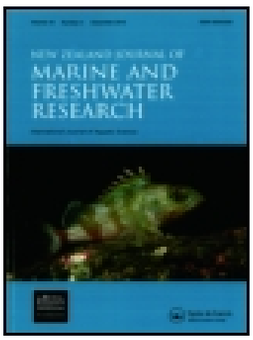

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Suffocation of pilchards (Sardinops sagax) by a green microalgal bloom in Wellington Harbour, New Zealand

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Short communication

Suffocation of pilchards (*Sardinops sagax*) by a green microalgal bloom in Wellington Harbour, New Zealand

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Abstract A bloom of the green microalga *Tetraselmis* sp. in a small lagoon in Wellington Harbour, New Zealand, is considered to have caused a fish kill of pilchards (*Sardinops sagax*). The fish deaths are attributed to anoxia brought about by a combination of reduced dissolved oxygen levels and the microalgae sticking to and clogging the secondary gill lamellae of the fish.

Keywords Prasinophyceae; microalgae; fish kill; histology

INTRODUCTION

On 10 December 1993 an estimated half tonne of pilchards (*Sardinops sagax*) were reported dying in a small lagoon in Wellington Harbour (Fig. 1). Reports from fishers indicate that most deaths occurred shortly after low tide at 0738 hrs. Swimming fish were very lethargic, mouthing at the surface, with most already dead. Only pilchards were affected, fish close to the bottom, including the spotty *Notolabrus celidotus* and triplefins (Tripterygiidae), were apparently unaffected. Dissolved oxygen concentrations in the water were low and large numbers of a small green microalga were observed in water samples and adhering to the fish gills.

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Three marine fish kills have been associated with algal blooms in New Zealand waters. In the summer of 1983 fish and shellfish died at Bream Bay, north of Leigh during a bloom dominated by the non-toxic diatom *Cerataulina pelagica*. The deaths were attributed to anoxia (Taylor et al. 1985) though the toxin producing prymnesiophyte *Prymnesium calathiferum* was also implicated (Chang & Ryan 1985). A small number of sea-caged quinnat salmon (*Oncorhynchus tshawytscha*) died at Akaroa in March 1987 during a bloom of the dinoflagellate *Gyrodinium aureolum* and mortalities of caged quinnat salmon at Stewart Island were associated with a bloom of *Heterosigma akashiwo*, a toxic flagellate (Boustead et al. 1987; Boustead et al. 1989; Chang et al. 1990; MacKenzie 1991). In Australia red tides caused by dinoflagellates similar to *Cochlodinium helix* have been implicated in fish kills in Queensland, and *Gymnodinium* spp. have been associated with fish kills in New South Wales, Tasmania and Victoria (Hallegraeff 1992). The Wellington Harbour fish kill is the first record of a green microalga suffocating fish through its size and adhesive properties.

MATERIALS AND METHODS

Ten moribund pilchards were taken for examination, and water samples were collected from five sites around the Frank Kitts Park lagoon in Wellington Harbour. Fish were autopsied, samples of gill tissue were fixed in 10% formalin, embedded in wax, sectioned and stained with Haematoxylin and Eosin using standard techniques. Water samples were taken from 5 locations around the lagoon and were fixed with 1% Lugols iodine. A further set of unfixed water samples were collected later in the day. Subsamples (10 ml) were preserved in buffered formalin (c. 2 % final concentration) and settled for 4 h in Utermöhl counting chambers. We tested the time required for settlement and found a minimum settling time of 4 h to be perfectly adequate. Organisms were identified and total counts of the

