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HARMFUL ALGAL BLOOMS OF THE SOUTHERN BENGUELA CURRENT: A REVIEW AND APPRAISAL OF MONITORING FROM 1989 TO 1997

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The Benguela upwelling system is subjected to blooms of harmful and toxic algae, the incidence and consequences of which are documented here. Red tides are common and usually attributed to members of the Dinophyceae, most of which are non-toxic. The incidence of these blooms varies spatially, with most blooms confined to the area west of Cape Agulhas. Cape Point forms the natural divide for species that dominate blooms of the west coast of South Africa as opposed to those that dominate the South Coast. Blooms occur most commonly from January to May, during the latter half of the upwelling season. Each red tide is associated with synoptic weather patterns, which dictate the onshore and offshore movement of dinoflagellate-dominated frontal blooms. There is also interannual variation, thought to be related to weather pattern changes. The harmful effects of high-biomass, non-toxic blooms include die-offs resulting from anoxia or hypoxia. Other effects of high biomass blooms include those that may cause mechanical or physical damage or those that may alter the foodweb. Recently, a bloom of the very small pelagophyte, *Aureococcus anophagefferens*, referred to as brown tide, in Saldanha Bay and Langebaan Lagoon resulted in growth arrest in both oysters and mussels. Toxic species cause mass mortalities of fish, shellfish, marine mammals, seabirds and other animals. Human illness is caused by contaminated seafood when toxic phytoplankton are filtered from the water by shellfish that accumulate toxins to levels that are potentially lethal to humans and other consumers. Of these shellfish poisoning syndromes, Paralytic (PSP) and Diarrhetic Shellfish Poisoning (DSP) are common in the Benguela. Confirmed cases of PSP have been attributed to the dinoflagellate *Alexandrium catenella*. Although shellfish are usually only marginally affected, in extreme cases of poisoning, mussel mortalities have been observed, and in most instances these have been attributed to blooms of *A. catenella*. Sardine *Sardinops sagax* mortalities in St Helena Bay have also been attributed to the ingestion of this PSP-producing dinoflagellate. Monitoring has revealed the presence of *Dinophysis acuminata*, *D. fortii*, *D. hastata*, *D. tripos* and *D. rotundata*, all of which have been reported to cause DSP. The dinoflagellate *Gymnodiniun* cf. *mikimotoi*, has been implicated in a type of Neurotoxic Shellfish Poisoning and human skin and respiratory irritations have been attributed to aerosol toxins produced by this species.

Although most of the phytoplankton of the Benguela upwelling system make a useful contribution to the production supporting the large fishery within this ecosystem, the region is also subjected to problems associated with blooms of harmful and toxic algae. Toxic species may cause mass mortalities of fish, shellfish, marine mammals, seabirds and other animals (Brongersma-Sanders 1948, 1957, Copenhagen 1953, Horstman 1981, Horstman *et al*. 1991), and human illness and death may result from contaminated seafood (Sapeika 1948, 1958, Grindley and Sapeika 1969, Popkiss *et al*. 1979, Horstman 1981, Horstman *et al*. 1991, Pitcher *et al*. 1993b, Pitcher and Matthews 1996). Blooms of non-toxic phytoplankton can also cause harm, as a consequence of either mechanical damage, such as the clogging of fish gill tissue (Grindley and Nel 1968, Brown *et al*. 1979), or the indirect effects of biomass accumulation, such as anoxia (Copenhagen 1953, Grindley and Taylor 1962, 1964, Pieterse and Van der Post 1967, Horstman 1981, Matthews and Pitcher 1996, Pitcher and Cockcroft 1998, Cockcroft *et al*. 2000). Both toxic and non-toxic blooms are also, in some instances, able to alter the trophic structure of marine systems, so causing trophic dysfunction (Smayda 1997, Pitcher *et al*. 1999). The scientific community now employs the term "Harmful Algal Bloom" (HAB) to describe this array of bloom phenomena (Smayda 1997).

Globally, there is a belief among many experts that the scale and complexity of the phenomenon of HABs is expanding (Anderson 1994). They note that the number of toxic blooms, the economic losses from them, the types of resources affected and the kinds of toxins and toxic species have all increased, and in many cases these increases have been related to human activities such as pollution. However, the magnitude of this expansion may be questioned and rather attributed to increased awareness and improved surveillance.

HABs are usually characterized by the proliferation and occasional dominance of a particular species of toxic or otherwise harmful alga, and in many instances these proliferations discolour the water, a phenomenon known as red tide. Such blooms are able to impact both commercial and recreational fisheries throughout

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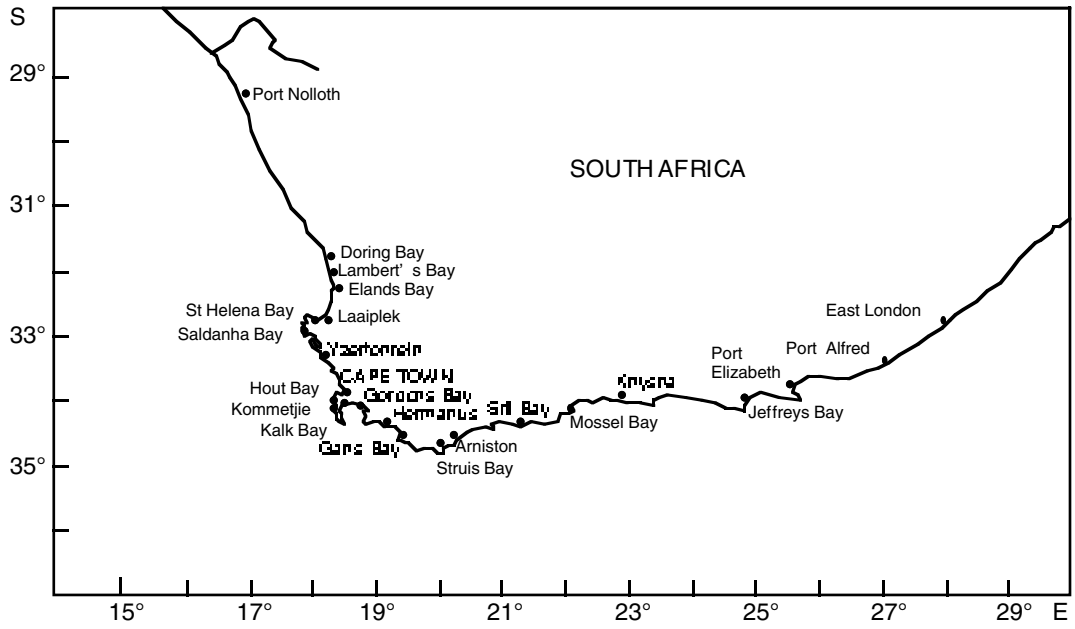

Fig. 1: Map of the south and west coasts of South Africa indicating the sites of daily sampling (Elands Bay and Gordons Bay) and the 24 stations at which Fisheries Control Officers are located

the coastal region of the Benguela, one of four major eastern boundary currents of the world's oceans. Here, wind-driven upwelling is a feature of ocean circulation and plays a critical role in the bloom dynamics of the harmful algae common to the region (Pitcher and Boyd 1996, Pitcher *et al.* 1993a, 1995, 1998). Furthermore, the coupling between this physical environment and the biological characteristics of the various harmful species holds the key to understanding many bloom phenomena.

Harmful algae bloom in the Benguela over a vast area, covering nearly 20 degrees of latitude, and most blooms are typically attributed to a group of phytoplankton known as the dinoflagellates. Their ability to swim makes them amenable to physical aggregation and, in some instances, species are capable of bioluminescence at night, further contributing to the high visibility of the blooms. There is great disparity in the amount of available information about HABs within the Benguela ecosystem, and although some of the harmful species are found throughout the region, others appear to be confined to particular areas within the region.

Since Gilchrist (1914) identified red tide as one of the factors causing fluctuations in the supply of fish in the Benguela, various research efforts have been undertaken to investigate the phenomenon. These efforts were, however, seldom sustained and only in 1989 was a HAB monitoring programme implemented in the southern Benguela. The primary objective of this programme was to provide a warning and information system to the public and the aquaculture and fishing industry. The intention of the programme was to identify those species responsible for harmful blooms and to establish the problems associated with each. It was not intended to serve the purpose of ensuring seafood safety. Information obtained on the incidence, distribution and duration of blooms, together with improved insight into the problems and risks associated with blooms, would, however, permit better planning and management options in different coastal regions. The opportunity to relate seasonal and regional occurrences of harmful blooms to particular hydrographic processes and meteorological events would be afforded and in the longer term also provide an opportunity to detect any increase or decrease in the incidence of harmful blooms.

