

PARALYTIC SHELLFISH POISONING: EFFECTS ON BREEDING TERNS

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ABSTRACT.—More than 70 Common Terns (*Sterna hirundo*) and lesser numbers of other terns and gulls were killed on 11–12 June 1978, by Paralytic Shellfish Poisoning (PSP; toxin of the dinoflagellate *Gonyaulax excavata*) at a breeding colony in Massachusetts. PSP toxin was detected at lethal levels in sand-lance (*Ammodytes americanus*), the terns' principal food. Almost all terns that died were females in pre-laying condition; other birds vomited and survived. Breeding performance of survivors was unaffected. Mortality was greatest in three-year-old birds; 10–25% of three-year-old females were killed. Although PSP toxin was present in local shellfish for about three weeks, tern mortality was limited to a few hours on two days. Much higher levels of toxin were detected in shellfish at other parts of the Massachusetts coast in 1978 and other years, but terns and other susceptible seabirds were unaffected.

Paralytic Shellfish Poisoning (PSP) results from ingestion of a toxin produced by marine algae and which accumulates primarily in molluscs. In the North Atlantic, the organism primarily responsible for producing the toxin is a dinoflagellate (*Gonyaulax excavata*, formerly known as *G. tamarensis*), whose periodic blooms are known as "red tides" (Loeblich and Loeblich 1975, Sweeney 1976, White 1980a). The toxin acts by blocking propagation of nerve impulses, leading to paralysis of skeletal muscles and, in sufficient doses, to death from respiratory failure (Schantz 1973). It has recently been discovered that the toxin can accumulate in marine zooplankton to levels that are lethal to planktivorous fish (White 1977, 1980b, 1981a, b). However, there are few reports of PSP in piscivorous fish or piscivorous birds. McKernan and Scheffer (1942) reported mortality of Common Murres (*Uria aalge*) and loons (*Gavia* spp.) in an outbreak off Washington state. Coulson et al. (1968) and Armstrong et al. (1978) reported two incidents in northeastern England, in which large numbers of breeding Shags (*Phalacrocorax aristotelis*), Great Cormorants (*P. carbo*) and Northern Fulmars (*Fulmarus glacialis*) and smaller numbers of Herring Gulls (*Larus argentatus*) and terns (*Sterna* spp.) were killed. Effects on the terns in the 1968 incident were described in more detail by Horobin (1970) and Dunn (1972). In this paper I describe a similar incident at a tern colony in Massachusetts, in which I was able to document differential effects upon age- and sex-classes.

OBSERVATIONS

THE TERN COLONY

The study was carried out in 1978 at Monomoy National Wildlife Refuge, Massachusetts

(41°38'N, 69°58'W). The colony contained 2,500–3,000 pairs of Common Terns (*Sterna hirundo*), about 220 pairs of Roseate Terns (*S. dougallii*), 18 pairs of Arctic Terns (*S. paradisaea*), and about 500 pairs of Laughing Gulls (*Larus atricilla*). A colony of more than 10,000 pairs of Herring Gulls occupied an adjacent area. The principal food of Common Terns at this colony is the sand-lance (*Ammodytes americanus*), which they catch mainly in water less than 40 cm deep over sand-bars within 2 km of the colony. Arctic and Roseate terns also feed primarily on sand-lance, but the Roseate Terns catch them mainly in deeper water. Some Herring Gulls feed on sand-lance with the Common Terns, while others feed at sea around fishing boats, along the shore, or on mainland refuse dumps. Laughing Gulls feed mainly on invertebrates, but a few feed on sand-lance with the Common Terns.

In 1978 I conducted a detailed study of the Arctic Terns. By 9 June I had marked 14 nests containing eggs and had located two more pairs of terns in the pre-laying phase of courtship-feeding (Nisbet 1977), and one male holding territory and displaying to a potential mate. By this date, most of the Common Terns had finished laying, although I was not checking them in detail.

MORTALITY ON 11–12 JUNE

I began to find dead and dying terns at Monomoy about 06:30 on 11 June 1978. All birds found on 11 June were freshly killed, including some lying dry on the sand below high water mark. This indicates that the birds probably began dying after high tide (05:00) on that day. By 14:00 I had found 15 Common Terns dead in the nesting area, 13 on the beach on the east side of the colony, and 7 on the flats on the

west side. On 12 June I found 17 more Common Terns dying or dead, of which only 8 appeared fresh. After 12 June I found only 5 more dead Common Terns, all of which appeared to have died on 11 or 12 June. Thus the incident appears to have been largely confined to a few hours on 11 June, with a minor recurrence the next morning.

