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Marine birds and harmful algal blooms: sporadic victims or under-reported events?

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This paper is dedicated to Harry, an 18-year-old Magellanic penguin, missing and presumed killed by a chance encounter with toxic algae off the coast of Argentina

Abstract

From the late Pliocene to now, blooms of toxic algae are associated with mortalities of marine birds. Given the long historical presence of harmful algal blooms (HABs) worldwide and the numbers of seabirds that feed on filter-feeding fish and shellfish, it is surprising that relatively few incidents of seabird deaths as a result of toxic algae have been reported. The limited information available tends to come from major events, whereas the rare events are missed and hence not reported. Much is anecdotal and still more probably is not published. We suspect that factors working in concert may lead to deaths and wrecks that might not occur as a result of anyone factor working independently, e.g. starvation tends to render birds more vulnerable to stress.

"Seabird wrecks", very much larger than usual concentration of seabird corpses washed ashore over a short period of time, often provide evidence of deleterious conditions in offshore populations, e.g. weather, food, pollution, fishing activities, and parasites. It is noted in the literature that wrecks caused by natural toxins such as botulism and algal toxins are apparently less common; however, this perception may be due to a combination of factors including the bird species involved, size of populations, location, and chance of discovery. Wrecks involving near-shore species probably provide a more accurate estimate of total mortality for any given event than offshore species.

A survey of available data on the impacts of toxic algae on seabirds revealed an array of responses ranging from reduced feeding activity, inability to lay eggs, and loss of motor coordination to death. Severe impacts on recruitment have been noted in some populations. There are few experimental studies; however, evidence has been provided for the ability of some species to 'learn' to avoid toxic food sources. We present a summary of available data on seabird/toxic algal interactions and suggestions of how impacts on seabirds during future blooms of harmful algae be recorded.

Keywords: Bird; Harmful algal blooms; Red tides

1. Introduction

Harmful algal blooms (HABs) or 'red tides' are a global phenomena and recent evidence indicates that their frequency and intensity are increasing

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(Shumway, 1990; Smayda, 1990; Hallagraeff, 1993; Burkholder, 1998). They are a serious threat to human health, aquaculture, fisheries, and ecosystem health. These HABs range from recurrent in some areas to episodic or, in some regions, persistent. It is the effects of the episodic blooms that tend to receive the most attention, because they are most often associated with lethal effects on adult stages of commercially important species (see Anderson, 1995 and references therein).

The algal toxins involved include domoic acid, saxitoxin, brevetoxin, okadaic acid, ciguatoxins, and their derivatives (Table 1). Toxins are moved through the food chain from the toxic phytoplankton to herbivorous consumers (molluscs, Crustacea or phytophagous fish) and then on to carnivorous fish, piscivorus and scavenging birds, and mammals (Matter, 1994). Little is known, however, about the impacts on higher trophic levels or how the movement and retention of these toxins influences ecological interactions and communities (Smayda, 1990; Shumway, 1995). The transfer and possible transformation of toxins as they move through the food web is an important consideration. Transfer of phycotoxins via food web interactions to higher trophic levels can have serious, deleterious effects (see Shumway, 1995 for review). Domestic animals (mostly cats and hens) died from eating discarded parts of shellfish being prepared for human consumption (Medcof et al., 1947; Tennant et al., 1955). Homing pigeons are sensitive to these toxins (Coulson et al., 1968a,b) (Table 2). The impact of these toxic events on marine life including marine mammals, fish, and shellfish have been well documented (see Landsberg, 2002 and references therein). The impacts on seabirds, one of the most common members of marine food chains and most likely to be consuming toxins already concentrated by other organisms, have not been summarized exclusively. It is surprising that the impacts of these toxins on seabirds have, for the most part, been overlooked or only casually mentioned in most field studies of HABs on marine communities, since seabirds are among the

Table 1
Illnesses associated with HABs in humans, causative organisms and clinical manifestations (Morris, 1999; Anderson et al., 2001)

