil.

The Australian Quarantine Inspection Service has released its Draft Coastal Ballast Water Guidelines for comment by the end of January 1994. Concern in Australia is no longer solely for international boating traffic but also from domestic traffic travelling from Australian ports contaminated with toxic dinoflagellates, exotic starfish and macroalgae (AQIS, 1993).

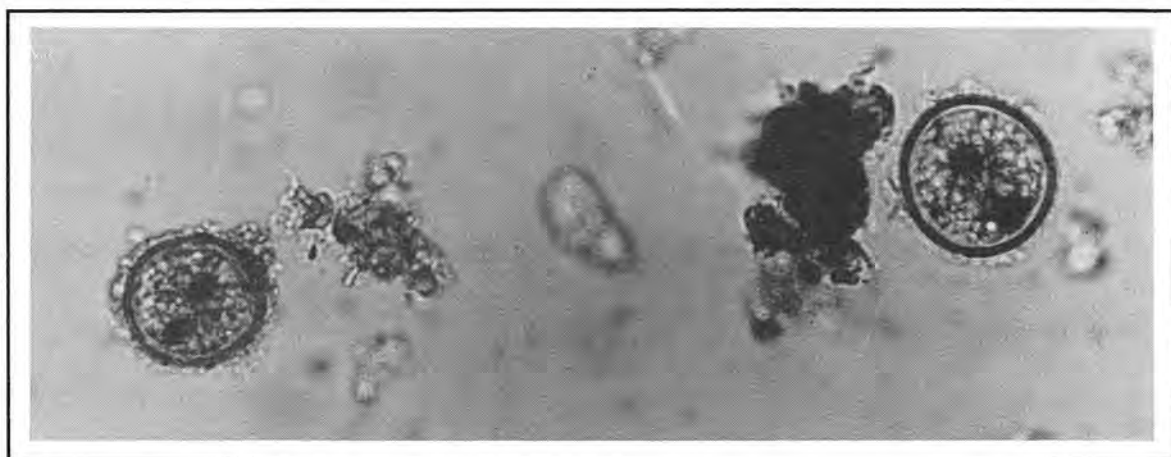
The Guidelines recommend that Australia should adopt domestic or ballast water guidelines and that a management system should be applied immediately to ballast water from ports affected by toxic algal blooms and other organisms (AQIS, 1993).



Photograph 24 Harbour sediment being sampled at Bunbury using an *Ekman* grab for a toxic dinoflagellate cyst survey.

With the pressure of increased exports from Western Australian shipping ports, it is important to establish the present status of all ports with respect to the presence of harmful species and their cysts. Once a toxic species is discovered in an area, it is difficult to pinpoint the exact time at which the species was introduced. The sediments of all Western Australian and Australian shipping ports should be sampled immediately, to provide a reference point to allow for future status comparisons to be made and to make management decisions regarding the risk to domestic shipping traffic.

Dredging operations (B Dale, pers. comm, 1988) and the transfer of shellfish from one area (Hallegraeff et al., 1988) have raised the concerns of scientists about the possibility of dinoflagellate cysts being spread from one area to another. Care should be exercised to ensure that any biological material or aquatic machinery transfers from one area to another are free from contamination by harmful species.



Photograph 25 Dinoflagellate cysts from Swan River estuary sediments. (Size : cyst diameter 20-28 μm)

4 TOXIC DIATOMS, RAPHIDOPHYTES, PRYMNESIOPHYTES AND OTHER RED TIDE ORGANISMS

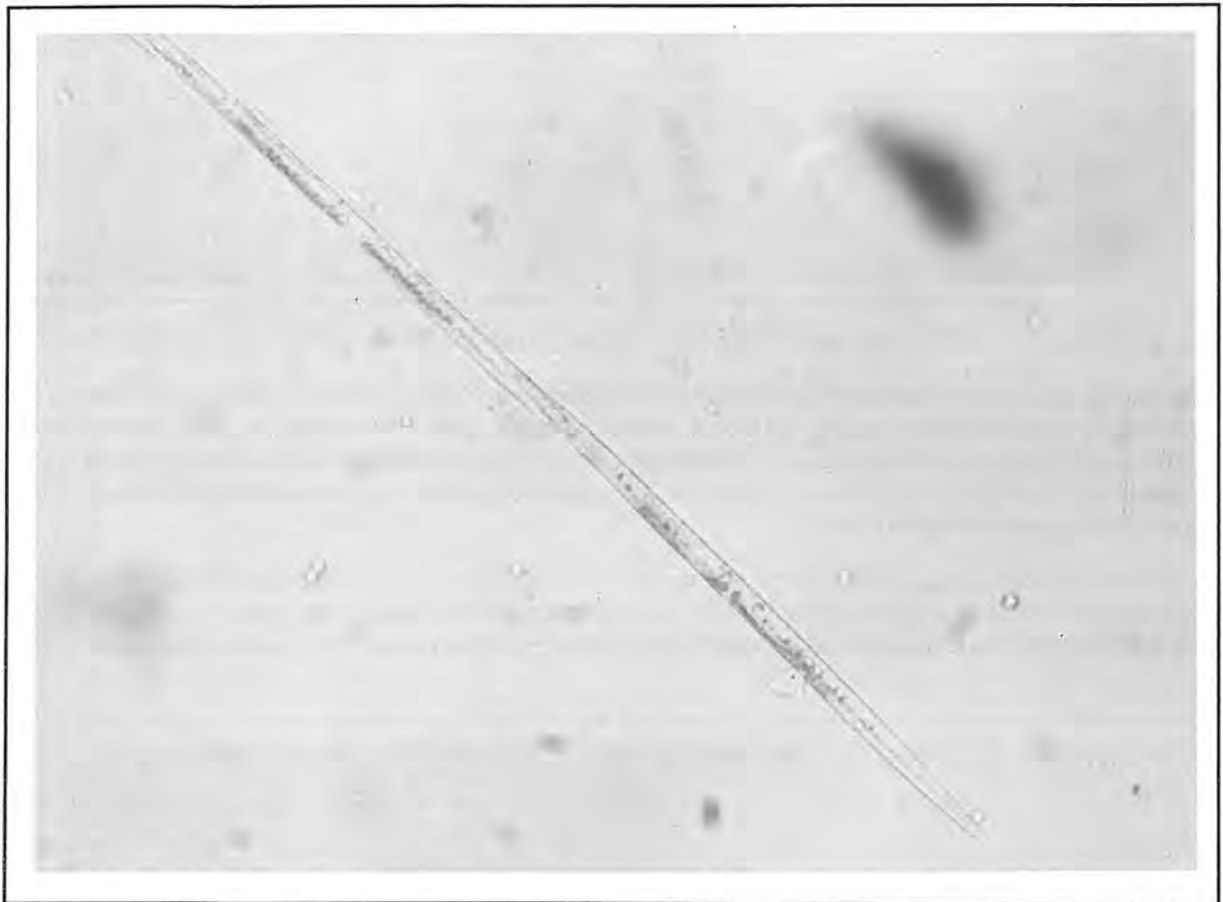
4.1 Diatoms

Diatoms are an essential part of the marine and freshwater ecosystems. This group is generally thought to be beneficial as it forms the base of most food webs. Several toxic diatoms, however, have recently been observed globally and have resulted in shellfish toxicity and deaths of human beings and pelicans.

4.1.1 *Pseudonitzschia*

An Australia-wide survey for the distribution of the toxigenic diatom genus *Pseudonitzschia* (ASP) was carried out by Hallegraef. In WA waters he found non-toxic *Pseudonitzschia pungens* f. *pungens* in the Swan River estuary (*P. australis*, John, 1983); non-toxic *P. pseudodelicatissima* in Cockburn Sound in Bunbury Harbour. The closely related species *Pseudonitzschia pungens* f. *multiseriis* which is toxic (Domoic acid), can only be distinguished from *Pseudonitzschia pungens* f. *pungens* clearly by electron microscopy (Hallegraef, 1994, in press).

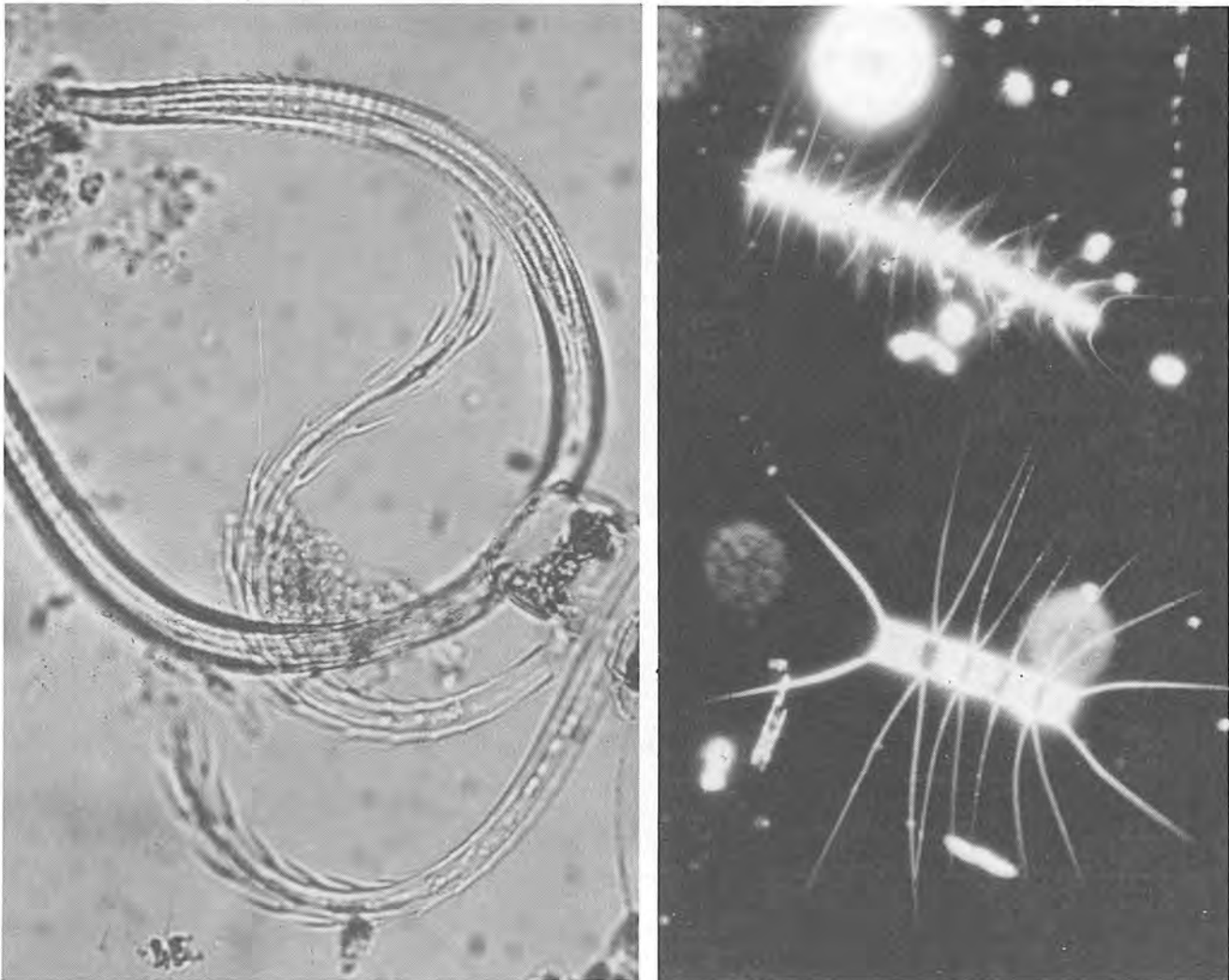
Preliminary ASP (Domoic acid -DA) testing has been carried out on mussels in Western Australia on a HPLC facility which is now operational at the Chemistry Centre of (WA).



Photograph 26 *Pseudonitzschia pungens* forma *pungens* from the Swan River estuary. (Size : cell length 80-140 μm)

4.1.2 *Chaetoceros*

Some large *Chaetoceros* species such as *C. convolutus* and *C. concavicornis*, having large barbed spines, cause gill damage and thus fish mortality in intensive aquaculture systems (Hallegraef, 1991). In the lower Swan River and Cockburn Sound one of the recognised potentially harmful species *Chaetoceros* cf. *danicus* occurs in low numbers in the summer/autumn period. Many dense blooms of *Chaetoceros* species occur in the Peel-Harvey estuary especially after the collapse of *Nodularia* blooms from December to January. There have been no confirmed cases of fish death or fish gill damage arising from this taxa in Western Australia.



Photograph 27 Examples of *Chaetoceros* diatoms with spines from WA. *C. coarctatum* (left).

4.1.3 *Rhizosolenia cf. chunii*

Rhizosolenia cf. chunii can contaminate, and make mussels taste bitter and decrease their growth rates. It has been associated with shellfish mortality 3 to 8 months after a bloom ceased (Parry et. al, 1989).

In Western Australia a similar *Rhizosolenia* occurs in moderately large densities in the Peel-Harvey estuary during summer (i. e., *Rhizosolenia cf. imbricata*).

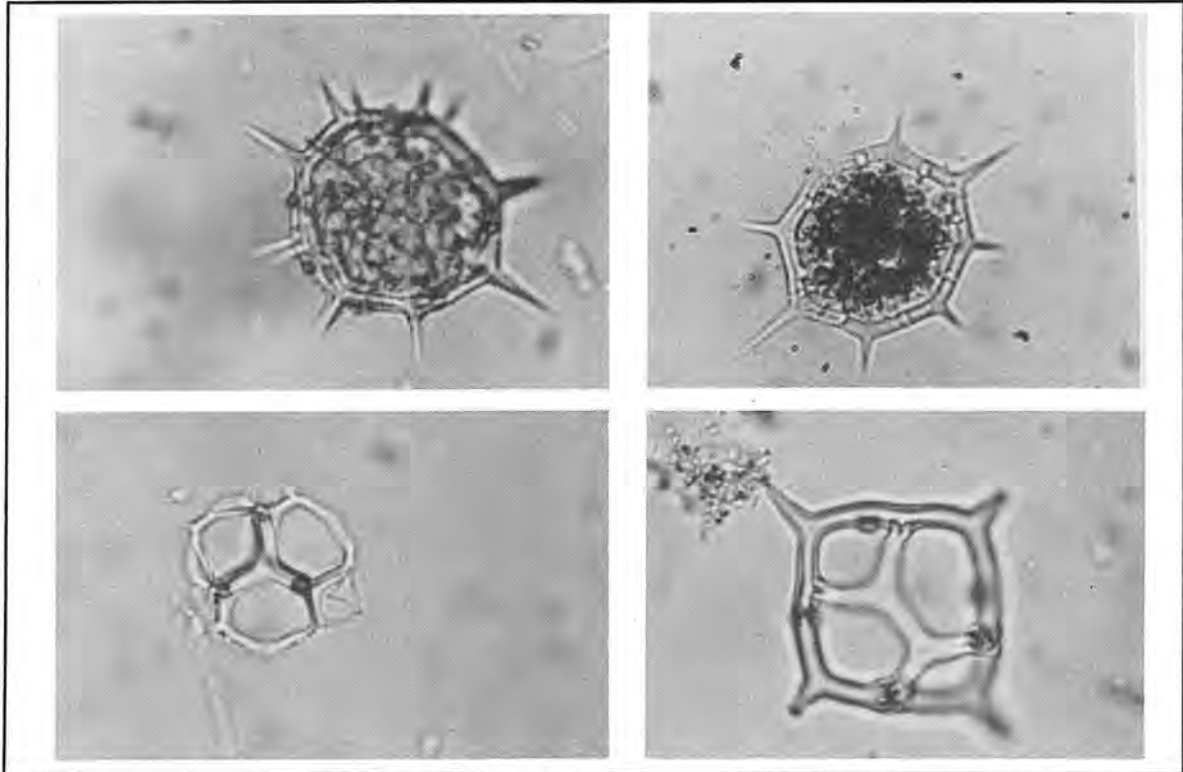
4.1.4 *Amphora coffeaeformis*

This species occurs in the Swan and Peel-Harvey areas (John, 1983). It has been linked to ASP (Domoic Acid) poisoning in shellfish (Hallegraeff, 1991). No ASP toxin testing has been carried on the Peel Inlet. A single shellfish sample tested from the Swan River proved negative for ASP in 1994 (Chemistry Centre, unpublished data).

4.2 Silicoflagellates

Very large blooms of *Distephanus speculum* occur in Cockburn Sound (J John, pers. comm). The species has also been observed in high densities in Albany, Peel-Harvey and Leschenault estuaries (Waterways Commission, unpublished data). *Dictyocha speculum* has been responsible for causing fish deaths in Europe (Hallegraeff, 1991). The complete life cycle and the significance of the recent build-up of this species in Western Australian coastal waters and estuaries are not fully understood. No toxic effects have been reported in Western Australian blooms of this species.

Other silicoflagellates found in Western Australia are *Ebria* and *Dictyocha*.



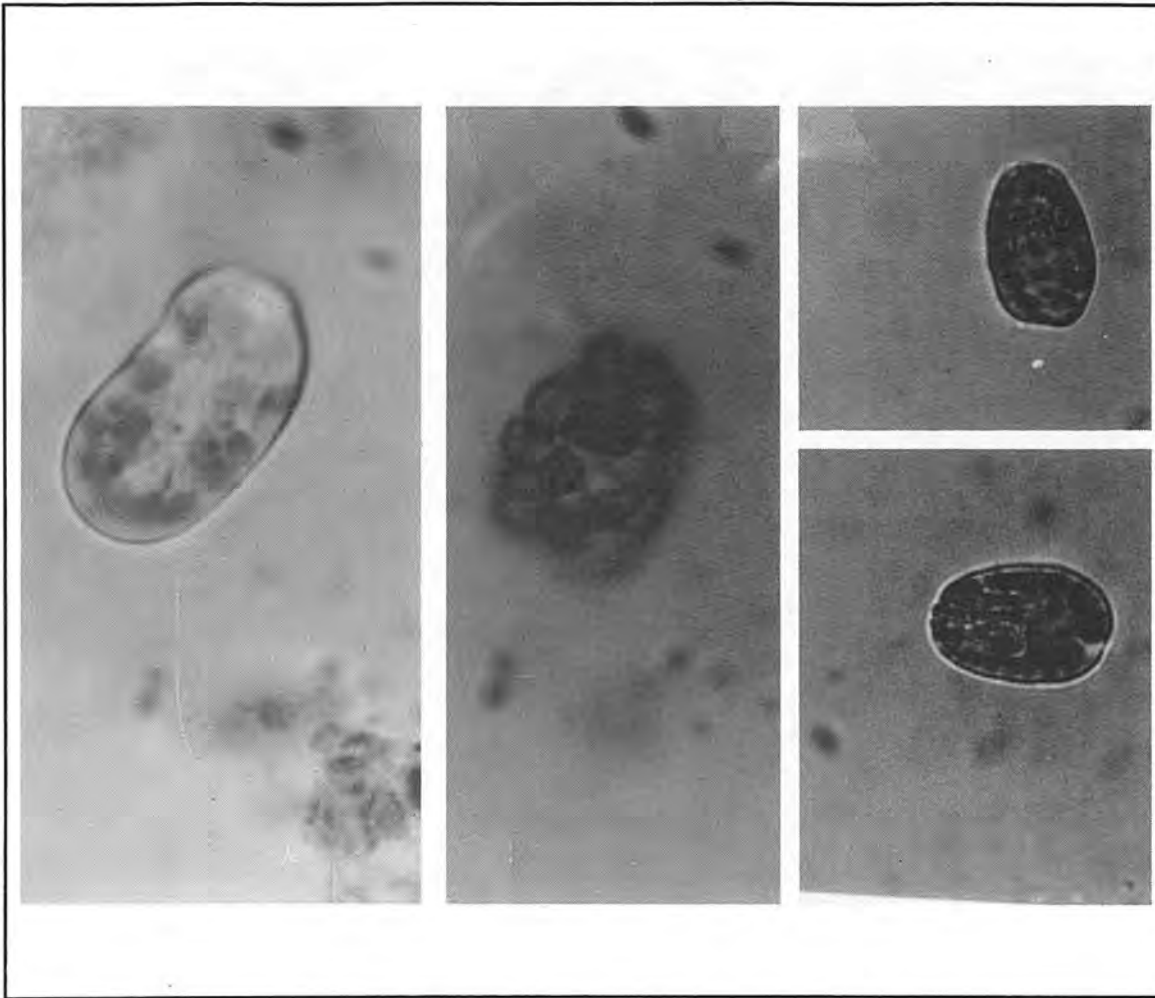
Photograph 28 The silicoflagellates *Distephanus speculum* (Size: cell diameter 10-20 μm) (top) and lower *Ebria tripartita* and *Dictyocha fibula* from the Oyster Harbour, Albany.

4.3 Raphidophytes

Heterosigma akashiwo is a Raphidophyte recognised internationally as a major killer of caged finfish. The killing mechanism of this species is thought to be a combination of clogging of fish gills by mucus secretion and the action of gill-damaging haemolytic substances (Hallegraeff, 1991). Major red tides of *Heterosigma akashiwo* have been recorded in the Swan and Canning River estuaries. In March 1994 a significant density of *Heterosigma* accompanied a *Cryptomonas* bloom in the lower Collie River. The impacts of this species on local wild fish stocks are poorly understood.

A closely related Raphidophyte species cf. *Heterosigma* occurs in Cockburn Sound. Another common Raphidophyte present in bloom quantities in the Swan River estuary is *Fibrocapsa* cf. *japonica*.

Chattonella species cause severe mass mortality problems in overseas fish farms. These very delicate species of Raphidophyte have not been recognised in samples of Western Australian waters.



Photograph 29 The Raphidophytes *Heterosigma* (left and centre) and *Fibrocapsa* (right). (Centre - Lugol preserved specimen.) (Size : *Heterosigma* length 11-20 μm , *Fibrocapsa* length 12-25 μm)

4.4 Prymnesiophytes

Prymnesium parvum has occurred in large blooms in the Vasse-Wonnerup estuarine system where fish kills have been a regular occurrence. This is the only isolation of this species in Australia to date.

Chrysochromulina polylepis caused a serious loss of fish in the Baltic and the North Sea in 1988. Many species of *Chrysochromulina* occur in the Swan and the Leschenault Inlet, and a bloom occurred the Peel-Harvey estuary in 1987 and in the Swan River in Autumn 1994. For positive species identification, these delicate species require specialised preservatives, shadow casting preparation, and electron microscopic techniques.

Cricosphaera caused a severe yellow-green scumming bloom (5,000,000 cells/L) in a canal off the Murray River at Mandurah in June 1993. The species was confirmed by electron microscopic examination of its scales. This bloom led to restrictions of the area for recreational contact activities. The same species was found mixed in a bloom of *Aphanizomenon* in the Blackwood River in January 1994.



Photograph 30 *Cricosphaera* scum from a canal in the Murray River at Mandurah in 1993.