This paper documents the incidence and consequences of HABs in the southern Benguela. In addition to providing a review of the literature, it incorporates much of the information gained from the HAB monitoring programme implemented in 1989, so providing

Table I: Algal species reported to form red tides or to be toxic

Dinophyceae
Alexandrium catenella (Whedon & Kofoid) Balech+*
Ceratium dens Ostenfeld & Schmidt+
Ceratium furca (Ehrenberg) Claparede & Lachmann+
Ceratium lineatum (Ehrenberg) Cleve+
Dinophysis acuminata Claparede & Lachmann*
Dinophysis fortii Pavillard*
Dinophysis hastata Stein*
Dinophysis tripos Gourret*
Dinophysis rotundata Claparede & Lachmann*
Gonyaulax grindeleyi Reinecke+*
Gonyaulax polygramma Stein+
Gonyaulax spinifera (Claparede & Lachmann) Diesing+
Gymnodinium cf. *mikimotoi* Miyake & Kominami ex Oda+*
Gymnodinium sanguineum Hirasaka+
Heterocapsa triquetra (Ehrenberg) Stein+
Noctiluca scintillans (Macartney) Kofoid & Swezy+
Prorocentrum balticum (Lohmann) Loeblich III+
Prorocentrum micans Ehrenberg+
Prorocentrum rostratum Stein+
Prorocentrum triestinum Schiller+
Polykrikos schwartzii Butschli+
Scrippsiella trochoidea (Stein) Loeblich III+
Bacillariophyceae
Chaetoceros convolutus Castracane‡
Pseudo-nitzschia sp. H. Peragallo‡
Raphidophyceae
Chattonella sp. Biecheler‡
Heterosigma akashiwo (Hada) Hada ex Sournia+‡
Pelagophyceae
Aureococcus anophagefferens Hargraves & Sieburth+
Haptophyceae
Chrysochromulina sp. Lackey‡
Emiliania huxleyi (Lohmann) Hay & Mohler+
Gephyrocapsa oceanica Kamptner+
Phaeocystis pouchetii (Hariot) Lagerheim‡
Syracosphaera pulchra Lohmann+
Euglenophyceae
Eutreptiella sp. da Cunha+
Ciliates
Mesodinium rubrum (Lohmann) Hamburger & Buddenbrock+

+ Red tide
* Toxic
‡ Potentially harmful or toxic

an evaluation of the threat of HABs in the region. An assessment of future monitoring and research requirements is also provided.

DATA COLLECTION

The monitoring programme has remained simple, consisting of three components:

(i) The investigation of all reported red tides. Fisheries Control Officers located at 24 stations around the South African coast have been instructed to report and sample all red tides (Fig. 1). Other reports are received from personnel of the research ships of Marine & Coastal Management, Oil Pollution vessels and aircraft, the Air Force and from members of the public. Data are presented for the period 1 July 1989–30 June 1997.

(ii) The daily collection of water samples from Elands Bay and Gordons Bay for the enumeration of phytoplankton (Fig. 1). Data are presented for the period 1 July 1992–30 June 1997.

(iii) Toxicity tests were seldom carried out routinely, but rather when toxic species were observed in water samples.

Samples for phytoplankton analysis were usually collected as surface bottle samples and fixed in buffered formalin and enumerated by the Utermöhl method (Hasle 1978). Although the focus of the monitoring programme was on the collection and analysis of phytoplankton samples, it was also necessary, particularly during toxic bloom events, to analyse for toxins. The mouse bioassay was applied to PSP-contaminated shellfish and fish by the procedure recognized by the Association of Official Analytical Chemists (AOAC 1984). A 24-h mouse bioassay, as described by Yasumoto *et al*. (1984), was used to assay collectively for the presence of DSP toxins. Alternately, a competitive enzyme-linked immunosorbent assay known as ELISA (Uda *et al*. 1988), marketed as a DSP-Check kit, was also used. Shellfish tested for NSP-type toxins were tested by means of a mouse bioassay, according to the procedures of Anon. (1985).

Although most of the time-series data presented here extend only to 30 June 1997, reference is made to events documented after this date.

RED TIDES

Species identified as responsible for red tides are listed in Table I.

Water discolourations, commonly referred to as red tides, occur throughout the southern Benguela. Although usually caused by members of the Dinophyceae, monitoring has revealed that blooms may be attributed to other species. Of these, the photosynthetic ciliate *Mesodinium rubrum* is particularly common. Notwithstanding that most of the species responsible for these blooms are non-toxic, they are nevertheless often perceived to be noxious and undesirable, and are also in some cases considered to be associated with polluted waters. Among the harmful effects of high-biomass, non-toxic blooms are die-offs resulting from anoxia or hypoxia following the decay of blooms.

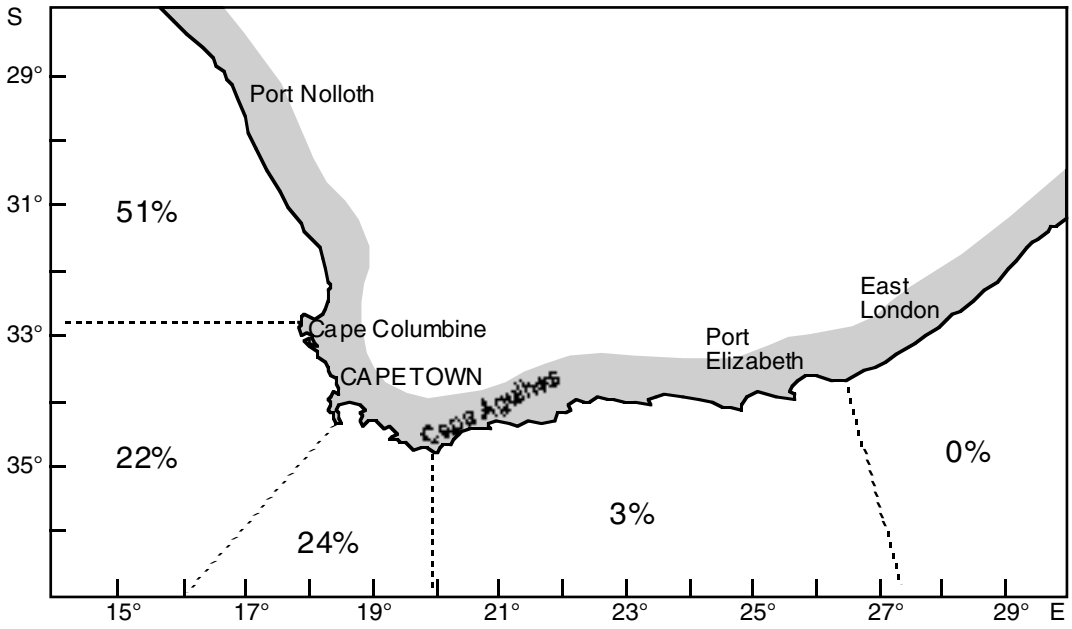

Fig. 2: Spatial distribution of reported red tides dominated by dinoflagellates (1 July 1989–30 June 1997)

Other effects include those that may cause mechanical or physical damage or those that may alter the food-web resulting in some form of perturbation to the ecosystem.

As with most phytoplankton blooms, the development of red tide results from a combination of physical, chemical and biological mechanisms, and interactions that are only partially understood. Given the diverse array of algae that produce toxins or cause problems, attempts to generalize the dynamics of harmful blooms may be ambitious. However, many red tides in the southern Benguela are attributed to various dinoflagellate species and therefore do share some common mechanisms of formation.

Dinoflagellates increase relative to diatoms as seasonal stratification increases during the course of the upwelling season (Horstman 1981, Pitcher *et al.* 1993a). Outside centres of upwelling in the southern Benguela, a broadening of the shelf is responsible for intensified stratification, so favouring a widespread distribution of dinoflagellates across the entire shelf (Pitcher and Boyd 1996, Pitcher *et al.* 1998). The population develops subsurface, in association with the thermocline. The inshore region is subjected to upwelling and the dinoflagellate population appears as a surface bloom in the vicinity of the upwelling front, which is displaced from the coast during the active phase of upwelling. There, increased light levels are responsible for enhanced production and result in dense dinoflagellate populations in and around the uplifted thermocline. Red tide forms and impacts on the coast, following relaxation of upwelling. As wind stress decreases during the quiescent phase of upwelling, cross-shelf currents become weaker and directed onshore. The return of warm, near-surface water corresponds to the onshore movement of the upwelling front and will often be accompanied by mean southward currents. Under these conditions, the dinoflagellate population accumulates inshore, where net poleward surface flow is responsible for the southward propagation of red tide.