In addition to 57 birds found dead around the colony, refuge personnel found 2 Common Terns freshly dead at the south end of Monomoy Island, 12 km to the SSW, on 11 June. The shellfish warden from the Town of Chatham found 14 Common Terns dead on the beach of Morris Island, 1 km north (downwind) of Monomoy, on 13 June. I was not able to search other beaches in the area.

The total of 73 Common Terns found dead represents about 1.3% of the number breeding at the colony. Other birds found dead at the colony on 11–12 June included two Arctic Terns (6% of the population), one Roseate Tern (0.2%) and two Laughing Gulls (0.3%). E. J. Fisk found 26 freshly dead Herring Gulls in their nesting colony on 11 June, about 3% of the number breeding in the area surveyed. The shellfish warden also found 12 dead Herring Gulls on Morris Island on 13 June.

SIGNS OF POISONING

Almost all dead terns were found lying belly down with their bills in the sand and their wings partly spread. Some had yellowish or greenish feces around the vent and several had protruding cloacas. One had an egg partly ejected from the cloaca.

During 3 hours' watching on 11 June, my colleagues and I saw 10 Common Terns collapse and die in a loafing flock on the east beach. They showed signs of poisoning within 2–10 minutes of joining the flock. They fell forward and beat one or both wings ineffectually against the sand; some stumbled or floundered across the sand while trying to take off (Fig. 1). When picked up, they appeared paralyzed; they were unable to stand or move their wings. The birds soon began to gasp, their heads drooped and their eyes closed. Most were dead within 5–10 minutes of showing signs of poisoning. Only about 5% of the terns in the flock were affected; the others behaved normally except for showing alarm or hostile reactions to the stricken birds.

One bird that appeared to have reached the terminal phase (with head drooped and eyes closed) was placed in a dark box. Two hours later, it appeared fully recovered so it was released and flew off normally. Another, whose wings and legs were paralyzed for at least 6 h (but whose eyes remained open and bright),

was held overnight and appeared fully recovered next morning.

In addition to the dead birds, I found piles of vomited sand-lance in many places in the colony on 11–12 June. To judge from the condition of the fish (heads and front third of the bodies partly digested), they had been vomited within 20–30 min after ingestion. The fish were juveniles, mostly in the 70–90 mm range of lengths. I found at least 150 piles of vomited fish, suggesting that more birds vomited than were killed. Most dead birds had empty stomachs, suggesting that they had vomited also, but I did not see any birds vomiting.

I watched the Arctic Terns from a blind for about 2 h on 11 June. The 14 pairs that had had eggs on 9 June continued incubating normally, changing over at regular intervals. However, the two females who had been in the pre-laying stage were both lying dead in their mates' territories when I arrived at 06:45. About 20 min later, one male flew in with a sand-lance, landed beside his mate, and extended the fish towards her. On finding her dead, he jerked back with raised head, flew up with an alarm call, circled round and landed again; this sequence was repeated several times before he flew off toward the sea with the fish. The apparently startled reaction of this bird suggests that his mate had died subsequent to his last visit. The other male whose mate had died was seen only a few times and landed near her for only a few seconds. The seventeenth male was present in his territory for most of the 2 hours and displayed repeatedly with fish, but no female was seen. I suspect that the female who had been displaying with him on 9 June had died also, since he remained unmated for at least two weeks thereafter. Only two Arctic Tern nests were started later in the season, on 28 June and 5 July, and these were in a distant part of the colony.

After 12 June, I observed no unusual behavior or other adverse effects that could be attributed to the poisoning incident. Of 28 Arctic Tern eggs laid before 11 June, 4 were taken by predators, 4 were washed out by a high tide, 2 failed to hatch, and 18 hatched (90% of those incubated to term). Hatching success also appeared very high in the Common Terns. Except in areas subject to predation, both species raised young to fledging in good numbers.

