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Paralytic Shellfish Poisoning in Papua New Guinea, 1972[1]

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ABSTRACT: Outbreaks of paralytic shellfish poisoning and simultaneous red tides (*Pyrodinium bahamense* Plate) in the Port Moresby district during 1972 are recorded. Bioassays of various bivalve shellfish during and subsequent to the red tide "season" revealed different retention times of toxin in different species. Samples of the red tide elicited the same paralytic poisoning effects in mice as did toxic shellfish extracts.

THERE HAVE BEEN seven known deaths and 125 recorded hospitalized cases from paralytic shellfish poisoning in Papua New Guinea up to 1971. All have been on the north coast and outer islands (Maclean, in press). The south coast of mainland Papua was thought to be free from the problem. However, in mid-1972 there occurred in Papua one of the most serious outbreaks of paralytic shellfish poisoning on record, resulting in the death of three indigenous children and hospitalization of 20 adults. These casualties of paralytic shellfish poisoning followed the consumption of the bivalve shellfish *Anadara maculosa* (Reeve) that had become contaminated with the dinoflagellate *Pyrodinium bahamense* Plate during a "red tide" bloom.

Seven distinct outbreaks of paralytic shellfish poisoning were reported during the period March to July 1972 among coastal people in the vicinity of Port Moresby. Three of these instances have been diagnosed retrospectively.

On 27 April 1972, at Walai, a coastal village 80 km southeast of Port Moresby, two indigenous boys, aged 4 and 6 years, died 9 and 15 hours respectively after eating boiled *Anadara maculosa*. Two other villagers were admitted to the hospital at this time and were diagnosed as suffering from the same form of poisoning. The symptoms exhibited were a tingling sensation in the lips, hyperactivity, ataxia, and mild convulsion. Death resulted from respiratory paralysis (Popei, Mills, and Rhodes 1972). These symptoms are those of paralytic shellfish poisoning (Halstead 1965: 174–185).

On 29 April 1972, at Walai, following the ingestion of the same shellfish species, a girl aged 6 years died and 18 patients with paralytic shellfish poisoning symptoms were admitted to hospital. In all, 23 of the 38 people who ate shellfish at Walai between 27 and 29 April were affected.

In mid-May 1972, four people, including two children, were admitted to hospital with symptoms of paralytic shellfish poisoning. All had eaten the bivalve shellfish *Anadara maculosa* and/or *Spondylus* sp. gathered near separate villages in the Port Moresby area.

In late July 1972, a single mild case of paralytic shellfish poisoning was reported following the ingestion of oysters, *Crassostrea echinata* (Quoy & Gaimard), collected from Port Moresby wharf.

METHODS

At the time of the fatalities shellfish samples were obtained from Walai village (both freshly collected and boiled samples from the 27 April death house), from a number of beaches in the Port Moresby area, and later from Port Moresby wharf. The shellfish collection included a wide range of species, for the greater part bivalves. Collection of selected toxic species was continued until bioassays proved negative.

Shellfish samples were extracted and analyzed following the Association of Official Ana-

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lytical Chemists procedure (Horwitz 1970: 305). From one to six specimens of each species were extracted, the number being determined by the specimens' size. Red tide was bioassayed by injection of an aqueous solution containing the organisms.

Interpretation of the toxin level in terms of micrograms per 100 grams of shellfish meat as suggested in the above analytical procedure was not possible in this study due to the nonavailability of sufficient mice of suitable weight, and the lack of a standard toxin sample.

Toxicity was rated on an arbitrary three-point scale. A mean death time of mice of less than 7 minutes was regarded as toxic (+ +). This level of toxicity was found in the initial samples from the Walai death meals. Death times between 7 and 20 minutes were regarded as representing mild toxicity (+); mice surviving longer than 20 minutes did not subsequently die from effects of the toxin (0).

For each test, 6 to 10 mice were injected with shellfish extract.

A permanent plankton station was commenced in Port Moresby harbor on 2 June 1972, after the red tides had ceased, to monitor concentrations of *Pyrodinium* each fortnight. A 48-micron, 15-cm-diameter net was used for plankton hauls.