As a consequence of these mechanisms of formation, red tide has been closely related to the prevailing winds of the Benguela, which govern most hydrodynamic processes on the continental shelf. Several meteorological patterns and cycles at the seasonal, event and interannual scales have therefore been identified with the development of red tide (Pitcher *et al.* 1995). Establishment of the importance of winds in driving the hydrodynamic processes critical to the formation of blooms provides the potential for physical parameters of environmental selection to be recognized and characterized, thereby providing a

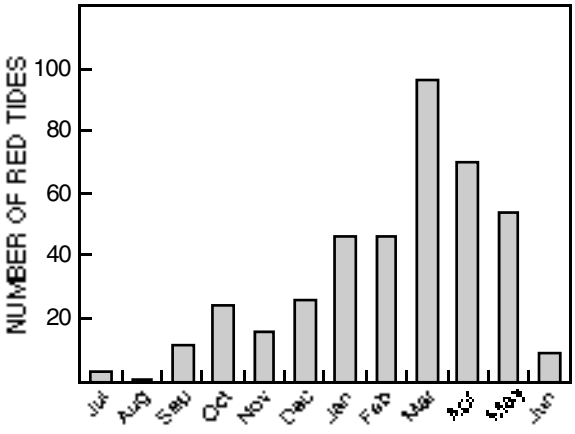

Fig. 3: Seasonal incidence of reported red tides dominated by dinoflagellates (1 July 1989–June 1997)

degree of predictive capability based on quantifiable responses to the physical environment, particularly meteorological forcings such as insolation and wind stress. The links between meteorological forcings and HABs, mediated through ocean physics and cell physiology, implies that the predictive capability for HABs should strive for, but can never exceed, the accuracy of weather and climate predictions. However, there are other forms of prediction that can also be useful, including delineation of areas susceptible to particular HAB species.

The incidence of blooms on the South African coast varies spatially (Fig. 2). Of the 403 dinoflagellate-dominated red tides investigated between 1 July 1989 and 30 June 1997, most were confined to the area west of Cape Agulhas, which is generally accepted as the southernmost extent of the upwelling system. Blooms are absent from the East Coast, whereas more than half of the blooms take place downstream of the upwelling centre off Cape Columbine, in an area where a broadening of the shelf is responsible for intensified stratification.

Of the dinoflagellate blooms reported between 1 July 1989 and 30 June 1997, most were observed during the latter half of the upwelling season, notably during the first five months of the year (Fig. 3), confirming the strong seasonal signal in the incidence of blooms in the southern Benguela. Once again, red tide activity is thought to be highest then, because thermal stratification, which favours dinoflagellates, is at a maximum as a consequence of increased solar irradiance (Pitcher *et al*. 1993a). The seasonal incidence of blooms does, however, vary spatially (Fig. 4). On the West Coast, blooms are generally first observed north of Cape Columbine. Blooms develop there from early in the upwelling season and peak in incidence in March. These blooms are generally absent farther south, until they are advected southwards, often as a "flood event", during the latter half of the upwelling season (Probyn *et al*. 2000). Consequently, the highest incidence of red tide in the vicinity of Cape Town is in April and May.

A daily time-series of dinoflagellate abundance at Elands Bay and Gordons Bay over a 5-year period provides further information on the timing and frequency of red tides (Fig. 5). At the event scale, each red tide is associated with synoptic weather patterns, which dictate the onshore and offshore movement of dinoflagellate-dominated frontal blooms (Pitcher *et al*. 1995, 1998), and hence their high variability. At the interannual scale too there is considerable variation in red tide activity, also believed to relate to weather pattern changes, including global weather patterns such as *El Niño*s in the Pacific Ocean (Pitcher *et al*. 1995). The mechanisms driving interannual variability appear to differ on the west and south coasts of South Africa. The timing of blooms on the South Coast is similar to that on the West Coast, with the frequency and magnitude of blooms greatest during the latter part of the upwelling season. However, there appears to be a tendency for blooms to persist longer in False Bay (here defined as the South Coast), into May and June.

In examining the species responsible for red tide in the southern Benguela, Cape Point appears to be the natural divide for blooms of the West and those of the South coasts. Not only do the incidence of blooms in these areas differ, but so do the dominant species (Fig. 6). On the South Coast, blooms of *Gymnodinium* cf. *mikimotoi*, *Prorocentrum rostratum*, *Prorocentrum triestinum* and *Scripsiella trochoidea* are common. On the West Coast, blooms of *Ceratium furca*, *Ceratium lineatum*, *Prorocentrum micans* and to a lesser extent *Alexandrium catenella* dominate. Only blooms of *Noctiluca scintillans* are common in both regions.

Low oxygen events

Anoxia in continental shelf waters may be ascribed to the advection of oxygen-deficient water from a remote source and from *in situ* formation. In coastal upwelling systems such as the Benguela, *in situ* formation resulting from the degradation of organic-rich material derived from phytoplankton blooms is generally thought to be the controlling factor (Bailey 1991). The inshore decay of red tides after the exhaustion of nutrients has in many instances been responsible for

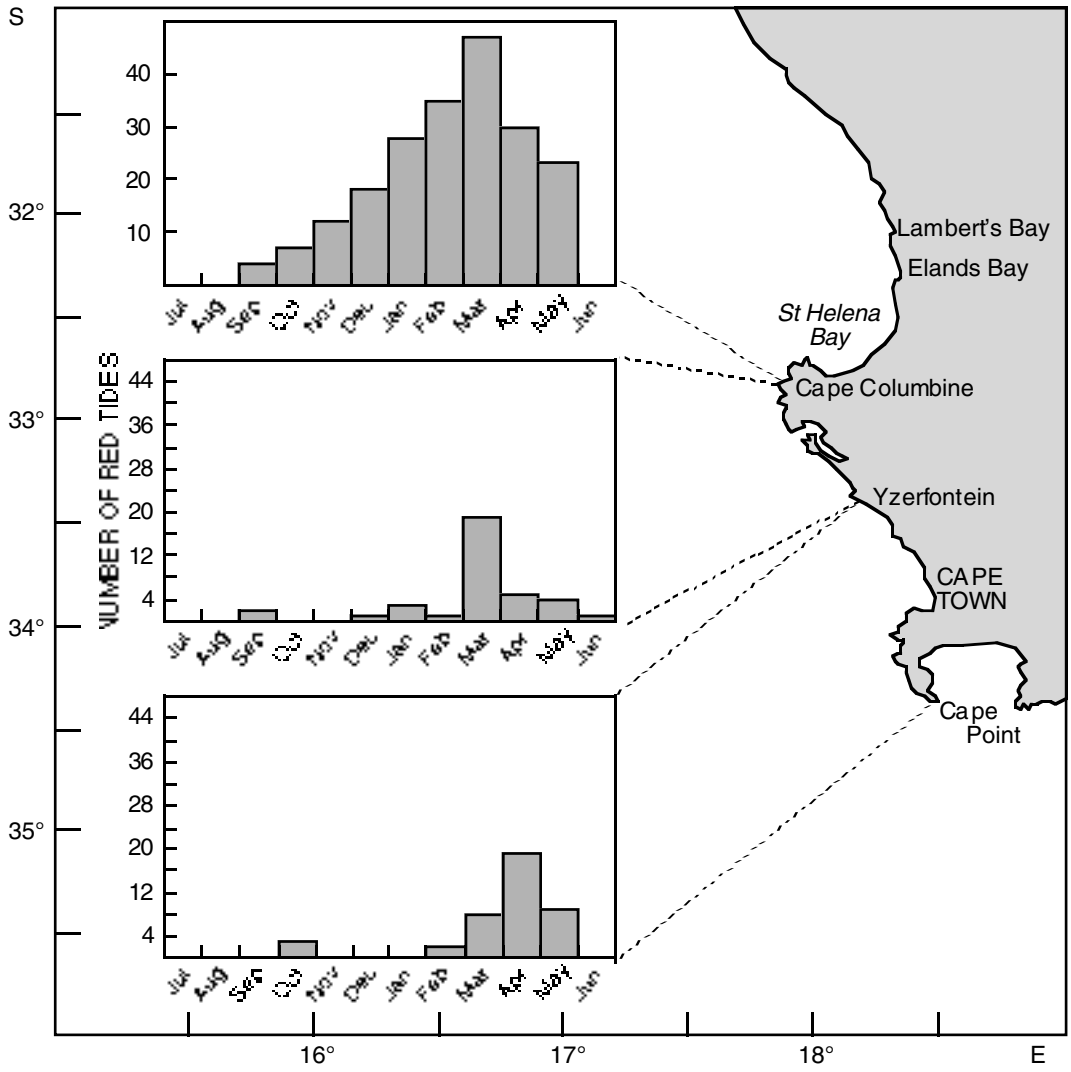

Fig. 4: Seasonal incidence of reported red tides dominated by dinoflagellates for three sectors of the West Coast (1 July 1989–30 June 1997)

marine mortalities as a consequence of oxygen depletion (Gilchrist 1914, Grindley and Taylor 1962, 1964, Horstman 1981, Matthews and Pitcher 1996, Pitcher and Cockcroft 1998, Cockcroft *et al*. 2000).