LABORATORY FINDINGS

Twenty-four Common Terns found dead on 11–12 June were autopsied at the National Fish and Wildlife Health Laboratory, 8 were autopsied at the Suburban Experiment Station of the University of Massachusetts, and 24 were

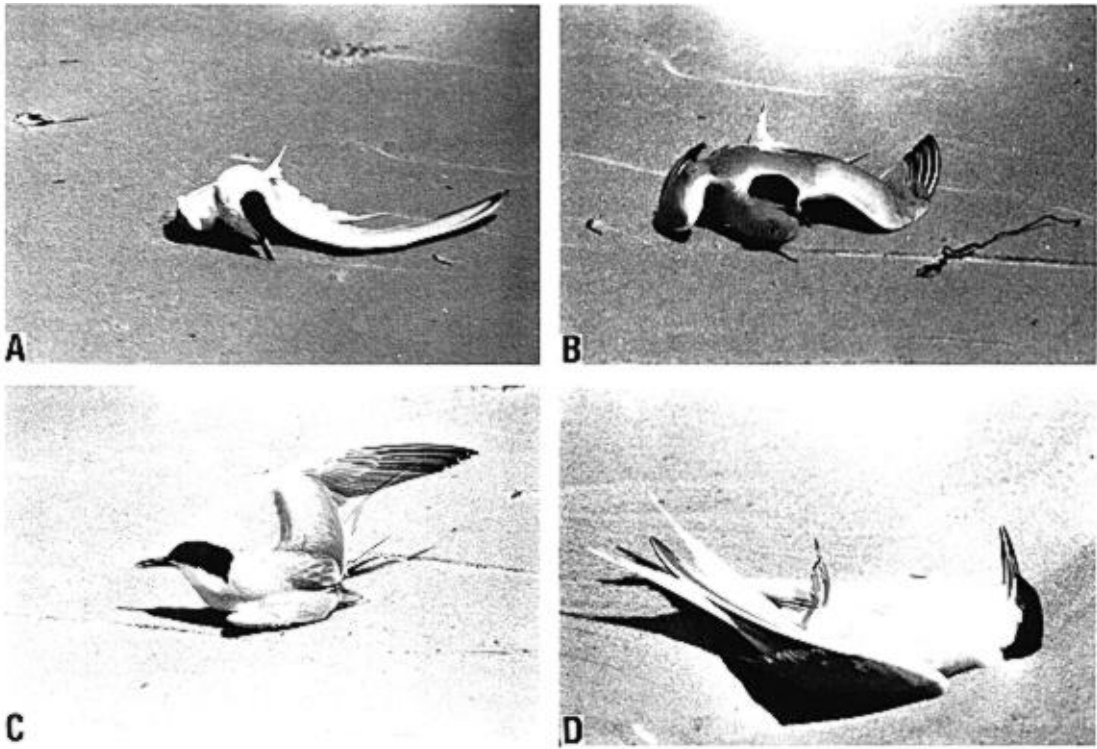


FIGURE 1. Common Tern dying from Paralytic Shellfish Poisoning. The four photographs (A–D) were taken in a sequence lasting about three minutes (from color transparencies by P. Trull).

dissected by me. All the birds were in good condition, and most had deposits of subcutaneous fat. The autopsies yielded no noteworthy findings except that in 13 birds the duodenal or intestinal mucosa were thickened and contained pale mucoid material, leading to a tentative diagnosis of enteritis. Inflammation and hemorrhaging of the duodenum, intestine and spleen, which were reported in earlier incidents of PSP in birds (McKernan and Schaffer 1942, Bicknell and Walsh 1975, Coulson et al. 1968, Hockey and Cooper 1980), were not seen in these terns. Stomachs were empty or contained small quantities of partly digested sand-lance.

A pooled sample of vomited fish picked up in the colony was analyzed by the Lawrence Experiment Station of the Massachusetts Department of Environmental Quality Engineering (DEQE). The analysis followed the standard procedure (Association of Official Analytical Chemists 1975), except that additional HCl was added to reduce the pH to 3 during extraction. PSP toxin was detected in the fish at an estimated concentration of 97 $\mu\text{g}/100\text{ g}$ (see note "a" to Table 3). This is probably well above the lethal concentration: assuming a meal size of 10 g, a tern would have ingested a dose of about 80 μg toxin/kg body weight, compared to the estimated lethal

dose of 10 $\mu\text{g}/\text{kg}$ body weight in humans (Schantz 1973). Two pooled samples of livers from dead Common Terns were also analyzed, but did not contain PSP toxin above the detection limit of 40 $\mu\text{g}/100\text{ g}$.