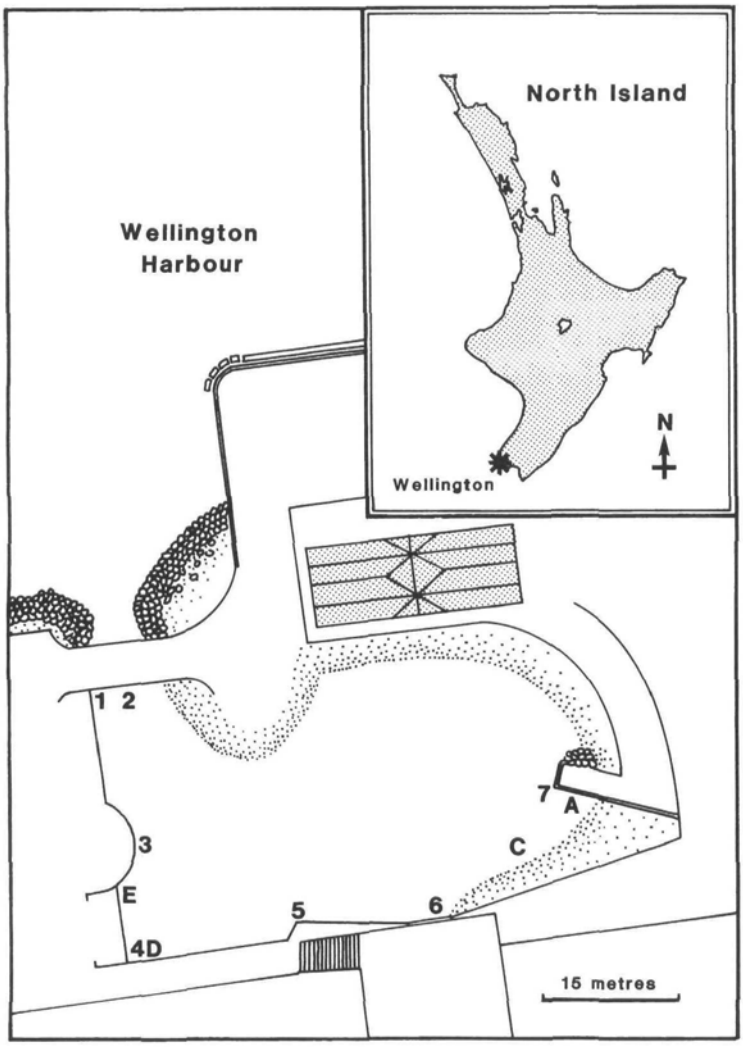

Fig. 1 Frank Kitts Park Lagoon, Wellington Harbour, showing sampling sites. Inset shows location of Wellington, North Island, New Zealand. Sampling sites 1 to 7 and A to E as in Tables 1 and 2. Site B (not shown) is beneath A.

dominant species carried out using an Olympus IMT–2 inverted microscope. Dissolved oxygen readings, obtained from a portable meter, were provided by Wellington Regional Council staff.

RESULTS

The fish, measuring 12–14 cm fork length, showed slight haemorrhage and moderate mucous production from the gills. Gills were a normal colour. Fresh smears of the gill showed the presence of large numbers of small green microalgae. No other pathology was evident. On examination of the sectioned gill tissue, the gill filaments on all fish were found to be clogged with microalgae stuck between the filaments (Fig. 2). Some hypertrophy and oedema of the epithelium was noted, particularly at the base of the secondary lamellae.

Wellington Regional Council measurements of dissolved oxygen levels in the lagoon at 1100 h are shown in Table 1. The highest reading, at the entrance to the lagoon on an incoming tide, was 7.0 to 7.5 ppm (temperature 15°C). The lowest readings were on the surface at the furthest point from the entrance (1.2 ppm), and 2.0 ppm where the bulk of the dead fish floated (between sites 4 and 5, Fig. 1).