Some of the earliest accounts of mortalities attributed to red-tide-induced low oxygen events were recorded at the turn of the 20th Century by Gilchrist (1914). An incident in Stompneus Bay around 1869 was described in which geelbek *Atractoscion aequidens* were picked up from dark-red water after being found swimming with their heads above the water, apparently in a "stupified" condition. Another incident in 1907 was described in which Saldanha Bay became filled with red water, known locally as flower water, following several days of north-westerly winds. The fish in the bay were floating belly upwards in a "disabled" condition and were cast ashore in large numbers, apparently on account of the presence of large quantities of decaying matter.

In April 1978, extensive faunal mortalities, including

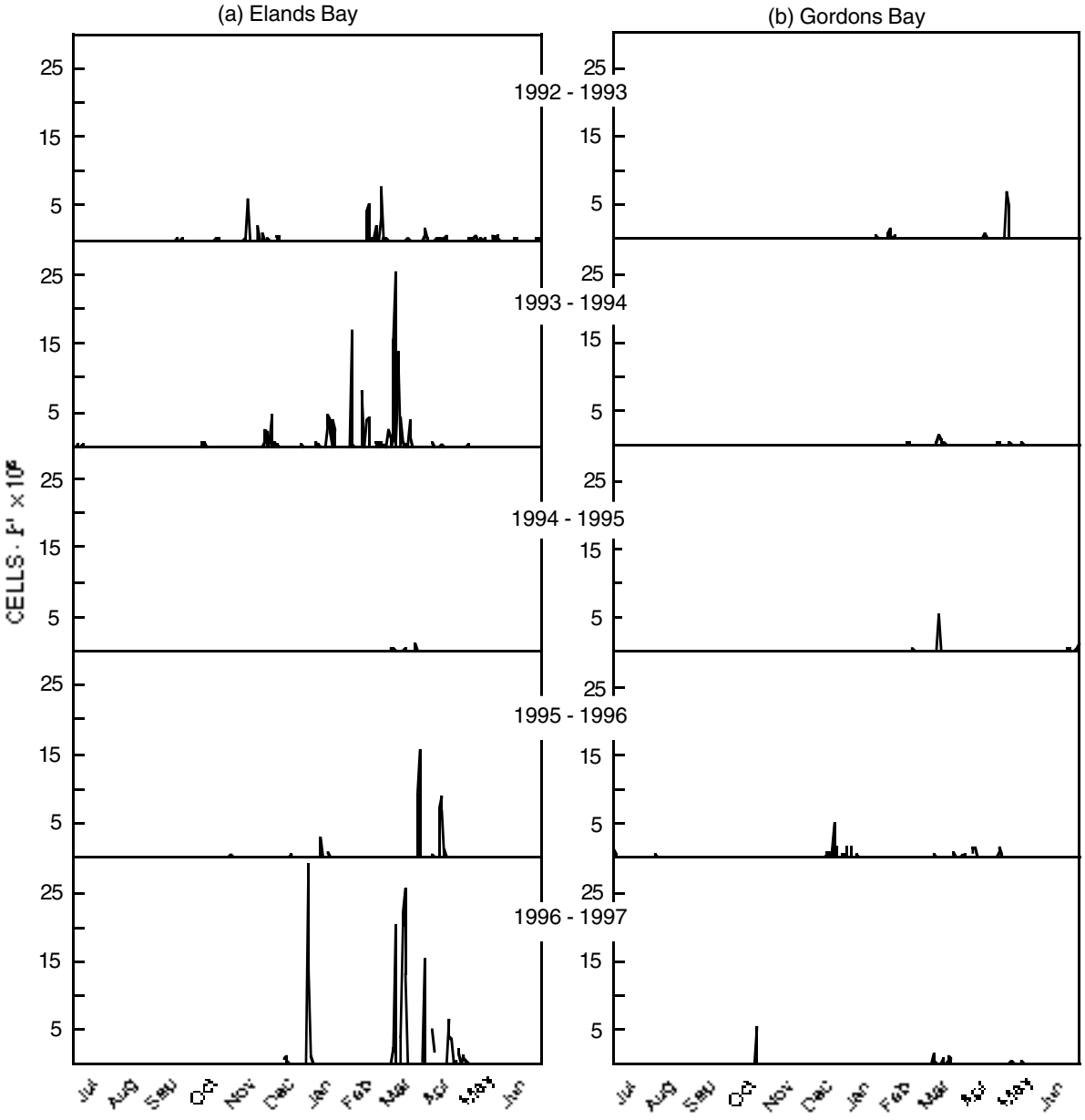

Fig. 5: Daily time-series of dinoflagellate abundance at (a) Elands Bay and (b) Gordons Bay (1 July 1992–30 June 1997)

large numbers of rock lobster *Jasus lalandii*, were attributed to the decay of a bloom of the photosynthetic ciliate *Mesodinium rubrum* in St Helena Bay (Horstman 1981). In extreme cases of oxygen depletion, the bacteria causing decay turn to sulphur as a substitute for oxygen and begin to produce poisonous hydrogen sulphide. In March 1994, a massive marine mortality was experienced in St Helena Bay as a consequence of the entrapment and subsequent decay of an expansive red tide dominated by *C. furca* and *P. micans* (Matthews and

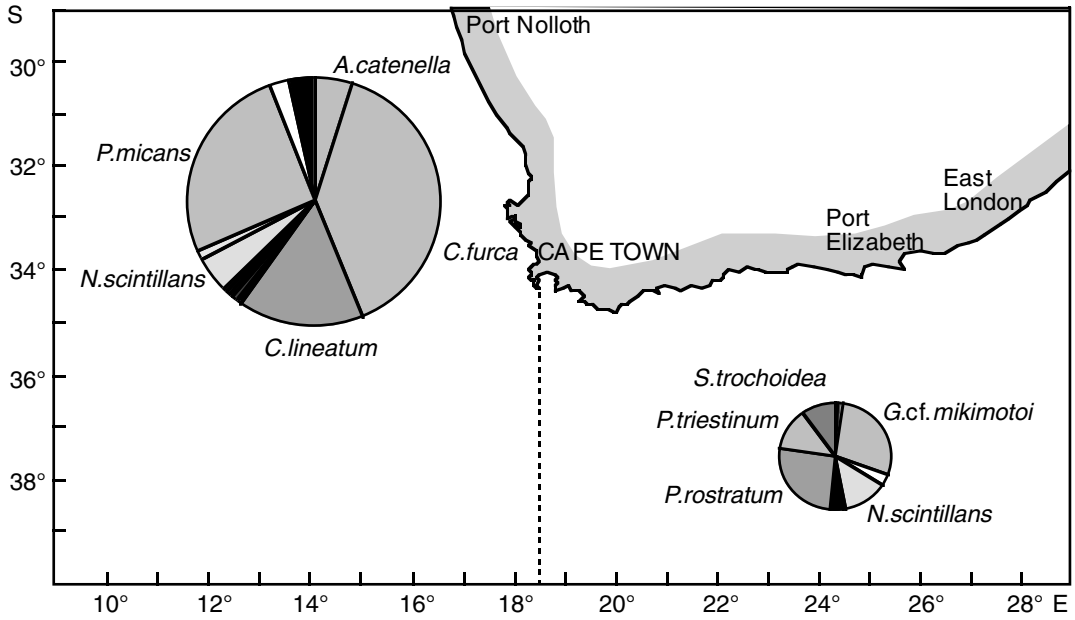

Fig. 6: Composition of reported red tides dominated by dinoflagellates on the West and South coasts (1 July 1989 to 30 June 1997)

Pitcher 1996). Marine life died from both suffocation and hydrogen sulphide poisoning. Oxygen concentrations were maintained at <0.5 m$\ell \cdot \ell^{-1}$ in the bottom waters of the bay and hydrogen sulphide, subsequently generated by anaerobic bacteria in the absence of dissolved oxygen, was recorded in excess of 50 µmol$\cdot \ell^{-1}$. Approximately 60 tons of rock lobster and 1 500 tons of fish, consisting of about 50 species, washed ashore. Mullet *Liza richardsoni* made up the bulk of the fish mortality, with the remainder dominated by sharks and bottom-dwelling fish. The production of hydrogen sulphide gas caused the sea to turn black, and the event was soon dubbed the "black tide" by the media; this was the first recorded incidence of hydrogen sulphide poisoning in the southern Benguela.