Syndrome	Causative organisms	Toxins produced	Clinical manifestations
Paralytic shellfish poisoning (PSP)	*Alexandrium* spp., *Gymnodinium catenatum*, *Pyrodinium bahamense*, and others	Saxitoxin and derivatives	Acute paresthesias and other neurological manifestations may progress rapidly to respiratory distress, muscular paralysis, and death
Neurotoxic shellfish poisoning (NSP)	*Karenia* (*Gymnodinium*) *brevis*	Brevetoxins	Gastrointestinal and neurological symptoms; respiratory and eye irritation with aerosols
Diarrhetic shellfish poisoning (DSP)	*Dinophysis* spp., *Prorocentrum* spp.	Okadaic acid and dinophysis toxins (DTXs)	Acute gastroenteritis
Amnesic shellfish poisoning (ASP)	*Pseudo-nitzschia* spp.	Domoic acid and isomers	Gastroenteritis, neurological manifestations, leading in severe cases to amnesia (permanent short-term memory loss), coma and death

Table 2
Symptoms noted in dying birds on Farne Islands in May 1968 (Coulson et al., 1968b)

Shags (*P. aristotelis*)	Terns (*Sterna* spp.)	Guillemot (*Uria aalge*)
–	Inability to lay eggs	–
Loss of equilibrium (inability to stand or even keep head up), motor incoordination, and restriction of pupil	Inability to stand and motor incoordination (convulsions)	Convulsions
Excess vomiting, abnormal green–brown faeces, and intestinal haemorrhage	Vomiting and abnormal faeces	–
Paralysis	Paralysis	Severe paralysis
Failure of circulatory system, and congestion of organs, including lungs	Failure of circulatory system, and frequent gasping and congestion of lungs	–

most valuable biological indicators for environmental perturbation (Swennen, 1977; Boersma, 1978, 1986).

The reproductive rate of seabirds is low compared to their terrestrial relatives and they have conservative reproductive traits (Lack, 1954). Seabirds are often 3–7 years of age before they are sexually mature and in many instances, females produce only one egg annually (Gill, 1995). This low rate of reproduction, coupled with low natural mortality of adults, results in long life spans, reaching 20–50 years for some species. Any event that significantly increases the mortality rate of adults will have major impacts on the populations. Many studies attest to the impacts of pollutants, especially oil, on seabirds (see Burger and Gochfeld, 2002). Other well-studied impacts include pesticides, heavy metals, plastics, and other man-made pollutants (see Burger and Gochfeld, 2002). Although the incidence of a HAB among naive birds can have disastrous impacts on the populations, the impacts of HABs on seabirds have received comparatively little attention.

Algal toxins and bird deaths are often linked. Emslie et al. (1996) described avian death assemblages in marine sediments from the late Pliocene of Florida. They hypothesized that the large number of cormorant and other seabirds were caused by toxic red tides of *P. bahamense* (see their discussion for explanation of species distribution and extinction). Mortalities of frigate birds, terns, gulls, ducks, and vultures from Tampa Bay to Key West Florida were reported by Glazier (1882), Moore (1882), and Walker (1884). Field data are, however, sparse and there have been relatively few experimental studies. This is in part due to the difficulty of keeping seabirds in captivity, the logistical difficulties of field studies, the permitting processes necessary to work with seabirds, the unpredictable nature of these outbreaks, the short time span of some outbreaks, the lack of awareness during outbreaks of HABs that birds might be affected, or that dead birds might be the victims of HABs. Swennen (1977) described what appears to be the first successful holding facilities for seabirds, including areas for experimental studies. Subsequent studies have not, however, included algal toxins.

In an early review, Steidinger et al. (1973) reported dead cormorants, ducks, frigate birds, gulls, terns, and vultures (specific species not given) as a result of blooms of *Gymnodinium breve* and noted that the birds could either have ingested toxic prey or drunk the toxic seawater. They pointed out the need for further studies on the impacts of red tides on migratory species, especially animals seeking out the protection and productivity of estuaries to breed or harbor juvenile stages; however few such studies have materialized.

Given the long historical presence of HABs worldwide and the numbers of seabirds that feed on filter-feeding fish and shellfish, it is surprising that there are not more records. The questions that should be asked are: “why are not more birds impacted by algal toxins?” and “is the apparent low impact a case of unnoticed or unrecorded incidents?”

