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RED TIDES IN THE AUSTRALASIAN REGION

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Abstract

Australian waters are not subject to "red tide" phenomena as a regular occurrence. Episodic water discolourations have been caused by blue-green algae (*Trichodesmium*), diatoms (*Thalassiosira*), prymnesiophytes (*Phaeocystis*) and especially dinoflagellates (*Noctiluca, Scrippsiella*). Most of these plankton blooms appeared to be harmless events, and sporadic fish kills most likely resulted from associated anoxic conditions. However, several potentially toxic dinoflagellates have also been recognised. *Gambierdiscus toxicus* is responsible for ciguatera fish poisoning in the Great Barrier Reef region. *Pyrodinium bahamense* var. *compressa* causes paralytic shellfish poisoning (PSP) in Papua New Guinea waters, but has not as yet been observed along the coasts of northern Australia. *Gymnodinium catenatum* (PSP) has necessitated institution of control measures for shellfish farms in southern Tasmanian waters, and the potentially toxic (PSP) *Protogonyaulax catenella* and *Alexandrium* (*Protogonyaulax*) *ibericum* have been recorded in southern Australian waters. *Dinophysis fortii* and *D. acuminata* which cause diarrhetic shellfish poisoning (DSP), are of concern in Tasmanian and New Zealand coastal waters.

CONTENTS

INTRODUCTION

Red tides are blooms of unicellular plankton algae that become so dense they discolour the sea (e.g. "Red Sea"). Plankton blooms may also appear yellow, brown, green, blue or milky in colour, depending upon the organism involved. Red-tide species in the Australasian region include representatives of the blue-green algae, ciliates and dinoflagellates, with diatoms and prymnesiophytes being only rarely involved. Most red tides are caused by motile or strongly buoyant species. Their high concentrations are achieved through a combination of high growth rates and vertical (behavioural) or horizontal (physical) aggregation. Dense plankton concentrations are most strongly developed under stratified stable conditions, at high temperatures and following high organic input from land run-off after heavy rains.

The majority of plankton blooms appear to be completely harmless events, but under exceptional conditions, non-toxic bloom-formers may become so densely concentrated that they generate anoxic conditions that cause indiscriminate kills of fish and invertebrates in sheltered bays (Grindley and Taylor, 1962). Oxygen depletion can result from high respiration by the algae (at night or in dim light during the day) but is more commonly caused by bacterial respiration during decay of the algal bloom.

An essentially different phenomenon is the production by dinoflagellates of potent toxins that find their way through fish or shellfish to man. In this case even low densities of the toxic algae in the water column may be sufficient to cause such illnesses in humans as paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning (DSP) and ciguatera fish poisoning. PSP can result from eating either bivalve shellfish or planktivorous fish (clupeotoxicity), while DSP is caused by eating shellfish, fand ciguatera by eating tropical fish. The toxins involved evoke a variety of gastro-intestinal and neurological symptoms in humans (Table 1) but rarely affect the nervous systems of fish or shellfish. Another group of toxins (ichthyotoxins) selectively kill fish by inhibiting their respiration.

On a global scale, close to 2000 cases of human poisoning through fish or shellfish consumption occur each year, and economic damage through reduced local consumption and reduced export of seafood products can be considerable.

Generally speaking, both the number and intensity of algal blooms seem to be on the rise and their geographic extent seems to be spreading. While toxic plankton blooms appear regularly on a seasonal basis in temperate waters of Europe, North America and Japan (Taylor and Seliger, 1979, Anderson *et al.*, 1985), they are still relatively rare in tropical and subtropical waters (White *et al.*, 1984).

The present paper illustrates and discusses several potentially hazardous organisms that have been recognised in Australasian waters. Clinical symptoms of various types of fish and shellfish poisoning are summarised to assist their diagnosis by medical practitioners, and illustrations of the plankton organisms are provided to improve identification by plankton workers. Blooms of the toxic species, in particular, need to be carefully monitored and fish and shellfish products from affected areas should be tested for toxins by public health officials who should, if necessary, issue warnings promptly and effectively.