4.5 Chlorophytes

No toxicity has been found in chlorophyte blooms so far (Moestrup, pers. com., 1993). In Western Australia chlorophyte blooms can be very dense without any observable toxic effects. Though blooms with densities of up to 3,500,000,000 cells/L of *Chlamydomonas* cf. *globosa*, have frequently been recorded in the Swan River in spring, they have not been implicated in fish deaths so far. Similarly, intense blooms of *Carteria* have been recorded in the Canning River upstream of the Kent St Weir. *Scenedesmus* at densities of 2,500,000,000 cells/L have been found entering the Preston River from a lake affected by an industrial wastewater discharge (LIMA, unpublished data).

4.6 Cryptophytes

Cryptomonads have not been shown to produce toxic blooms so far. Spring-summer red tides of *Cryptomonas* species frequently occur in the Swan-Canning, Serpentine, Murray and Collie Rivers, especially when the river is stratified in summer.

A blue-green coloured bloom of *Hemiselmis virescens* was recorded in the Swan Estuary. The estuarine mussels, *Xenostrobus securis*, sampled then frozen from the bloom-affected area, released a bluish pigment when allowed to defrost.

4.7 Euglenophytes

Euglenophyte blooms are not known to pose a toxic algal problem (Moestrup, pers. comm., 1993). In the Swan-Canning, Avon and Collie rivers blooms of *Eutreptiella* have been recorded in the brackish waters.

Frequently, bright red scums of the freshwater species *Euglena rubrum* have been observed in the upper reaches of the Canning River, farm dams, freshwater lakes, open stormwater drains. This species can change its colour from red to green at different times of the day. On the highly tannin-coloured waters of the Canning River, a powdery surface film, composed of rounded off *Euglena* resting cysts, is recorded in summer which frequently causes complaints by river users. Over twenty species of Euglenophytes (*Euglena*, *Eutreptiella*, *Trachelomonas* and *Phacus*) have been recorded in WA waterbodies at various densities.



4.8 Chrysophytes

Chrysophytes do not pose many problems except for *Aureococcus* which can form extraordinary brown tides. These are very small cells of about 2-3 μm in diameter. *Aureococcus* has caused mussel and fish deaths (Hallegraeff, 1991), reduced the fecundity in anchovies, caused a reduction in polychaete larvae and the amount of macroalgae attached to mussels (Smayda and Fofonoff, 1989). No blooms of *Aureococcus* have been confirmed in Western Australia.

Apedinella blooms have occurred in the upper Swan River estuary in 1993. The spines of this species from the Swan River bloom were found to quickly clog filter papers used for chlorophyll analyses. These spines of *Apedinella* could potentially pose problems to fish gills during blooms.

4.9 Coccolithoporids

Coccolithoporids have caused spectacular white water blooms in New South Wales. This affected the ability of marine animals to feed. Over 300 species of Coccolithoporids are known oceans world-wide (Hallegraeff, 1988). These species can be easily overlooked by their small size and in incorrect preservation techniques. They can produce white water blooms which have deleterious effects on feeding by some marine animals. In Western Australia the densities of these species appear to be relatively low.



Photograph 31 Coccolithoporids from the lower Swan River estuary in summer/autumn. (Size : cell diameter 6-10 μm)

4.10 Cholera, amoebae and other threats

There are increasing identifications of cholera in ballast water samples especially from South America. Increasing cholera outbreaks in India and Bangladesh may have implications for human health in Australia.

Frequently, surface water temperatures over 30°C are recorded in the upper Swan River (Swan River Trust unpublished data). It is likely that the amoebic meningitis causing protozoa *Naegleria fowleri* is present in other fresh to brackish exposed, untreated waterbodies including rivers which are used for swimming. This can cause a risk to river users, especially by schools and social groups in the summer months.

The numbers of species of crustacea, bivalves, fish, crabs, starfish and macroalgae introduced into Australian waters is increasing. This has increased concern for the new threat of the potential transfer of these organisms in domestic coastal shipping movements especially from Tasmanian waters.

The recent discovery of a potentially harmful worm colony (*Sabella spallanzanii*) in Cockburn Sound, highlights the concerns of introductions of organisms in both domestic and international ballast.



5 CURRENT MECHANISMS FOR SURVEILLANCE IN WESTERN AUSTRALIA

There is clearly a potential human health risk from consumption of shellfish, or fish flesh that may have been contaminated with toxins from various phytoplankton species. Species that have been confirmed to have, or are suspected of having caused toxicity in edible tissues are present in many coastal and estuarine waters.

Blue-green algae and other potentially toxic phytoplankton species have become well established in many coastal, estuarine and inland waterways of Western Australia. These species may contaminate farm and stock water supplies with potentially serious health consequences.

A number of phytoplankton species have been identified which may compromise the commercial viability of various aquacultural enterprises. This industry is in its infancy in Western Australia and needs to be supported.

An appropriate surveillance program is needed to monitor waters, sediments and ballast waters for potentially harmful phytoplankton, to assess their health and aquacultural implications, and to develop, implement and co-ordinate appropriate management strategies.

A documented protocol for response to toxic and potentially toxic phytoplankton outbreaks needs to be established in Western Australia. This need was recently highlighted when a dense bloom of the blue-green *Trichodesmium erythraeum* was observed off metropolitan coastal waters in April 1994. This resulted in many samples of the algae being sent to the Waterways Commission, Department of Agriculture, Chemistry Centre of WA, Fisheries Department and Universities by various concerned agencies. None of the samples collected included healthy intact cells but comprised partly decomposed samples which were leaking their intracellular contents into the surrounding water. This added problems to the significance of and interpretation put on the toxicity test results obtained from tests carried out.

5.1 Surveillance

A comprehensive cost effective phytoplankton sampling program needs to be established to monitor the presence of harmful species and toxicity in Western Australian waters. The results from the current study will allow for widespread recognition of bloom species presence and their intensity. The presence of cysts from harmful dinoflagellates in marine and estuarine sediments, including harbour sediments, needs to be determined. There need to be improvements of toxic phytoplankton identification and detection methods to enable rapid turn around of results. Facilities for the isolation and culture of suspected harmful phytoplankton species need to be established to enable the toxicity of strains to be tested when their presence is in low numbers. Appropriate microscopic equipment and techniques are required for the identification of dinoflagellates and other potentially harmful species.

The PSP, DSP, NSP and ASP toxicities of wild and cultured shellfish need to be more regularly and completely monitored as part of this program. Some Western Australian mussel farmers are intending to export to European and US markets. Bacteriological and chemical testing of the products is required in order to satisfy the Australian Quarantine Inspection Service (AQIS) export requirements and those of the American Food and Drug Authority (FDA).

In the sub-tropical coastal region a surveillance program for ciguatera and PSP-causing dinoflagellates needs to be carried out.

5.2 Toxicity testing

Toxicity testing of mussels (both wild and cultured) for PSP and ASP can currently be undertaken by the Chemistry Centre (WA) using High Performance Liquid Chromatography (HPLC).

Bioassay using mice can be set up for DSP but this method of testing is increasingly under pressure from animal welfare organisations. Mouse bioassay could be continued for blue-green algal toxin testing by Animal Health Laboratories at the Western Australian Department of Agriculture. Worldwide resistance to the use of laboratory mice is creating a demand for alternative chemical analytical methods for algal toxins. The Chemistry Centre of WA has recently established a *Microtox* testing facility where bacteria are used to indicate potential human toxins. The unit was recently used to test a blue-green algal bloom sample from the Canning River to successfully confirm its toxicity. Similarly the *Microtox* was used to test the dinoflagellate red tide of *Gymnodinium simplex* from the Swan River, which proved negative for toxins.

A data base should be established whereby actual phytoplankton densities can be related to toxicity effects of the various species.

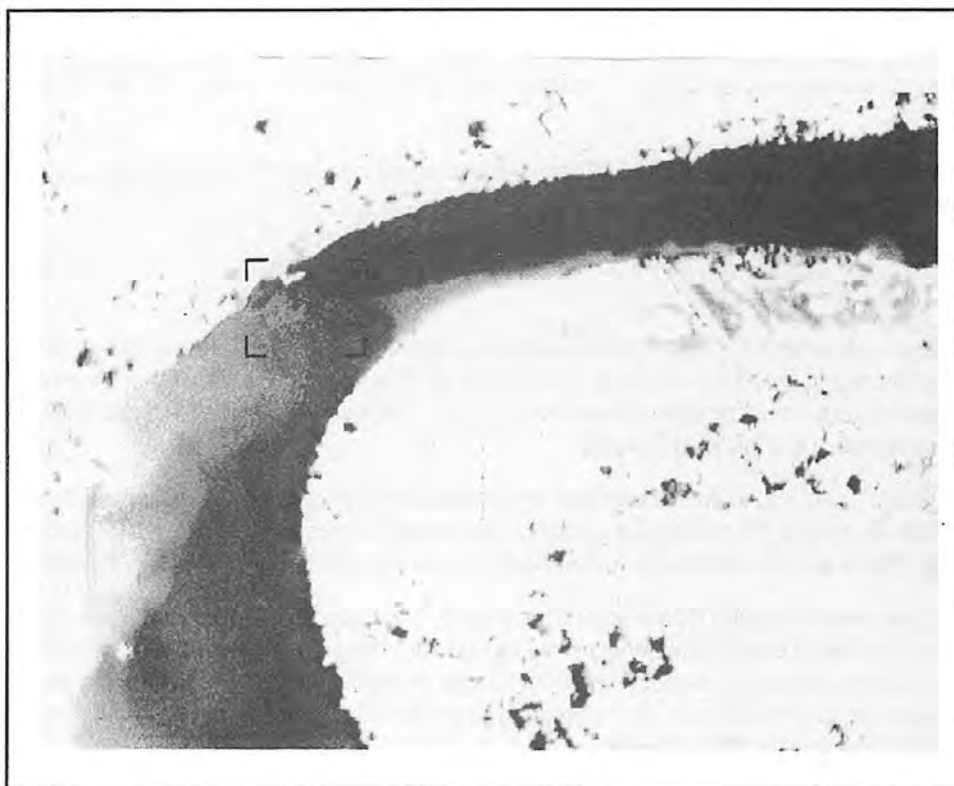
The World Health Organisation has recognised the need to establish immuno-assay techniques to complement other techniques.



Photograph 32 *Microtox* toxicity testing facility at the Chemistry Centre of WA.

5.3 Surveillance of toxic blooms in WA

The Waterways Commission and the Swan River Trust have been monitoring major phytoplankton species in some Western Australian estuaries (Swan-Canning, Peel-Harvey and Leschenault) and associated rivers and drains, for a number of years. The CSIRO in conjunction with the Swan River Trust has begun the remote sensing of blooms using the Compact Airborne Spectrographic Imager (CASI). Flights over the Swan River were flown in February 1993 in both spectral and spatial modes. Early analysis results of the CASI spectral mode data showed a strong relationship between spectra and the suite of phytoplankton species (Hick et al, 1994). Collaborative WWC/CSIRO work has continued in 1993/94 to carry out the remote sensing of the common blooming species using the Digital Multi Spectral Video (DMSV) system in the Swan and Canning Rivers. The technique should eventually permit quick and efficient surveillance of all waterways experiencing blooms of similar species.



Photograph 33

Remote sensing image of a *Eutreptiella* bloom in the Rivervale area of the Swan River in 1994 acquired using the Digital Multi Spectral Video system. Courtesy of Dr F Honey (Spec Terra Systems Pty Ltd).



Some potentially harmful and toxic species have been identified. Dinoflagellate species may be toxic at very low cell densities and can be easily overlooked during routine environmental sampling. With the establishment of the Avon River (ARMA), Albany Waterways (AWMA) and most recently the Wilson Inlet (WIMA) Management Authorities, phytoplankton species present in those estuaries and rivers are gradually being examined more closely by the Waterways Commission.

Many other waterways however remain untested for harmful species presence and for shellfish toxicity. Some of these waterways have been used for various commercial shellfish aquaculture projects. These may require a transfer of biological materials such as shellfish and algae. This then could facilitate the transfer of toxic dinoflagellate cysts and other unwanted harmful organisms/diseases from infected areas into areas presently free of these organisms. Organic materials and nutrients can accumulate in the sediments below these aquaculture sites. These accumulated nutrients are subsequently released to the overlying water column, when low oxygen levels develop at the bottom promoting the rapid increase of normally rare or sub-visible harmful species, which in turn can decimate or ruin the mariculture project itself.

The Waterways Commission frequently receives red tide and blue-green samples from various locations outside its management areas. This way most of the suspected toxic phytoplankton species from WA so far have been identified by the Waterways Commission and confirmed by Dr Hallegraeff. The Port Authorities of WA are moving to determine the present status in regard to exotic dinoflagellate cysts in their harbour sediments and have requested the assistance of the Waterways Commission in future sampling programs. Dredging operations in harbours located in the mouths of tidal estuaries and allow benthic dinoflagellate cysts to be remobilised upstream, during incoming tides into nutrient-rich waters. This would allow blooms of these species to develop.

Closures of recreational and shellfish harvesting in Mandurah during *Nodularia* blooms are effected jointly by the local government health departments and the Waterways Commission. No such closures or monitoring have occurred in the Vasse-Wonnerup estuary. The most effective measure to control toxin exposure in the general public is through environmental monitoring programs at the source. Only when these developments are in place can safe seafood products be available for human consumption.

A comprehensive surveillance and testing program would establish the necessity for closing commercial and wild shellfish sites over summer months when phytoplankton activity is at its peak. A surveillance program will enhance and supply necessary information on the diversity of harmful phytoplankton present in Western Australian waters and sediments including locations, life cycles and toxicity. Base-line studies will aid in the potential identification of exotic introduced species from foreign and national shipping ballast waters.

Other advantages include the provision of a data base for the placement of new fishing and mariculture leases. The relationships between Western Australian organisations involved in this area are shown in Appendix 6.

5.4 Gaps in current efforts

There is a long coastline and many waterbodies in Western Australian which are potentially at risk and are currently not being monitored. The Waterways Commission's monitoring program operates at a spot check or event response level, and is not giving the complete picture of the risks associated with potential harmful phytoplankton throughout Western Australia. In many instances, the responsibility for monitoring and the management of toxic algal species is not clear with a number of State, Federal and local government agencies having overlapping interests and responsibilities. Clearly an integrated approach by all agencies having some role in this problem is needed.

In WA there is the need for a phased response when a potentially toxic or harmful bloom event is indicated including the means for conservative closures of recreational and aquacultural areas.

5.4.1 Surveillance

The monitoring of phytoplankton in marine and estuarine environments by itself does not provide sufficient protection for public health, but does provide useful early warnings for finfish and shellfish enterprises. An appropriate Statewide surveillance program needs to combine regular phytoplankton and cyst monitoring, including algal culture and toxicity evaluation using direct techniques on algae, shellfish and mussels and using indirect mammalian and bacterial bioassay.

Because of the threat to aquaculture, to public health and to commercial and recreational fisheries, an accurate, quantitative, cost-effective system which is able to provide an early warning of harmful phytoplankton events needs to be established. This should provide the ability to detect with speed the presence of toxic species and changes in their numbers, and to determine human health implications.

A regular phytoplankton monitoring program would establish the presence of potentially toxic species in the estuarine and coastal waters, in ballast waters and in sediments. This would need to be complemented by toxicity testing of commercial and wild mussel, oysters and fish. A thorough assessment of the ecology of potentially harmful species would help refine current limited mussel monitoring programs. The third component of an appropriate surveillance program would identify the human health implications of toxic phytoplankton through analytical and biological toxicity evaluation.



A survey of State health records may identify past occurrences of shellfish poisoning resulting from toxic phytoplankton now that a better understanding of clinical symptoms has been gained globally. This information would then form the basis of management strategies for both short and long term response, and the development of longer term management.

Significant economic benefits and enhanced export potential for Western Australian shellfish would be stimulated through international knowledge of an established phytoplankton monitoring program. It is already a requirement for export to an increasing number of countries. A clean and safe product could be produced on the world market arena.

Many waterways, lakes and inlets such as the Blackwood River, Avon River, Vasse-Wonnerup, Hardy Inlet etc. are not being routinely monitored for the presence of harmful species. Many areas are only examined if by chance a bloom has been noticed by chance. The same is largely true for small wetlands in the metropolitan area. Bottom to surface phytoplankton net tows are required to ensure that harmful species will be sampled when blooms are situated metres below the water surface.

5.5 Solutions for Western Australia

The various stakeholders in Western Australia and the many issues and areas of Harmful Algal Surveillance are given in Appendices 6 and 7. From these tables it is apparent that an integrated approach to the problem of harmful algal blooms was needed.

A working party of government officers from the various departments met to co-ordinate action required for algal (*Nodularia*) blooms in Western Australia in November 1993. A working group was set up to prepare a report to Government, recommending a coordinated strategy to control and manage harmful algal bloom outbreaks in inland and coastal Western Australian waterways.

The management issues to be addressed by the working group are:

- * surveillance
- * toxicity testing
- * risk assessment
- * public education
- * impact minimisation
- * research and co-ordination

5.6 Toxicity testing and risk assessment

In Western Australia various algal and shellfish toxicity tests can be carried out by the Department of Agriculture and at the Chemistry Centre of WA. These can be expanded to encompass more toxicity parameters.

5.6.1 Detection and quantification of algal toxins

Analysis techniques for the presence of toxins in shellfish and finfish must be sensitive, specific, precise and cost effective and must be shown to be consistent through extensive inter laboratory calibration and comparison with a variety of other methods.

5.6.2 Paralytic Shellfish Poisoning (PSP)

Biological methods

a) Mouse bioassay

The mouse bioassay involves intraperitoneal injection of an acidified extract (1 mL) of shellfish tissue into mice to determine acute toxicity. Though 'mouse units' have been used, the preferred toxin level units are the $\mu\text{g STX eq}/100\text{g}^{-1}$. The lowest detectable PSP level is about $40 \mu\text{g STX eq}/100\text{g}$ wet weight of tissue. The coefficient of variation is about 20%. The method is non-specific but is useful for broad screening. The mouse test is a quick test for a run of lots of samples.

b) Housefly testing

This uses the same acidified extract injected by microsyringe into 20 domestic houseflies first anaesthetised by cold treatment. The lowest detectable PSP level is about $20 \mu\text{g STX eq}/100\text{g}$ wet weight of tissue, which is more sensitive than the mammalian bioassay. The loss of specificity of potential mammalian toxins reduces the effectiveness of this technique.



Chemical methods

a) Fluorometric detection

This chemical method for PSP analysis relies on the alkaline oxidation of toxins to fluorescent derivatives. Total toxicity ratios can be either under or over estimated depending on the fluorescence of the toxin profiles. This method can give cost-effective assessment of toxicity once a data base of toxin profile and derivative profile behaviour is established.

The High Performance Liquid Chromatography (HPLC) methods for PSP enables the quantification and resolution of individual toxin components, based on the use of post-column oxidation of toxins to fluorescent products.

HPLC offers advantages as a monitoring tool because it is very cost effective once the methodology is established. Its precision can be around 3%, its reliability is good, it is highly specific to PSP toxins and it is from 4-400 times more sensitive (<1 µg STXeq/100g tissue) than mouse bioassay.

Sullivan and Oshima HPLC methods.

The Sullivan method offers the potential for high volume sample screening as part of a PSP regulatory program. Its LC column has longevity and tolerance to relatively impure shellfish samples. A single injection separates most significant shellfish toxins. Many worldwide HPLC facilities have been configured using this method and comparisons between mouse bioassay have been carried out.

The Oshima HPLC method has the advantage of a more simplified toxin chromatogram. It reduces the occurrence of co-eluting toxins and subsequent underestimation of toxicity (Soames and Stewart (CCWA) pers. comm, 1993).

5.6.3 Diarrhetic Shellfish Poisoning (DSP).

Though not reported to cause death DSP toxin contamination of shellfish causes severe gastrointestinal disturbances including acute diarrhoea, nausea, vomiting and abdominal pain. The toxins involved in DSP effects are polyether fatty acids, Okadaic acid (OA), dinophysins (DTX1, DTX3) and the polyether lactone Pectenotoxins (PTX1, PTX2, and PTX3). Yessotoxin (YTX) has recently been described.

Biological Methods

a) Mouse bioassay for total DSP toxicity using intra peritoneal injection

Homogenates from the soft tissues and digestive glands of shellfish extracted with acetone, transferred to diethylether, and subsequently evaporated, are tested by intra peritoneal injection of 1 mL into three (18-20g) mice. These are observed for 24 hours or until death. Dilutions provide the number of mouse units per g of original tissue. A mouse unit (MU) is the least amount of tissue required to kill two of three mice in a 24 hour period.

b) Mouse Bioassay-for Total DSP toxicity using regular ingestion

Extracts prepared as above are force fed into 4-5 day old mice. The effects are measured as the "fluid accumulation ratio" (FAR).

Tissue can be fed without pre-treatment to experimental rats, with food avoidance and faecal consistency taken as indicative of gastrointestinal toxin.

Chemical methods

The extraction of toxins from tissue is successively carried out using methanol, petroleum ether and chloroform steps. Esterification of DSP toxins is followed by a clean up using a Sep-pak silica cartridge column, then detection and quantification of fluorescent esters of OA and DTX1 by HPLC. The detection of concentrations is good but some analytical interference still exists.

5.6.4 Amnesic Shellfish Poisoning (ASP)

Domoic acid

DA is a secondary amino acid which acts as a glutamate antagonist on the kainate receptors of the central nervous system.