AGE- AND SEX-DISTRIBUTION

Of the 56 Common Terns that were dissected, 54 (96%) were females. All of these were in pre-laying condition, either with fully-formed eggs in the oviduct or shell gland (17) or with large follicles (mostly 7–26 mm in diameter) in the ovary (37). All but two weighed between 125 and 175 g, outside the normal range of 105–125 g for non-gravid females (Nisbet 1977).

The two dead Arctic Terns were also females in pre-laying condition. One weighed 136 g, well above the normal range of 95–120 g for non-gravid females (Hatch and Nisbet, unpubl. data), and had a fully-formed egg in the oviduct plus several large ovarian follicles. The other weighed 92 g and was at an earlier stage in egg-formation, with largest follicle 9 mm in diameter. I did not dissect dead birds of other species.

Since most Common Terns had finished laying before 11 June, the high susceptibility of laying females led to differential mortality of the birds that were laying later. Data obtained

TABLE 1. Laying dates of Common Terns of known age* at Monomoy National Wildlife Refuge, Massachusetts, 1973–1980.

Age (years)	Number trapped	Dates of laying first egg		Median
		Range		
2	6	20 June–23 July		30 June
3	21	30 May–12 July		12 June
4	19	24 May–17 June		2 June
5	11	23 May–14 June		1 June
6	6	23 May–30 May		27 May
7–10	9	21 May–31 May		25 May

* Records include both males and females. Most birds were banded as chicks at Monomoy; the paucity of birds older than five reflects the relatively small numbers of chicks banded there prior to 1975. In 1978 laying at Monomoy was probably three to four days earlier than the average for 1973–1980. Common Terns at other colonies in Massachusetts lay up to 10 days earlier than at Monomoy (Nisbet 1977).

in other years, summarized in Table 1, indicate that most Common Terns laying around 11 June are three years old; a minority of four-year-olds is expected to be still laying at this time, and some two-year-olds are preparing to lay. The age-distribution of banded birds found dead (Table 2) conforms to this expectation. Although one of two dead males was five years old, all banded females were either three years old (9) or two years old (2). However, because few birds had been banded before 1975, the difference in the frequency of recovery of the females was not quite statistically significant (11/358 vs. 0/103, $P = 0.060$, Fisher exact test).

About 60% of the 1975 year-class had been banded as chicks (Nisbet et al. 1978). Hence there were probably about 220 three-year-old females in the colony in 1978 (see Table 2). Assuming that most of the 73 birds found dead were three years old (see above), the mortality rate of three-year-old females may have been as high as 25%. It was probably over 10%, since at least 7% of the estimated number of banded birds were recovered, and some of the dead birds were not checked for bands.

The two dead Arctic Terns would have been the 15th and 16th to lay among the population of 18 females. However, in contrast to Common Terns, there is no consistent tendency for younger Arctic Terns to lay later in the season (Coulson and Horobin 1976). At Monomoy in 1978, the only Arctic Terns of known age were two three-year-old females, which started laying on 30 May and 4 June, 4th and 14th in the population of 18. Thus, it is not clear that younger birds in this species were disproportionately affected.

MEASUREMENTS OF PSP TOXIN IN SHELLFISH

Shellfish in Massachusetts are monitored for PSP toxin continuously from April to October each year by DEQE. The program combines periodic sampling of shellfish at a number of sites with intensive local studies when toxin

TABLE 2. Ages of banded female Common Terns killed by Paralytic Shellfish Poisoning, Monomoy National Wildlife Refuge, Massachusetts, 11–12 June 1978.

Year class	Age in 1978	Number of chicks banded at Monomoy and Tern Island ^a	Number of banded birds expected breeding in 1978 ^b	Number of banded females killed in 1978
1969	9	109	6	0
1970	8	112	7	0
1971	7	157	11	0
1972	6	257	21	0
1973	5	313	29	0 ^c
1974	4	266	29	0
1975	3	2,404	260	9 (3.5%)
1976	2	3,388	98	2 (2.0%)

^a About half of the birds banded in 1969–1973 were at a satellite colony at Tern Island, 5 km N of Monomoy; the terns from Tern Island moved to Monomoy in 1973–1974.