Seabirds are generally sensitive to environmental pollutants and toxins and have on several occasions been the ‘alarm’ or sentinel that signaled the presence of algal toxins. During the first recorded outbreak of a toxic dinoflagellate in Massachusetts in 1972, one dead and several obviously ailing seagulls on a dock were noted (Hurst, Maine Department of Marine Resources, personal communication) and a simultaneous kill of ∼100 birds (black ducks and gulls) were the first clues that something was amiss. Autopsies of the birds showed the same extensive hemorrhaging of the internal organs as seen in other episodes of paralytic shellfish poisoning (PSP) (Bicknell and Collins, 1972; see for description of symptoms associated with PSP). The causative organism was positively identified as *Gonyaulax tamarensis* and toxic shellfish were noted in some gut contents of the dead birds (Sasner et al., 1975). Sasner et al. also noted that “many other birds apparently perished after feeding on toxic shellfish”, but were not recorded.

‘Wrecks’ are not the only indicators of impacts of environmental factors on birds and often times only a few individuals of near-shore species are impacted. There are also numerous reports of unexplained bird mortalities and several of these strongly implicate but do not demonstrate algal toxin poisoning (see Environment Agency, 2000a,b). The most obvious problem in establishing a cause and effect is the fact that bird deaths are usually noted well after the ‘toxic’ event. Further, many of these ‘events’ take place in offshore waters and by the time dead birds begin appearing on shorelines the cause has dissipated or cannot be sampled.

Many authors continue to use local shellfish toxicity as an indicator of the levels of exposure that the

birds are experiencing. Only for the birds feeding directly on shellfish in the area monitored, is this a true measure. Not only are the majority of bird deaths attributable to accumulation of toxins by filter-feeding fishes, especially sand lances and herring, birds are highly mobile and may be feeding in areas distant from where the mortalities are noted. Further, levels of toxicity in shellfish are not necessarily indicative of toxin levels in other organisms, most especially mobile species such as fish. Finally, the toxin levels in shellfish, unless monitored constantly, are a good indicator of a bloom occurrence but not necessarily of bloom duration.

The current review summarizes reported impacts of HABs on birds. The impacts of freshwater cyanobacteria on birds are not covered here and the reader is referred to Landsberg (2002) for a recent review.

2. Historical records

The available information about seabird species and toxic algal blooms is summarized in Tables 3 and 4, with more detailed information given here. As indicated, it is mostly fragmented or anecdotal. During a spate of toxic shellfish in Washington coastal waters, McKernan and Scheffer (1942) reported cats and chickens dying from eating viscera of razor clams. The toxicity of clams was attributed to the dinoflagellate *Gonyaulax catenella*. They also noted long windrows of pelagic barnacles, *Lepas fascicularis*, and speculated that these too may have concentrated the toxic algae. Gooseneck barnacles have been reported as toxic by other investigators (see Shumway, 1995). The authors noted that in the chickens, the entire length of the intestine was greatly inflamed and blood vessels were enlarged and sharply outlined. In conjunction with the outbreak of toxic shellfish, several dead seabirds (see Table 3) were found and the dissected intestines of gulls (species not specified) presented the same inflamed condition as that seen in the chickens. During this time, fishermen also reported seeing large numbers of dead birds floating about 10–20 miles off the Washington coast and large numbers of murres and shearwaters floating dead about 30 miles offshore in British Columbia. While some of these birds washed ashore, many probably did not, so mortality is underestimated. While the authors could not provide true cause and effect, there appears little doubt that these bird deaths were attributable to PSP toxins ingested via prey items including shellfish, small crustaceans, and planktivorous fish.

In the late spring of 1969, an outbreak of paralytic shellfish poisoning (*Gonyaulax* = *Alexandrium*) *tamarense* occurred on the northeast coast of UK. Because such outbreaks were rare in this region (only one such outbreak had been reported in the previous 40 years), considerable effort was expended in studying the impacts (Adams et al., 1968; Coulson et al., 1968a,b). High cell concentrations were monitored up to 10–15 miles offshore. In addition to human illnesses, these blooms were responsible for the sudden death of numerous seabirds (see Tables 1 and 2), primarily shags (*Phalacrocorax aristotelis*). It was fortuitous that Coulson and co-workers had been studying seabirds in this region for several years and were able to provide one of the few in-depth reports on the impacts of HABs on bird populations to date (see also Nisbet, 1983). It was estimated that some 80% of the breeding population in Northumberland died (Coulson et al., 1968a). During the initial stages of the outbreak, large numbers of dead sand eels (*Ammodytes* spp.), the normal diet of shags, were caught in nets and washed on shore. Birds were seen to lose equilibrium and stagger, many vomited sand eels (Wood and Mason, 1968).