Extraction

Acidic aqueous extraction of DA in 0.1N HCl with 5 minutes of boiling and stirring produces the best compromise of reproducibility,



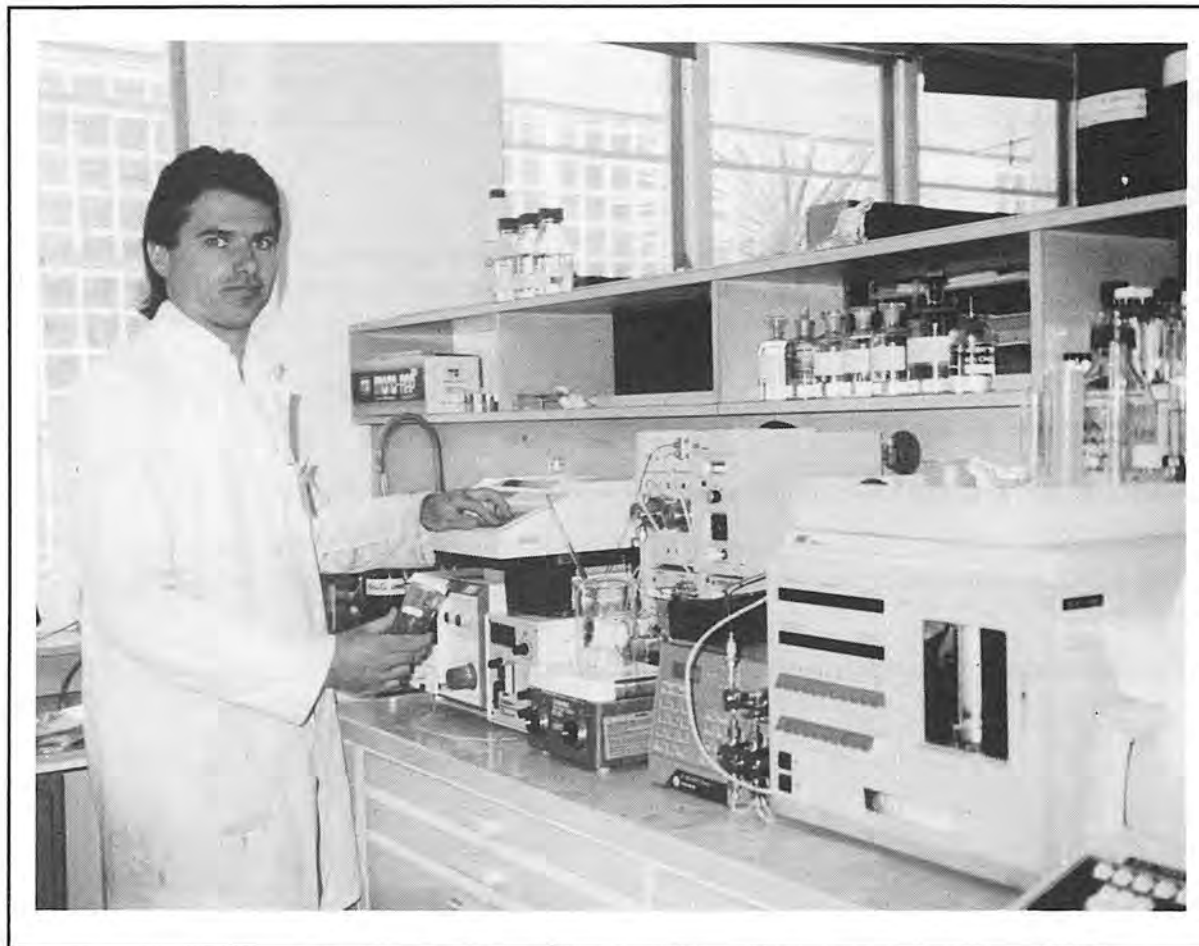
recovery (75% DA) and cost effectiveness. Other neutral aqueous and aqueous/methanol extractions may produce more efficient DA recovery.

Mouse bioassay

Intra peritoneal injection into three mice produces typical symptoms over 18 hours, of scratching the eye and ear regions by the hind legs on both sides alternatively, uncoordinated limb movements and stupor. Sensitivity is not reliable below 25mg/100g.

Chemical methods

HPLC using UV absorbency detection (242nm) has been developed for DA. The detection limit is 75µg/100g of sample. Diode array detectors or scanning UV detectors provide better characterisations of DA peaks.



Photograph 34 High Performance Liquid chromatography (HPLC) unit at the Chemistry Centre of WA

5.6.5 Neurotoxic Shellfish Poisoning (NSP)

Symptoms are similar to and milder than PSP. These NSP toxins are known as brevetoxins. They are lipid soluble long chain polycyclic ether compounds. Brevetoxins B (BTXB) and C (BTXC) and GB1, GB3 and GB6 have been characterised.

Biological method.

A mouse bioassay has been developed for NSP. The method is similar to that for PSP with a more elaborate extraction and clean-up procedure, followed by 24 hours of observation.

Chemical methods

HPLC is currently not widely used because of the labile nature of brevetoxins.



Sample Handling Problems

There may be problems obtaining sufficient weight or volume of sample. Proper storage is critical especially for NSP and DSP samples. Samples are to be frozen as rapidly as possible and for DSP, boiled first before freezing to reduce fatty acid interference.

Biological methods

Apart from the mouse bioassay for PSP, the other bioassays are semi-qualitative at best. In most bioassay methods toxin specific symptoms are poorly defined. The exceptions are the characteristic symptoms of PSP, DSP-diarrhoea and intestinal fluid accumulation, and the scratching symptom of DA.

Summary of chemical methods

HPLC methods are a good quality control for bioassay methods and can provide identification of specific toxins. The occurrence of non-toxic fluorescent artefacts, however, is a major drawback in auto-analyser methods for PSP toxicity by fluorescence.

There has been a problem with the limited availability of high quality standard toxins for calibration. Efficient sample processing is most readily obtained when the HPLC is operated continuously and dedicated to a specific task and not employed for multiple purposes.

Immunological techniques

No immunological test kits are available for general use in regulatory programs though several prototypes have been developed. The use of immunological techniques for monitoring phytotoxins is advantageous because they can be adapted to yield high sensitivity and toxin specificity. They offer the possibility of being used in the field by the shellfish producers themselves. Sample extraction for immunological assay is simple.

Methods using cell cultures

Cell cultures may be used as a guideline in monitoring the presence of toxins in shellfish and algae.

HPLC methods are now available for the detection and quantification of PSP toxins, for the common components of DSP for NSP and Domoic Acid. For the near future the bioassay method will continue to be employed for regulatory surveillance, with confirmation with LC analysis.

With the changing attitude to the use of experimental animals immunoassays may become more important.



6 CONCLUSIONS

To date, many microalgal organisms which are known to produce harmful red tides, neurotoxins or hepatotoxins or have contributed to fish deaths through anoxia or other mechanisms have been identified and characterised. The fact that their numbers, along with their frequency and geographic distribution of toxic blooms, have been increasing, is of major economic, environmental and public health concern.

Toxic dinoflagellate blooms are no longer an occasional phenomenon for a number of reasons including eutrophication and transfer of harmful organisms in ships ballast etc. They pose a major economic and a serious health threat to the afflicted country. Losses of export and local markets due to contaminated seafood has the potential to affect many world economies. As well as the public health problems associated with increases in regular acute poisoning, the chronic effects of long term exposure to these dinoflagellate toxins are unknown.

Australia has already experienced the impact of at least one introduced toxic dinoflagellate species and other organisms, so control measures are required to reflect the risk to Australia's economy, public health and environment. Ballast water is a serious threat not just from international ports but now also from within Australia. The world-wide trend of increased phytoplankton blooms and the discovery of harmful species is being repeated in Western Australian waters. Much of this trend is due to the greater concern and awareness by the scientists and public alike and the increasing nutrient enrichment of waterways. The threat of an increased discharge of nutrients in wastewater to coastal waters, which have been shown to contain potentially toxic *Alexandrium*, *Dinophysis* and *Prorocentrum* species is also of concern. The potential increase of these species' densities could have a harmful impact on the shellfish in the area. Raphidophytes and other presently sub-visible harmful species, could be increasing their presence to harmful numbers at depth and could express themselves as a problem to fish and shellfish alike before they were detected by surface plankton monitoring.

The enforcement of dinoflagellate toxin regulations, which are currently being considered by the National Food Authority, ultimately depends on the availability of reliable analytical techniques and surveillance programs.

The presence of an ever increasing frequency of potentially toxic species of cyanobacterial blooms occurring in many waterways in which they were not previously found indicates that further more intensive and complete routine surveillance and toxicity testing of all waterbodies and needs to be carried out.

Without a National or local shellfish monitoring program in place, Western Australia could be caught by surprise when shellfish (recreational) become toxic and officials are unprepared to handle sampling, testing and communicating results from tests. Response time determines how many people become adversely affected

In Western Australia these areas transcend the boundaries of many management agencies. A central unit or protocol for Statewide surveillance and testing of marine, estuarine, riverine and wetland harmful phytoplankton, which combines the knowledge, expertise and facilities of the various agencies, would provide the most efficient use of the available resources. This unit could be used by local, State and private management bodies in areas where there is little previous experience in the areas of blooms and their management.

The Harmful Algal Committee, under the auspices of the Health Department of WA, has begun to address the major issues relating to harmful algal surveillance and management of toxic blooms in Western Australia, taking into account the requirements of the various affected management authorities.

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Appendix 1 Potentially harmful phytoplankton blooms and red tides observed in Western Australian waterways excluding the Swan-Canning River

Type	Waterbody	Location	Species	Impact
Blue-green bloom	Blackwood River	Bridgetown	<i>Aphanizomenon</i>	Max 4,800,000/mL-Closures to recreation, dominantestic use Feb-Mach 1993 &1994
Blue-green bloom	Blackwood R		<i>Nodularia</i>	31,000/mL Closures in the area Feb 1994
Blue-green bloom	Blackwood R		<i>Anabaena circinalis</i>	8,000/mL Closures in the area Feb 1994
Blue-green bloom	Sleeman River	Denmark	<i>Anabaena circinalis</i>	132,500/mL. Warning to farmers Feb 1994
Chlororphyte	Sleeman River	Denmark	<i>Chlamydomonantas</i>	950,500/mL. Green Water Feb 1994
Chlororphyte	Pallinup River		<i>cf Nannochloris</i>	September 1989
Chlororphyte	Lake	Dunsborough	<i>Carteria</i>	218,000/mL Feb 1994
Blue-green	Nornalup Est.		<i>Oscillatoria</i>	7,000/mL June 1993
Euglenophyte	Collie River	Bunbury	<i>Eutreptiella</i>	787,000/mL-April 1987
Red Tides	Collie River	Bunbury	<i>Cryptomonas</i>	80,000-108,000/mL April 1987
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	80,000-166,000/mL April 1989
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	50,000-61,000/mL Feb-March 1990
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	37,000 /mL-loss of amenity, Jan 1992
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	7,000-23,000/mL. April 1991
Raphidophytes	Collie River	Bunbury	<i>Heterosigma akashiwo</i>	40,000/mL March 1994
Dinoflagellate bloom	Collie River	Bunbury	<i>Gymnodinium simplex</i>	18,600-453,000/mL-Dinoflagellate Red Tide
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	15,000 /mL-loss of amenity, Mar 1992
Red tides	Collie River	Bunbury	<i>Cryptomonas</i>	23,000/mL April 1991
Diatom bloom	Collie River	Bunbury	<i>Skeletonema potamos</i>	20,000-65,000/mL Jan 1992
Prymnesiophytes	Vasse-Wonnerup Estuary	Busselton	<i>Prymnesium parvum</i>	240,000/mL. Fish kills in area. Feb 1990
Diatoms	Vasse-Wonnerup Estuary	Busselton	<i>Nitzschia closterium</i>	110,000/mL. Jan 1990
Dinoflagellate	Vasse-Wonnerup Est.	Busselton	<i>Prorocentrum minimum</i>	186,000/mL June 1990
Dinoflagellate	Vasse-Wonnerup Estuary	Busselton	<i>Prorocentrum minimum</i>	180,000/mL July 1990
Diatoms	Wilson Inlet	Denmark	<i>Chaetoceros</i>	10,000-20,000/mL

Type	Waterbody	Location	Species	Impact
Dinoflagellate	Wilson Inlet	Denmark	<i>Prorocentrum</i> cf. <i>lima</i>	Tested mussels negative/ Cultured by CSIRO Hobart negative DSP
Dinoflagellate-benthic bloom	Parker Point	Rottneest Island	<i>Prorocentrum</i> cf. <i>lima</i>	Unknown - dense benthic bloom growth (Massini)
Dinoflagellate-red tide	Collie	Bunbury	<i>Gymnodinium simplex</i>	400,000/mL-loss of amenity, March 1989.
Dinoflagellate-red tide	Mindarie Keys	Indian Ocean	<i>Scrippsiella</i>	68,000/mL. Complaints Feb 1992. Two more unidentified blooms since
Blue-green	Blackwood River		<i>Microcystis</i> cf. <i>incerta</i>	50,500,000/mL 1993
Dinoflagellate-red tide	Collie River	Upstream	cf. <i>Peridinium</i> sp.	Fish kill in fresh water pool. 46,200/mL. Mucus in sample? May 1993
Raphidophyte-benthic bloom	Cockburn Sound	Mandurah	<i>Heterosigma</i>	Sediment covered with jelly-like coating
Yellow-green tide	Blackwood River	Bridgetown	<i>Cricosphaera</i>	200,000 /mL
Diatoms	Wilson Inlet	Denmark	<i>Chaetoceros</i>	10,500-20,500 associated with low oxygen in bottom water Sept. 1985
Dinoflagellates	Leschenault Estuary	Bunbury	<i>Katodinium</i>	10,950/mL red tide
Blue-greens	Harding Dam		<i>Anabaena/ Microcystis</i>	Low cell numbers- taste & odour problems experienced occasionally
Blue-greens	Dodson Wetlands	Bunbury	<i>Anabaena/ Oscillatoria</i>	Toxic-recreational and health concerns
Blue-green bloom	Warnbro-Fremantle	Indian Ocean	<i>Trichodesmium erythraeum</i>	Health warnings and some crayfish deaths April 1994
Fish deaths	Kalgan River	Albany	no apparent bloom	700 six-lined trumpeter Jan 1994

Appendix 2 Potentially harmful phytoplankton blooms observed in the Swan-Canning and Avon rivers in Western Australia.

Month	Species	Location	Chlor a µg/L	x10 ³ cells/Litre
1-Mar-78	Blue-green scum	Middle Swan Bridge	-	
4-May-78	Microalgal bloom	East Perth Waters	-	
19-Oct-78	<i>Chlamydominantonas</i>	U/s Sandy Beach	-	
1-May-79	<i>Cryptomonas</i>	Hill St Perth Water	20-160	3,100-340,000
16-Oct-79	<i>Chlamydominantonas</i>	Success Hill	687	1,060,000
22-Oct-80	<i>Skeletonema</i> /Mixed	Como Jetty	26	3,400
8-23 Oct-80	<i>Chlamydominantonas</i>	Causeway	100-2310	
15-Apr-81	<i>Peridinium</i>	Barrack St Jetty	840	
30-Apr-81	<i>Gonyaulax</i>	Barkers Bridge	81	9,400
2-Nov-81	<i>Microcystis littoralis</i>	Riverton Bridge		Green scum
11-Nov-81	<i>Chlamydominantonas</i>	Ascot-Guildford	76-160	46,000-65,000
11-Nov-81	<i>Euglena</i> / <i>Peridinium</i>	Maylands	21	4,700
23-Dec-81	<i>Euglena</i> scum	Nicholson Bridge u/s	-	
23-Dec-81	<i>Nodularia</i>	West Midland Pool	-	Scum visible
10-Feb-82	<i>Skeletonema</i> <i>Chlamydominantonas</i> <i>Glenodinium</i>	Barkers Bridge	20	19,000
10-Feb-82	<i>Skeletonema</i>	Hill St Perth Water	40	17,000
10-Feb-82	<i>Euglena</i> / <i>Glenodinium</i> / Dinoflagellates	Forbes St	300	37,000
10-Feb-82	<i>Chlamydominantonas</i>	Barkers Bridge	2500	
4-May-82	<i>Cryptomonas</i>	U/s Nicholson Rd Bridge	2600	
19-May-82	<i>Crypt</i> / <i>Gleno</i> / <i>Gonyaul</i>	Kingsley St	64	2,750
19-May-82	<i>Crypt</i> / <i>Gyrodinium</i>	Forbes St	30	2,300
27-Oct-82	<i>Chlamydominantonas</i>	Ascot-Maylands	150-950	
20-Dec-82	<i>Euglena</i>	Eden Hill-Guildford	61-1000	

Month	Species	Location	Chlor a µg/L	x10 ³ cells/Litre
12-Jan-83	<i>Euglena/Skeletonema</i>	Barkers Bridge	24	3,900
12-Jan-83	<i>Perid/Mixed</i>	Forbes St	47	2,900
28-Mar-83	<i>Cryptomonas</i>	Forbes St	50	
27-Apr-83	<i>Pyramimonas</i>	Kent St Weir	48	
20-Apr-83	<i>Cryptomonas</i>	Barkers Bridge	150	18,000
17-May-83	<i>Cryptomonas</i>	Barkers Bridge	900	61,000
26-Oct-83	<i>Heterosigma akashiwo</i>	Hill St Perth Water	1000	
1-Nov-83	<i>Chlamydomontonas</i>	Success Hill	630	787,400
1-Oct-88	<i>Prorocentrum minimum</i>	Perth Water-Melville Water	-	9,286-25,500
1-Oct-88	<i>Prorocentrum minimum</i>	Pelican Point U/S	-	25,077
1-Oct-88	<i>Prorocentrum minimum</i>	Barrack St Jetty	-	31,300
1-Nov-88	<i>Chlamydomontonas</i>	Success Hill	-	515,500-1,083,000
10-Nov-88	<i>Chlamydomontonas</i>	Forbes St	-	247,600
14-Nov-88	<i>Chlamydomontonas</i>	White Rocks	-	60,600
1-Jan-89	<i>Scrippsiella</i>	Forbes-Sandy Beach	190-300	26,000-41,000
2-Feb-89	<i>Scripps/Heterosigma Eutrept/Cryptomonas</i>	Milne St	-	15,100
14-Feb-89	<i>Scripps/Heterosigma Cryptomonas</i>	Forbes St	-	70,800
14-Feb-89	<i>Scrippsiella and Heterosigma</i>	White Rocks-Bassendean	-	16,200-77,000
21-Apr-89	<i>Heterosigma akashiwo</i>	Causeway	-	142,550
26-Apr-89	<i>Eutreptiella/Skelet</i>	Causeway	81	38,000
26-Apr-89	<i>Heterosigma akashiwo/DF's</i>	Kingsley St	72	35,300
1-Nov-89	<i>Chlamydomontonas</i>	Milne St	-	247,000
21-Nov-89	<i>Skeletonema costatum</i>	Maylands	40	182,00
23-Jan-90	cf. <i>Scrippsiella</i> (mucus-producer)	Forbes St	150	21,429
13-Feb-90	<i>Skeletonema costatum</i>	Narrows	-	22,600
8-Mar-90	<i>Hemiselmis virescens</i>	Milne St	68	276,400

Month	Species	Location	Chlor a µg/L	x10 ³ cells/Litre
8-Mar-90	Mixed	Hill St Perth Water	-	15700
19-Mar-90	<i>Crypto</i> /mixed	Barkers Bridge	48	18900
1-Apr-90	<i>Cryptomonas</i> , 66%	Maylands	48	18880
1-Apr-90	<i>Cryptomonas</i> , 98%	Maylands	340	98000
3-Apr-90	<i>Cryptomonas</i>	Rivervale-Bassendean	150-340	34,000-100,000
1-May-90	<i>Cryptomonas</i>	St Johns	260	90000
1-May-90	Unidentified Df	Salter Pt	N/A	34900
27-Nov-90	<i>Eugl/Chlamy/Skelet</i>	White Rocks	-	21286
27-Nov-90	<i>Euglena</i>	Maylands Pool	-	10230
31-Jan-92	<i>P. minimum</i> <i>/Scripp/Cylind/Nitz</i>	St Johns	-	18200
4-Feb-92	Mixed Div	Ascot-White Rocks	65-110	24,700-27,400
4-Feb-92	Mixed Div	Hill St Perth Water	-	36600
4-Feb-92	Mixed Div Diats dominant	St Johns	30	34900
4-Feb-92	Mixed Div Diats dominant	Maylands Pool	52	50400
4-Feb-92	Mixed Diatoms	Milne St	66	18700
4-Feb-92	<i>P min/Eutrept/Crypt</i>	Success Hill	62	25444
4-Feb-92	<i>P min/Skeletonema</i>	RMPark	28	9400
4-Feb-92	<i>Prorocentrum</i> <i>minimum/Eutreptiella</i>	Barkers Bridge	78	27200
7-Feb-92	Mixed Div Diats dominant	Forbes St	10	36800
7-Feb-92	<i>Prorocentrum minimum</i>	Caversham Jetty	-	29000
12-Feb-92	Mixed Div with <i>Cryptomonas</i> dominant in't	Causeway-Maylands	32-43	12900-14,200
20-Feb-92	Mixed divisions- <i>P.</i> <i>mininum</i> dominant in't	Forbes St	81	93300
20-Feb-92	Mixed divisions <i>P min</i> & /Diats dominant in't	Rivervale-Bayswater	43-110	15,300-70,100
26-Feb-92	Mixed Div & <i>Hetero</i>	Eden Hill-Guild'fd	77	48150-49,000
26-Feb-92	Mixed Div <i>P min</i> dominant	Kingsley St	44	61400
26-Feb-92	Mixed divisions <i>P</i> <i>minimum</i> & Chlorophytes dominant	White Rocks	130	59800
26-Feb-92	Mixed Div <i>P min</i> /Diat dominant	Forbes St	82	58344

Month	Species	Location 1	Chlor a	x10 ³ cells/Litre
5-Mar-92	Mixed Div Diat dominant	Causeway-Rivervale	12-29	37500-220,950
5-Mar-92	Mixed Div Diat dominant	Maylands-Bassendean	25-32	233,600-482,050
5-Mar-92	Mixed Div <i>P min</i> /Diat dominant	Kingsley St	130	90900
5-Mar-92	Mixed Divisions	Eden Hill-Guildford	26-34	13,700-40,600
15-Mar-92	Mixed Divisions Diat /DF dominant	Causeway-Bayswater	-	119,150-248,200
15-Mar-92	Mixed Div Diat/DF dominant	Forbes St	-	148,900
15-Mar-92	Mixed Div <i>P min</i> /Diat dominant	Bassendean-Eden Hill-Guildford	-	23,750-100,650
30-Mar-92	Mixed Div Diat /Crypt dominant	Barkers Bridge	-	16,150
30-Mar-92	Mixed Div Diat /Crypt dominant	Kingsley St	-	91,460
30-Mar-92	Mixed Div Diat /DF dominant	White Rocks	-	170,600
30-Mar-92	Mixed Div Diat dominant	Causeway-Success Hill	-	50,900-280,600
14-Oct-92	<i>Scrippsiella</i>	Hill St Perth Water	-	500,000
29-Oct-92	<i>Chlamydomonantonas</i>	Maylands Pool	12-17	23,630-37,000
30-Oct-92	<i>Chlamydomonantonas</i>	Success Hill	-	3,315,200
26-Nov-92	Diatoms	White Rocks-Bassendean	36	25,840-61,950
10-Dec-92	DF/ <i>Cryptomonas</i>	Forbes St	19	29,581
7-Mar-93	DF/Diatoms	Ascot-Bassendean	-	28,988-29,600
18-Mar-93	Dinoflagellates	Ascot-Bassendean	-	23,154-54,500
17-May-93	<i>Prorocentrum dentatum</i>	Hill St Perth Water	-	300,000
Jan-Mar 1994	<i>Anabaena spiroides</i>	u/s Kent St Weir	-	20,000-480,000
Feb-Apr 1994	<i>Anabaena circinalis</i>	u/s Kent St Weir	-	10,000-2,000,000
Feb-Mar 1994	<i>Microcystis</i>	u/s Kent St Weir	-	10,000-20,000
Feb-Mar 1994	<i>Gymnodinium simplex</i>	White Rocks-Midland	-	10,000-1,600,000
18 Jan 1994	<i>Eutreptiella</i>	Avon-Katrine Bridge	-	36,000

Month	Species	Location 1	Chlor a µg/L	x10 ³ cells/Litre
18 Jan 1994	<i>Katodinium</i>	Glen-Avon Pool	-	10,000
18 Jan 1994	<i>Nitzschia closterium</i>	Avon D/S Northam	-	143,800
18 Jan 1994	<i>Chaetoceros</i>	Avon D/S Northam	-	70,000
19 Apr 1994	Dinoflagellates	Maylands	-	640,000
Jan 1989	Blowfish kill	Pt Walter	-	
1-Feb-83	Boney Herring kill	Midland Pool	-	
31-Jan-92	Fish Kill	Maylands to Guildford	-	
10-May-93	Fish Kill	Barkers Bridge	-	
25-28 April 1994	Cobbler, Flathead, shrimp deaths	Bayswater-Ascot	-	

Appendix 3 Harmful algal blooms and red tides (>circa 9,000/mL) in the Mandurah area (excluding the Peel-Harvey estuary).