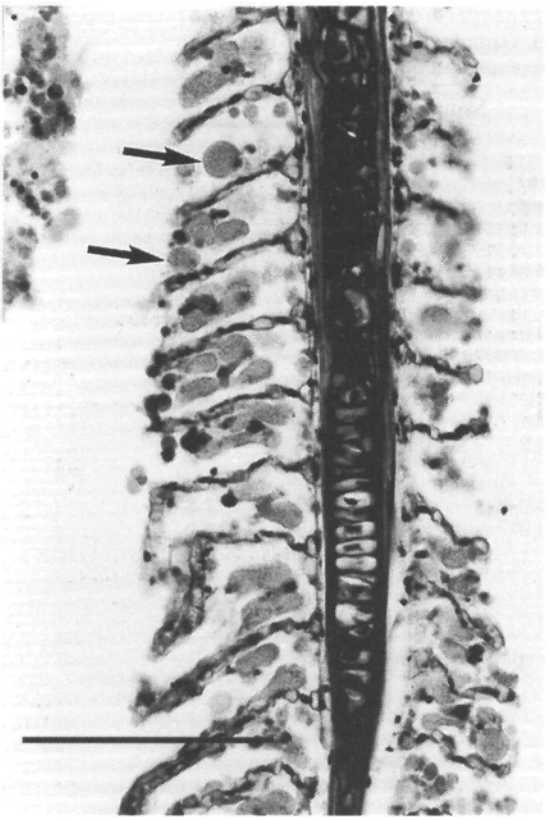

Fig. 2 Section of affected gill filament showing microalgae (arrows) adhering to filaments. scalebar = 80 µm.

Table 1 Dissolved oxygen measurements from Frank Kitts Lagoon, Wellington Harbour, 1100 h on 10 December 1993. Sampling sites as shown on Fig. 1. Temperature range 15.0–15.8°C. Repeat measurements in brackets. (Data supplied by Wellington Regional Council).

Sample site	Location	Dissolved Oxygen (ppm)
1	under bridge (shoreline)	3.9
2	under bridge (channel)	7.4 (7.0) (surface) 7.2 (7.5) (1 m depth)
3	footpath	2.3 (surface) 3.4 (1 m depth)
4	beside boat ramp	2.0 (0.5 m depth)
5	by overbridge	1.8 (surface) 1.8 (1 m depth) 2.6 (bottom)
6	between bridge and beach	1.9 (surface) 1.2 (1 m depth)
7	small wharf	1.2 (1.2) (surface) 1.9 (1.9) (1 m depth)

The water samples contained large numbers of small (7–10 µm), green, quadriflagellate microalgae which fitted the description of the genus *Tetraselmis* Stein, 1878 (Class: Prasinophyceae in Chrétiennot-Dinot (1990, p 66–74)). The genus was not *Pyramimonas*, although there were cells of this genus present (Table 2). In live samples the microalgae were observed, under the inverted microscope, attaching to the walls of the plastic sample bottles. Low numbers of the ichthyotoxic species *Amphidinium carterae* (Dinophyceae) and *Heterosigma akashiwo* (Raphidophyceae) were also present (Table 2).

DISCUSSION

Phytoplankton blooms are a major international problem for fish and shellfish industries. Five mechanisms whereby microalgae can kill fish have been identified in the literature: mechanical damage to gills caused by spines of algae species such as *Chaetoceros* sp. (Yang & Albright 1992); asphyxiation caused by oxygen depletion (Holmes & Lam 1985); gas bubble trauma from extreme oxygen supersaturation (Renfro 1963); chemical toxicity caused by ichthyotoxins (Roberts et al. 1983; Black et al. 1991); and increased seawater viscosity due to secretion of mucilages (Hallegraeff 1992).