Aerial red tide surveys were made fortnightly from May to December 1972, between Port Moresby and Walai village (Figure 1).

RESULTS

The results of the toxin bioassays are shown in Table 1. Affected mice exhibited hyperactivity, convulsions, ataxia, and death by respiratory paralysis. Autopsies were performed to ensure that death was not due to other causes. Control injections of extractant acid and of mackerel (*Cybium commersoni* [Lacépedè]) flesh extract produced no toxic symptoms or death.

Toxin was not detected in the gastropods examined, but most bivalves from all areas sampled at the time of fatalities were toxic, as was the red tide solution. Three bivalves were not toxic at this time: *Tapes hiantina* (Lamarck), *T. literata* (Linnaeus), and *Gafrarium tumidum* (Röding). Of the toxic species, *Anadara maculosa* and *Modiolus auriculatus* (Krauss) were non-toxic when bioassayed in July 1972. However, *Spondylus* was still highly toxic in July and *Crassostrea echinata* was still mildly toxic until at least September.

Red tides were found during the first aerial survey (17 May) in coastal waters between Port Moresby and Walai. The extent of water discoloration is shown in Figure 1. Approximately 40 kilometers of the coastline were tinged faintly brown (diffuse zone) interspersed with orange-brown streaks from 0.1 to more than 1.0 kilometers long and 10 to 50 meters wide, running generally parallel and close to the coast.

A second flight on 25 May revealed that red tide had disappeared from the coastal waters and was confined to Port Moresby harbor. The third flight on 6 June and subsequent fortnightly flights to December 1972 failed to locate any further red tide blooms.

Near-surface samples of red tide were taken during the first aerial survey by lowering a Nansen bottle from the survey helicopter. The organism responsible, *Pyrodinium bahamense* Plate (Figure 2), was present in concentrations of about 600,000 per liter in the streaks, whereas a sample in the diffuse zone contained 37,000 per liter.

The concentration of *P. bahamense* in plankton hauls in Port Moresby harbor from June to December 1972 was estimated to be less than one per liter and formed less than 0.1 percent of the plankton samples.

DISCUSSION

The coincidence of the red tide blooms with initial shellfish toxicity, coupled with the identical clinical effects of *Pyrodinium bahamense* and bivalve shellfish meat extracts in bioassay studies, indicates that the dinoflagellate was the source of the toxin responsible for the human deaths and sickness. *P. bahamense* has not previously been connected with paralytic shellfish poisoning.

Since no red tide blooms were observed in the collection areas later than May 1972, and *Pyrodinium* was a rare phytoplankter in Port Moresby harbor from June to December 1972,