Species	Type	Date of occurrence	Max Density x10 ³ cells/Litre	Location
<i>Cyclotella atomus</i>	Diatoms	July 1990	200,500	Yunderup Canals
<i>Prorocentrum minimum</i>	Dinoflagellates	October 1989	74,000	Yunderup Canals
<i>Rhizosolenia minima</i>	Diatoms	February 1989	92,000	Yunderup Canals
<i>Ocillatoria</i>	Blue-green	February 1989	Scum	Cox Bay
<i>Chaetoceros</i>	Diatoms	August 1989	11,000	Yunderup Canals
<i>Skeletonema costatum</i>	Diatoms	March 1989	171,000	Yunderup Canals
unidentified	Dinoflagellates	February 1989	9,500	Yunderup Canals
<i>Chaetoceros</i>	Diatoms	June 1984	42,000	Yunderup Canals
<i>Chaetoceros radians</i> , <i>Chaetoceros perpusillum</i>	Diatoms	July 1984	44,000	Yunderup Canals
<i>Chaetoceros socialis</i>	Diatoms	Sept 1984	23,000	Yunderup Canals
<i>Chaetoceros radians</i> , <i>Chaetoceros socialis</i>	Diatoms	Oct 1984	26,000	Yunderup Canals
<i>Chaetoceros socialis</i>	Diatoms	Nov 1984	21,000	Yunderup Canals
<i>Skeletonema costatum</i>	Diatoms	Des 1984	27,000	Yunderup Canals
<i>Chaetoceros</i>	Diatoms	Oct 1985	9,000	Yunderup Canals
<i>Scrippsiella</i> , <i>Oxyrrhis marina</i>	Dinoflagellates	Jan 1986	12,000	Yunderup Canals
<i>Chaetoceros gracile</i>	Diatoms	Feb 1986	145,000	Yunderup Canals
mixed diatoms	Diatoms	March 1989	171,000	Yunderup Canals
<i>Cyclotella atomus</i>	Diatoms	Sep 1984	270,000	Yunderup Canals
<i>Chaetoceros socialis</i>	Diatoms	Oct 1983	17,800	Yunderup Canals
<i>Enciculifera</i>	Dinoflagellate	Nov 1983	500	Yunderup Canals
<i>Trichodesmium</i>	Blue-green	April 1994	Red Stain	Sticks Channel
<i>Synechococcus</i>	Blue-green	Feb-July 1991	36,000,000	Mandurah
<i>Nodularia</i>	Blue-green	Feb 1990-94	9,000,000	Serpentine
<i>Anabaena</i> and <i>Microcystis</i>	Blue-greens	Jan 1992& 93		Serpentine
<i>Prorocentrum minimum</i>	Dinoflagellate	1989	300,000	Peel-Estuary Canals
<i>Cricosphaera</i>	Prymnesiophyte	May 1993	540,000	Murray River Canal
Centric Diatoms		June 1992	91,950	Murray River

Appendix 4

 Harmful algal blooms and red tides (> circa 9,000/mL) excluding *Nodularia spumigena* in the Peel-Harvey estuary 1983-85 (based on individual site data).

Species	Type	Date of occurrence	Max Density 000's Litre	Estuary
<i>Cerataulina pelagica</i>	Diatoms	June-1983	11,000-18000	Harvey
<i>Cerataulina pelagica</i>	Diatoms	June-1983	8,500-10,000	Peel
<i>Prorocentrum minimum</i>	Dinoflagellates	Jun 1983	14,500	Harvey
<i>Prorocentrum minimum</i>	Dinoflagellates	Jul 1983	9,000-14,000	Peel
<i>Cyclotella atomus</i>	Diatoms	Aug 1983	50,000	Harvey
<i>Prorocentrum minimum</i>	Dinoflagellates	Aug-Sep 1983	9,700-23,000	Peel
<i>Prorocentrum minimum</i>	Dinoflagellates	Aug 1983	11,500-13,800	Harvey
<i>Cyclotella atomus</i>	Diatoms	Aug 1983	14,000-17,000	Harvey
<i>Cyclotella atomus</i> , <i>Skeletonema potamos</i>	Diatoms	Sep 1983	9,000-13,000	Harvey
<i>Chaetoceros</i>	Diatoms	Nov 1983	24,000-50,000	Harvey
<i>Chaetoceros</i>	Diatoms	Nov 1983	54,000	Peel
<i>Asterionella glacialis</i>	Diatoms	Nov 1983	24,600	Harvey
<i>Asterionella glacialis</i>	Diatoms	Nov 1983	10,700-39,600	Peel
<i>Chaetoceros</i>	Diatoms	Jan 1984	10,500-32,000	Harvey
<i>Chaetoceros perpusillum</i> / <i>Chaetoceros. radians</i>	Diatoms	Jul 1984	9,000	Harvey
<i>Chaetoceros perpusillum</i> / <i>Chaetoceros. radians</i>	Diatoms	Jul 1984	10,000	Peel
<i>Chaetoceros radians</i>	Diatoms	Jul 1984	15,500-27,000	Harvey
<i>Chaetoceros radians</i>	Diatoms	Jul 1984	16,000-86,300	Peel
<i>Chaetoceros radians</i>	Diatoms	Aug 1984	19,500-52,000	Harvey
<i>Cerataulina pelagica</i>	Diatoms	Nov-1984	13,500	Harvey
<i>Cerataulina pelagica</i>	Diatoms	Nov-1984	10,500-44,500	Peel
<i>Cerataulina pelagica</i>	Diatoms	Jan-1985	35,700-53,000	Peel
<i>Chaetoceros radians</i>	Diatoms	Jan 1985	10,500	Peel
<i>Chaetoceros</i>	Diatoms	Jan 1985	15,200-15,800	Harvey
<i>Cerataulina pelagica</i>	Diatoms	Jan-1985	9,200-16,000	Harvey
<i>Nitzschia closterium</i>	Diatoms	Jan-1985	18,500-21,000	Harvey
<i>Entomoneis</i>	Diatoms	Jan-1985	5,500-12,400	Harvey

Appendix 5 Harmful algal blooms and red tides (>circa 10,000/mL) excluding *Nodularia spumigena* in the Peel-Harvey estuary 1985-1992, based and attenuated by being the composite mean integrated densities of 3 sites in each estuary:

Species	Type	Date of occurrence	Max Density x10 ³ cells/Litre	Estuary
<i>Asterionella glacialis</i>	Diatom	Jan 1985	12,500	Harvey
<i>Chaetoceros gracilis</i>	Diatom	Feb 1985	139,500	Harvey
<i>Asterionella glacialis</i>	Diatom	Nov 1985	15,500	Peel
<i>Asterionella glacialis</i>	Diatom	Dec 1985	31,000	Peel
<i>Asterionella glacialis</i>	Diatom	Dec 1986	22,500	Peel
<i>Chaetoceros socialis</i>	Diatom	Dec 1986	133,000	Harvey
<i>Chaetoceros socialis</i>	Diatom	Dec 1986	9,000	Peel
<i>Chaetoceros</i>	Diatom	Jan 1987	14,500	Harvey
<i>Chaetoceros</i>	Diatom	Jan 1987	22,500	Peel
<i>Skeletonema costatum</i>	Diatom	Sep 1987	19,500	Peel
<i>Chaetoceros radians</i>	Diatom	Sep 1987	9,000	Peel
<i>Chaetoceros socialis</i>	Diatom	Oct 1987	16,800	Harvey
<i>Chaetoceros radians</i>	Diatom	Jul 1988	57,000	Peel
<i>Chaetoceros radians</i>	Diatom	Jul 1988	9,000	Harvey
<i>Skeletonema costatum</i>	Diatom	Aug 1988	9,200	Peel
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Aug 1988	13,300-136,000	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Sep 1988	15,300-35,600	Harvey
<i>Chaetoceros</i>	Diatom	Dec 1988	20,000	Harvey
<i>Prorocentrum minimum</i>	Dinoflagellate	Aug 1989	9,200	Peel
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Aug 1989	13,000	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Sep 1989	9,000-12,000	Harvey
<i>Skeletonema costatum</i>	Diatom	Oct 1989	9,200	Peel
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Oct 1989	9,000	Harvey

Species	Type	Date of occurrence	Max Density $\times 10^3$ cells/Litre	Estuary
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Oct 1989	14,000	Peel
<i>Chaetoceros</i>	Diatom	Nov 1989	10,000	Peel
<i>Chaetoceros</i>	Diatom	Dec 1989	10,500-12,700	Peel
<i>Asterionella glacialis</i>	Diatom	Dec 1989	20,200	Peel
<i>Chaetoceros</i>	Diatom	Nov 1989	10,000	Peel
<i>Chaetoceros</i>	Diatom	Mar 1990	11,000	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Aug 1991	14,100-257,800	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Aug 1991	9,000-15,400	Peel
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Sep 1991	145,100-269,000	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Sep 1991	24,200	Peel
<i>Nitzschia</i>	Diatom	Sep 1991	14,400-54,400	Harvey
<i>Nitzschia</i>	Diatom	Sep 1991	9,000	Peel
<i>Nitzschia closterium</i>	Diatom	Dec 1991	18,700	Peel
<i>Rhizosolenia minima</i>	Diatom	Feb 1992	106,000	Harvey
<i>Oscillatoria</i>	Blue-green	Feb 1992	11,200	Harvey
<i>Cyclotella atomus/ Skeletonema potamos</i>	Diatom	Aug/Sep 1992	11,000 150,200	Harvey
<i>Cyclotella atomus / Skeletonema potamos</i>	Diatom	Aug 1992	12,000-72,000	Peel

Appendix 6 Relationships between WA stakeholders and issues of harmful phytoplankton surveillance

WA Stakeholders	Phytoplankton cyst, ballast monitoring & identification	Toxicity testing	Human health risk assessment	Aquaculture animal risk assessment	Impact minimisation strategies	Education and dissemination	Research	Co-ordination
WA Health Dept	X	X	X	-	X	X	-	X
Aquaculture industry	X	X	X	X	X	X	-	?
Fisheries Dept	X	X	-	X	X	X	-	?
Australian Quarantine Inspection Service WA	X	-	-	-	X	X	-	?
Dept of Environmental Protection	X	X	X	X	X	X	-	?
Water Authority WA		X	X	X	X	X	-	-
WA Port Authorities	X	-	-	-	X	X	-	?
WA Dept of Agriculture Animal Health	X	X	-	X	X	X	-	?
Chemistry Centre of WA	X	X	-	-	-	-	-	?
Statutory Development Authorities	X	?	X	X	X	-	-	?
Local Government Authorities	X	X	X	X	X	X	-	?
Tertiary institutions	-	-	-	-	-	X	X	?
WA Waterways Commission	X	X	X	-	X	X	-	?
Swan River Trust	X	X	X	-	X	X	-	?



Appendix 7 Relationships between WA stakeholders and issues relating to areas affected by harmful phytoplankton surveillance

WA Stakeholders	Oceans	Ports and harbours	Estuaries/Rivers	Lakes and Wetlands	Farm dams	Drinking Water Dams
WA Health Dept	X		X	X	X	X
Aquaculture industry	X		X	X	X	
WA Fisheries Dept	X	X	X	X	X	
Australian Quarantine Inspection Service WA	X	X	X	-	-	
Dept. Environmental Protection	X	X	X	X	X	
WA Port Authorities	X	X	X	-	-	
WA Dept of Agriculture Animal Health	X	X	X	X	X	
WA Water Authority	X	-	X	X	-	X
Chemistry Centre of WA	-	-	-	-	-	-
Statutory Development Authorities	X	X	X	?	-	-
Local Government Authorities	-	-	X	X	-	-
Tertiary institutions	X	X	X	X	X	X
WA Waterways Commission	X	X	X	X	-	-
Swan River Trust	X	X	X	X	-	-



PHYCOLOGICAL REVIEWS 13

A review of harmful algal blooms and their apparent global increase*

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CONTENTS

I. Introduction	80
II. Global increase of algal blooms	81
III. Increased scientific awareness of toxic species	82
Diarrhetic Shellfish Poisoning (DSP)	82
Amnesic Shellfish Poisoning (ASP)	83
IV. Increased utilization of coastal waters for aquaculture	83
V. Increase of algal blooms by cultural eutrophication	89
Ciguatera fishfood poisoning and coral reef disturbance	91
VI. Stimulation of algal blooms by unusual climatological conditions	92
Toxic <i>Pyrodinium bahamense</i> blooms in the tropical Indo-West Pacific	92
Fossil blooms of <i>Gymnodinium catenatum</i> in the Kattegat-Skagerrak	93
VII. Transport of dinoflagellate cysts in ships' ballast water or associated with the transfer of shellfish stocks	94

* Text of a plenary lecture presented at the Fourth International Phycological Congress, Duke University, North Carolina, USA, 5-9 August 1991, updated with new algal bloom reports which have become available during the Fifth International Conference on Toxic Marine Phytoplankton, Newport, Rhode Island, USA, 28 October-1 November 1991.

VIII. Conclusions	96
IX. Acknowledgements	97
X. References	97

I. INTRODUCTION

The microscopic planktonic algae of the world's oceans are critical food for filter-feeding bivalve shellfish (oysters, mussels, scallops, clams) as well as the larvae of commercially important crustaceans and finfish. In most cases, the proliferation of plankton algae (so-called 'algal blooms'; up to millions of cells per litre) is therefore beneficial for aquaculture and wild fisheries operations. However, in some situations algal blooms can have a negative effect, causing severe economic losses to aquaculture, fisheries and tourism operations and having major environmental and human health impacts. Among the 5000 species of extant marine phytoplankton, some 300 species can at times occur in such high numbers that they obviously discolour the surface of the sea (so-called 'red tides'),

Table 1. Different types of harmful algal blooms

- (1) Species which produce basically harmless water discolorations; however, under exceptional conditions in sheltered bays, blooms can grow so dense that they cause indiscriminate kills of fish and invertebrates due to oxygen depletion. Examples: dinoflagellates *Gonyaulax polygramma* Stein, *Noctiluca scintillans* (Macartney) Ehrenberg, *Scrippsiella trochoidea* (Stein) Loeblich III, cyanobacterium *Trichodesmium erythraeum* Ehrenberg.
- (2) Species which produce potent toxins that can find their way through the food chain to humans, causing a variety of gastrointestinal and neurological illnesses, such as:
 - Paralytic Shellfish Poisoning (PSP)
(Examples: dinoflagellates *Alexandrium acatenella* (Whedon et Kofoed) Balech, *A. catenella* (Whedon et Kofoed) Balech, *A. cohorticula* (Balech) Balech, *A. fundyense* Balech, *A. fraterculus* (Balech) Balech, *A. minutum* Halim, *A. tamarense* (Lebour) Balech, *Gymnodinium catenatum* Graham, *Pyrodinium bahamense* var. *compressum* (Böhm) Steidinger, Tester et Taylor)
 - Diarrhetic Shellfish Poisoning (DSP)
(Examples: dinoflagellates *Dinophysis acuta* Ehrenberg, *D. acuminata* Claparède et Lachmann, *D. fortii* Pavillard, *D. norvegica* Claparède et Lachmann, *D. mitra* (Schütt) Abé vel Balech, *D. rotundata* Claparède et Lachmann, *Prorocentrum lima* (Ehrenberg) Dodge)
 - Amnesic Shellfish Poisoning (ASP)
(Examples: diatoms *Nitzschia pungens* f. *multiseries* Hasle, *N. pseudodelicatissima* Hasle, *N. pseudoseriata* Hasle)
 - Ciguatera Fishfood Poisoning
(Examples: dinoflagellate *Gambierdiscus toxicus* Adachi et Fukuyo, ?*Ostreopsis* spp., ?*Prorocentrum* spp.)
 - Neurotoxic Shellfish Poisoning (NSP)
(Example: dinoflagellate *Gymnodinium breve* Davis)
 - Cyanobacterial Toxin Poisoning
(Examples: cyanobacteria *Anabaena flos-aquae* Brébisson ex Bornet et Flahaut, *Microcystis aeruginosa* Kützing, *Nodularia spumigena* Mertens ex Bornet et Flahaut)
- (3) Species which are non-toxic to humans, but harmful to fish and invertebrates (especially in intensive aquaculture systems) by damaging or clogging their gills. Examples: diatom *Chaetoceros convolutus* Castracane, dinoflagellate *Gymnodinium mikimotoi* Miyake et Kominami ex Oda, prymnesiophytes *Chrysochromulina polylepsis* Manton et Parke, *C. leadbeateri* Estep, Davis, Hargraves et Sieburth, *Prymnesium parvum* Carter, *P. patelliferum* Green, Hibberd et Pienaar, raphidophytes *Heterosigma akashiwo* (Hada) Hada ex Hara et Chihara, *Chattonella antiqua* (Hada) Ono.

while only 40 or so species have the capacity to produce potent toxins that can find their way through fish and shellfish to humans (Table 1).

It is believed that the first written reference (1000 years BC) to a harmful algal bloom appears in the Bible: '... all the waters that were in the river were turned to blood. And the fish that was in the river died; and the river stank, and the Egyptians could not drink of the water of the river' (Exodus 7: 20–21). In this case, a non-toxic bloom-forming alga became so densely concentrated that it generated anoxic conditions resulting in indiscriminate kills of both fish and invertebrates. Oxygen depletion can be due to high respiration by the algae (at night or in dim light during the day) but more commonly is caused by bacterial respiration during decay of the bloom.

One of the first recorded fatal cases of human poisoning after eating shellfish contaminated with dinoflagellate toxins happened in 1793, when Captain George Vancouver and his crew landed in British Columbia in an area now known as Poison Cove. He noted that for local Indian tribes it was taboo to eat shellfish when the seawater became phosphorescent due to dinoflagellate blooms (Dale & Yentsch 1978). The causative alkaloid toxins, now called paralytic shellfish poisons (PSP) (Fig. 1a), are so potent that a pinhead-size quantity (about 500 µg), which can easily accumulate in just one 100 g serving of shellfish, could be fatal to humans. On a global scale, close to 2000 cases of human poisoning (15% mortality) through fish or shellfish consumption are reported each year and, if not controlled, the economic damage through reduced local consumption and reduced export of seafood products can be considerable. Whales and porpoises can also become victims when they receive toxins through the food chain via contaminated zooplankton or fish (Geraci *et al.* 1989). Poisoning of manatees by dinoflagellate brevetoxins contained in tunicates attached to seagrass (in Florida; Steidinger, personal communication), and of pelicans by diatom domoic acid contained in anchovies have also been reported (in California; Work *et al.* 1991).

The third type of harmful algal bloom has become apparent only as a result of our increased interest in intensive aquaculture systems for finfish. Some algal species can seriously damage fish gills, either mechanically or through production of hemolytic substances. While wild fish stocks have the freedom to swim away from problem areas, caged fish appear to be extremely vulnerable to such noxious algal blooms. In 1972 in Japan, a bloom of the raphidophyte flagellate *Chattonella antiqua* thus killed 500 million dollars worth of caged yellow-tail fish in the Seto Island Sea (Okaichi 1989).

Table 1 summarizes the above three different types of harmful algal bloom problems, together with representative examples of causative algal species, ranging from dinoflagellates, diatoms, prymnesiophytes and raphidophytes to cyanobacteria. Clinical symptoms of various types of fish and shellfish poisoning are listed in Table 2 and the diversity of chemical structures of algal toxins is illustrated in Fig. 1. Unfortunately, there is no clear-cut correlation between algal concentrations and their potential harmful effects. Dinoflagellate species such

Table 2. Clinical symptoms of various types of fish and shellfish poisoning

Paralytic Shellfish Poisoning (PSP)	Diarrhetic Shellfish Poisoning (DSP)	Amnesic Shellfish Poisoning (ASP)	Ciguatera
Causative organism <i>Alexandrium catenella</i> ; <i>Alexandrium minutum</i> ; <i>Alexandrium tamarense</i> ; <i>Gymnodinium catenatum</i> ; <i>Pyrodinium bahamense</i>	<i>Dinophysis acuminata</i> ; <i>Dinophysis fortii</i>	<i>Nitzschia pungens</i> f. <i>multiseriis</i> ; <i>Nitzschia pseudodelicatissima</i> ; <i>Nitzschia pseudoseriata</i>	<i>Gambierdiscus toxicus</i> ; ? <i>Ostreopsis siamensis</i> J. Schmidt; ? <i>Prorocentrum lima</i>
Symptoms <i>Mild Case</i> Within 30 min: tingling sensation or numbness around lips, gradually spreading to face and neck; prickly sensation in fingertips and toes; headache, dizziness, nausea, vomiting, diarrhoea <i>Extreme Case</i> Muscular paralysis; pronounced respiratory difficulty; choking sensation; death through respiratory paralysis may occur within 2–24 h of ingestion	After 30 min to a few h (seldom more than 12 h): diarrhoea, nausea, vomiting, abdominal pain Chronic exposure may promote tumour formation in the digestive system	After 3–5 h: nausea, vomiting, diarrhoea, abdominal cramps Decreased reaction to deep pain; dizziness, hallucinations, confusion; short-term memory loss; seizures	Symptoms develop within 12–24 h of eating fish. Gastrointestinal symptoms: diarrhoea, abdominal pain, nausea, vomiting Neurological symptoms: numbness and tingling of hands and feet; cold objects feel hot to touch; difficulty in balance; low heart rate and blood pressure; rashes. In extreme cases, death through respiratory failure
Treatment Patient has stomach pumped and is given artificial respiration. No lasting effects	Recovery after 3 d, irrespective of medical treatment		No antitoxin or specific treatment is available. Neurological symptoms may last for months and years. Calcium and mannitol may help relieve symptoms

as *Dinophysis* and *Alexandrium* can contaminate shellfish with toxins, even at very low cell concentrations. The prymnesiophyte *Chrysochromulina* produces only moderate biomass levels but has a very high toxic potency. Finally, the prymnesiophyte *Phaeocystis* is non-toxic but its nuisance value is caused by very high biomass levels.