Table 1. Clinical symptoms of various types of fish and shellfish poisoning

	PARALYTIC SHELLFISH POISONING	DIARRHETIC SHELLFISH POISONING	CIGUATERA
Causative organism	*Pyrodinium bahamense var. compressa*, *Protogonyaulax tamarensis*; *Protogonyaulax catenella*; *Gymnodinium catenatum*	*Dinophysis fortii*; *Dinophysis acuminata*	*Gambierdiscus toxicus*
Symptoms mild case:	Tingling sensation or numbness around lips, gradually spreading to face and neck; prickly sensation in fingertips and toes; headache, dizziness, nausea, vomiting, diarrhoea.	After 30 min to a few hours: diarrhoea, nausea, vomiting, abdominal pain.	Symptoms develop within 12 - 24 hrs of eating fish. Gastro-intestinal symptoms: diarrhoea, abdominal pain nausea, vomiting.
severe case:	Incoherent speech; progression of stiffness and non coordination of limbs; general weakness and feeling of lightness; slight respiratory difficulty; rapid pulse.		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.
extreme case:	Muscular paralysis; pronounced respiratory difficulty; choking sensation; death through respiratory paralysis may occur within 2 to 24 h after ingestion.		In extreme cases, death through respiratory failure.
Treatment	Patient has stomach pumped and is given artificial respiration. No lasting effects.	Recovery after 3 days, irrespective of medical treatment.	No antitoxin or specific treatment is available. Neurological symptoms may last for months and years. Calcium may help relieve symptoms.

DESCRIPTION OF RED-TIDE ORGANISMS

Nowhere is the need for correct taxonomic identification of plankton organisms more critical than in the study of the toxic species. As red tides are often monospecific blooms, elucidating the autecology of the constituent species becomes crucial not only to understanding the bloom event but also to deciding on possible measures for its control. Some dinoflagellates (e.g. *Protogonyaulax*) produce benthic cysts that can seed further blooms; the monitoring for these species must therefore also take into account benthic cyst populations and sedimentary processes.

Three categories of "red tide organisms" are distinguished: species that produce mostly harmless water discolourations; species that are non-toxic to man, but harmful to fish and invertebrates by damaging or clogging their gills; and species that produce potent toxins that can find their way through the food chain to man.

Species Producing Harmless Water Discolourations

A wide variety of organisms have produced water discolourations in Australasian waters, but only in exceptional cases have such plankton blooms caused fish kills in sheltered bays due to the generation of anoxic conditions.

Trichodesmium (blue-green alga)

This tropical filamentous alga produces seasonal (February-April) water blooms in the Java, Banda, Arafura and Coral Seas. The East Australian Current and the Leeuwin Current Transport the algal masses as far south as Sydney and Perth (Creagh, 1985). These algae appear as yellow-grey (early bloom) or reddish-brown (late bloom) coloured windrows, occupying up to 40,000 square kilometers. The long filaments mass together to form raft-like (*T. erythraeum*, Fig. 1a,b) or radiating or bundle-like aggregations (*T. thiebautii*, Fig. 1c,d).

At the start of the bloom the filaments usually appear throughout the water column, but during late-bloom stages the strong gas vacuoles cause a massive rise of the alga to the surface layers. Differentiated cells within the centre of the colony are capable of fixing atmospheric nitrogen, which allows the alga to thrive under nutrient-impoverished oceanic conditions, where they readily outcompete other phytoplankton. Wave action can break up the bundles and inactivate the central nitrogenase enzyme (Carpenter and Price, 1976), which is why calm seas are a prerequisite for *Trichodesmium* blooms. The alga can be a nuisance to swimmers, but harmful effects on fish are seldom observed except in sheltered bays where the decaying bloom may generate anoxic conditions that can cause indiscriminate kills o fish and other marine fauna.

An unusual death of corals caused by the decomposition of masses of *Trichodesmium* driven ashore by the wind has been recorded from New Caledonia (Baas Becking, 1951). *Trichodesmium* red tides ("sea sawdust) were observed as early as 1770 during Captain Cook's voyage through the Coral Sea. There is no evidence of a relationship with industrial pollution.