In April 1997, the largest ever stranding of rock lobster on the South African west coast followed the decay of a massive bloom of *C. furca* (Pitcher and Cockcroft 1998, Cockcroft *et al*. 2000). Exhaustion of nutrients and eventual decay of the bloom inshore led to oxygen depletion throughout the water column. As a consequence, around 1 500 tons of rock lobster were stranded as they concentrated in the shallow surf zone in their attempt to escape concentrations of oxygen on the sea bed that were below detectable limits. During the ensuing weeks, several further strandings were observed south of Elands Bay, and the total loss was eventually estimated to be 2 000 tons of rock lobster. These strandings raised concern about the economic future of the stocks in the area. To provide perspective, the total allowable catch of *Jasus lalandii* for the entire South African coast in 1997 was 1 700 tons, and of particular concern was that almost all the stranded animals were smaller than the legal minimum size. Strandings of rock lobster as a consequence of low oxygen are common on the West Coast and are most frequent in the vicinity of Elands Bay. Because rock lobsters are relatively slow-growing and long-lived, the fishery there is likely to recover slowly, unless there is extensive immigration from other areas.

Although most mortalities attributed to anoxia have been reported on the West Coast, a mass mortality of fish and invertebrates in False Bay and Walker Bay on the South Coast in April and May 1962 was caused by oxygen depletion following the decay of a bloom of *Gonyaulax polygramma* (Grindley and Taylor 1962, 1964). The sea in the Strand and Gordons Bay area was reported to have become slimy with rotten plankton, and an estimated 100 tons of dead fish and invertebrates

were removed from the beach in that area. Blooms of *Noctiluca scintillans* have also caused localized fish mortalities in False Bay, but these mortalities have been attributed to the production of ammonia (Horstman 1981).

Mechanical damage

Fish are particularly susceptible to physical damage of their gills by certain phytoplankton. For example, the diatom *Chaetoceros* has been linked to many fish kills for this reason (Bell 1961, Rensel 1993). Species such as *C. convolutus* and *C. concavicornis* have long, barbed spines, which may lodge between gill tissue and trigger the release of large quantities of mucous, which may result in death from reduced oxygen exchange. These impacts occur in various coastal regions of the world, at aquaculture sites where caged fish are unable to escape the blooms. Although the above-mentioned *Chaetoceros* species are found in the Benguela (Pitcher 1988), they have not been implicated in any fish mortalities. It is likely that these species will become problematic in the event of local development of finfish aquaculture.

In August and September 1976, a bloom of a particular *Gymnodinium* species resulted in the mortality of a wide variety of fish in False Bay (Brown *et al*. 1979). Attempts to identify the small, highly mobile species failed as efforts to preserve the organism did not succeed. Examination of the gills of the affected fish revealed clogging by the dinoflagellate as the most likely cause of the mortality. Although mice survived intraperitoneal injection of an acidic extraction of the phytoplankton, acute morbidity indicated a degree of toxicity and a possible alternative cause of the mortality.

Alteration of the foodweb

It is now recognized that the impacts of HABs extend beyond the obvious manifestations of poisonous shellfish and dead fish and include more subtle effects on trophic structure, which include processes and interactions that can alter or destroy ecosystems through time. All trophic compartments of the marine foodweb are believed to be vulnerable to harmful blooms and it has been suggested that chronic, sublethal impacts of HABs may be more important than acute impacts in altering foodwebs or causing trophic dysfunction (Smayda 1997).

Because ecosystem effects from HABs can be subtle and difficult to document, their true extent or impact is often not established. In 1985, blooms of a small pelagophyte, *Aureococcus anophagefferens*, (referred to as "brown tide" because of the resulting water colour), appeared suddenly in embayments along the U.S. mid-Atlantic Coast (Bricelj and Lonsdale 1997). These blooms caused several problems, including mortalities, recruitment failure and growth inhibition of suspension feeding bivalves. Initially, it was believed that the problems were caused by starvation as a result of inefficient feeding on the small cells, but it now seems likely that grazing avoidance was caused by the production of toxic or possible inhibitory substances on the cell surface. In addition to these effects, eelgrass beds were also decimated as a consequence of severe light attenuation caused by the blooms (Bricelj and Lonsdale 1997). During 1997, 1998 and 1999, a bloom of the same pelagophyte occurred in Saldanha Bay and Langebaan Lagoon on the west coast of South Africa (Pitcher *et al*. 1999). These blooms occurred in the latter months of summer and persisted for several weeks at unprecedented high cell densities ($>10^9$ cells$\cdot\ell^{-1}$). Their presence had similar detrimental affects on the aquaculture industry in Saldanha Bay, causing growth arrest in both oysters and mussels.

TOXIC BLOOMS

Of the many species of microscopic algae at the base of the marine food chain, a small number are able to produce toxins (Sournia 1995). Those known to be toxic or harmful in in the southern Benguela are listed in Table I. The class Dinophyceae contains the majority of toxic species, and they make their presence known in many ways, ranging from massive blooms of cells that discolour the water to dilute, inconspicuous concentrations of cells, noticed only because of the harm caused by their highly potent toxins.

Fish-killing species

In some instances marine fauna are killed by microalgal species that release toxins and other compounds into the water. The ways in which phytoplankton kill fish and other marine organisms in this manner are poorly understood. Generally, neurotoxins or, more commonly, haemolytic and haemaglutinating compounds are produced by these algae, which may cause physiological damage to gills, major organs, circulatory or respiratory systems or interfere with osmoregulatory processes (Rensel 1995).

A toxic, fish-killing gymnodinoid dinoflagellate, *Gymnodinium* cf. *mikimotoi*, has bloomed regularly

on the south coast of South Africa since it was first observed in False Bay in 1988. In 1989, it caused extensive mortalities of sub- and intertidal fauna, including an estimated 30 tons of abalone *Haliotis midae* in the HF Verwoerd Marine Reserve on the South Coast (Horstman *et al*. 1991). Faunal mortalities attributed to subsequent blooms have been small. However, in 1996, several land-based abalone farmers in Walker Bay experienced larval mortalities associated with a bloom of *G*. cf. *mikimotoi*, and the potential impact of this organism remains a particular concern to this industry (Pitcher and Matthews 1996).

Attempts to identify this species have revealed that, although it shares the characteristics of several toxic gymnodinoid dinoflagellates, it may have features that distinguish it from those species (Horstman *et al*. 1991, Pitcher and Matthews 1996). For example, the False Bay species produce aerosol toxins that cause respiratory distress in humans. Toxins of this type have been associated with *Gymnodinium breve* blooms off the west coast of Florida for many years (Steidinger 1993). The False Bay species is, however, morphologically dissimilar in that it does not possess the apical crest so distinctive of *G. breve*. Instead, it bears a greater resemblance to *Gymnodinium mikimotoi*, a common species in Japanese waters, and to the north European species *Gyrodinium aureolum*, which are now regarded as conspecific (Anderson *et al*. 1998). Morphologically, the resemblance to *G. mikimotoi* is particularly strong in that the cells are similar in size, are dorsoventrally flattened, an apical groove is sculptured on the epicone, and girdle displacement and the number and shape of chloroplasts are essentially identical. The local species, however, differs from *G. mikimotoi* in the shape and position of the nucleus. In *G. mikimotoi*, an ellipsoidal or reniform nucleus is longitudinally orientated in the left hand side of the cell, whereas the nucleus of the local species lies horizontally in the hypocone. Furthermore, although *G. mikimotoi* and *G. aureolum* have been responsible for mortalities of marine fauna, aerosol toxins such as those encountered in False Bay have apparently not been associated with these species.

Other harmful phytoplankton species known or suspected of causing fish mortalities, particularly in aquaculture, include members of the Raphidophyceae. The raphidophyte *Heterosigma akashiwo* is renowned for its ichthyotoxic blooms, and farmed fish are especially vulnerable because of their inability to avoid such blooms (Honjo 1994). *H. akashiwo* blooms in the southern Benguela, but it has yet to be associated with any harmful effects. Smayda (1998) indicated that blooms of *H. akashiwo* are frequently associated with water masses that are chemically conditioned by river runoff. The blooms reported off the South African coast appear to be associated with freshwater runoff, 75% of the blooms having been observed off the mouth of the Berg and Storms rivers or in the Knysna Lagoon. The incidence of the blooms is also unusual in that 50% of them were observed between June and September, before the main upwelling season.