Another extensive PSP incident occurred in 1975 and again, Coulson and co-workers (Armstrong et al., 1978) were able to provide comprehensive coverage of the event and its impacts on the bird colonies. While the 1975 red tide was not as severe as that of 1968, over 60% of the breeding shags (*P. aristotelis*) died from the toxin. The authors counted 156 dead birds but estimated that over four times that many had perished. They also noted that shags are the most sensitive seabird to the toxins. Because few immature birds were present during the time of toxin outbreak, re-colonization of the breeding sites was possible.

Furphy et al. (1971) presented a detailed accounting of large numbers of unoiled seabirds either dead or dying, on or near beaches around the Irish Sea and Firth of Clyde. They estimated a total of ~15,000 dead birds, but pointed out that the actual kill may have been considerably higher. Species involved included guillemots (*Uria aalge*), razorbills (*Alca torda*), gulls, cormorants (*Phalacrocorax caarbo*) and shags

Table 3
A summary of recorded impacts of HAB species on birds

Bird species	Algal species	Location/date	Description of incident	Toxicity	Notes	Reference
Kittiwake (*Rissa tridactyla*), herring gull (*Larus argentatus*), guillemot (*Uria aalge*)	*Dinophysis* spp. (4.4–6.8 × 10^3 cells/l)	Northeast coast, UK/June–September 1995, May–September 1996, May–October 1997	Birds haemorrhaged from gut and in considerable distress	Not reported; DSP toxin found in livers	Deaths not attributed to a single toxic event; livers analyzed by HPLC	Krokowski (personal communication)
California murre (*Uria aalge californica*), pacific loon (*Gavia arctica pacifica*), white-winged scoter (*Melanitta fusca deglandi*), tufted puffin (*Lunda cirrhata*), sooty shearwater (*Puffinus griseus*), herring gull (*Larus argentatus*), western gull (*Larus occidentalis*), pacific fulmar (*Fulmarus glacialis*), black footed albatross (*Diomedea nigripes*)	*Gonyaulax catenella*	Washington/May 1942	Approximately 225 dead birds identified		Reports of cats and chickens dying from eating viscera of razor clams; bird deaths suspected due to consumption of contaminated small fish and crustaceans; large numbers of gooseneck barnacles also noted in area	McKernan and Scheffer (1942)
Black oyster-catcher (*Haematopus moquini*), southern blackbacked gull (*Larus dominiccus*), hartlaub's gull (*Larus hartlaubii*)	*Gonyaulax catenella*, *Gonyaulax grindleyi*	Lambert's Bay and Bloubergstrand, South Africa/May 1979	Birds consumed black mussels (*Choromytilus meridionalis*) and white mussels (*Donax serra*); pathological lesions and bleeding of the stomach and intestine; stomachs empty, birds probably starved to death	Not reported	Oyster–catcher populations were approximately halved following outbreaks	Hockey and Cooper (1980), Horstman (1981, personal communication), Popkiss et al. (1979)
Shag (*Phalacrocorax aristotelis*) (see Table 4 for complete listing of other species)	*Gonyaulax tamarensis* (>7 × 10^4 cells/l)	Farne Islands, UK/May 1968; Farne Islands, UK/May 1975	199 shags killed; intestinal hemorrhaging commonly observed; before death, birds were seen to lose equilibrium and stagger; many vomited sand eels (*Ammodytes* spp.), 156 dead shags; several other species found dead, similar to 1968 outbreak (see Table 2 for summary)	Toxicity not determined in birds or sand lance; shellfish samples collected nearby in excess of 7500 MU/100 g, approximately 6000 MU/100 g mussel meat	80% of shag breeding population killed; authors estimate total deaths probably four times higher than recorded	Wood and Mason (1968), Coulson et al. (1968a,b), Armstrong et al. (1978)
Black duck (*Anas rubripes*)	*Gonyaulax tamarensis*	New Hampshire coast/mid-September 1972	Approximately 1600 duck deaths occurred after feeding on toxic shellfish (e.g. *Mytilus*, *Siliqua*, *Ensis*)	2000–4000 μg STX eq/100 g at peak	Also noted 620 waterfowl, gulls, and shorebirds representing 13 different species (not specified) dead at Plum Island, MA	Bicknell and Collins (1972), Bicknell and Walsh (1975), Sasner et al. (1975)
Herring gull (*Larus argentatus*)	*Alexandrium tamarense*	St. Lawrence Estuary, Que., Canada/July 1996		360 μg STX eq/100 g in sand lance; 110 and 48 μg STX eq/100 g in bird intestine and brain, respectively	Sand lance (*Ammodytes hexapterus*) also found dead; cats that ate dead fish also exhibited symptoms of PSP	Levasseur et al. (1996)