Noctiluca (dinoflagellate)

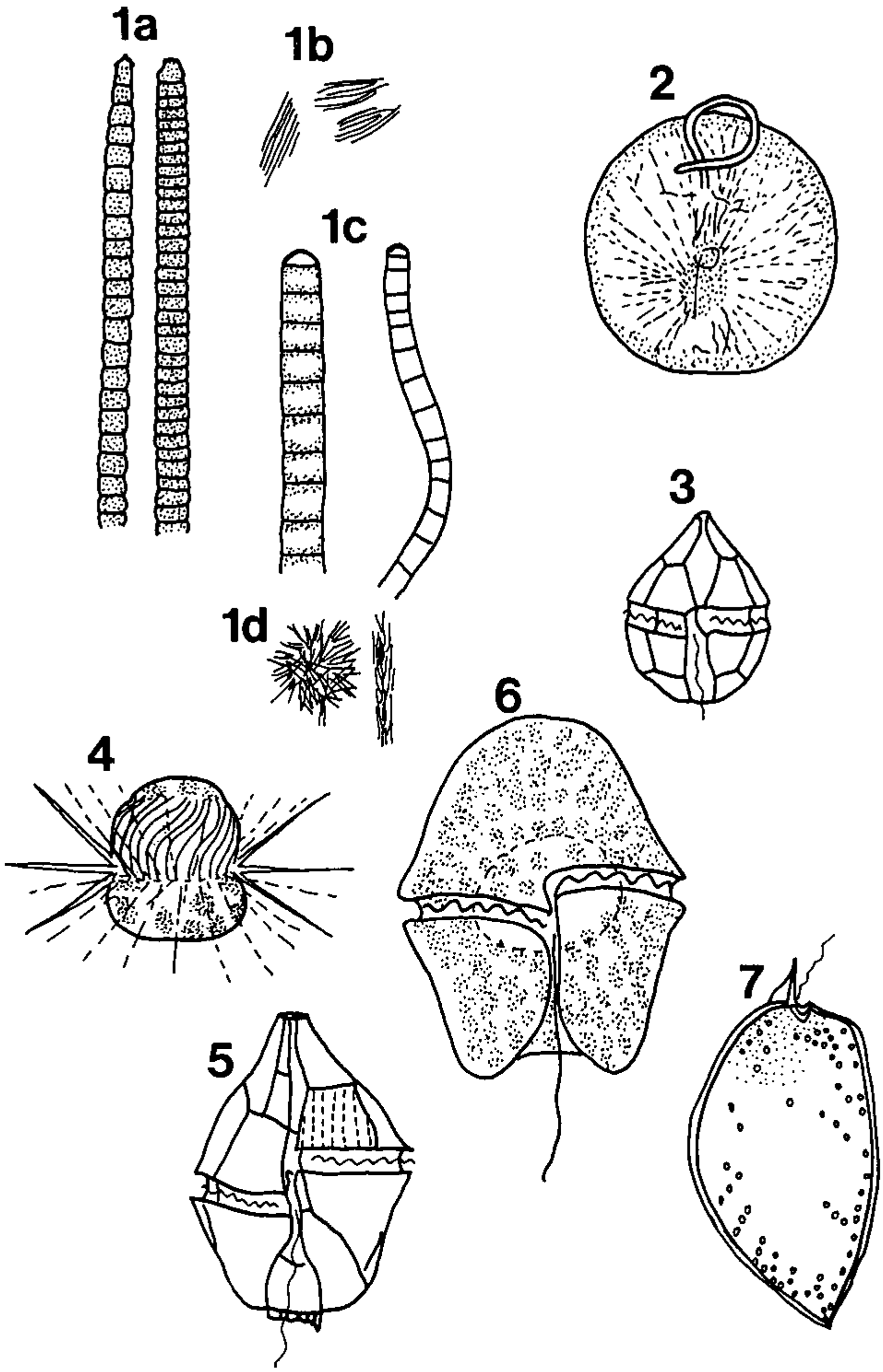
The large, strongly buoyant, non-photosynthetic alga *Noctiluca scintillans* (Fig. 2) has caused spectacular red and pink water discolorations e.g. in Lake Macquarie, New South Wales. This species is mostly restricted to coastal waters and occurs especially in the vicinity of river mouths and following heavy rainfalls. No toxic effects are known but the high ammonia content of the vacuole may irritate fish, which generally avoid the bloom areas.

Scrippsiella (dinoflagellate)

The small flagellate *Scrippsiella trochoidea* (Fig. 3) has produced red water and associated fish kills in West Lakes, South Australia (Steffensen and Hallegraeff, 1984, unpublished data). This organism has also been implicated in causing anoxic conditions and massive fish kills in Sydney Harbour in the 1890's (Whitelegge, 1891).