^b Assumptions: 90% survival from banding to fledging; 4% of fledglings return and breed at age two; 15% return and breed at age three; 15% return and breed at age four; 87% annual survival thereafter; 80% of survivors breed at Monomoy (see Austin and Austin 1956, Nisbet 1978, DiConstanzo 1980).

^c One five-year-old male was killed in 1978.

concentrations start to exceed actionable levels (80 μg toxin/100 g edible tissue). Most samples are of edible mussels (*Mytilus edulis*), ribbed mussels (*Modiolus demissus*) or soft-shelled clams (*Mya arenaria*). Peak concentrations of toxin in *M. edulis* tend to be two to three times higher than those in the other two species (Bicknell and Collins 1972, White 1982). Most samples are collected from bays and inlets rather than from open shores.

PSP toxin was first recorded at hazardous levels in Massachusetts shellfish in 1972 (Bicknell and Collins 1972, Bicknell and Walsh 1975), and has subsequently been detected each year. Levels measured in 1978 were the highest in any year except 1972. As in previous years, positive findings were limited to five discrete areas (see Fig. 2):

1. Salt ponds at Falmouth, 40–50 km WSW of Monomoy; PSP toxin was found in shellfish from 24 April to 8 June, with a peak of 1,460 $\mu\text{g}/100$ g in *Mya* in early May.

2. The coast and inlets between Salisbury and Manchester, 100–125 km NNW of Monomoy; PSP toxin was found in shellfish from 22 May to 11 July, with peaks of 420–2,070 $\mu\text{g}/100$ g in *Mytilus* and 770–1,500 $\mu\text{g}/100$ g in *Mya* between 1 and 13 June.

3. Estuaries in Scituate and Marshfield, 70–90 km NW of Monomoy; PSP toxin was found from 31 May to 12 June, with a peak of 130 $\mu\text{g}/100$ g in *Mytilus* on 5 June.

4. Nauset Inlet, 12–20 km N of Monomoy; PSP toxin was found in shellfish from 23 May to 19 June, with peaks of 66–385 $\mu\text{g}/100$ g in *Mytilus* in the period 30 May–4 June.

5. Pleasant Bay and Monomoy Island, Chatham; PSP toxin was found in shellfish between 6 and 25 June, with peaks of 42–375