Shellfish poisoning

One major category of public health impact from HABs occurs when toxic phytoplankton are filtered from the water by shellfish such as clams, mussels, oysters or scallops, which then accumulate the algal toxins to levels that are potentially lethal to humans or other consumers. Typically, the shellfish themselves are only marginally affected. These shellfish poisoning syndromes have been described as Paralytic (PSP), Diarrhetic (DSP), Neurotoxic (NSP) and Amnesic Shellfish Poisoning (ASP). Except for ASP, a new syndrome that results in short-term memory loss in victims, all are caused by biotoxins synthesized by the dinoflagellates. The ASP toxin is produced by diatoms, a group of phytoplankton that until recently was considered free of toxins.

The toxins responsible for these syndromes are not single chemical entities, but are families of compounds having similar chemical structures and effects. Most algal toxins cause human illness by disrupting electrical conduction, uncoupling communication between nerve and muscle, and impeding critical physiological processes. Symptoms do vary among the different syndromes, but are generally neurological or gastrointestinal, or both.

PARALYTIC SHELLFISH POISONING (PSP)

PSP-type illnesses in humans have been documented since the 1700s, but the cause was unknown until the late 1920s, when researchers from California connected this type of shellfish poisoning to a dinoflagellate now in the genus *Alexandrium*. Currently, 12 dinoflagellate species in the genera *Alexandrium*, *Pyrodinium*, *Gonyaulax* and *Gymnodinium* are known to produce PSP-causing toxins. Considerable progress has been achieved in the structural elucidation of PSP toxins. Since the structure of the parent compound, saxitoxin, was first described, approximately two dozen naturally occurring derivatives have been found among various organisms (Shimizu 1996). Individual dinoflagellate species do not contain all the toxins; rather they contain suites of toxins, and the combination and potency can vary depending on the geographic isolate and environmental conditions (Cembella 1998). In the Benguela, PSP is associated with the dinoflagellate

Alexandrium catenella (Sapeika 1948). This species is widely distributed in cold, temperate waters and is consistently toxic. The toxins disrupt normal nerve functions by blocking voltage-gated sodium channels, which prevent the inward flow of sodium ions, and block the generation and propagation of action potentials in individual nerve axons and selected muscle fibres, resulting in paralysis of the neuromuscular system. The resulting symptoms include tingling and numbness of the mouth, lips and fingers, accompanied by general muscular weakness and incoordination. Acute doses inhibit respiration, and death may result from respiratory paralysis.

One of the earliest possible cases of PSP in the Benguela was in 1888, when many people became ill and some died after eating shellfish collected near Cape Town (Gilchrist 1914). The waters of Table Bay were described at the time as being luminescent after dark. Also, many baboons were found dead, having apparently been poisoned after eating white mussels *Donax serra*. Species of *Alexandrium* are both toxic and capable of emitting light in response to the mechanical stimulation generated by breaking waves, indicating that *A. catenell*a was possibly the causative species.

Confirmed cases of PSP in the region, however, were only described much later, in 1948, when groups of people became ill after eating mussels collected north of Blouwberg Strand near Cape Town (Sapeika 1948). One man subsequently died and fowls that had eaten the same mussels also died. The sea was observed to have an unusual red colour the fortnight prior to the poisonings, and extracts from the mussels were later found to contain an extremely toxic principle, which was attributed to the dinoflagellate *Gonyaulax catenella* (now *Alexandrium catenella*). Ten years later, several cases of PSP were again reported, one victim having died after eating black mussels *Mytilus meridionalis* collected during May at Paternoster on the West Coast (Sapeika 1958). Following an outbreak of PSP near Cape Town in May 1978, when at least 30 cases of PSP were diagnosed (Popkiss *et al*. 1979), public health officials were warned that they were faced with two choices: to monitor shellfish toxicities or to close the coast to shellfishing altogether.

PSP bioassays conducted since 1 July 1989 indicate that PSP is confined to the West Coast, the highest incidences occurring north of St Helena Bay. A single incidence of mussel poisoning on the East Coast was reported in 1958, after a family had eaten black mussels collected north of Durban in December (Mann and Winship 1958). One of the children died 6 h after eating the mussels. The symptoms were very similar to those of PSP and, although this incidence of shellfish poisoning was ascribed to *G. catenella* (Mann and Winship 1958), it is a cold, temperate water species and unlikely to have been found on the East Coast. Observations from the present monitoring programme indicate that mussel collectors east of Cape Point are not at risk of PSP poisoning.

Daily monitoring at Elands Bay has revealed that *A. catenella* blooms in the latter half of the upwelling season during most years, although it seems to have failed to bloom during the 1992–1993 season (Fig. 7). Following implementation of the monitoring programme, some of the highest toxin concentrations in shellfish (>2 500 µg STX equiv.·100 g^{-1}) were recorded during the 1996–1997 upwelling season, possibly as a consequence of prolonged exposure to the bloom. During that period, 22 cases of PSP were recorded in people eating mussels from localities between Cape Town and Lambert's Bay on the West Coast (Muller *et al*. 1998, Pitcher and Cockcroft 1998). Two people died and one became apnoeic, but survived after being intubated and ventilated for 26 h. In most of the 22 cases, intoxication appeared 1–5 h post-ingestion and symptoms and signs cleared within 36 h. These incidents of PSP occurred despite a ban being placed on the collection of wild shellfish at that time. These cases of PSP highlight the difficulties of communicating a restriction on the collection of shellfish in a country in which many languages are spoken and in which there is a high level of illiteracy. In addition to monitoring, it is therefore crucial to communicate effectively, police and enforce these bannings.

Although shellfish are usually only marginally affected by toxic phytoplankton, in extreme cases of poisoning, mortalities of both white and black mussels have been observed on the West Coast, and in most instances these have been attributed to blooms of *A. catenella* (Horstman 1981). In one such event in 1967, a bloom extending from Elands Bay to north of the Olifants River was believed to have wiped out the entire adult white mussel population there. In a similar incident in December 1966, the death of hundreds of thousands of white mussels on the West Coast was associated with a bloom of an unknown dinoflagellate species (Grindley and Nel 1968, 1970, De Villiers 1979). The responsible dinoflagellate was subsequently described as a new species, *Gonyaulax grindleyi* (Syn: *Protoceratium reticulatum*) by Reinecke (1967). However, it was not clear what mechanism was involved in the deaths and, although toxicity tests revealed that the mussels were toxic, it was concluded that the source of the toxin was *A. catenella* because it had been observed within the vicinity. In 1974, a similar mussel mortality was again associated with a bloom of *G. grindleyi*, and in that instance mussels remained toxic for 6 months without any

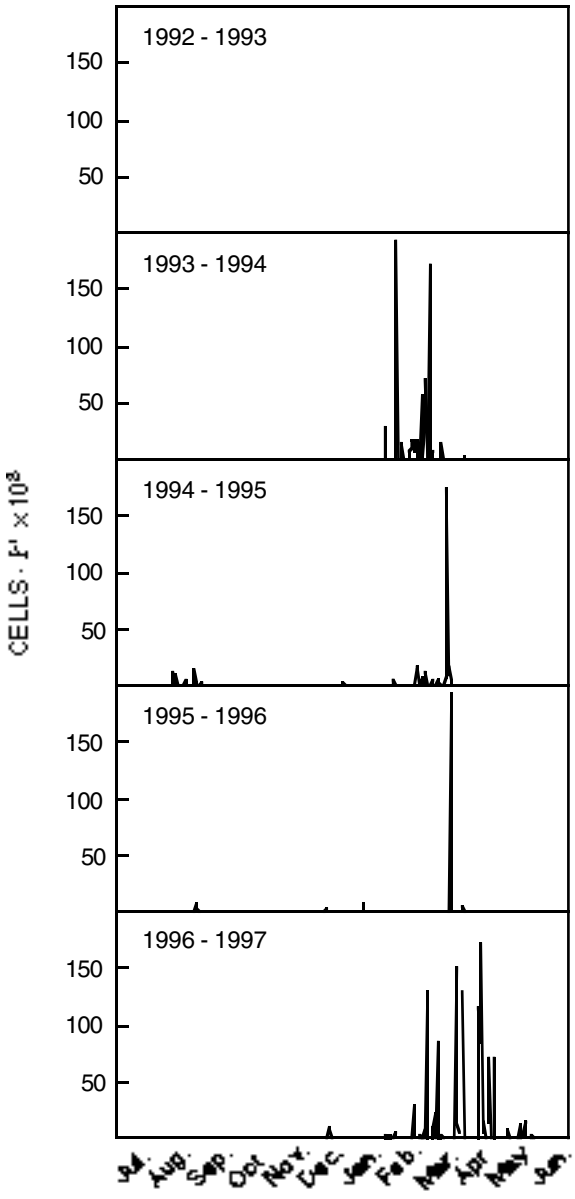

Fig. 7: Daily time-series of *Alexandrium catenella* at Elands Bay (1 July 1992–30 June 1997)

trace of *A. catenella*, indicating that *G. grindleyi* was the probable source of the toxin (Horstman 1981). Recently, the toxic properties of *G. grindleyi* have been confirmed, yessotoxins having been identified in field samples and cultured material (Yasumoto and Satake 1998).