Other water discolourations

A wide range of other non-toxic plankton blooms are known from Australasian waters. The ciliate *Mesodinium rubrum* (Fig. 4) (containing cryptomonad algal symbionts) has caused purple water in Wellington Harbour, New Zealand (Bary, 1953) and in Parramatta River, New South Wales. The dinoflagellate *Gonyaulax polygramma* (Fig. 5) has produced red water in New South Wales estuaries, the dinoflagellate *Gymnodinium sanguineum* (Fig. 6) has produced red water in New Zealand sounds (Bary, 1953), and the dinoflagellate *Prorocentrum micans* (Fig. 7) has caused brown water in the Karamea Bight, New Zealand (Cassie, 1981).



Figs 1-7 Apart from the unarmoured flagellates, the illustrations show diagnostic features of the outer cell wall only. Flagella and cellular inclusions are not included.

Fig. 1a Blue-green alga Trichodesmium erythraeum Ehrenberg; filament 7-12 µm wide, 60-750 µm long

Fig. 1b Blue-green alga Trichodesmium erythraeum; raft-like aggregations, 1 cm diam.

Fig. 1c Blue-green alga Trichodesmium thiebautii Gomont; filament 3-16 µm wide, 1-2 mm long

Fig. 1d Blue-green alga Trichodesmium thiebautii; radiating and bundle-like aggregation, 1 cm diam.

Fig. 2 Dinoflagellate Noctiluca scintillans (Macartney) Kofoid; 260-720 µm diam.

Fig. 3 Dinoflagellate Scrippsiella trochoidea (Stein) Loeblich; 16-36 µm long, 20-23 µm broad

Fig. 4 Ciliate Mesodinium rubrum Lohmann; 10 µm diam.

Fig. 5 Dinoflagellate Gonyaulax polygramma Stein; 29-66 µm long

Fig. 6 Dinoflagellate Gymnodinium sanguineum Hirasaki; 40-80 µm long

Fig. 7 Dinoflagellate Prorocentrum micans Ehrenberg; 35-70 µm long

Species Damaging or Clogging the Gills of Fish and Invertebrates

Some species of diatoms, prymnesiophytes and dinoflagellates can harm fish or invertebrates by damaging or clogging their gills.

Mucus-producing species

Diatoms are not usually included among the red tide organisms, but several species of the genus *Thalassiosira* (Fig. 8, e.g. *T. mala, T. partheneia, T. weissflogii*) can form gelatinous masses that may harm farmed oysters by clogging their gills (Takano, 1956). Gelatinous diatom blooms are well-known from New South Wales coastal waters and the Gulf of Carpentaria (Hallegraeff, 1984). Similarly, colonies of the prymnesiophyte *Phaeocystis pouchetii* (Fig. 9) form irritant substances (acrylic acid) and mucilage. The latter can interfere with fishing by clogging gills of fish and bivalves and by fouling fishing nets (most recently in New Zealand; Chang, 1983).

Species damaging fish gills

The small, unarmoured dinoflagellate *Gyrodinium aureolum* (Fig. 10) is one of the most common red-tide organisms in North European waters (Tangen, 1983), where its coffee-brown blooms have been associated with mortality of marine farmed fishes and benthic invertebrates. The alga has a destructive effect on the lamellar epithelium of fish gills (Roberts *et al.* 1983). This dinoflagellate has been positively identified in New Zealand and Tasmanian waters, but is not known to have caused fish kills in that region. Massive fish kills in Port Phillip By in the early fifties (Wood, 1964) were associated with brown water discolourations caused by a small, unidentified *Gymnodinium* species (now thought to have been *G. nagasakiense*). Episodic fish deaths have occurred in Port Phillip Bay in recent years, but their precise cause is still unknown (Anonymous, 1984).

Toxic Species

Some twenty out of the total number of 1500 extant species of dinoflagellates are known to produce potent toxins that can find their way through the food chain to man. Seven toxic dinoflagellate taxa have been recognised in the Australasian region. In addition, toxic blue-green algae occur in eutrophic Australian freshwaters and a toxic golden-brown flagellate has caused fish kills in New Zealand.