Table 3 (*Continued*)

Bird species	Algal species	Location/date	Description of incident	Toxicity	Notes	Reference
Common tern (*Sterna hirundo*), arctic tern (*Sterna paradisaea*), roseate tern (*Sterna dougallii*), laughing gull (*Larus atricilla*), herring gull (*Larus argentatus*)	*Gonyaulax excavata*	Massachusetts/June 1978	73 common terns dead (1.3% of breeding colony); 2 arctic terns; 1 roseate tern; 2 laughing gulls; 38 herring gulls dead; most common terns were females in pre-laying condition	97 μg STX eq/100 g in sand lance		Nisbet (1983)
Brown pelican (*Pelecanus occidentalis*), Brandt's cormorant (*Phalacrocorax penicillatus*), double-crested cormorant (*Phalacrocorax auritus*), western gull (*Larus occidentalis*), pelagic cormorant (*Phalacrocorax pelagicus*)	*Pseudonitzschia australis*	Santa Cruz, CA/September 1991	95 Brandt's cormorants and 43 brown pelicans died after ingesting northern anchovies (*Engraulis mordax*) which had grazed on *P. australis*; other species represented <7% of total carcass count; hemorrages and necrosis of the skeletal muscle; serum blood urea nitrogen and creatinine phosphokinase elevated in affected birds	69% case fatality rate; domoic acid levels 150 μg/g wet weight	First documentation of domoic acid poisoning outside Atlantic Canada; cells identified in fish guts	Fritz et al. (1992), Work et al. (1993a,b)
Brown pelican (*Pelecanus occidentalis*)	*Pseudonitzschia* sp.	Cabo San Lucas, Baja, CA/January 1996	Over 150 pelicans killed after eating contaminated mackerel (*Scomber japonicus*)	37.2 μg/g domoic acid in bird digestive tract; ~47 μg per fish	Deaths represent 50% of colony	Ochoa et al. (1996), Beltrán et al. (1997)
King penguin	*Pseudo-nitzschia australis*	Zoo in Kentucky/summer 2000	Four penguins died after being fed herring and anchovies collected from Monterrey Bay, CA	20 ppm domoic acid and 30–35 MU/100 g PSP toxins in anchovy guts; trace amounts of domoic acid in herring guts		Naar et al. (2002)
Lesser scaup (*Aythya affinis*), double-crested cormorant (*Phalacrocorax auritus*), red-breasted merganser (*Mergus merganser*)	*Gymnodinium breve*	Tampa, FL/February–March 1974	12000 and possibly 20000 lesser scaup died		Four times more male than female lesser scaup died; no direct link between *G. breve* and bird death demonstrated but circumstantial evidence very strong	Quick and Henderson (1975), Forrester et al. (1977), Schreiber et al. (1975)
Double-crested cormorant (*Phalacrocorax auritus*)	*Karenia brevis* (*Gymnodinium breve*)	Sanibel Island, FL/1995–1999	360 birds admitted to Clinic for Rehabilitation of Wildlife suffering from ataxia	Illnesses significantly correlated with presence of *K. brevis*	Brevetoxin positively identified in organs of four birds in 1997	Kreuder et al. (2002)

See text for further explanation and discussion. There are also numerous reports of unexplained bird mortalities and several of these strongly implicate but do not demonstrate algal toxin poisoning. Reassessment of historical data on bird kills coupled with records of HAB species/blooms might explain some of the bird kills.