II. GLOBAL INCREASE OF ALGAL BLOOMS

While harmful algal blooms, in a strict sense, are completely natural phenomena which have occurred throughout recorded history, in the past two decades the public health and economic impacts of such events appear to have increased in frequency, intensity and geographic distribution. One example, the increased global distribution of paralytic shellfish poisoning, is illustrated in Fig. 2. Until 1970, PSP-producing dinoflagellate blooms (of *Alexandrium (Gonyaulax) tamarense* and *Alexandrium (Gonyaulax) catenella*) were only known from temperate waters of Europe, North America and Japan (Dale & Yentsch 1978). By 1990, PSP was well documented from throughout the Southern Hemisphere, in South Africa, Australia, India, Thailand, Brunei, Sabah, the Philippines and Papua New Guinea. Other species of the dinoflagellate genus *Alexandrium*, such as *A. cohorticula* and *A. minutum*, as well as the unrelated dinoflagellates *Gymnodinium catenatum* and *Pyrodinium bahamense* var. *compressum*, have now also been

implicated (Fig. 3). On a smaller, regional scale, between 1978 and 1982 the number of areas affected by PSP in Japan increased from 2 to 10 (Fig. 4; Anraku 1984). Unfortunately, there are very few long-term records of algal blooms at any single locality. Probably the best dataset refers to the concentration of PSP toxins (μg saxitoxin equivalent/100 g shellfish meat) in Bay of Fundy clams, which has been monitored by mouse bioassay since 1944 (White 1987). Shellfish containing more than 80 μg PSP/100 g shellfish meat are considered unfit for human consumption. Figure 5 shows some tendency for an increased frequency of dinoflagellate blooms at this site in the 1970s and early 1980s, but no major outbreaks have occurred since then.

The issue of a global increase in harmful algal blooms has been raised previously in specialized conference proceedings only (Anderson 1989; Hallegraeff *et al.* 1990; Smayda 1990), while the biology of toxic dinoflagellates has been reviewed by Steidinger (1983) and the impacts of algal blooms on aquaculture have been summarized by Shumway (1990). In the present review, four explanations for this apparent increase of algal blooms will be explored: increased scientific awareness of toxic species; increased utilization of coastal waters for aquaculture; stimulation of plankton blooms by cultural eutrophication and/or unusual climatological conditions; and transport of dinoflagellate resting cysts either in ships' ballast water or associated with movement of shellfish stocks from one area to another.

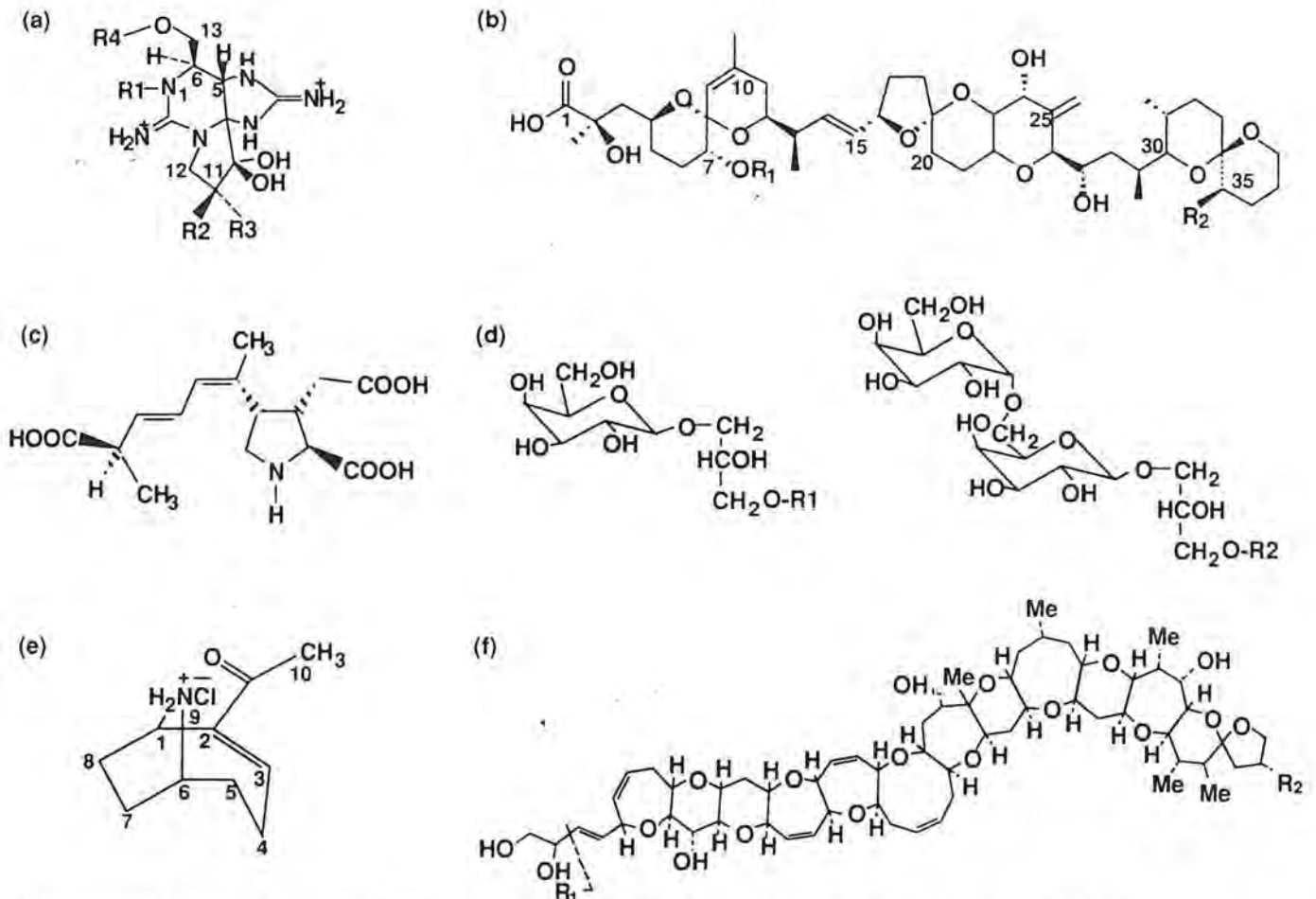


Fig. 1. Diversity of chemical structures of algal toxins. (a) Paralytic shellfish poisons from the dinoflagellates *Alexandrium* spp., *Gymnodinium catenatum* and *Pyrodinium bahamense*, $R_1 = \text{H}$ or OH , $R_2 = \text{H}$ or OSO_3^- , $R_3 = \text{H}$ or OSO_3^- , $R_4 = \text{CONH}_2$ (carbamate toxins), CONHSO_3^- (sulfamate toxins) or H (decarbamoyl toxins). (b) Diarrhetic shellfish poisons from the dinoflagellates *Dinophysis* spp. or *Prorocentrum lima*; $R_1 = \text{H}$, $R_2 = \text{H}$ (okadaic acid) or CH_3 (dinophysin toxin-1). (c) Domoic acid from the diatom *Nitzschia pungens* f. *multiseriata*. (d) Hemolysins from the dinoflagellates *Amphidinium carterae* Hulbert, *Gyrodinium aureolum* and prymnesiophyte *Chrysochromulina polyylepis*; $R_1 = \text{acyl}$ (C_{18-4w3}); $R_2 = \text{acyl}$ (C_{18-3} , C_{20-3}) or acyl (C_{20-3}). (e) Anatoxin-a from the cyanobacterium *Anabaena flos-aquae*. (f) Ciguatera toxin from the dinoflagellate *Gambierdiscus toxicus*; $R_1 = \text{CH}_2 = \text{CH}-$; $R_2 = \text{H}$ (*Gambierdiscus*) or OH (moray eels).

III. INCREASED SCIENTIFIC AWARENESS OF TOXIC SPECIES

Reports of harmful algal blooms, associated human illnesses or damage to aquaculture operations are receiving increased attention in newspapers, the electronic media and the scientific literature. Consequently, more and more researchers are now surveying their local waters for the causative organisms. Increased reports on the occurrence of dinoflagellates of the genus *Alexandrium* are a good example. Until 1988, the type species *A. minutum* was only known from Egypt (Halim 1960); it has now been reported from Australia, France, Spain, Portugal, Italy, Turkey and the east coast of North America (Hallegraeff *et al.* 1991). Other examples are in the recent description of the newly recognized phenomena of Diarrhetic Shellfish Poisoning (since 1976) and Amnesic Shellfish Poisoning (since 1987).

Diarrhetic Shellfish Poisoning (DSP)

This phenomenon was first documented in 1976 from Japan where it caused major problems for the scallop fishery (Yasu-

moto *et al.* 1978). The first dinoflagellate to be implicated was *Dinophysis fortii* (in Japan), soon followed by *D. acuminata* (in Europe) (Figs 6A, B), *D. acuta*, *D. norvegica* (in Scandinavia), *D. mitra*, *D. rotundata* and the benthic dinoflagellate *Prorocentrum lima*. The recent detection of DSP toxins in the heterotrophic dinoflagellates *Protoperdinium oceanicum* (Vanhoffen) Balech and *P. pellucidum* Bergh (Lee *et al.* 1991) may reflect their feeding on *Dinophysis*. Between 1976 and 1982, some 1300 DSP cases were reported in Japan, in 1981 more than 5000 cases were reported in Spain, and in 1983 some 3300 cases were reported in France. In 1984 in Sweden, DSP problems caused a shutdown of the mussel industry for almost a year. The clinical symptoms of DSP (Table 2) may often have been mistaken for those of bacterial gastric infections and the problem may be much more widespread and serious than previously thought. Unlike PSP, no human fatalities have ever been reported and patients usually recover within three days. However, some of the polyether toxins involved (okadaic acid, dinophysin toxin-1; Fig. 1b) may promote stomach tumours (Suganuma *et al.* 1988) and thus produce chronic problems in shellfish consumers. Shellfish

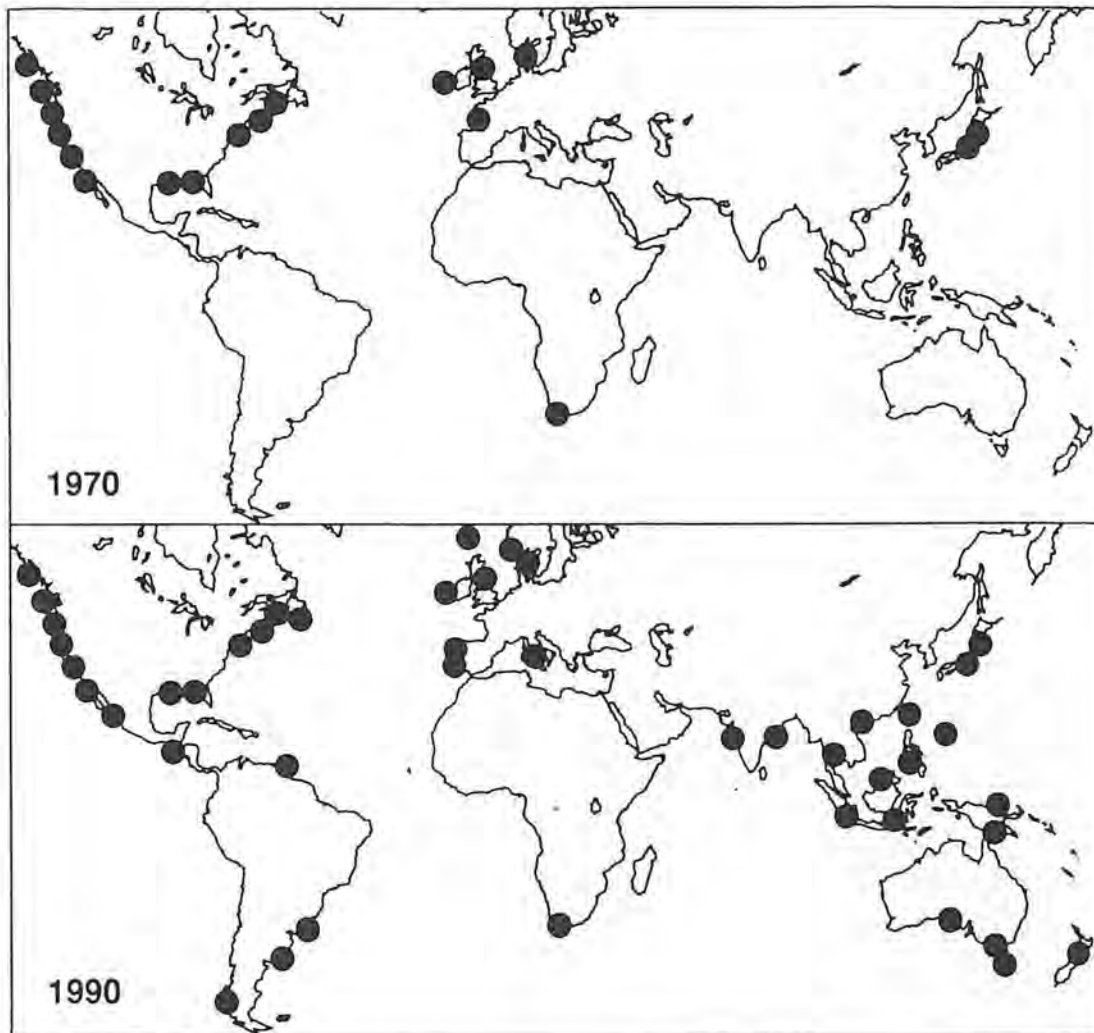


Fig. 2. Known global distribution of paralytic shellfish poisoning (PSP) in 1970 and 1990.

containing more than $2 \mu\text{g}$ okadaic acid and/or $1.8 \mu\text{g}$ dinophys toxin-1 per gram of hepatopancreas are considered unfit for human consumption (Lee *et al.* 1987). The known global distribution of DSP (Fig. 7) includes Japan, Europe, Chile, Thailand, Nova Scotia and possibly Tasmania (Australia) and New Zealand.

Amnesic Shellfish Poisoning (ASP)

This phenomenon was first recognized in 1987 in Prince Edward Island, Canada, where it caused 3 deaths and 105 cases of acute human poisoning following the consumption of blue mussels (Bates *et al.* 1989). The symptoms (Table 2) include abdominal cramps, vomiting, disorientation and memory loss (amnesia). Most unexpectedly, the causative toxin (the excitatory amino acid domoic acid; Fig. 1c) is produced by a diatom and not by a dinoflagellate. Shellfish containing more than $20 \mu\text{g}$ domoic acid per gram of shellfish meat are considered unfit for human consumption. To date, *Nitzschia pungens* f. *multiseriata*, *N. pseudodelicatissima* (Fig. 6C) and *N. pseudoseriata* (*Pseudonitzschia australis* Frenguelli) have been implicated (Subba Rao *et al.* 1988; Martin *et al.* 1990; Work *et al.* 1991). These diatoms produce toxins only after the onset

of the stationary growth phase, induced by either phosphate or silicate deficiency (Bates *et al.* 1991). Their known global distribution is shown in Fig. 8. Again, the problem may be much more widespread than is currently recognized.

IV. INCREASED UTILIZATION OF COASTAL WATERS FOR AQUACULTURE

With increased problems of overfishing of coastal waters, more and more countries are looking towards aquaculture as an alternative. Indeed, some fisheries scientists predict that within the next 10–20 years, the increasing value of world aquaculture production may well approach the decreasing value of the total catch of wild fish and shellfish. Aquaculture operations act as sensitive 'bioassay systems' for harmful algal species and can bring to light the presence in water bodies of problem organisms not known to exist there before. The increase in shellfish farming worldwide is leading to more reports of paralytic, diarrhetic and amnesic shellfish poisoning. On the other hand, increased finfish culture is drawing attention to algal species which can cause damage to the fishes' delicate gill tissues.

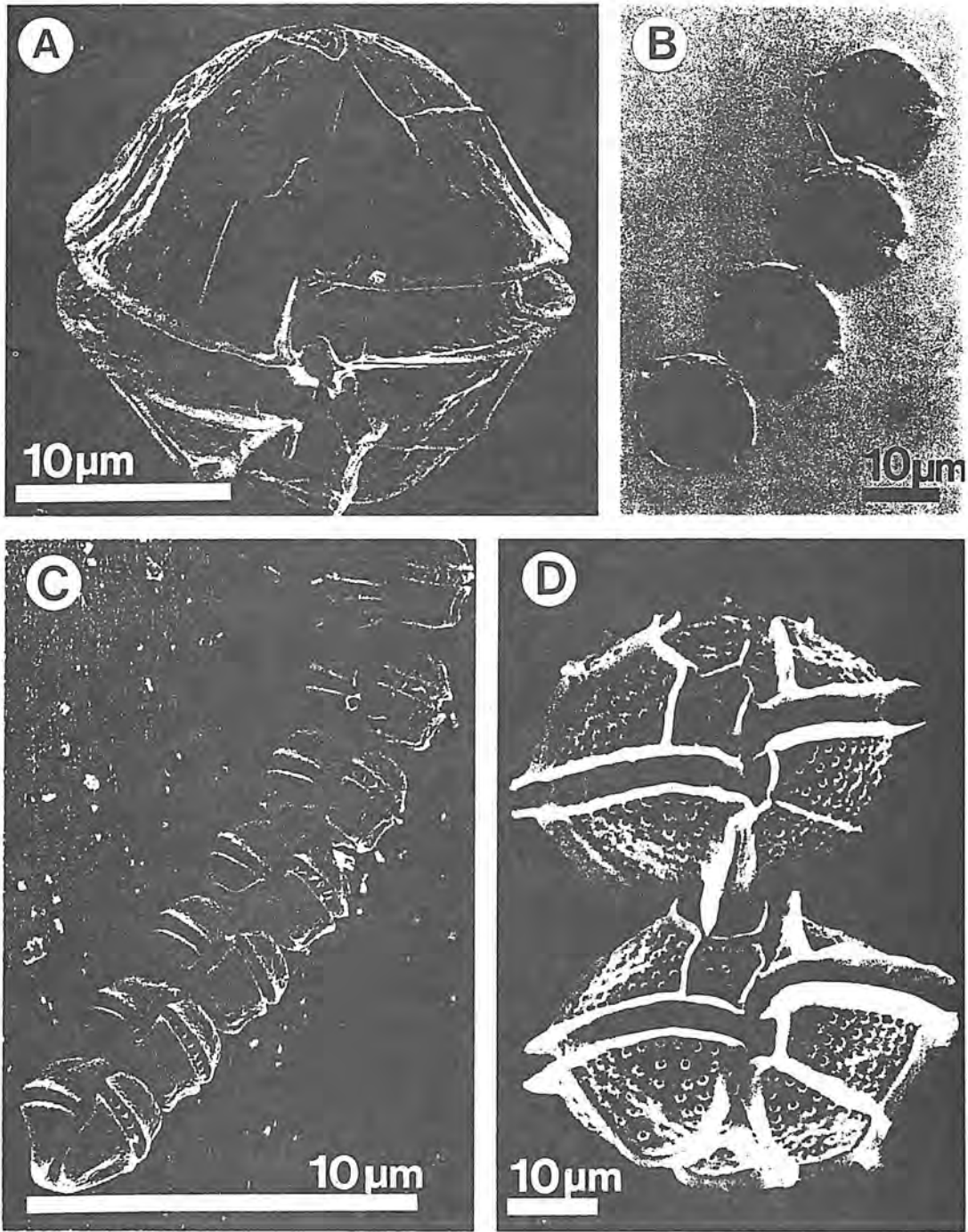


Fig. 3. Causative organisms of paralytic shellfish poisoning. (A) SEM, *Alexandrium minutum* from South Australia. (B) LM, *Alexandrium catenella* from Australia. (C) SEM, *Gymnodinium catenatum* from Tasmania, Australia. (D) SEM, *Pyrodinium bahamense* var. *compressum* from Papua New Guinea (Dinophyceae).

In fish pens in British Columbia, deaths of lingcod, sockeye, coho, chinook and pink salmon have been caused by dense concentrations (5000 cells per litre) of the diatoms *Chaetoceros convolutus* and *C. concavicornis* Mangin (Fig. 9A). The diatoms' long hollow spines (setae) are studded with smaller barbs along their length. The setae can break off and penetrate the gill membranes of fish, with the smaller barbs preventing them from coming out. Fish death may be caused by capillary hemorrhage, dysfunction of gas exchange at the gills, suffocation

from an overproduction of mucus, or even from secondary infection of the damaged tissue (Bell 1961; Rensel 1991).

A more widespread problem for fish farmers is the production by various algal groups of fatty acids or galactolipids which damage the epithelial tissues of the gills. In experimental assay systems these substances destroy red blood cells and therefore have been provisionally termed 'hemolysins' (Fig. 1d). Algal species as diverse as the raphidophytes *Heterosigma akashiwo* (Fig. 9C) and *Chattonella antiqua*, the prymnesiophytes *Chryso-*

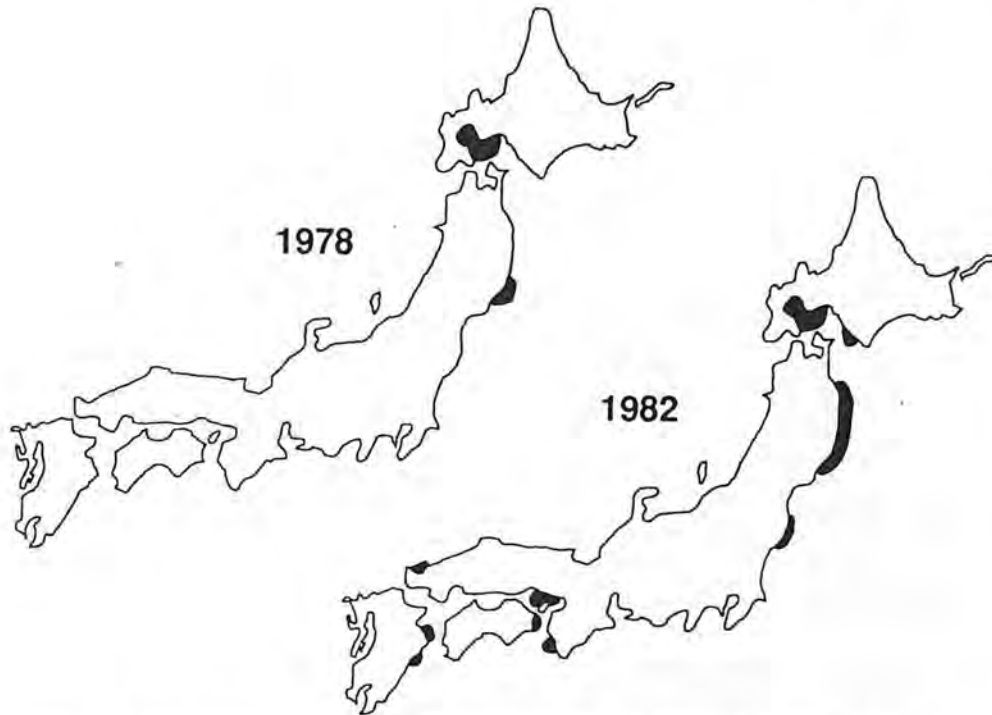


Fig. 4. Expansion of areas affected by paralytic shellfish poisoning in Japan between 1978 and 1982 (from Anraku 1984).

chromulina polylepis and *Prymnesium parvum* (Fig. 9D), and the dinoflagellate *Gymnodinium mikimotoi* (= *G. nagasakiense* Takayama et Adachi = ?*Gyrodinium aureolum* Hulburt; cf. Fig. 9B) have been implicated. *Heterosigma* has killed caged fish in Japan, Canada, Chile, and New Zealand, while *Chattonella* is a fish killer confined mainly to Japan (Seto Inland Sea). With these two raphidophyte flagellates, both physical

clogging of gills by mucus excretion as well as gill damage by hemolytic substances may be involved. In January 1989, a *Heterosigma* bloom in Big Glory Bay, Stuart Island (New Zealand) killed cage-reared chinook salmon worth 12 million NZ dollars (Chang *et al.* 1990). The two prymnesiophyte flagellates *Chrysochromulina* and *Prymnesium* produce substances that affect gill permeability, leading to a disturbed ion balance.