Gambierdiscus toxicus (dinoflagellate)

The lens-like, benthic alga *Gambierdiscus toxicus* (Fig. 11) is common in the Great Barrier Reef region, where it appears in epiphytic association with bushy red, brown and green seaweeds and also free in sediments and coral rubble. Captain Cook suffered from the tropical fish poisoning "ciguatera" when visiting New Caledonia in 1774, but the causative dinoflagellate organism was only identified in 1978 (Adachi and Fukuyo, 1979). The potent neurotoxins ciguatoxin and maitotoxin accumulate through the food chain, from small fish grazing on the coral reefs into the organs of bigger fish that feed on them. Control measures include eating only smaller fish and not eating such species as red bass, coral trout, chinaman fish, barracuda, moray eel and Spanish mackerel. Nearly 500 cases of ciguatera food poisoning are known from Queensland (Tonge et al. 1967, Gillespie, 1980, Gillespie et al. 1985, 1986), and a few poisonings have also been reported from north-west Australia. No adequate treatment is yet available. The symptoms persist for months and may recur up to several years later (Table 1).

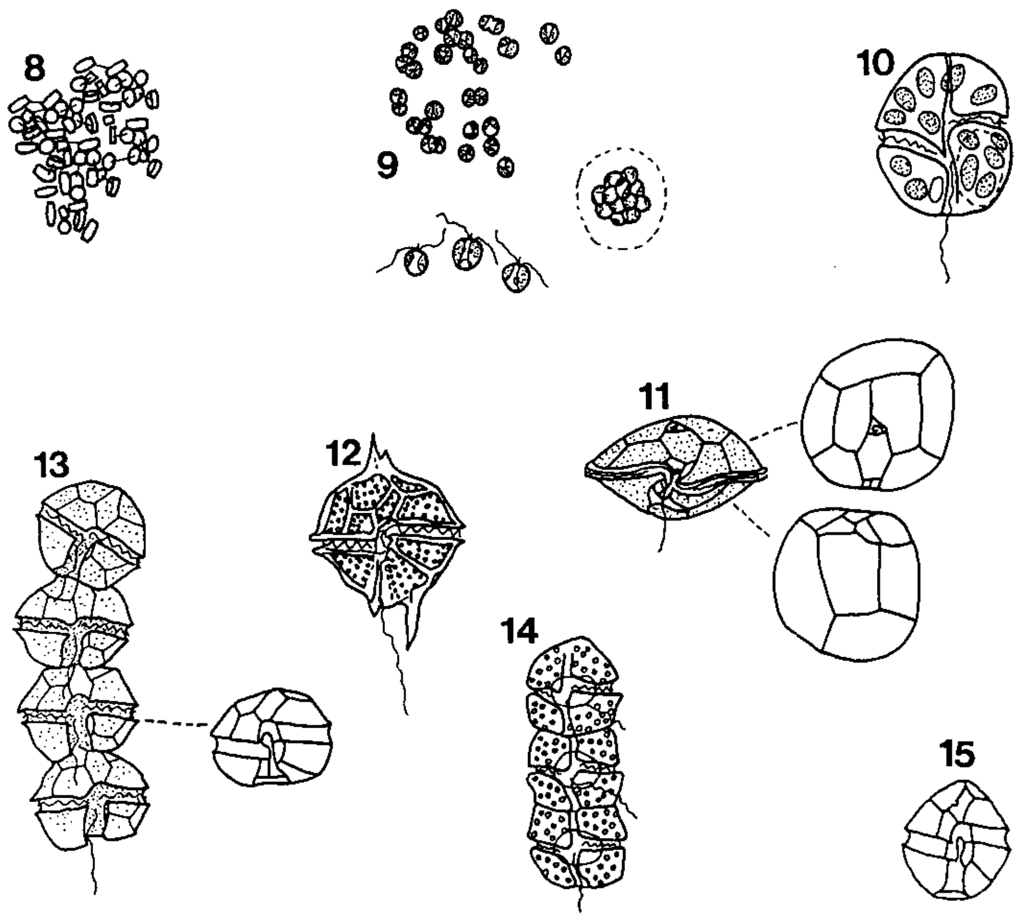
Pyrodinium bahamense var. compressa (dinoflagellate)

The tropical alga *Pyrodinium bahamense* var. *compressa* (Fig. 12) produces seasonal red tides (patches up to 300 km long) in Papua New Guinea waters (Maclean, 1977, 1979), but has not yet been observed around the coasts of northern Australia. This species can appear as single cells or in chains, and produces a spinose resting cyst that is involved in initiating the blooms. At present only the Indo-Pacific populations (var. *compressa*) are known to produce toxins, while non-toxic populations (var. *bahamense*) have been found in the tropical Atlantic (Steidinger *et al.*, 1980). The toxins include a weak ichthyotoxin (which kills fish) and an array of PSP toxins. Because of its high toxic potential and its apparent spreading through the West Indies (Indonesia, Malaysia, Phillipines), this species is considered to be the "number one" red-tide danger in the Indo-Pacific region, with over 700 human illnesses and 50 deaths attributed to it to date. Paralytic shellfish poisoning can result from eating bivalve shellfish and also planktivorous fish such as sardine and anchovy (clupeotoxicity). The toxicity of fish can be reduced by removing their gills and intestines before consumption, but shellfish can remain toxic for up to 5 months after *Pyrodinium* has disappeared from the water column.