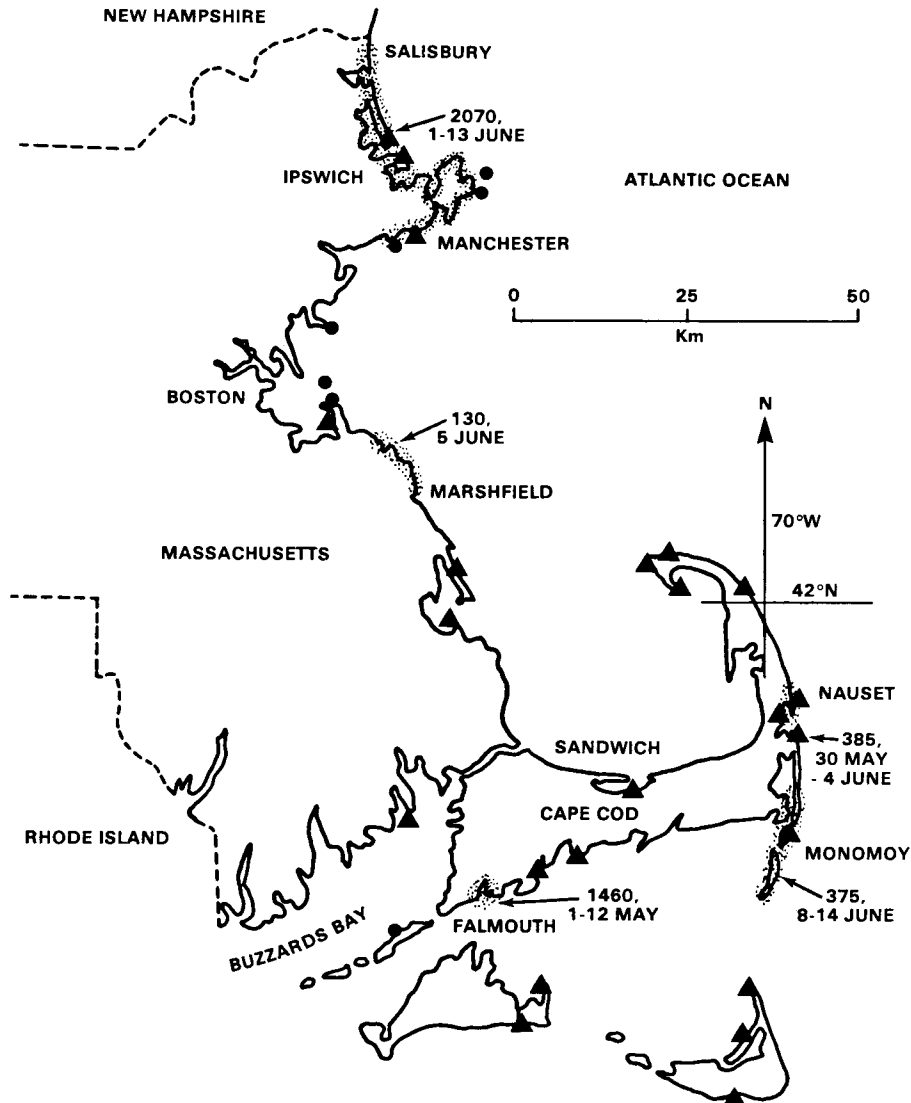


FIGURE 2. Map of the Massachusetts coastline. Areas where PSP toxin was detected in shellfish in 1978 are shaded; figures denote the peak concentrations (in $\mu\text{g}/100$ g edible tissue) and the dates when high concentrations were recorded. Triangles mark tern colonies that were monitored in 1978 without signs of mortality (except at Monomoy). Circles mark cormorant colonies that increased in numbers between 1972 and 1979.

$\mu\text{g}/100$ g in *Modiolus* and *Mya* between 8 and 14 June.

Shellfish were sampled from 25 other stations around Cape Cod throughout May–July, but PSP toxin was not detected outside the limited areas specified in paragraphs 1, 4, and 5 above. Table 3 summarizes measurements of PSP toxin from area 5, including Monomoy Island. PSP toxin was first detected on 6 June and concentrations appear to have peaked by 8 June, at least at stations north of Monomoy. At the more southern stations, highest concentrations were measured on 13–14 June, but no samples were collected earlier. Unfortunately, no samples were collected at any station on 10–11 June. However, it is unlikely that a

sharp peak in toxin levels on those days could have been missed, since toxin is retained in shellfish for several weeks after exposure ceases (Bicknell and Collins 1972), so that such a peak should have been reflected in subsequent measurements.

FISH KILLS

Two unusual fish kills took place on Cape Cod during the same period. On 13 June, the shellfish warden found about 60 bluefish (*Pomatomus saltatrix*), 2–6 kg in weight, together with a few spiny dogfish (*Squalus acanthias*), skates (*Raja* spp.) and monkfish (*Lophius americanus*), dead on the beach at Morris Island. On 15 June, P. Trull found many thousand sand-

TABLE 3. Measurements of paralytic shellfish toxin^a in shellfish, Chatham, Massachusetts, June 1978.

Location	Distance from tern colony	Species ^b	Date (June)														
			6	8	9	12	13	14	15	19	21	25	26	27	28		
Ryder's Cove	5 km N	<i>Modiolus</i>				ND											ND
Fish Pier	4 km NNE	<i>Mya</i>	63	90					ND	ND	ND					ND	ND
Horne's Marina	2 km NNE	<i>Mya</i>		73	67	44				ND	ND						ND
Stage Harbor	2 km N	<i>Modiolus</i>		42	ND					ND	ND						
Nauset Beach, S tip	1 km E	<i>Mya</i>							95		60	50	41				
Monomoy, NW	0.5 km W	<i>Modiolus</i>										44					
Shark Hole	3 km SSW	<i>Modiolus</i>						ND			46				ND		ND
Monomoy, S tip	12 km SSW	<i>Modiolus</i>							375				108				
Monomoy, S tip	12 km SSW	<i>Mya</i>							255								

^a Paralytic shellfish toxin is measured by a bioassay technique (Association of Official Analytical Chemists 1975), in which the biological activity in mice of extracts from shellfish is measured on a scale calibrated against a standard of saxitoxin. The measure given is the equivalent quantity of saxitoxin in μg per 100 g of edible tissue (Schantz 1973). ND, not detected (less than 40 $\mu\text{g}/100\text{g}$). Source: Massachusetts Department of Environmental Quality Engineering.