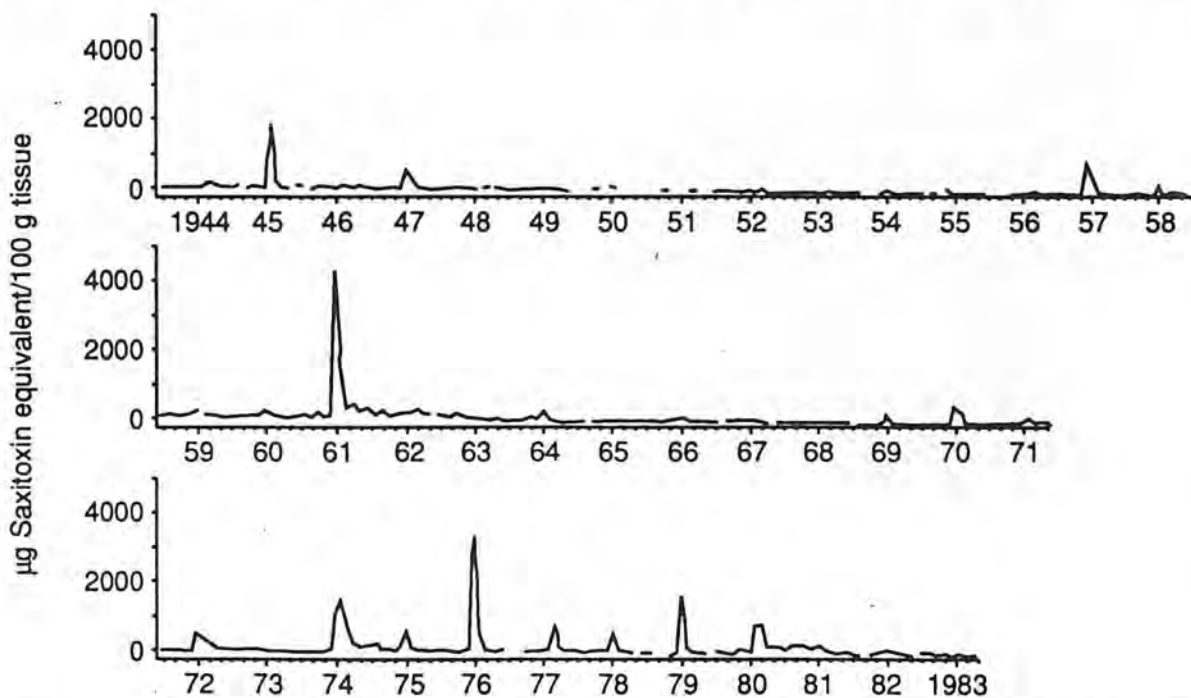


Fig. 5. Concentration of PSP toxins in Bay of Fundy clams (μg saxitoxin equivalent/100 g tissue) in the period 1944–1983 (from White 1987).

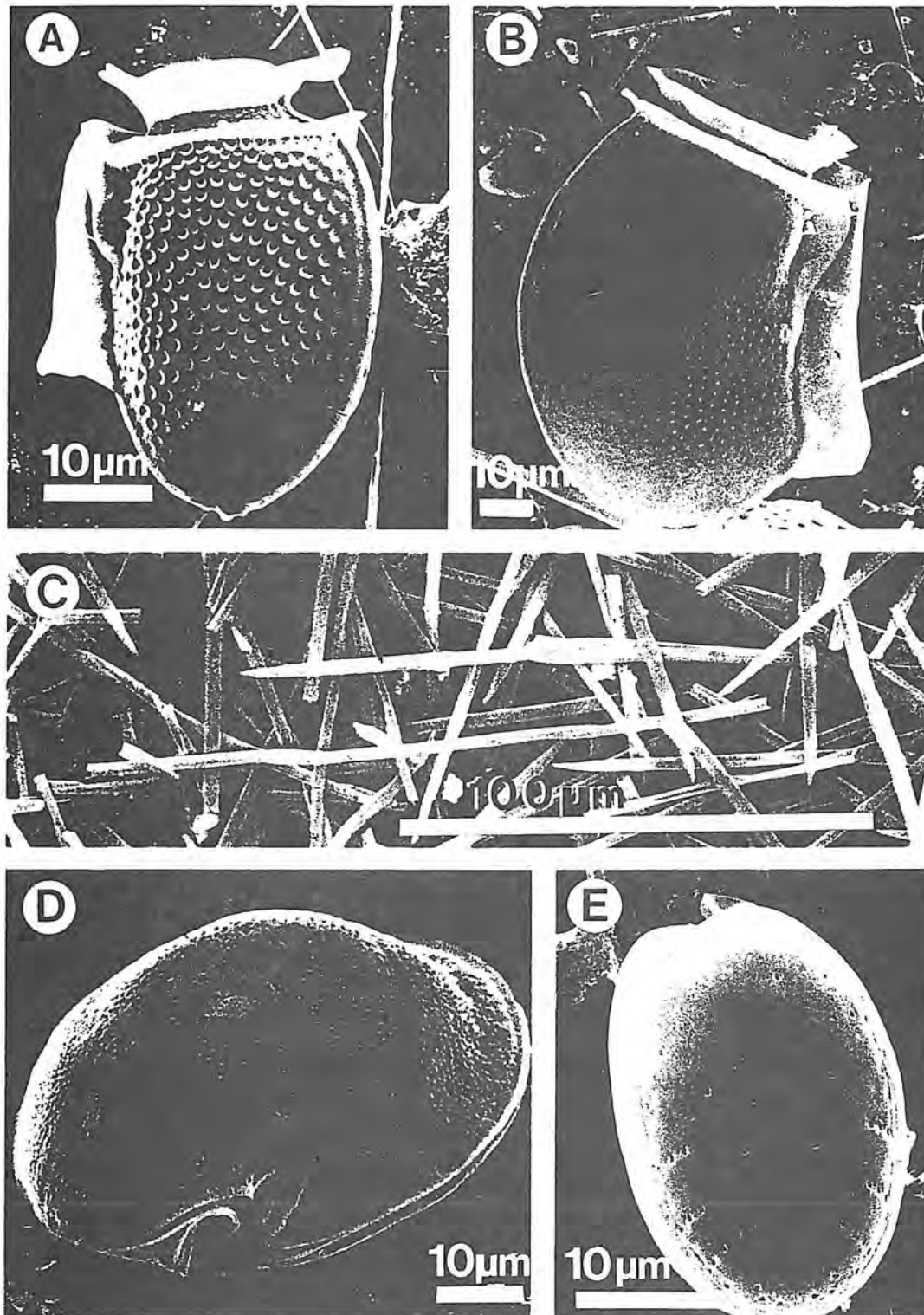


Fig. 6. *A and B.* Causative organisms of diarrhetic shellfish poisoning. (A) SEM. *Dinophysis acuminata* from Tasmania. (B) SEM. *D. fortii* from Tasmania (Dinophyceae). (C) SEM. Causative organism of amnesic shellfish poisoning, the diatom *Nitzschia pseudodelicatissima* from Tasmania. *D and E.* Causative organisms of ciguatera fishfood poisoning. (D) SEM. *Gambierdiscus toxicus* from Australia. (E) SEM. *Prorocentrum mexicanum* Tafall from Palau (Dinophyceae).

Toxicity by these species is promoted by phosphorus deficiency. A massive bloom (60 000 km²; 10⁷–10⁸ cells l⁻¹) of *Chrysochromulina polylepis* occurred in May–June 1988 in the Skagerrak, the Kattegat, the Belt area and the Sound between

Denmark, Norway and Sweden (Rosenberg *et al.* 1988; Kaas *et al.* 1991). Fish deaths occurred due to damage of gill membranes which produced a lethal increase in the chloride concentration in the blood, and fish cages moved into less saline

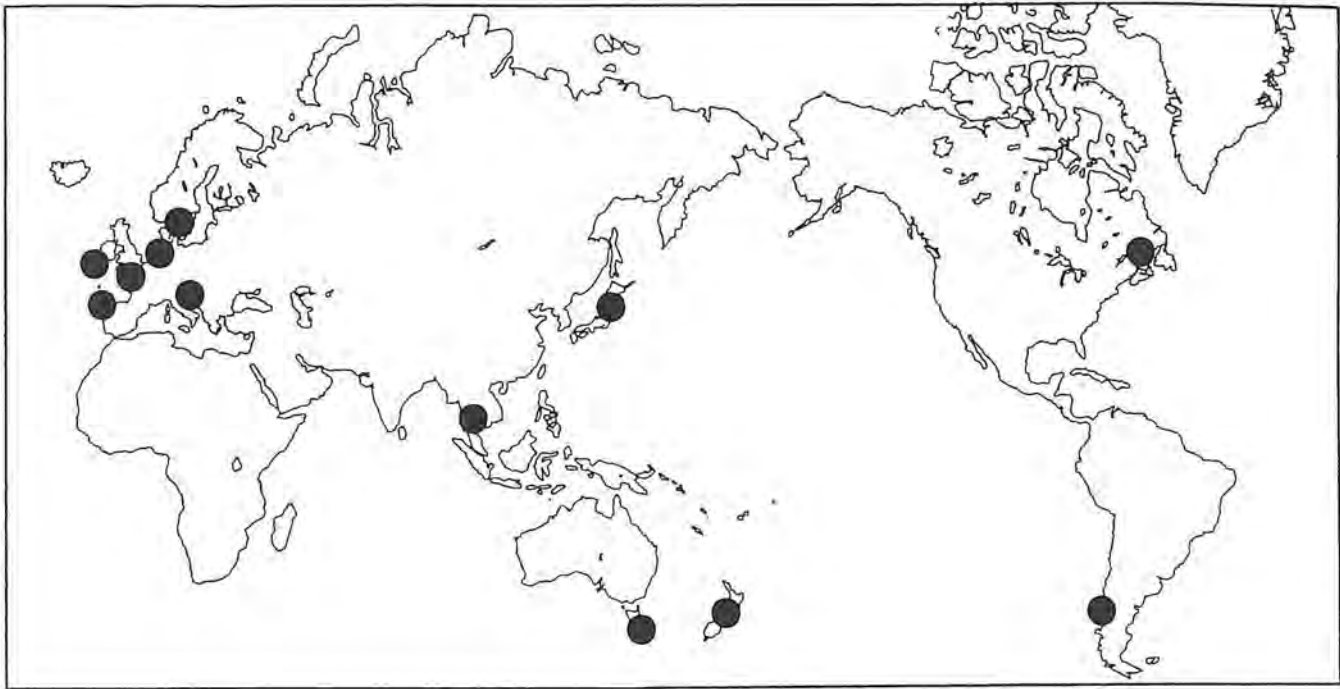


Fig. 7. Global distribution of diarrhetic shellfish poisoning (DSP).

fjords were therefore less affected. *Prymnesium parvum* has caused mortality of *Tilapia* fish in brackish water culture ponds in Israel (Shilo 1981), as well as mortality of salmon and rainbow trout in net-pens in Norway (Johnsen & Lein 1989). However, probably the greatest problem for Norwegian fish farms

is blooms of the unarmoured dinoflagellate *Gyrodinium aureolum* (first reported in 1966) (Tangen 1977). Similar dinoflagellates are common in Ireland and Scotland, as well as Japan and Korea (under the name *Gymnodinium nagasakiense* but now more appropriately called *G. mikimotoi*). Character-

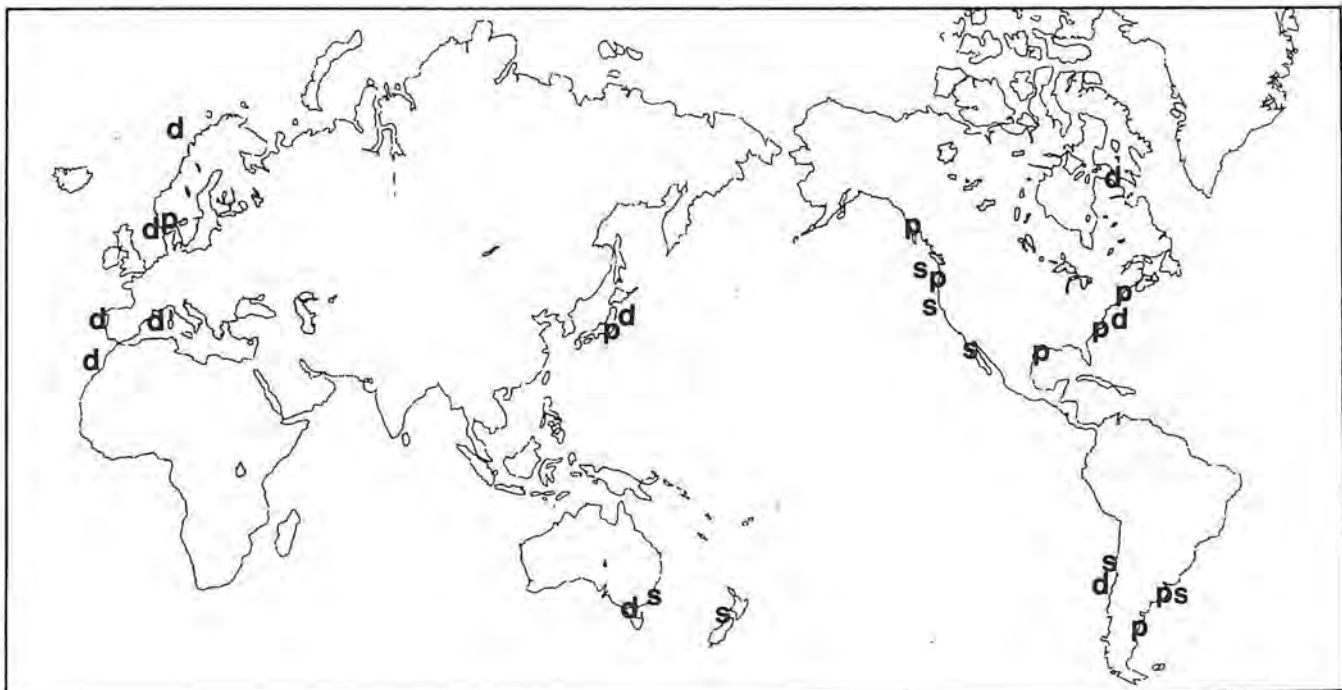


Fig. 8. Global distribution of *Nitzschia pungens* f. *multiseries* (p), *N. pseudodelicatissima* (d) and *N. pseudoseriata* (s), potential causative organisms of amnesic shellfish poisoning (from data in Fryxell *et al.* 1990 and Hasle 1965).

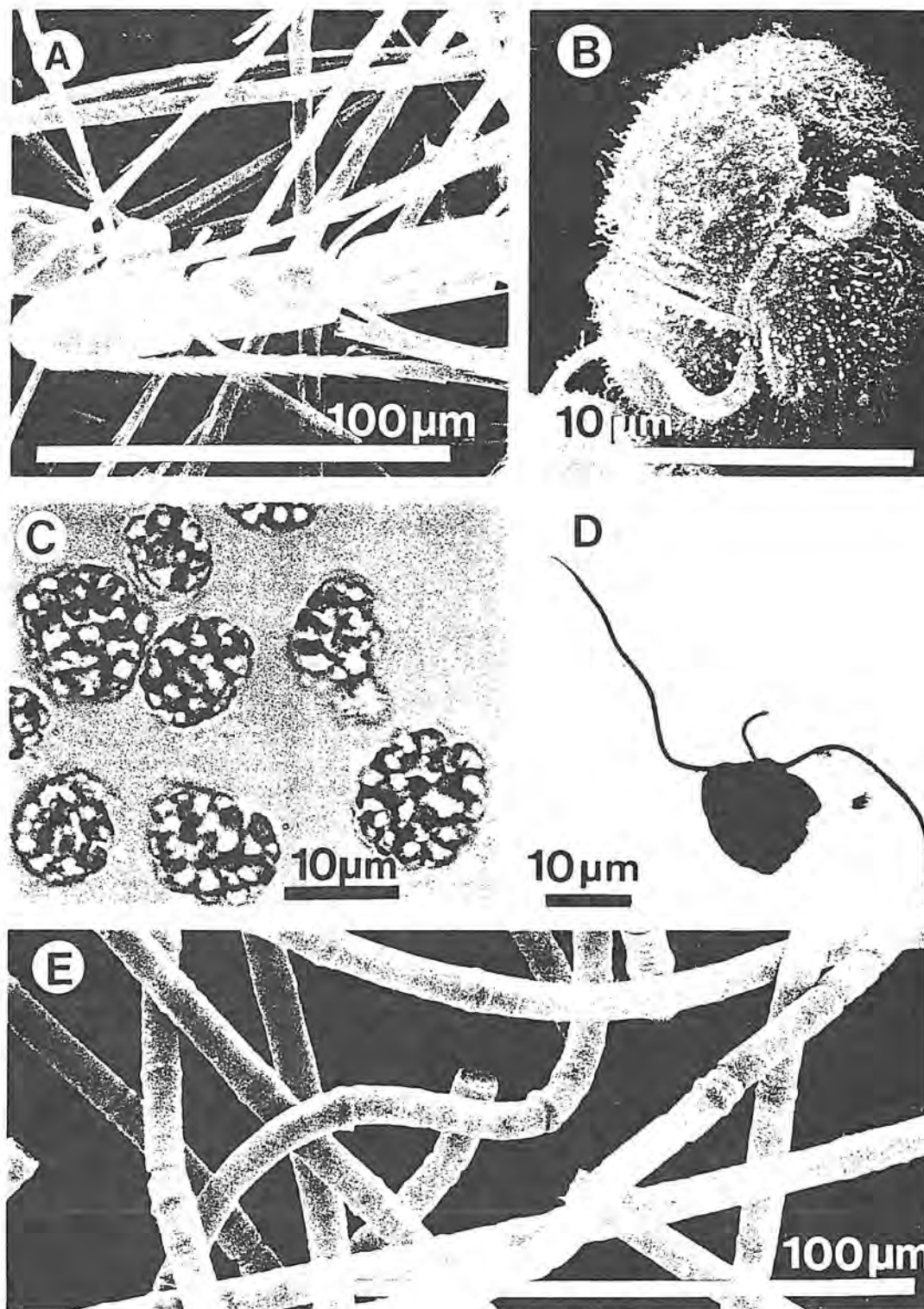


Fig. 9. A–D. Algal species which can kill cage-reared fish. (A) SEM. Diatom *Chaetoceros concavicornis* from British Columbia. (B) SEM. Dinoflagellate *Gymnodinium galatheanum* from Norway (closely related to *Gyrodinium aureolum*). (C) LM. Raphidophyte *Heterosigma akashiwo* from New Zealand. (Lugol preserved sample.) (D) TEM. Prymnesiophyte *Prymnesium parvum* from Australia. (E) SEM. Hepatotoxic filamentous cyanobacterium *Nodularia spumigena* from Australia.

istic histopathological symptoms to fish are a severe necrosis and sloughing of epithelial tissues of the gills and digestive system (Roberts *et al.* 1983).

Sophisticated monitoring systems using buoys with fibre

optical sensors and data transfer by satellites (the MARINET system) are in place on the Norwegian coast to allow cages being towed away from bloom-affected areas. During the 1988 *Chrysochromulina* bloom, more than 26 000 tons of fish in

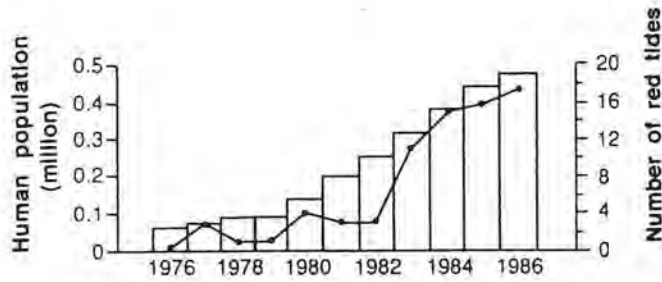


Fig. 10. Correlation between the number of red tide outbreaks per year in Tolo Harbour (continuous line) and the increase of human population in Hong Kong (bar diagram), in the period 1976–1986 (from Lam & Ho 1989).

1800 cages were thus moved from their permanent site into inland fjords. Fish losses in cages can also be reduced by stopping feeding fish since feeding attracts the fish to the surface and increases oxygen demand. In some cases, pumping of water to dilute the algal concentration, or immediate harvesting of marketable fish before they can be killed by algal blooms, may also be an option. The hemolytic toxins do not accumulate in fish flesh. Virtually all algal blooms, even of non-toxic species, reduce the fishes' appetite and reduced oxygen concentrations stress the fish and make them more vulnerable to disease.