Protogonyaulax catenella and related dinoflagellate species

The cosmopolitan dinoflagellates *Protogonyaulax tamarensis* and *Protogonyaulax catenella* (Fig. 13) cause widespread outbreaks of PSP in North America, Europe and Japan, but until recently these organisms or their relatives were unknown from the Australasian region. A toxic species similar to *P. tamarensis* (to be described as a new taxon) has been linked with fish and shellfish kills in 1983 in North Island, New Zealand (Taylor, 1984); the toxic *Alexandrium (Protogonyaulax) ibericum* (Balech, 1985; Fig. 15) caused water discolourations in 1986 in Port River (South

Australia); and *P. catenella* was identified in 1986 from Port Phillip Bay. The related chain-forming dinoflagellate *Gymnodinium catenatum* (Fig. 14) has necessitated control measures for shellfish farms in southern Tasmanian waters, and has been implicated in causing two incidents of mild human poisoning (Hallegraeff and Sumner, 1986). *G. catenatum* resembles *Protogonyaulax catenella* in morphology and PSP toxins but does not have a cell wall divided into plates (Morey-Gaines, 1982; Oshima *et al.*, 1987). PSP toxins are also known from wild mussels collected near Bateman's Bay, New South Wales (Le Messurier, 1935), but the causative dinoflagellate species was not identified. All the above species produce cysts that may seed further blooms. The toxicity of these cysts may be ten times higher than that of the motile dinoflagellate cells (Dale *et al.*, 1978).



Figs 8–15 Apart from the unarmoured flagellates, the illustrations show diagnostic features of the outer cell wall only. Flagella and cellular inclusions are not included.

Fig. 8 Diatom *Thalassiosira mala* Takano; cells 4–9 µm diameter, up to 2000 cells per colony

Fig. 9 Prymnesiophyte *Phaeocystis pouchetii* (Hariot) Lagerheim; cells 4–8 µm diam., colonies up to several cm

Fig. 10 Dinoflagellate *Gyrodinium aureolum* Hulburt; 27–34 µm long, 17–32 µm wide

Fig. 11 Dinoflagellate *Gambierdiscus toxicus* Adachi et Fukuyo; 42–140 µm diameter; ventral view and details of epithecal and hypothecal tabulation

Fig. 12 Dinoflagellate *Pyrodinium bahamense* Plate var. *compressa* Steidinger, Tester et Taylor; 34–68 µm diameter

Fig. 13 Dinoflagellate *Protogonyaulax catenella* (Whedon et Kofoid) Taylor; chain; cells 30–37 µm long, 37–49 µm diameter

Fig. 14 Dinoflagellate *Gymnodinium catenatum* Graham; chain; cells 24–34 µm long

Fig. 15 Dinoflagellate *Alexandrium ibericum* Balech; 22–29 µm long, 15–22 µm diameter

Dinophysis fortii and related dinoflagellate species

The dinoflagellates *Dinophysis fortii* and *Dinophysis acuminata* (Figs. 16, 17) are commonly encountered in temperate and tropical coastal waters, but they are seldom abundant (up to 10^3-10^4 cells l^{-1}).