^b Ribbed mussel, *Modiolus demissus*; soft-shelled clam, *Mya arenaria*.

launce dead and dying along the shore at North Sandwich, 45 km WNW of Monomoy. Unfortunately none of these fish could be analyzed for PSP toxin. The exact coincidence of the first kill with the tern mortality, together with the fact that bluefish and spiny dogfish feed on sand-launce, provide circumstantial evidence that PSP toxin may have been the causative agent. However, no findings of PSP toxin in shellfish were reported from the Sandwich area.

DISCUSSION

This paper is the fourth report of PSP in fish-eating birds (McKernan and Scheffer 1942, Coulson et al. 1968, Armstrong et al. 1978). Although the test for PSP toxin is a bioassay for neurotoxicity rather than a specific chemical test, the rapidity of action of the toxin, the similarity of the toxic signs in each case, and the temporal and geographical coincidence of each incident with blooms of *Gonyaulax* spp. and with measurements of PSP toxin in shellfish, leave no reasonable doubt that PSP toxin was the causative agent. In the case reported here, the identification of PSP toxin in the fish on which the birds had been feeding, at levels high enough to be lethal to the birds, provides confirmation that was lacking in the earlier cases. PSP toxin could not be detected in the livers of the affected birds, but this conforms to the earlier finding by Armstrong et al. (1978).

White (1977, 1980b, 1981a, b) has shown that PSP toxin can accumulate in marine zooplankton to levels that are lethal to planktivorous fish. Several kills of Atlantic herrings (*Clupea harengus*) have been recorded off eastern Canada (White 1977, 1980b), and there is one report of a kill of sand-launce (Adams et al. 1968). This paper now confirms the hypothesis of Coulson et al. (1968) that sand-launce can accumulate PSP toxin to levels that are lethal to birds. It also provides circum-

stantial evidence for mortality of piscivorous fish.

Two puzzling features of the 1978 incident were its brief duration and its limitation to an area where toxin levels in shellfish were relatively modest. Although PSP toxin was present in shellfish near Monomoy for at least 19 days (Table 3), bird mortality was confined to a few hours on two days; I observed no signs of sublethal effects before or afterwards. In contrast, in the 1968 incident in England, tern mortality occurred over a period of 19–33 days, and laying patterns and incubation behavior were disrupted, resulting in unusual losses of eggs to predators and low breeding success (Coulson et al. 1968, Horobin 1970, Dunn 1972, Coulson and Horobin 1976). At the Farne Islands on the northeast coast of England, food shortage attributed to mortality of the sand-launce was also implicated as a factor in the low breeding success of Arctic Terns (Coulson and Horobin 1976).

Although concentrations of PSP toxin were at least as high in shellfish at Nauset Inlet as at Monomoy, three tern colonies at Nauset were checked at regular intervals throughout the 1978 season without sign of mortality or disruption of the normal laying pattern (Minsky 1978, Trull 1978). Furthermore, toxin concentrations have been much higher (frequently exceeding 1,000 $\mu\text{g}/100\text{g}$ in *Mytilus* and *Mya*) in the Salisbury-Manchester area of Massachusetts in each year since 1972, but the only bird mortality reported to date has been of molluscivorous birds in 1972 (Forward 1973, Bicknell and Walsh 1975). A colony of Least Terns (*Sterna antillarum*) at Crane's Beach, Ipswich, was monitored almost daily through the 1978 season (Thompson 1978), but no mortality was seen, despite PSP toxin levels of up to 2,070 $\mu\text{g}/100\text{g}$ in *Mytilus* nearby. B. Blodget (pers. comm.) visited a tern colony at Tinker's Island, Manchester, on 15 June

1978, and found breeding progressing normally, with no sign of mortality. Although other seabirds in this area have not been studied in detail, numbers of Double-crested Cormorants (*Phalacrocorax auritus*) are known to have increased considerably between 1972 and 1979, despite annual outbreaks of PSP (Drury 1973–1974, Erwin 1979, Erwin and Korschgen 1979, Hatch 1982; see Fig. 2).