V. INCREASE OF ALGAL BLOOMS BY CULTURAL EUTROPHICATION

Evidence is accumulating from diverse areas such as Hong Kong Harbour, the Seto Inland Sea in Japan and North European coastal waters that 'cultural eutrophication' from domestic, industrial and agricultural wastes can stimulate harmful algal blooms. Figure 10 illustrates an 8-fold increase in the number of red tides per year in Hong Kong Harbour in the period 1976–1986 (Lam & Ho 1989). This increase (mainly *Gymnodinium nagasakiense*, *Gonyaulax polygramma*, *Noctiluca scintillans* and *Prorocentrum minimum* (Pavillard) Schiller) shows a striking relationship with the 6-fold increase in human population in Hong Kong and the concurrent 2.5-fold increase in nutrient loading, mainly contributed by untreated domestic and industrial waste. As of 1 April 1988, all new industrial and sewage effluents in Hong Kong Harbour have needed to meet strict environmental quality objectives. A similar experience was noted in the Seto Inland Sea, one of the major fish farm areas in Japan (Okaichi 1989) (Fig. 11). Between 1965 and 1976 the number of confirmed red tide outbreaks (mainly *Chattonella antiqua*, since 1964; and *Gymnodinium nagasakiense*, since 1965) progressively increased 7-fold, concurrent with a 2-fold increase in the COD (chemical oxygen demand) loading, mainly from untreated sewage and industrial waste from pulp and paper factories. During the most severe outbreak in 1972, a *Chattonella* red tide killed 14 million cultured yellow-tail fish. Effluent controls were then initiated to reduce the chemical oxygen demand loading by about half, to introduce secondary sewage treatment, and to remove phosphate from household detergents (Seto Inland Sea Environment Conservation Law). Following a time-lag of 4

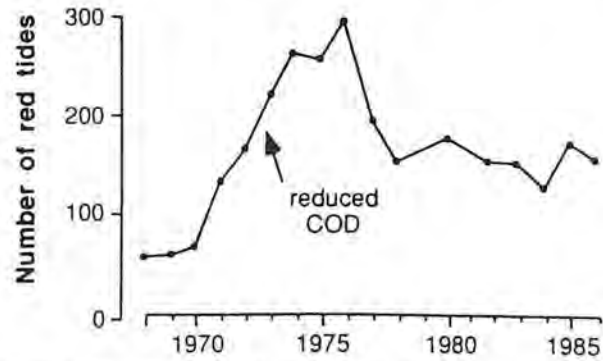


Fig. 11. Long-term trend in the frequency of red tide outbreaks in the Seto Inland Sea, Japan, in the period 1965–1986 (from Okaichi 1989).

years, the frequency of red tide events in the Seto Inland Sea then decreased by about 2-fold to a more stationary level.

A similar pattern of a long-term increase in nutrient loading of coastal waters is evident for the North Sea in Europe (van Bennekom & Salomons 1981) (Fig. 12). Since 1955 the phosphate loading of the River Rhine has increased 7.5-fold, while nitrate levels have increased 3-fold. This has resulted in a significant 6-fold decline in the Si : P ratio, because long-term reactive silicate concentrations (a nutrient derived from natural land weathering) have remained constant. More recently, improved wastewater treatment has been causing increases in the ammonia : nitrate ratio of River Rhine discharge (Riegman & Noordeloos 1991). The nutrient composition of treated wastewater is never the same as that of the coastal waters in which it is being discharged. There is considerable concern (Ryther & Dunstan 1971; Officer & Ryther 1980; Smayda 1990) that such altered nutrient ratios in coastal waters may favour blooms of nuisance flagellate species which replace the normal spring and autumn blooms of siliceous diatoms.

The remarkable increase of foam-producing blooms of the prymnesiophyte *Phaeocystis pouchetii* (Hariot) Lagerheim, which first appeared in Dutch coastal waters in 1978, is probably the best-studied example of this phenomenon (Lancelot *et al.* 1987). Algal species which are not normally toxic (*Chrysochromulina*, *Nitzschia pungens* f. *multiseriis*, *Prymnesium parvum*) may be rendered toxic when exposed to atypical nutrient regimes (e.g. phosphate deficiency) resulting from cultural eutrophication (Shilo, 1987; Edvardsen *et al.* 1990; Bates *et al.* 1991). The 1988 bloom in the Kattegat of the prymnesiophyte *Chrysochromulina polylepis*, not unusual in terms of biomass but unusual in terms of its species composition and toxicity, has been related to a change in the nutrient status from nitrogen to phosphorus limitation (Maestrini & Granéli 1991). Another *Chrysochromulina* bloom, this time of the species *C. leadbeateri*, occurred in north Norway in May–June 1991 and killed 700 tonnes of Atlantic salmon (Tangen 1991). As in Hong Kong and Japan, several North European countries have now agreed to reduce phosphate and nitrate discharges by 50% in the next several years, but their efforts will almost certainly be in vain if neighbours such as Poland continue polluting. Furthermore, such indiscriminate reductions in nutrient discharges are not addressing the problem of changing nutrient ratios of coastal waters. In the Black Sea a long-term decrease in the Si : P ratio has been associated with an increase of dinoflagellate blooms of *Prorocentrum cordatum* (Ostenfeld)

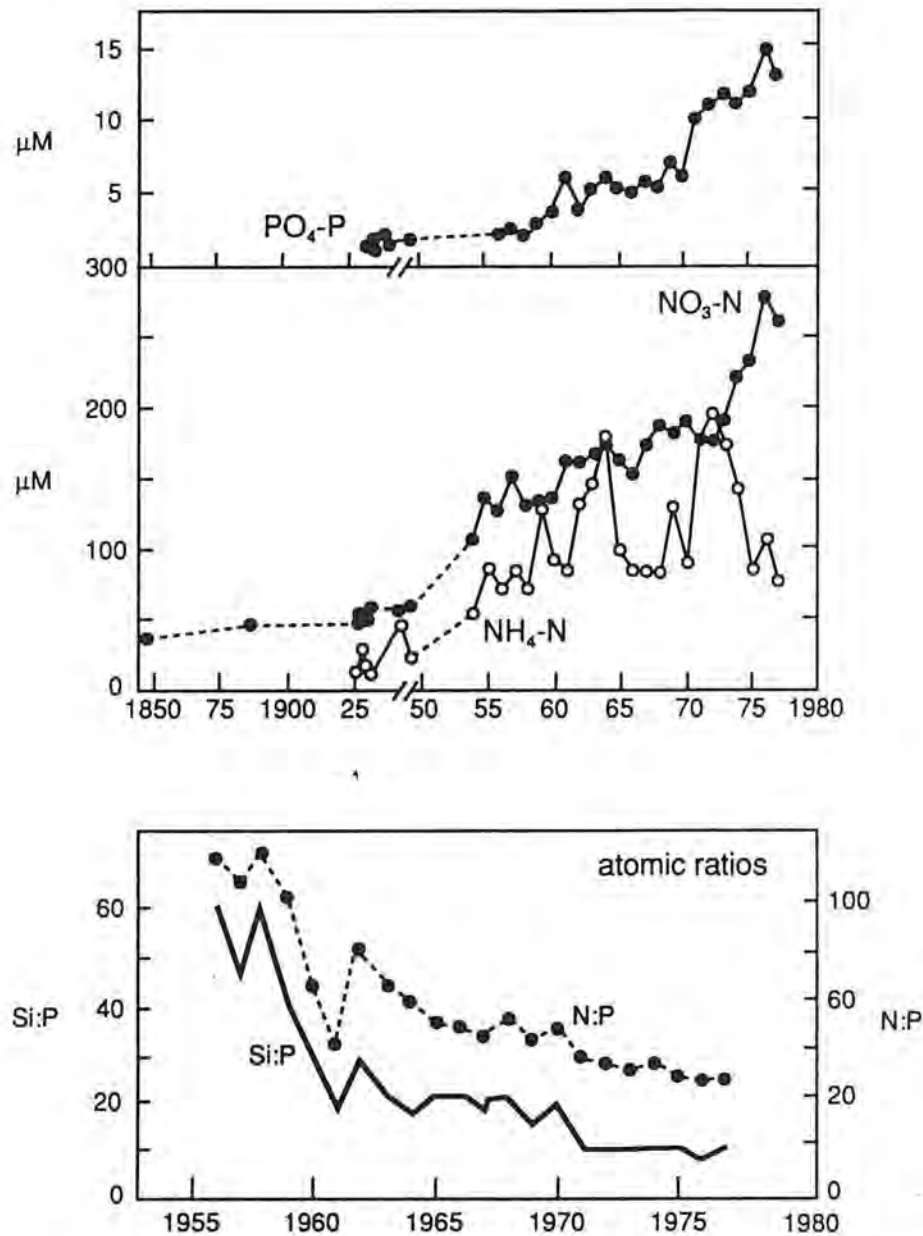


Fig. 12. Long-term trend in the phosphate, nitrate and ammonia loading of the River Rhine (top) and concurrent changes in the N:P and Si:P nutrient ratios (bottom) (from van Bennekom & Salomons 1981).

Dodge (Bodeanu & Usurelu 1979; Fig. 13). Changed patterns of land use, such as deforestation, can also cause shifts in phytoplankton species composition by increasing the concentrations of humic substances in land run-off. Acid precipitation can further increase the mobility of humic substances and trace metals in soils. Experimental evidence from Sweden indicates that river water draining from agricultural soils (rich in N and P) stimulates diatom blooms but that river water draining from forest areas (rich in humic and fulvic acids) can stimulate dinoflagellate blooms of species such as *Prorocentrum minimum* (Granéli & Moreira 1990). Agricultural run-off of phosphorus can also stimulate cyanobacterial blooms, for example of *Nodularia spumigena* in the Baltic Sea and in the Peel-Harvey Estuary, Australia (Figs 9E, 14). These species produce hepatotoxic peptides (*Nodularia*, *Microcystis*) and neurotoxic

alkaloids (*Anabaena*, *Aphanizomenon*) (Fig. 1E), which can kill domestic and wild animals that drink from the shores of eutrophic ponds, lakes and reservoirs (Edler *et al.* 1985). Cyanobacteria also pose public health problems through the production of teratogens and tumour promoters, which are not acutely toxic (Carmichael 1989). The toxins can accumulate in mussels and have been reported to contaminate drinking water (for example, during a 1000 km long *Anabaena circinalis* bloom in the Darling River, Australia, in 1991). A neurotoxic factor has also been associated with some strains of the common marine, bloom-forming tropical cyanobacterium *Trichodesmium thiebautii* Gomont (Hawser *et al.* 1991). While this organism can thrive under nutrient-impovertished oceanic conditions, it is possible that coastal eutrophication can prolong or enhance bloom phenomena.

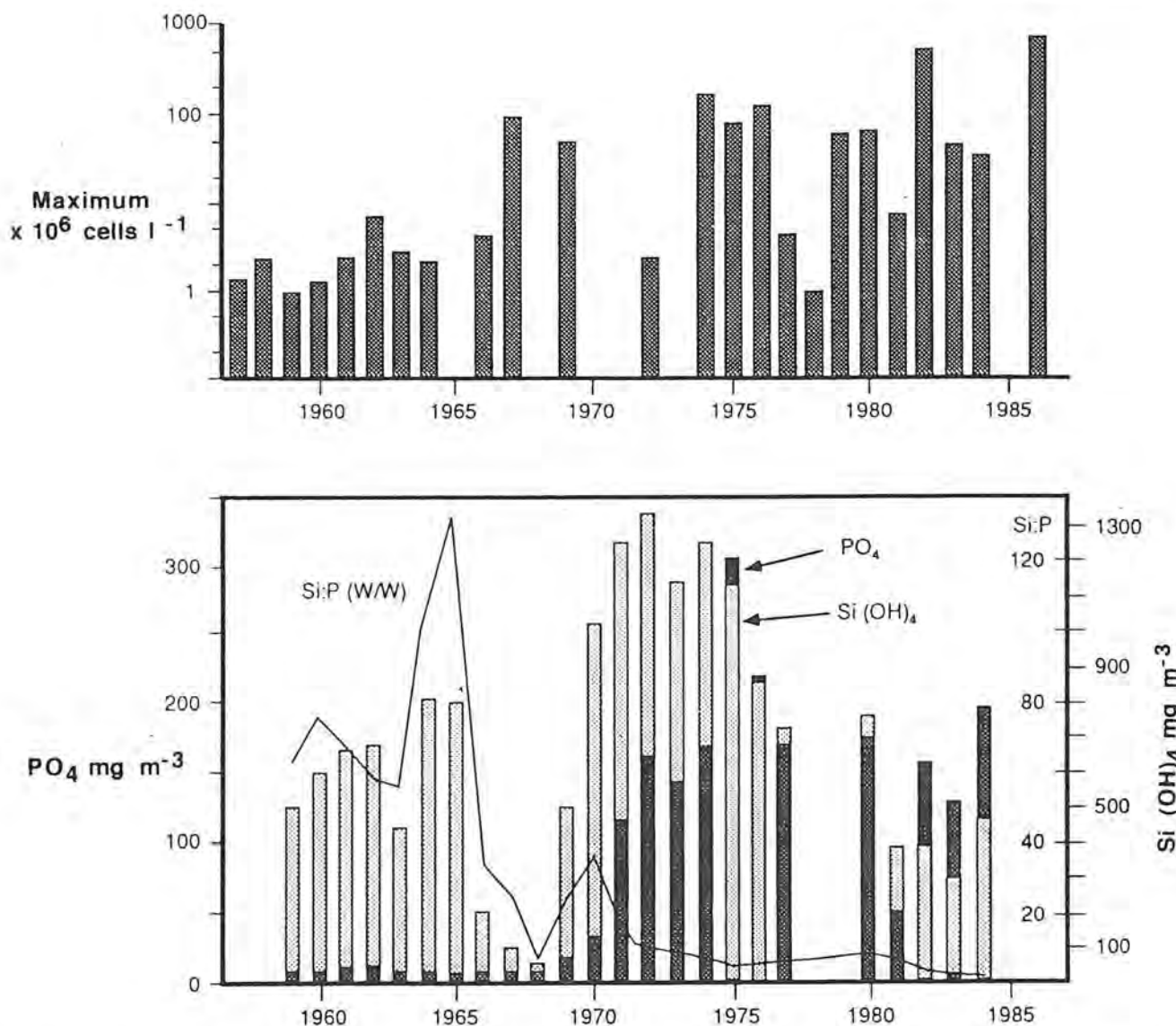


Fig. 13. Long-term trend in the incidence of *Prorocentrum cordatum* dinoflagellate blooms in the Black Sea (top) and concurrent changes in the phosphate and silicate loadings and Si:P nutrient ratios (adopted from Smayda 1990; partially based on Bodeanu & Usurelu 1979).

A much more complex 'cultural eutrophication' scenario has emerged in Long Island waters, where an unusual 'brown tide' (caused by the chrysophyte picoplankton *Aureococcus anophagefferens* Hargraves et Sieburth) has been related to the discharge of chelators (such as citric acid) in detergents and lawn treatments, together with a suppression of zooplankton grazing by pesticides (Cosper *et al.* 1989, 1991). This bloom was responsible for a reduction in the extent and biomass of eelgrass beds and caused starvation and recruitment failure in commercial scallop populations.

Ciguatera fishfood poisoning and coral reef disturbance

Ciguatera is a tropical fishfood poisoning syndrome well known from coral reef areas in the Caribbean, Australia, and especially French Polynesia. Humans consuming contaminated fish such as red bass, chinaman fish, moray eel, and paddle tail can suffer from gastrointestinal and neurological illnesses and in

extreme cases can die from respiratory failure (Table 2: Gillespie *et al.* 1986). The causative organisms are benthic dinoflagellates such as *Gambierdiscus toxicus*, and possibly *Ostreopsis siamensis*, *Coolia monotis* Meunier, *Prorocentrum lima* and related species (Figs 6D, E), that live in epiphytic association with bushy red, brown and green seaweeds (up to 200 000 cells/100 g of algae) and also occur free in sediments and coral rubble. These dinoflagellates produce the potent neurotoxins gambiertoxin (Fig. 1f) and maitotoxin, which accumulate through the food chain, from small fish grazing on the coral reefs into the organs of bigger fish that feed on them (the principle toxin fraction in fish is ciguatoxin). While in a strict sense this is a completely natural phenomenon (Captain Cook suffered from this illness when visiting New Caledonia in 1774), from being a rare disease two centuries ago ciguatera now has reached epidemic proportions in French Polynesia. In the period 1960–1984 more than 24 000 patients were reported from this area, which is more than six times the average for the

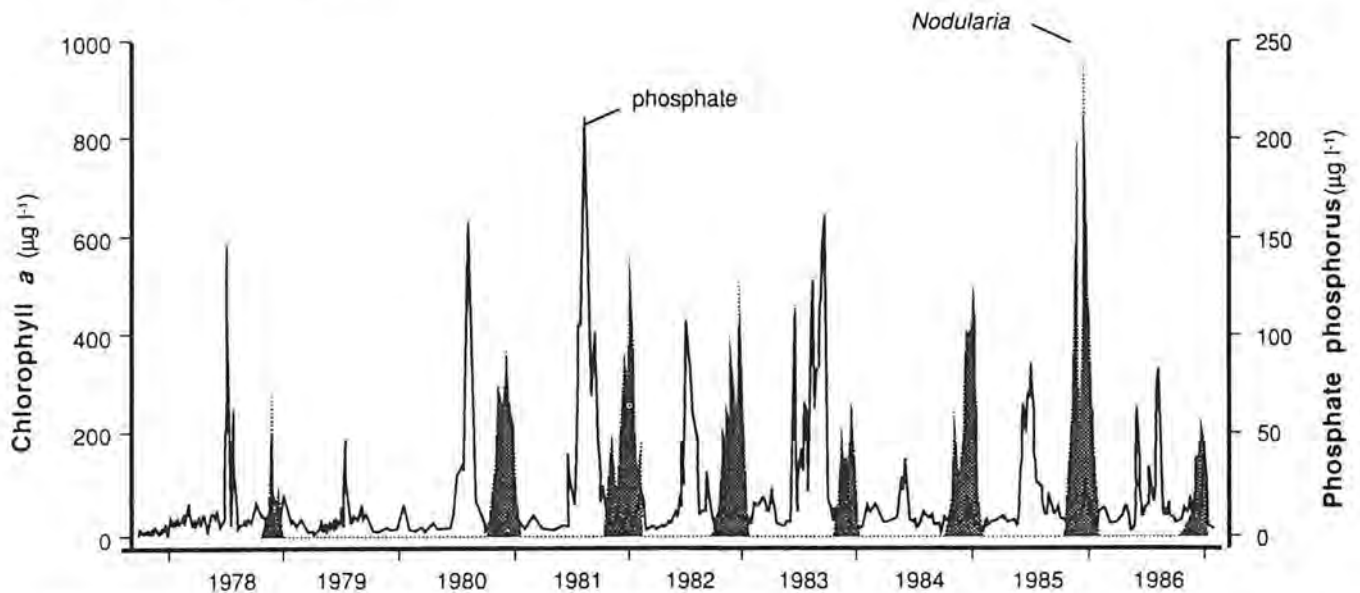


Fig. 14. Relationship between *Nodularia spumigena* cyanobacterial blooms (as chlorophyll concentration) in the Peel-Harvey Estuary, Australia, and its relationship to riverine phosphate loading from agricultural run-off (from Hillman *et al.* 1990).

Pacific as a whole. Evidence is accumulating that reef disturbance by hurricanes, and military and tourist developments are increasing the risk of ciguatera by increasing benthic substrate for dinoflagellate growth (Fig. 15; Bagnis *et al.* 1985).

VI. STIMULATION OF ALGAL BLOOMS BY UNUSUAL CLIMATOLOGICAL CONDITIONS

Toxic *Pyrodinium bahamense* blooms in the tropical Indo-West Pacific

At present the dinoflagellate *Pyrodinium bahamense* is confined to tropical, mangrove-fringed coastal waters of the Atlantic and Indo-West Pacific. A survey of fossil occurrences of its resting cyst *Polysphaeridium zoharyi* (Rossignol) Bujak *et al.* (Figs 16A, 17) (records go back to the Eocene, 50 million years ago) indicates a much wider range of distribution in the past. For example, in the Australasian region at present the

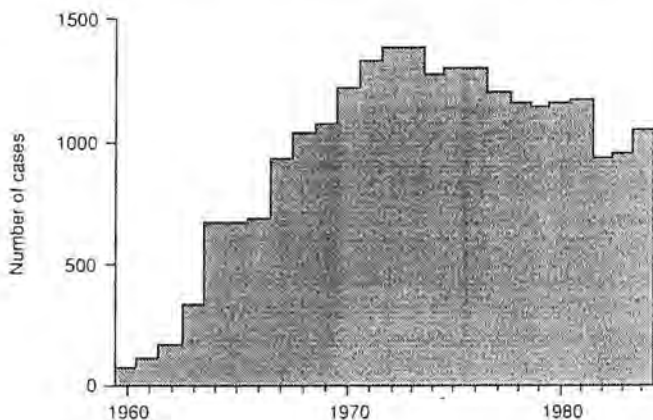


Fig. 15. Incidence of ciguatera fishfood poisoning in French Polynesia in the period 1960–1984 (from Bagnis *et al.* 1985).

dinoflagellate does not extend further south than Papua New Guinea, but in the Pleistocene it ranged as far south as Sydney Harbour (McMinn 1989). There is genuine concern that with increased greenhouse effect and warming of the oceans, this species may return to Australian waters. In the tropical Atlantic, in areas such as Bahia Fosforescente in Puerto Rico and Oyster Bay in Jamaica, this species forms persistent luminescent blooms which are a major tourist attraction. Both plankton bloom material and oysters and mussels attached to mangrove roots in Bahia Fosforescente appeared to be non-toxic (Hallegraeff, unpublished observations). The first harmful implications of *Pyrodinium* blooms became evident in 1972 in Papua New Guinea. Red-brown water discolorations coincided with the fatal food poisoning of three children, and mouse bioassays on shellfish from a house in the affected village subsequently established *Pyrodinium bahamense* as a source of paralytic shellfish poisons (Maclean 1977). Since then, toxic *Pyrodinium* blooms have apparently spread to Brunei and Sabah (1976), the central Philippines (1983) and the northern Philippines (1987). Maclean (1989) presented strong circumstantial evidence for a coincidence between *Pyrodinium* blooms and El Niño-Southern Oscillation (ENSO) climatological events. El Niño is caused by an imbalance in atmospheric pressure and sea temperature between the eastern and western parts of the Pacific Ocean and results in a shoaling of the thermocline. The recent 1991–92 ENSO event and recurrence of dinoflagellate blooms in the Philippines tend to substantiate these claims (Fig. 18).