Fish deaths have not previously been recorded in the Frank Kitts Park lagoon. On the morning of the kill the oxygen levels in the lagoon were low, particularly at the surface. This indicates that the reduction was brought about by the green microalgae rather than decomposition of benthic material which would create low oxygen levels near the substrate. Dissolved oxygen concentrations in fish ponds are usually lowest near dawn, increase during daylight hours to a peak in the afternoon (Boyd 1982). Normally fish are able to avoid areas of low oxygen concentration unless physically caged (Richardson 1989). In this incident fish were free to swim out of the lagoon, the entrance to which the fish would have located by swimming away from the areas of lower oxygen saturation. However, the clogging of the gills by the adhesive microalgae would have rapidly reduced oxygen absorption across the gill surfaces and would possibly overcome the ability of the fish to detect and avoid the oxygen depleted water. The distinctive breaks in the gill lamellar epithelium reported for trout (*Salmo trutta*) gills exposed to low oxygen levels (Drewett & Abel 1983) were

Table 2 Counts of microalgae in water samples from Frank Kitts Lagoon, Wellington Harbour, 10 December 1993. Sample sites as shown in Fig. 1. Cells $\times 10^3$ per litre. Sample B from 0.5 m depth, all others from 0.1 m depth.

	Sample site				
	A	B	C	D	E
	small wharf	small wharf	beach	boat ramp	footpath
Prasinophyceae					
Tetraselmis sp.	1484.2	773.0	1303.1	1987.7	1674.8
Raphidophyceae					
Heterosigma akashiwo	0.2	0.0	0.0	0.0	0.0
Dinophyceae					
Alexandrium sp.	0.0	0.2	0.4	0.4	0.2
Amphidinium carterae	0.4	0.4	0.2	2.4	5.4
Ceratium furca/tripos	0.4	0.8	0.2	0.8	0.0
Diatomophyceae					
Cerataulina pelagica	3.4	0.0	1.4	6.2	1.8
Benthic diatoms	16.0	10.0	8.0	6.5	4.0
Euglenophyceae					
Euglena sp.	0.0	0.2	2.3	0.8	0.2
Other small flagellates*	348.0	386.5	326.0	497.0	558.5

*small flagellates included *Cryptomonas* sp. and *Pyramimonas* sp.

not seen in the pilchards examined but the slight haemorrhage seen at autopsy indicates that some disruption of the epithelium had occurred.

In the incident reported here, *Amphidinium carterae* and *Heterosigma akashiwo* were also present but occurred in low numbers (Table 2). Exposure of juvenile *Oncorhynchus tshawytscha* to the ichthyotoxin of *Heterosigma akashiwo* has been described by Black et al. (1991). They found no pathological changes to the gills, no excess mucous, and though algae were present in the interlamellar and interfilamental spaces, they were not in direct contact with the lamellar epithelium. The authors hypothesised that *H. akashiwo* kills by production of an ichthyotoxin which leaves no histological changes to the gills of affected fish. However, Chang et al. (1990) reported that gills of salmon (*Onchorhynchus tshawytscha*) exposed to *Heterosigma akashiwo* showed a pathology ranging from mild oedema and epithelial cell hypertrophy to severe acute exudative and degenerative changes. No algal cells were found between the primary lamellae. Exposure of *Oncorhynchus mykiss* to *Gyrodinium aureolum* (= *G. mikimotoi*) causes a similar gill lesion consisting of a generalised necrotizing degeneration of the gill epithelium of the secondary lamellae with associated sloughing. This was accompanied by swelling and pyknosis of the primary lamellar epithelium and congestion of the branchial vessels (Jones et al. 1982; Roberts et al. 1983).

The involvement of an algal ichthyotoxin in the mortality cannot be completely dismissed due to the presence of the ichthyotoxic dinoflagellate *Amphidinium carterae*, although this species was present in very low numbers. However the green microalgae is implicated by its small size and its abundance. The cells were smaller than the interlaminar distance of the pilchard gill filaments and so could enter and clog the filaments. The adherent properties of the microalgae flagella, as observed in the live cultures, would have aided the clogging. The related species *Platymonas* (=*Tetraselmis*) *convolutae* is noted for the adherent nature of its flagella (Boney 1970). The oxygen level readings in the lagoon and the involvement of only the pilchard school are all indicative of a kill caused by a combination of mechanical asphyxiation and reduced oxygen levels caused by the green microalgae.

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