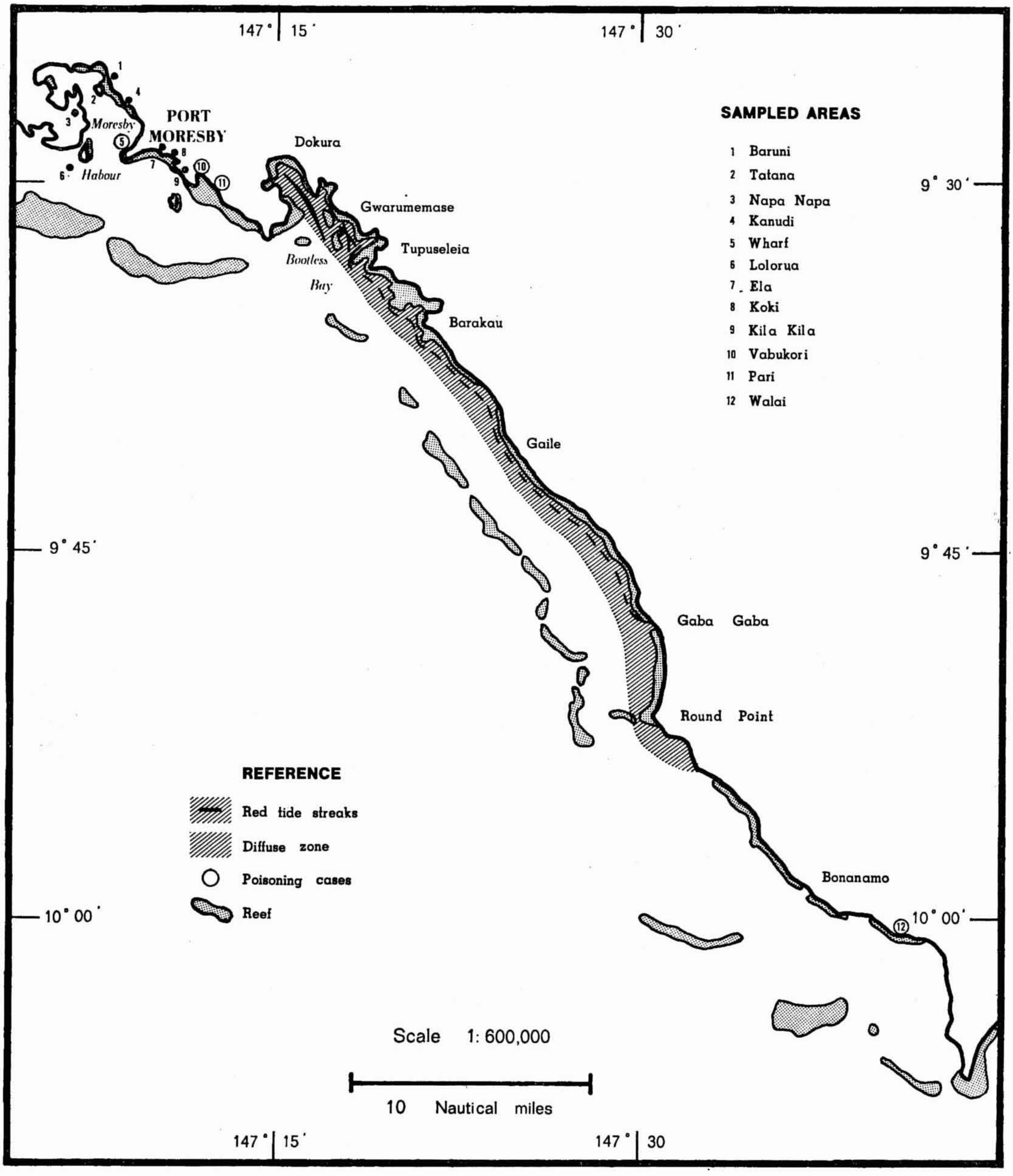

FIGURE 1. Map of coastal area between Port Moresby harbor and Walai Village showing location of red tide on 17 May 1972. Also shown are the shellfish sampling areas and villages where paralytic shellfish poisoning was reported.

the toxicity detected in the bivalves presumably originated before June.

Modiolus auriculatus and *Anadara maculosa* then were free from toxin within 6 weeks of the disappearance of *Pyrodinium* blooms, while *Spondylus* retained a high level of toxicity during this period, and *Crassostrea echinata* remained mildly toxic for at least 4 months.