Similar findings have been reported from northeastern England, where high levels of PSP toxin (ranging up to 6,300 "mouse units," or about 1,000 $\mu\text{g}/100\text{ g}$) have occurred in *Mytilus* in years when no effects on breeding terns or cormorants could be discerned (Armstrong et al. 1978). If levels in shellfish provide a reliable index of the concentration of PSP toxin in ambient water, these results suggest that the circumstances that lead to uptake of the toxin by fish to toxic concentrations are relatively infrequent.

Another striking feature of the incident at Monomoy was the high susceptibility of laying females. Coulson et al. (1968) similarly noted disproportionately high mortality of laying females, and attributed this to secondary effects of paralysis of the oviduct. However, I found only one bird that had died while laying an egg. Many others were at earlier stages in egg-formation, and most died too quickly for Coulson et al.'s explanation to apply. Although females eat a great deal while amassing nutritional reserves in the period before egg-laying, at least some birds were killed immediately before egg-laying, when their rate of food intake is reduced (Nisbet 1977). Thus, some other explanation for the susceptibility of laying females is required.

The concentration of mortality on three-year-old birds (Table 2) appears to have been a simple consequence of the occurrence of the toxin in the fish in mid-June (Table 1). Presumably, if the incident had occurred earlier, at the peak of laying, it would have affected older birds. Although the fish apparently were toxic for only a few hours on two days, I estimate that 10–25% of the three-year-old females were killed. PSP is potentially a serious threat to colonies of fish-eating birds in this area, because the sand-lance is the staple food of many species. Further investigation is needed of the factors leading to accumulation of the toxin by fish.

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RECENT PUBLICATIONS

A Coded Workbook of Birds of the World, Volume 1, Non-passerines.—Ernest P. Edwards. 1982. Published by the author. 134 p. Paper cover. \$10.00 plus \$1.00 handling (plus 50¢ handling for each additional copy) on prepaid orders; postage extra if billed. Source: Ernest P. Edwards, Box AQ, Sweet Briar, VA 24595; if ordering from outside U.S.A., please write for proforma invoice. This is a revised and enlarged edition of the non-passerine portion of the author's *A Coded List of Birds of the World* (1974); a volume on passerines is to come. The sequence of taxa has been rearranged, following several recent authorities. At the core of the book is a systematic list of all living or recently extinct species of birds, coded for computer usage. The entry for each species gives its alphanumeric code; possibly a symbol showing that it is extinct or denoting taxonomic or nomenclatural changes; the scientific and English names; a set of distribution symbols; and a symbol designating if the species is endangered. All this is on one line, arranged in a columnar format, making the information exceptionally easy to find. Detailed taxonomic notes, keyed to the symbols, are given after most family lists. Indexes are provided for genera, scientific names, important subspecific units (taxa that have been considered full species elsewhere), and English names. There follows a cross-reference list of codes in this edition and those in the first edition. References, maps. Now improved, this manual appears to be a strong contender against the several existing lists of birds of the world, e.g., Morony, Bock, and Farrand (1975; noted in *Condor* 77:521), Wolters (1975–1982; *Condor* 84:398), Gruson and Forster

(1976; *Condor* 78:279), Howard and Moore (1980; *Condor* 83:47), and Walters (1980; *Condor* 82:141). It will soon be available in a Magtape version and possibly a Decmate diskette version also; write to the author for details and prices.

World Inventory of Avian Skeletal Specimens 1982 and World Inventory of Avian Spirit Specimens 1982.—D. Scott Wood, Richard L. Zusi, and Marion Anne Jenkinson. 1982. American Ornithologists' Union and Oklahoma Biological Survey, Norman, Oklahoma 73019. 224 p., 181 p. Paper cover. \$25.00 apiece, including surface mail postage. For instructions on ordering, see *Condor* 85:262. These two oversize, computer-produced volumes are inventories of skeletons and embalmed specimens ("alcoholics") of birds in most of the major museum collections of the world. As preliminary information, each gives a list of the museums included, addresses and names of curators, and indexes to taxa. The principal content is a running tabulation that shows the holdings of each museum for every species. A detailed description of this project, an analysis of its data, and references can be found in Zusi, Wood, and Jenkinson (1982; *Auk* 99:740–757). Researchers in need of avian anatomical specimens will be immensely aided by these documents. Certain investigations that were hindered for lack of material may now be able to proceed, knowing whence it can be borrowed. The present authors deserve thanks for their labors, plus encouragement to continue and produce supplements.