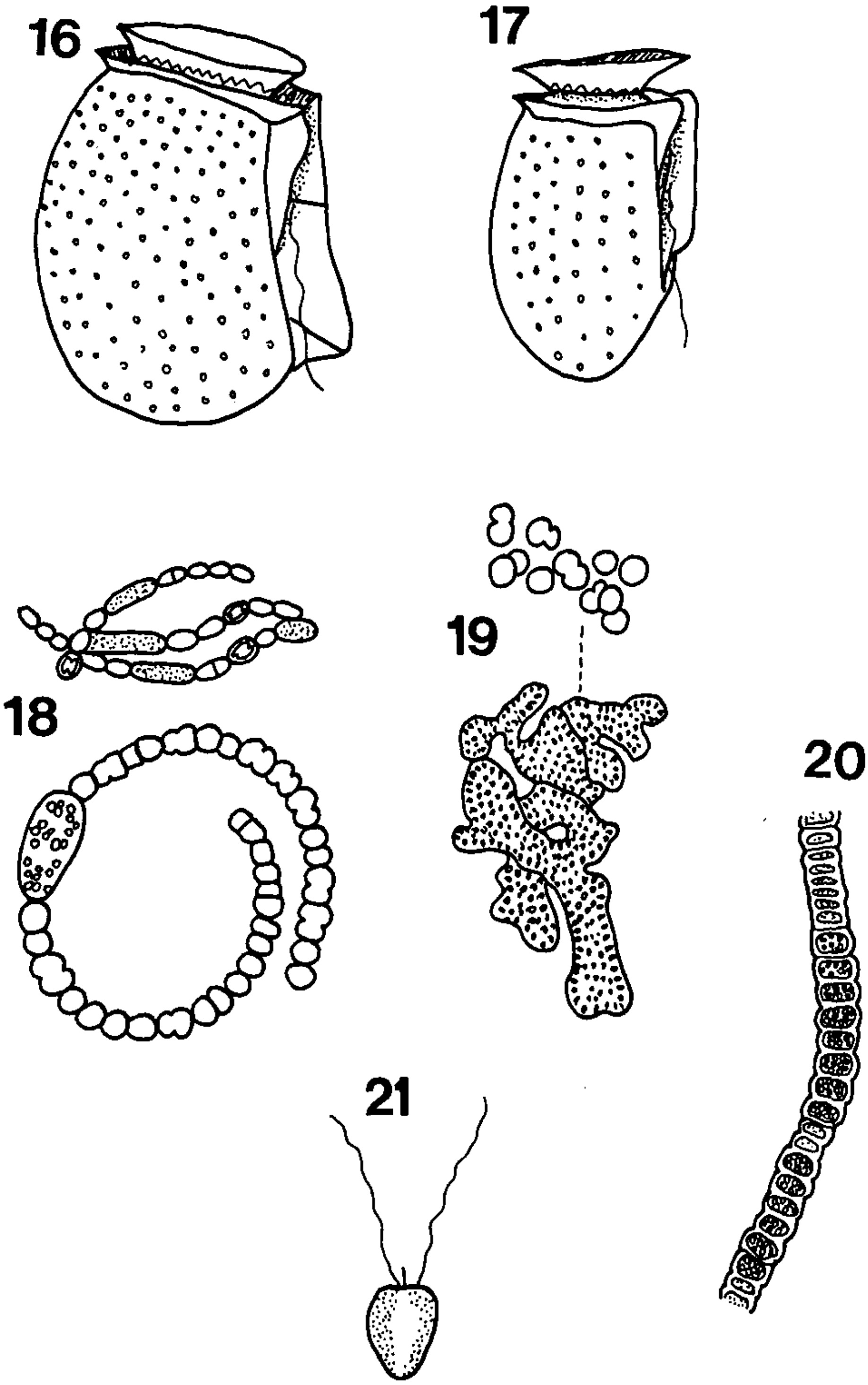
Seasonal population increases of *D. fortii* in Japan (Yasumoto *et al.*, 1978), and of *D. acuminata* in Holland, Spain, France and Ireland (Kat, 1983) have been implicated as the cause of diarrhetic shellfish poisoning (DSP). Dense *D. fortii* and *D. acuminata* blooms have also been observed in southern Tasmanian waters, and okadaic acid and dinophysis-toxin have been detected in Tasmanian mussels. Humans can be poisoned through eating mussels and, to a lesser extent, scallops and oysters. The clinical symptoms (Table 1) are easily confused with those of bacterial gastric infections; DSP (first described in 1976 in Japan) may therefore be much more common than realised at present. Control measures include seasonal closures of the shellfishery, purification of shellfish in laboratory tanks, and removal of the toxin-accumulating hepatopancreas from scallops before eating (not practicable with mussels and oysters).

Toxic blue-green algae in Australian freshwaters

The filamentous blue-green algae *Anabaena flos-aquae* (Fig. 18), *Microcystis aeruginosa* (Fig. 19) and *Nodularia spumigena* (Fig. 20) can produce water-soluble peptide and alkaloid toxins, including compounds identical to dinoflagellate saxitoxins. Water blooms of these species can kill domestic and wild animals that drink from the shores of eutrophic ponds, lakes and reservoirs (Francis, 1878; Main *et al.*, 1977).