Commercially important pelagic fish such as anchovy, herring, mackerel and sardine can also accumulate PSP toxins by filter-feeding (Haya *et al*. 1990, Anderson and White 1992). From a human health standpoint, it is fortunate that such fish are sensitive to these toxins and, unlike shellfish, die before the toxins reach dangerous levels in their flesh. In 1997, sardine *Sardinops sagax* mortalities in the St Helena Bay area were attributed to the ingestion of the PSP-producing *A. catenella*. Between 60 and 80 $\times$ 10^3 *Alexandrium* cells were found in the stomachs of these fish and analysis of the viscera revealed toxin concentrations slightly exceeding the harvestable limit of 80 µg STX equiv.·100 $g^{-1}$of viserca. This was the first confirmed case of a PSP-induced mortality of sardine in the Benguela. However, it is likely that this form of poisoning has accounted for several previously unexplained mortalities.

Just as human consumers of shellfish contaminated with biotoxins of algal origin are at risk, toxic algae may move through various components of the foodweb, affecting marine mammals and others at the top of the foodweb (Anderson and White 1992). In St Helena Bay in 1901, a particularly interesting observation of seabird mortalities was documented when hundreds of dead cormorants *Phalacrocorax* spp. were found floating close to tons of dead sardine that were drifting in "muddy-coloured water" (Gilchrist 1914). It is reasonable to conclude that these birds died after eating the sardine, which in turn had died after filtering toxic dinoflagellates. Several cases of paralysed or dead seabirds, following consumption of contaminated shellfish during incidences of PSP, have been reported by Horstman (1981).

Whales and seals may also be possible victims by ingesting PSP toxins through the foodweb via contaminated zooplankton or fish (Anderson and White 1992). In 1837, there were reports of strandings of millions of fish on the beaches of Table Bay (Gilchrist 1914). People eating the dead fish died, as did a large numbers of whales that were cast ashore around the Cape Peninsula. It is feasible that these whales died subsequent to the transfer of algal biotoxins through the foodweb. Similarly, toxic dinoflagellate blooms have been considered to be the most likely precursor to mass mortalities of seals on the Namibian coast (Wyatt 1980).

DIARRHETIC SHELLFISH POISONING (DSP)

DSP has been recognized in many parts of the world since it was first identified in 1978. Studies have shown that the DSP syndrome is usually triggered by dinoflagellates from the genus *Dinophysis* (Yasumoto 1990). Confirmation of their toxigenicity has, how-

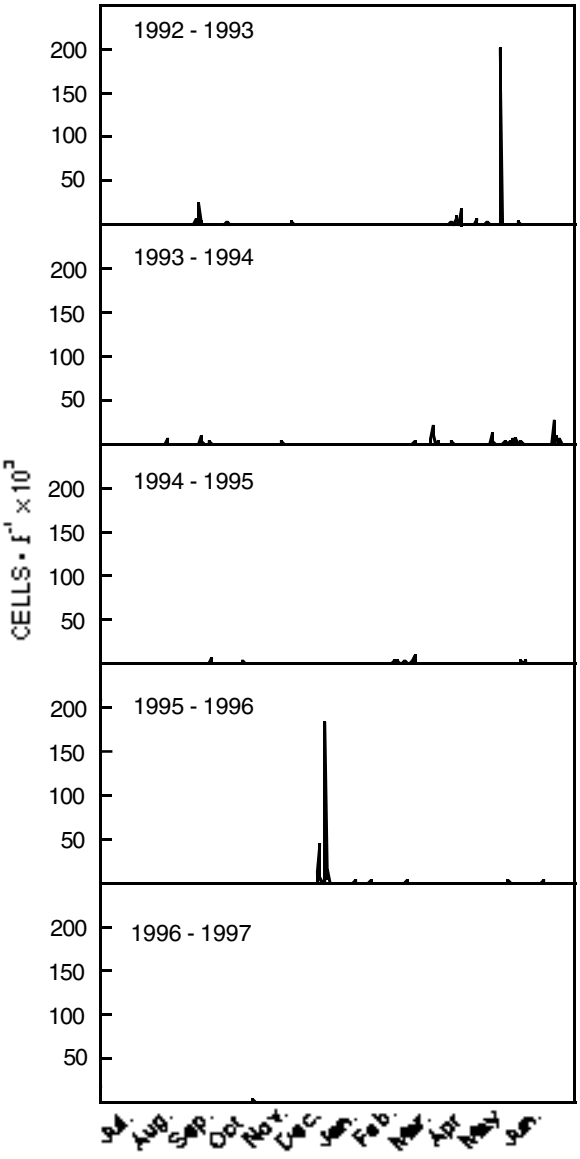

Fig. 8: Daily time-series of *D. acuminata* at Gordons Bay (1 July 1992–30 June 1997)

ever, been very difficult, because of the problems in culturing them. The DSP toxins are made up of several toxic compounds, which have been divided into three groups: okadaic acid and its derivatives, pectenotoxins and yessotoxins (Yasumoto 1990). Okadaic acid rapidly stimulates protein phosphorylation in cells and acts as a specific inhibitor of protein phosphatase in several metabolic processes. Diarrhoea is caused because okadaic acid is thought to stimulate the phosphorylation of proteins that control sodium secretion by intestinal cells (Cohen *et al*. 1990). The dominating symptoms, which typically develop within 4 h and may last up to three days, include diarrhoea, nausea, vomiting and abdominal pain (Yasumoto *et al*. 1978). It is likely that DSP has gone unreported on many occasions because of the relatively mild nature of the symptoms and the similarity of the symptoms with gastroenteritis, associated with the consumption of polluted shellfish.

DSP was first identified on the South African coast in 1991, and attributed to *Dinophysis acuminata* (Pitcher *et al*. 1993b). Monitoring has revealed that DSP is common on both the West and South coasts, and several other *Dinophysis* species that are known to cause DSP have also been recognized as a component of the phytoplankton, including *D. fortii*, *D. hastata*, *D. tripos* and *D. rotundata*.

A time-series of *Dinophysis* concentrations at Gordons Bay and Elands Bay (Figs 8 and 9) reveals their intermittent presence throughout the upwelling season, occurring in relatively low concentrations as a component of multispecific populations. Despite considerable interannual variation in cell densities, concentrations tend to peak in the latter half of each upwelling season. Analysis of mussels on the West Coast for okadaic acid, one of the toxins responsible for DSP, has shown that toxin concentrations exceed the harvestable limit of 2 $\mu g \cdot g^{-1}$ hepatopancreas for a considerable part of the year (Pitcher *et al.* in prep.). Periods of higher toxin concentrations have corresponded reasonably well with periods when *Dinophysis* is prevalent in the plankton, indicating that a concentration of approximately 2 000 cells$\cdot \ell^{-1}$ is sufficiently high to contaminate shellfish.

The widespread occurrence of *Dinophysis* on the South African coast, their presence throughout the upwelling season and the ability of low cell concentrations to render shellfish toxic demonstrates the potential for DSP to severely restrict growth in the local shellfish industry. The identification of DSP toxins as cancer promoters (Cohen *et al*. 1990) emphasizes the potential impact on public health, and further endorses the need for monitoring programmes to detect DSP.