TABLE 1

BIOASSAY RESULTS FROM PARALYTIC SHELLFISH POISONING SAMPLES

SPECIES	1972 DATE	COLLECTION SITE*	MICE DEATHS (PERCENTAGE)§	TOXICITY†
Lamellibranchia				
Crassostrea echinata (Quoy & Gaimard, 1835)	16 May	Pari	75	+
	20 July	Moresby Wharf	100	+
	7 September	Moresby Wharf	100	+
	15 December	Moresby Wharf	0	0
Crassostrea amasa (Iredale, 1939)	16 May	Moresby harbor	100	+ +
	4 December	Moresby harbor	0	0
Modiolus auriculatus (Krauss, 1848)	16 May	Pari	100	+ +
	16 May	Kila Kila	80	+
	12 July	Pari	0	0
Spondylus sp. C88801‡	15 May	Beruni	100	+ +
	16 May	Ela Beach	100	+ +
	16 May	Kila Kila	100	+ +
	16 May	Pari	100	+ +
	16 May	Vabukori	100	+ +
	10 July	Pari	100	+ +
	10 July	Koki	100	+ +
Anadara maculosa (Reeve, 1844)	1 May	Walai (boiled)	100	+ +
	1 May	Walai (fresh)	100	+ +
	16 May	Ela Beach	80	+ +
	10 July	Koki	0	0
	12 July	Pari	0	0
	17 July	Koki	0	0
Pinna sp.	16 May	Vabukori	100	+ +
Barbatia parvivillosa Iredale, 1939	16 May	Pari	100	+ +
Chama sp. C88799‡ C88800‡	15 May	Beruni	100	+ +
Tapes hiantina (Lamarck, 1835)	16 May	Kila Kila	0	0
	16 May	Ela Beach	0	0
Tapes literata (Linnaeus, 1758)	16 May	Kanudi	0	0
Gafrarium tumidum (Röding, 1798)	16 May	Ela Beach	0	0
Gastropoda				
Cyprea sp.	16 May	Vabukori	0	0
Lambis lambis (Linnaeus, 1758)	16 May	Vabukori	0	0
Conomurex sp.	16 May	Vabukori	0	0
Trochus niloticus Linnaeus, 1767	16 May	Vabukori	0	0
Trochus fenestratus Gmelin, 1791	16 May	Kila Kila	0	0
Pyrodinium bahamense Plate, 1906	26 May	Napa Napa	100	+ +

* For locations, see Figure 1.
† + +, death occurred in less than 7 minutes; +, death occurred from 7 to 20 minutes; 0, animal did not die.
‡ Australian Museum registration number.
§ For each test, the number of mice used varied between 6 and 10.

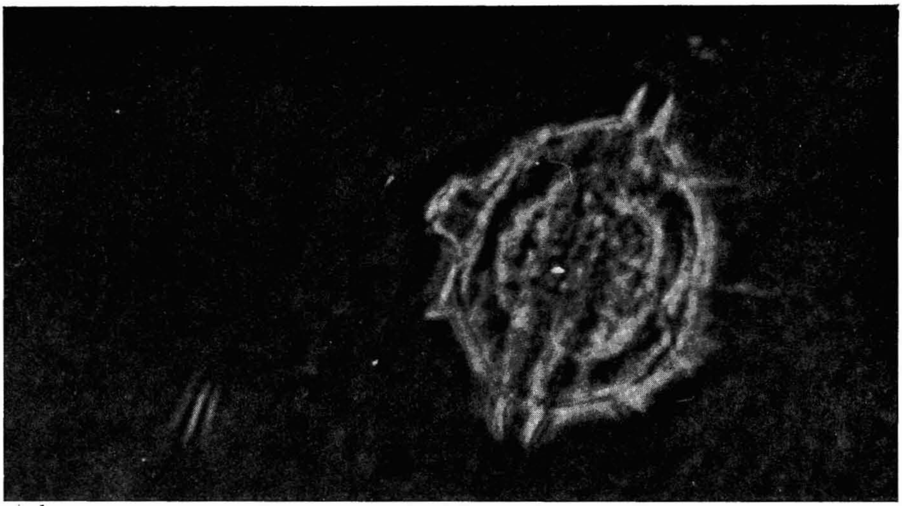

FIGURE 2. *Pyrodinium bahamense* Plate. Phase contrast photograph. Diameter 46 microns.

By contrast, *Crassostrea virginica* in Canada has been shown to lose the toxin from *Gonyaulax tamarensis* as soon as the latter disappears from the plankton (Prakash, Medcof, and Tennant 1971). *Crassostrea gigas* also loses *Gonyaulax* toxin very rapidly (Quayle 1969). Since a new source of toxin was responsible for the paralytic shellfish poisoning outbreaks in Papua, the difference in retention time of toxin in *Crassostrea* may indicate a difference in the *Pyrodinium* toxin structure and/or the shellfish binding mechanism. These problems are at present under study.

The bivalves that did not contain toxin were collected from areas where other bivalve species were toxic at the time and it is unlikely that they escaped exposure to the dinoflagellate. The reason for their lack of toxicity is not understood and is the subject of a current investigation.

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