Prymnesium (golden-brown flagellate)

The small flagellate *prymnesium parvum* produces fat-soluble ichthyotoxins, which have caused extensive fish mortalities in brackish waters in Europe and the Middle East (especially Israel). The related species *P. calathiferum* (Fig. 21) has been implicated in fish kills in New Zealand coastal waters (Chang and Ryan, 1985).



Figs 16–21 Apart from the unarmoured flagellates, the illustrations show diagnostic features of the outer cell wall only. Flagella and cellular inclusions are not included.

Fig. 16 Dinoflagellate Dinophysis fortii Pavillard; 60–70 µm long

Fig. 17 Dinoflagellate Dinophysis acuminata Claparede et Lachman; 25–47 µm long

Fig. 18 Blue-green alga Anabaena flos-aquae (Lyngby) de Brebisson; filaments 4–8 µm wide, cells 6–8 µm long

Fig. 19 Blue-green alga Microcystis aeruginosa Kützing; individual cells 3–7 µm diameter; colonies up to 1 cm

Fig. 20 Blue-green alga Nodularia spumigena Mertens; filaments 8–12 µm wide, 3–4 µm long

Fig. 21 Prymnesiophyte Prymnesium calathiferum Chang et Ryan; cells 6–10 µm long; 4–8 µm wide, flagella 11–18 µm long

CONTROL OF RED TIDES

Red tides can cause great economic damage, affecting both the seafood market and tourism. Destruction of fish and shellfish by anoxia and poisoning of fish and shellfish by toxin-producing dinoflagellates are especially critical in countries that depend heavily on mariculture for protein. Cooking and other treatments of fish and shellfish do not destroy the toxins.

The question arises as to whether these events can be artificially controlled. The various chemical and biological control methods that have been attempted have had mostly negative results. Copper sulphate was sprayed from planes to combat Florida red tides of *Ptychodiscus brevis* (Steidinger, 1983), but the killed algal cells released their endotoxins into the seawater and the decomposing algal mass generated anoxic conditions. Seed cultures of various predators, parasites or pathogens have been considered as candidates for biological control. However, the successful predator would accumulate the toxin and thus become a highly toxic vector to higher trophic levels in the food chain. Moderate-scale coastal engineering involving deepening or filling affected areas or reshaping the coastline or diverting rivers may be a viable option to combat anoxic conditions in sheltered bays. Marine dredging operations should be alerted to the possible danger of seeding benthic dinoflagellate cysts into the water column, and caution should be used when transferring shellfish from one area to another because resistant microscopic cysts could easily be carried with them. Red tides that are related to increasing eutrophication can be controlled by reducing discharge of sewerage and industrial wastes. On the other hand, phenomena such as PSP and ciguatera, which were known centuries before the modern industrial world developed, should be regarded as completely natural events.

Careful monitoring of the causative dinoflagellates, of their cysts and of associated seafood products appears to be the only solution at present. Depending upon the results, control measures may include elimination of the toxin-accumulating organs from fish or shellfish, avoidance of certain seafood species, depuration of shellfish in laboratory tanks and, in extreme cases, seasonal closures of particular fisheries.

Acknowledgments I am greatly indebted to Drs F. J. R. Taylor and K. A. Steidinger for introducing me to toxic dinoflagellate taxonomy and ecology. Professor E. Balech identified the two *Protogonyaulax (Alexandrium)* species. The following persons contributed material to this investigation: D. Cannon (Metropolitan Water Sewerage and Drainage Board, NSW), Dr N. Gillespie (Southern Fisheries Research Centre, Deception Bay, Qld), B. McGrath (NSW State Pollution Control Commission), Dr R. Lukatelich (Botany Department., University of Western Australia), Dr D. Steffensen (Engineering and Water Supply Department, SA), and Dr R. Wetherbee and Dr J. Larsen (School of Botany, Melbourne University). The Institute of Medical and Veterinary Science (Adelaide) carried out mouse bioassays, and Professor T. Yasumoto (Tohoku University, Japan) elucidated the toxin chemistry of Australian shellfish samples. Dr S. W. Jeffrey provided constructive comments on an earlier draft of the manuscript. This study was partially supported by Grant 86/84 from the Fishing Industry Research Trust Account (FIRTA).

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