NEUROTOXIC SHELLFISH POISONING (NSP)

NSP has been reported for many years from the south-eastern United States and eastern Mexico (Steidinger 1993). The causative species has been identified as *G. breve*, which produces nine or more polyether toxins that affect sodium channels by binding

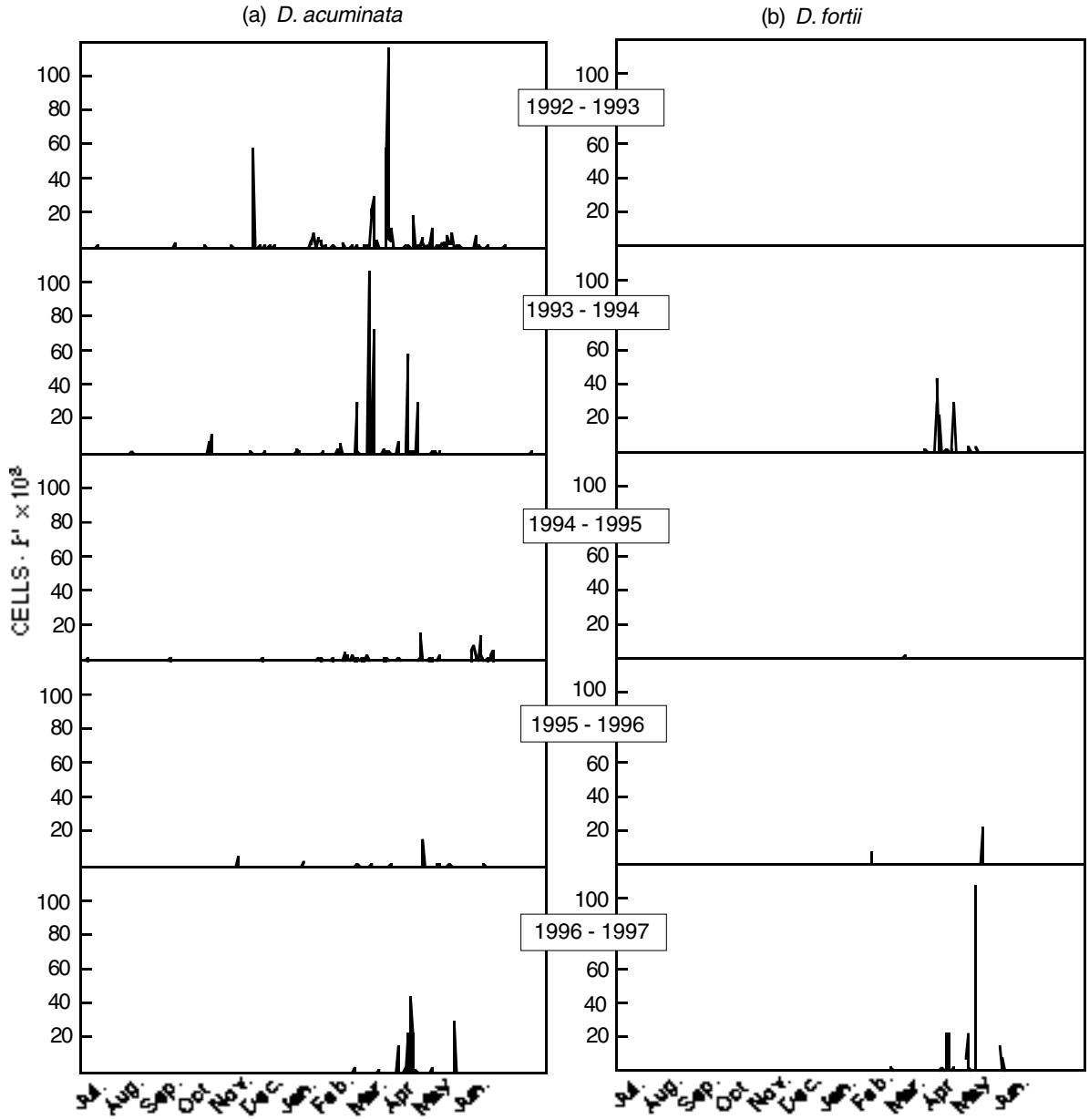

Fig. 9: Daily time-series of (a) *D. acuminata* and (b) *D. fortii* at Elands Bay (1 July 1992–30 June 1997)

to recognizable membrane receptor sites, so interfering with the transmission of nerve impulses. The symptoms of NSP are typically sensory abnormalities, but include nausea, vomiting, diarrhoea, dizziness, numbness, tingling sensations and hot-cold reversals. Another mode of human exposure is by inhalation of contaminated sea spray (Baden *et al.* 1985). Persistent coughing and sneezing are the primary debilitating

effects of the toxic spray, which causes severe irritation of conjunctivae and mucous membranes (Baden 1989).

Although *G. breve* has never been observed on the South African coast, the dinoflagellate *G.* cf. *mikimotoi*, which was recorded in local waters for the first time in 1988, has been implicated in a type of NSP (Horstman *et al*. 1991). Following a particularly conspicuous bloom in the summer of 1995–1996, mussels collected in False Bay were found to be toxic, using a mouse test for NSP toxins according to Anon. (1985). In addition to toxic shellfish, human skin and respiratory irritations have been attributed to *G.* cf. *mikimotoi*. These noxious effects to humans were particularly evident during the summer of 1995–1996. The bloom occurred at the height of the holiday season and beachgoers and seaside residents in False Bay were overcome by the discomfort of coughing, burning of the nasal passages, difficulty in breathing, stinging eyes and irritation to the skin (Pitcher and Matthews 1996). The noxious gases associated with this bloom eventually spread to Walker Bay and, although the discomforts experienced were considerable, symptoms usually subsided on leaving the area and no long-term effects were noted. Frequent or persistent blooms of *G.* cf *mikimotoi* could, however, cause considerable disruption to recreational activities on that region of the coastline. As a result of the sudden appearance of the species in 1988 in the plankton of False Bay, there were many unsubstantiated reports attributing the bloom to either the increased discharge of sewage into the bay or to the introduction of the species through ballast water discharge by damaged oil tankers seeking shelter and undergoing repair in the bay.

AMNESIC SHELLFISH POISONING (ASP)

ASP was recorded for the first time off the Canadian coast in 1987 (Subba Rao *et al*. 1988). The toxic agent was identified as domoic acid, a neuroexcitatory amino acid produced by the pennate diatom *Pseudo-nitzschia multiseries*. Symptoms of ASP include abdominal cramps, vomiting and neurological responses, involving disorientation and memory loss. Following the first outbreak of ASP, other species of *Pseudo-nitzschia* have been reported to produce domoic acid, including *P. australis*, *P. delicatissima*, *P. fraudulenta*, *P. pseudodelicatissima*, *P. pungens* and *P. seriata* (Bates *et al*. 1998). Species of the genus *Pseudo-nitzschia* are notoriously difficult to identify and, although ASP has not been recorded in the Benguela, many of the species responsible for ASP have a worldwide distribution and several are thought to occur in local waters (see Hasle 1972). Verification of ASP in the Benguela is further complicated, because the ecophysiological factors that control the production of domoic acid are not entirely understood and it is likely that the same *Pseudo-nitzschia* species that may be toxic in one part of the world are non-toxic in another (Bates *et al*. 1998).

CONCLUSION

The considerable economic, public health and ecosystem impacts of HABs are strong practical motivations for the establishment of monitoring programmes, made all the more pressing by the apparent escalating trend in their incidences. The direct benefits to society are many, as such programmes provide the information necessary to manage affected marine resources efficiently, protect public and ecosystem health, encourage and support aquaculture development, and contribute to policy decisions on coastal zone issues. Worldwide, importing countries are imposing increasingly more stringent controls on imported fisheries and aquaculture products, requiring seafood-producing countries to impose more rigorous monitoring procedures to prevent toxic seafood from entering the market (Fernández 2000). The need for, and the benefits of, the establishment and maintenance of monitoring programmes are therefore explicit.

It is now recognized that there can be impacts from harmful blooms in virtually all components of the marine foodweb, but the extent and magnitude of these impacts have yet to be established. It is therefore important to understand the physiological, toxicological and ecological mechanisms underlying the growth and proliferation of these algae and the manner in which they cause harm. Consequently, it is advantageous that monitoring programmes be accompanied by scientific programmes designed to increase understanding of the fundamental processes underlying the population dynamics and impact of HABs. This involves recognition of the many factors at the level of the organism that determine how HAB species respond to, and potentially alter their environment, the manner in which they affect or are affected by foodweb and community interactions, and how their distribution and abundance is regulated by the environment. More information is required on the ecology and oceanography of HABs, which will contribute to the ultimate goal of determining their principal causes, in order to achieve better skills at forecasting their occurrence. It is clear that regional research projects are required to address specific problems, whereas trans-boundary studies will derive benefit from comparisons among regions, and will enable common principles or mechanisms

underlying many of these phenomena to be identified. This understanding can ultimately be used to further improve the efficiency of monitoring programmes.

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