Pyrodinium is a serious public health and economic problem for the tropical countries that are affected, since they depend heavily on seafoods for protein and have little prior experience in toxic dinoflagellate research. The first PSP outbreak in the Philippines in 1983 caused a complete ban on the harvest and sale of all shellfish for a period of 8 months. The 'halo' effect of bad publicity also led Japan and Singapore to ban shrimp imports from the Philippines. Altogether, this organism has

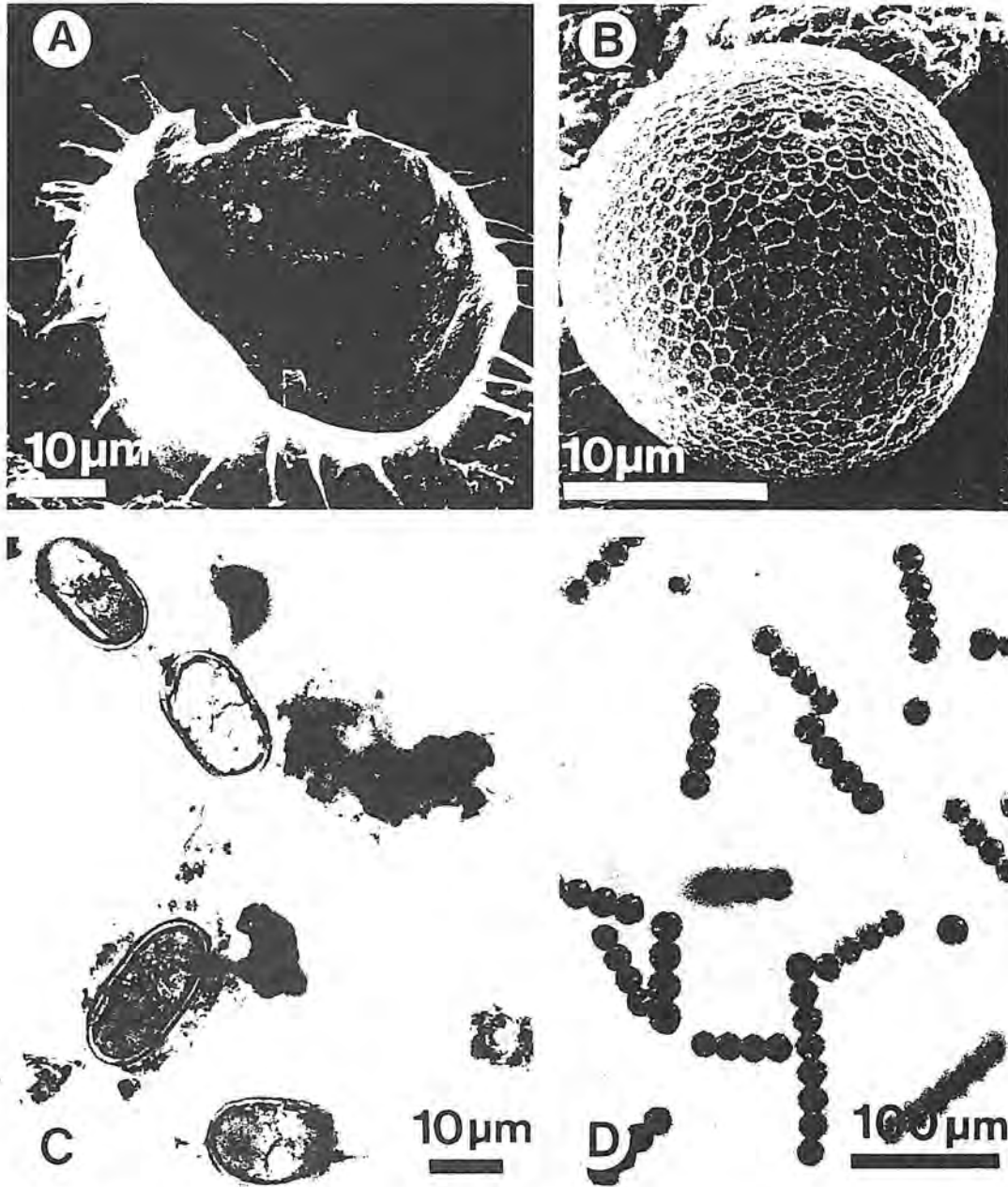


Fig. 16. Toxic dinoflagellate cysts from recent and fossil sediments and ships' ballast tank sediments. (A) SEM. Benthic resting cyst of the dinoflagellate *Pyrodinium bahamense*, known to geologists as *Polysphaeridium zoharyi*, from Papua New Guinea. (B) SEM. Fossil cyst of *Gymnodinium catenatum* from 2000-year-old Kattegat sediments. (C) LM. *Alexandrium tamarense* cysts which have been recovered from the ballast tank of a ship arriving in Eden, Australia, from Muroran, Japan. (D) LM. Motile cells of *Alexandrium catenella* cultured from cysts contained in the ballast tank of a ship arriving in Port Hedland, Australia, originating from Kashima, Japan.

now been responsible for more than 1000 human illnesses and 60 fatalities resulting from the consumption of contaminated shellfish as well as planktivorous fish such as sardines and anchovies. Most unexpectedly, during a *Pyrodinium* bloom in 1987 on the Pacific coast of Guatemala 187 people had to be hospitalized and 26 people died. In 1989 another bloom swept northward along the Pacific coast of Central America, again causing illness and death. The Guatemala populations are morphologically more similar to the Indo-West Pacific populations (sometimes distinguished as a separate variety *compressum*) than to the Caribbean morphospecies (var. *baha-*

mense) (Rosales-Loessener *et al.* 1989) and could well have crossed the Pacific, for example via cargoships operating between the Philippines and Panama.

Fossil blooms of *Gymnodinium catenatum* in the Kattegat-Skagerrak

The present-day distribution of the paralytic shellfish poison-producing dinoflagellate *Gymnodinium catenatum* includes the Gulf of California, Gulf of Mexico, Argentina, Japan, the Philippines, Palau, Tasmania, the Mediterranean and the Atlantic

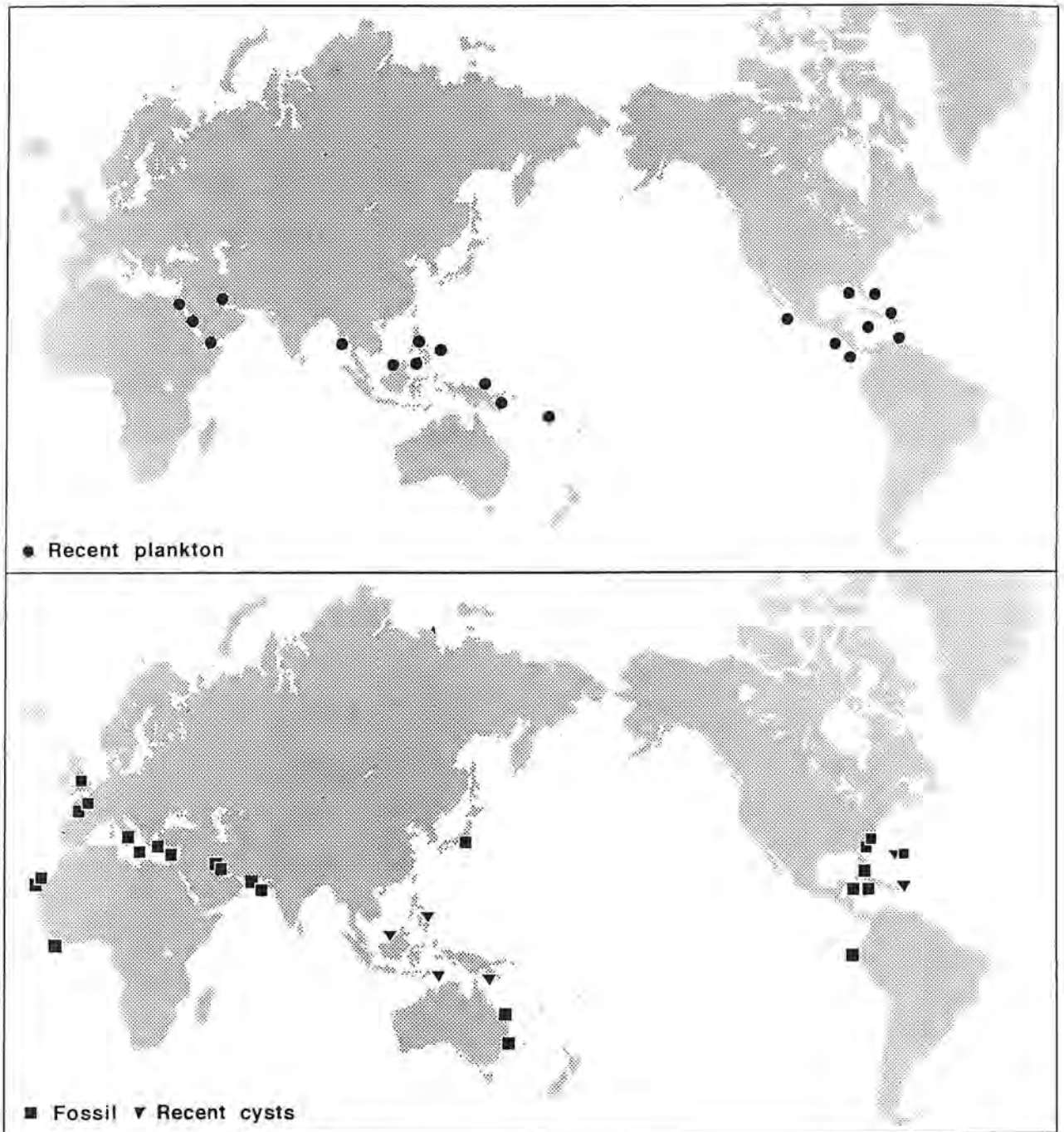


Fig. 17. Global distribution of the tropical dinoflagellate *Pyrodinium bahamense* in recent plankton (top) and of the fossil cyst *Polysphaeridium zoharyi* (bottom) (from Hallegraeff & Maclean 1989).

coast of Spain and Portugal (Fig. 19). This species has never been recorded in the living phytoplankton of the Kattegat-Skagerrak region of Scandinavia, even though living cysts have recently been detected in bottom sediments from the Danish coast (Moestrup, personal communication). Fossil cysts of this species were present in unusually large amounts in pollen records from Kattegat sediments (Fig. 16B; Nordberg & Bergsten 1988). A multidisciplinary study (Dale & Nordberg 1991) to reconstruct the prevailing paleoenvironment has suggested the following scenario: (1) the migration of *G. catenatum* into the area about 5000 years BP; (2) its establishment as part of the

local plankton; (3) a major blooming phase about 2000 to 500 years BP, of a magnitude that has not been seen since; and (4) its disappearance during the 'Little Ice Age'.

VII. TRANSPORT OF DINOFLAGELLATE CYSTS IN SHIPS' BALLAST WATER OR ASSOCIATED WITH THE TRANSFER OF SHELLFISH STOCKS

Cargo vessel ballast water was first suggested as a vector in the dispersal of non-indigenous marine plankton some 90 years

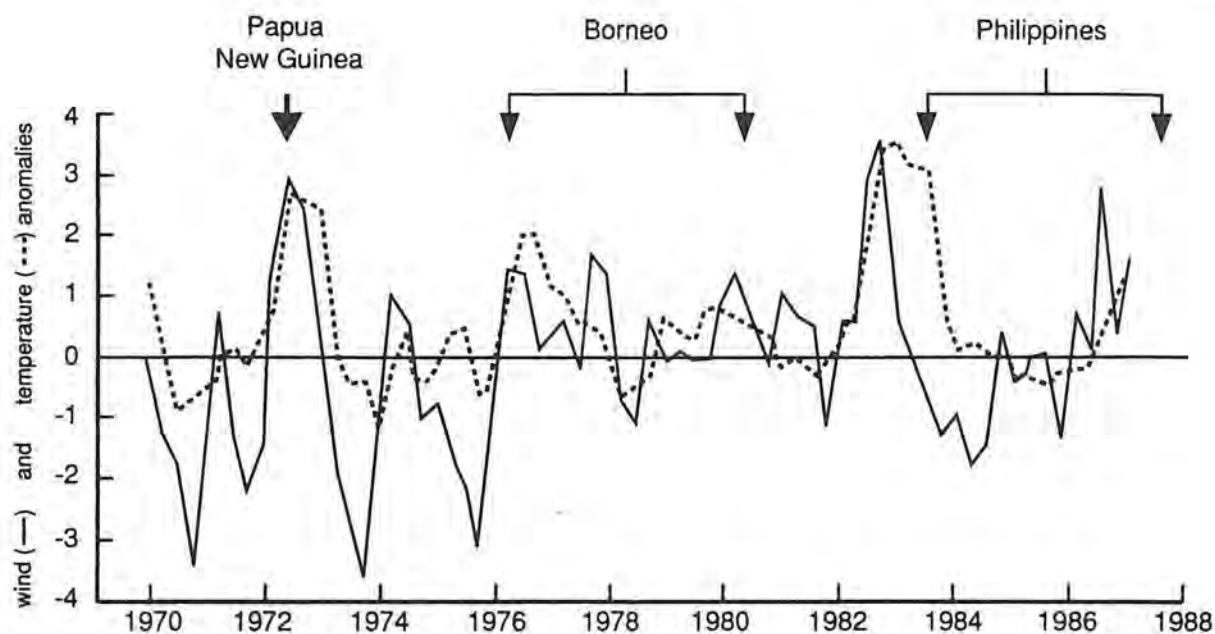


Fig. 18. Relationship between *Pyrodinium bahamense* blooms (arrows) in the tropical Indo-West Pacific and El Niño-Southern Oscillation (ENSO) climatological events in the period 1970–1988. The graph shows empirical orthogonal functions of zonal wind anomalies and zonal mean surface temperature anomalies over the near-equatorial eastern Indian and western Pacific Oceans. Strong positive anomalies are indicative of ENSO events (from Maclean 1989).

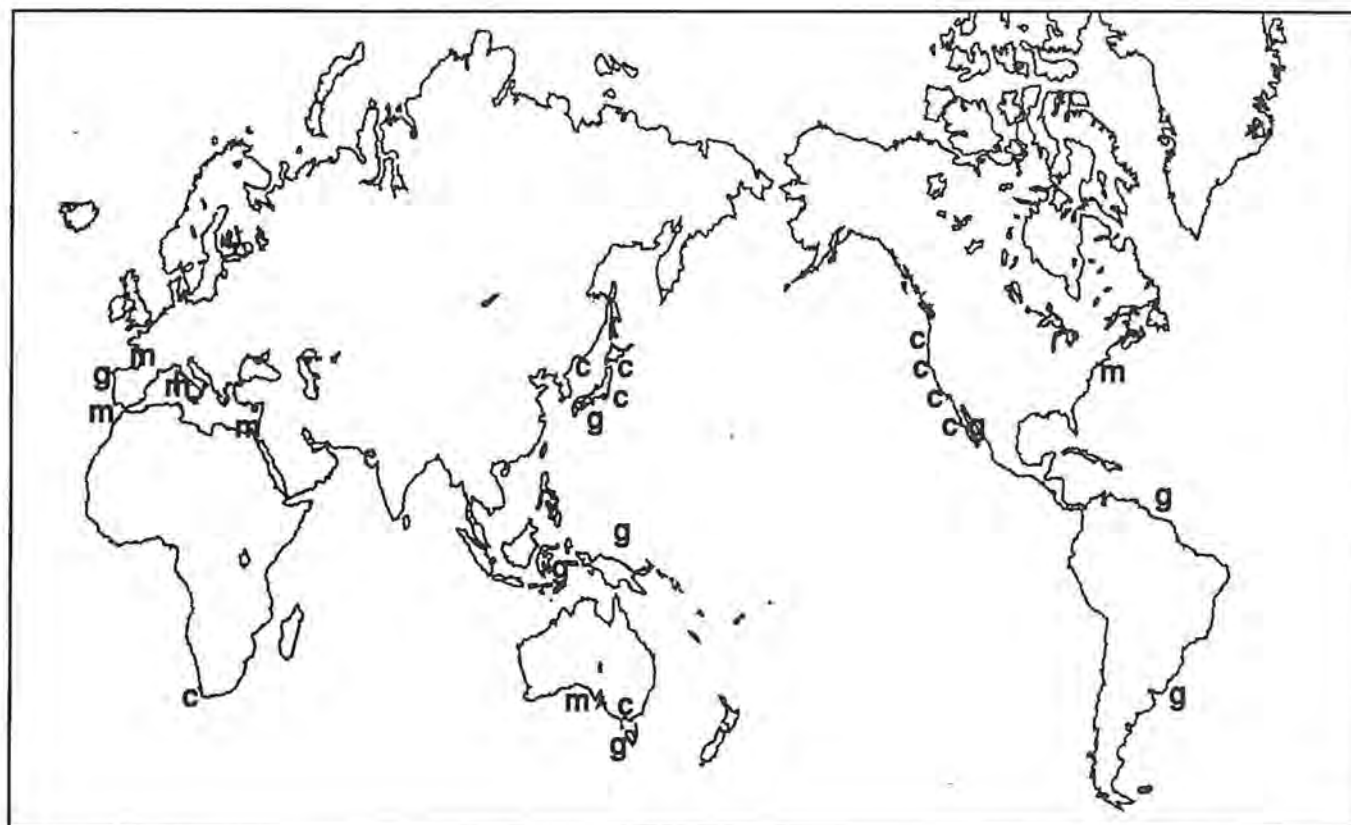


Fig. 19. Global distribution of the three PSP dinoflagellates *Gymnodinium catenatum* (g), *Alexandrium minutum* (m) and *Alexandrium catenella* (c) (from Hallegraeff *et al.* 1988).

ago. The diatom *Odontella (Biddulphia) sinensis* (Greville) Grunow, well-known from the tropical and subtropical coasts of the Indo-Pacific, had not been reported in European waters until 1903 when it produced dense plankton blooms in the North Sea. Since it appeared unlikely that this large diatom could have been overlooked previously and impossible that it could have been carried by currents from distant oceans, Ostefeld (1908) suggested that this species was introduced via the water or sediment contained in ships' ballast tanks. Subsequently, Hallegraeff *et al.* (1990) confirmed this possibility by culturing the related diatom species *Odontella aurita* (Lyngbye) C. Agardh from a ballast water sample collected at the end of a voyage from Japan to Australia. Whereas the introduction of *O. sinensis* was apparently without harmful effects, the more recent introduction into the North Sea of the diatom *Coscinodiscus wailesii* Gran et Angst (Bolch & Harbour 1977; Rincé & Paulmier 1986) has caused problems due to the clogging of fishing nets by extensive diatom mucus production.

In the 1980s the problem of ballast water transport of plankton species gained considerable interest when evidence was brought forward that non-indigenous toxic dinoflagellate species had been introduced into Australian waters into sensitive aquaculture areas, with disastrous consequences for commercial shellfish farm operations (Hallegraeff *et al.* 1988). While the planktonic stages of diatoms and dinoflagellates show only limited survival during the voyage in dark ballast tanks, their resistant resting spores are well suited to survive these conditions. One single ballast tank thus was estimated to contain more than 300 million toxic dinoflagellate cysts which could be germinated into confirmed toxic cultures (Fig. 16C, D; Hallegraeff & Bolch 1991). Paralytic shellfish poisoning was unknown from the Australian region until the 1980s when the first outbreaks appeared in the ports of Hobart (*Gymnodinium catenatum*), Melbourne (*Alexandrium catenella*) and Adelaide (*A. minutum*) (Fig. 19). In Hobart, Tasmania, an examination of historic plankton samples (Hallegraeff *et al.* 1988), cyst surveys in ²¹⁰Pb dated sediment depth cores (Bolch & Hallegraeff 1990; McMinn & Hallegraeff, unpublished observations) and genetic studies using enzyme electrophoresis and sexual compatibility experiments (Blackburn *et al.* 1989) provided strong circumstantial evidence that the toxic dinoflagellate *G. catenatum* was introduced in the last 10–20 years. Subsequently, Hallegraeff & Bolch (1992) confirmed the presence of *G. catenatum* cysts in four ballast water samples entering Australian ports from both Korea and Japan. The organism is now well established in southern Tasmania, benthic cyst beds of this species are widespread and dense dinoflagellate blooms in 1986, 1987 and 1991 necessitated the closure of 15 shellfish farms for periods up to 6 months (Hallegraeff & Sumner 1986). Similarly, the toxic dinoflagellate *Alexandrium catenella*, which has caused the closure of shellfish farms in Port Phillip Bay, Melbourne, was not known from the area before 1986. Hallegraeff & Bolch (1992) confirmed that viable cysts of this species were present in ballast water being discharged into this port, and rRNA sequencing indicated a remarkable match between ballast water and harbour water cultures of this dinoflagellate (Scholin & Anderson 1991). Finally, the toxic dinoflagellate *Alexandrium minutum* first appeared in the Port River, Adelaide, in 1986 in an area where cyst surveys carried out in 1983 failed to detect resting cysts in sediments (Bolch *et al.* 1991). The port of Adelaide has a shipping link with the

Mediterranean, which has the only other known global population of this dinoflagellate (Fig. 19). Moreover rRNA sequencing has indicated a remarkable match between Australian and Spanish cultures of this species complex (Scholin & Anderson 1991).

The evidence of ballast water transfer of marine organisms other than microscopic algae is considerable and includes species of fish, crustaceans, polychaete worms and molluscs (see review by Carlton 1985). As of 1 November 1991 the International Maritime Organisation (IMO) has ratified the introduction of voluntary guidelines for ballast water handling procedures by bulk cargo vessels. These measures aim to reduce the risk of harmful introductions by encouraging a range of practices such as reballasting at sea (only feasible for vessels up to 40 000 dead weight tonnage), ballasting in deep water, and disposal of ballast tank sediments away from sensitive aquaculture or marine park areas. The most effective measure to prevent the spreading of dinoflagellate cysts via ships' ballast water would be to avoid ballasting during toxic dinoflagellate blooms in ports. Other options using heat or chemical treatment (chlorine, hydrogen peroxide) of ballast water, either in hold or in onshore facilities, are now also being investigated (Rigby *et al.* 1991; Bolch & Hallegraeff 1993).

Another vector for the dispersal of algae (especially their resting cysts) is with the transfer of shellfish stocks from one area to another. The faeces and digestive tracts of bivalves can be loaded with viable *Alexandrium* cells (Bricelj *et al.* 1991) and sometimes can also contain resistant resting cysts. The Japanese seaweeds *Sargassum muticum* (Yendo) Fensholt, *Undaria pinnatifida* (Harvey) Suringer (Atlantic coast of Europe, Mediterranean), and *Laminaria japonica* Areschoug (Mediterranean) are thus thought to have been introduced into European waters via sporophyte stages contained with introduced Japanese oyster spat (van den Hoek 1987).

VIII. CONCLUSIONS

The question of whether the apparent global increase in harmful algal blooms represents a real increase or not, we will probably not be able to conclusively answer for some time to come. There is no doubt that our increasing interest in utilizing coastal waters for aquaculture is leading to an increased awareness of toxic algal species. As a result, what we are faced with today is that the effects on public health and economic impacts of harmful algal blooms are now showing signs of a truly global 'epidemic' (Smayda 1990), and we should start to respond to this problem as such. In countries such as Australia and New Zealand which pride themselves on their disease- and pollution-free status for aquaculture, every effort should be made to quarantine sensitive aquaculture areas against the unintentional introduction of non-indigenous harmful algal species (Hallegraeff & Bolch 1992).

Most importantly, people responsible for management decisions on pollutant loadings of coastal waters (including decisions on agricultural and deforestation activities in catchment areas) should be made aware that one probable outcome of increased nutrient loading will be an increase in harmful algal blooms. Finally, global climate change studies (El Niño, greenhouse effect, ozone depletion) need to consider possible impacts on algal bloom events. A number of new international

programmes [e.g. by the Intergovernmental Oceanographic Commission (IOC) of UNESCO, the Food and Agriculture Organization (FAO), the Scientific Committee on Oceanic Research (SCOR), and the International Council for Exploration of the Sea (ICES)] are now being created to study and manage harmful algal blooms and their linkages to environmental changes in a manner consistent with the global nature of the phenomena involved.

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