Table 4
Numbers of dead seabirds found on the Northumberland and north Durham coasts (25–31 May 1968) in relation to the populations at risk in Northumberland (from Coulson et al., 1968b, see also Armstrong et al., 1978)

Species	Population at risk (birds)	Total found dead	At risk died (%)
Fulmar	400	21	5
Gannet	–	11	–
Cormorant	600	24	4
Shag	1200	199	17
Common scoter	–	2	–
Eider	2500	60	2
Great black-backed gull	–	1	–
Lesser black-backed gull	3000	2	<1
Herring gull	1300	26	2
Common gull	–	1	–
Black-headed gull	200	1	<1
Kittiwake	7000	50	1
Common tern	3000	41	1
Arctic tern	7000	71	1
Sandwich tern	4000	58	1
Roseate tern	300	3	1
Razorbill	40+	6	1
Guillemot	4000	53	1
Puffin	45000	6	<1
Total dead birds	–	636	–

(*P. aristotelis*). While the authors do not attribute the kill to harmful algae, the timing and the species involved point to HAB as the cause of mortality. A few birds were found dead in mid-July, with numbers increasing in early September, the bulk of the mortalities occurring during the latter part of that month. Peak mortality was mid-September to mid-October, the peak time for algal blooms in these waters. Some of the bird species mortalities were unprecedented (razorbills), perhaps indicative of naive birds. All of the species involved are fish-eating birds, most of which consume their food whole and would be particularly prone to the algal toxins. It is unfortunate that water samples were not checked for the presence of toxic algae, but understandable as HABs were not a concern at the time.

Large numbers of lesser scaup (12,000–20,000) and low numbers of double-crested cormorants (*Phalacrocorax auritus*) and red-breasted mergansers (*Mergus merganser*) died during red tides (*G. breve*) in the Tampa, Florida area in 1975. Laymen quickly blamed the red tides for the deaths; however, experiments by Quick and Henderson (1975) did not demonstrate a link between *G. breve* and the death of the lesser scaup. They attempted to feed toxic clams (*Mercenaria campechiensis*) to lesser scaup. Ducks were given a 50:50 tapwater:red-tide water mix to drink for 11 days. The following 8 days the drinking water was continued plus each duck was given 20 g of toxic clams per day. The ducks readily ate the clams and at the end of the 2-week period showed no abnormalities and even an increased vigor. The ducks seemed healthy, although four times as many males as females died. Subsequent controlled experiments with Peking ducklings (Forrester et al., 1977) and *G. breve* produced illness and death with symptoms comparable to some of those seen in field kills of lesser scaup. The 20 g ration fed to the lesser scaup was apparently a sub-lethal dose of toxin, hence did not result in death. If the diet had continued for a longer period of time or the ducks experienced some additional stress, birds would have likely died. Finally, Quick and Henderson note that the heaviest kill occurred where little or no red tide penetrated but where the largest flocks congregated and they found indications of mild *G. breve* intoxication in some lesser scaups. The birds may have fed at another site where they ingested the toxins. Forrester et al. labeled the lethal disease idiopathic, although brevetoxin certainly seems a strong possibility in this case.

This episode seems to be an unfortunate case of the ducks being in the wrong place at wrong time. Lesser scaup are diving ducks and regularly feed on bivalve molluscs, organisms known to accumulate high concentrations of algal toxins. Red-tide events in the Tampa region of Florida generally occurred during spring and summer (in more recent years, blooms have extended throughout the year; see Steidinger and Penta Melton, 1999) and would not normally have been present in the winter months when the scaup were migrating and in need of more food. Further, the blooms were not common in Tampa Bay proper. In this particular year, the bloom was extensive, ‘out of season’ and, while no direct cause and effect could be demonstrated, it seems to be the most likely cause of the mass mortality of the lesser scaup.

During the winter of 1981/1982 approximately 3600 Eider ducks of the Skagerak population died (Wrånes, 1988). This represents about 20% of the spring

population and may have been the result of algal poisons in mussels. In the fall of 1981, a severe bloom of *Gyrodinium aureolum* ($\sim$10–20 $\times$ 10^7 cells/l) was noted followed by large numbers of mussel and tubeworm deaths from Norway, Sweden, and Denmark water surrounding the Skaggerak. Bird deaths were first attributed to parasites; however, equal numbers were found in dead and healthy birds. Cold was also considered but bird deaths in Sweden were noted before the onset of extreme cold. Some frostbite was noted on birds' feet. In addition, duck bills were excessively worn. Most of the eiders found in the Sorlandet showed evidence of diarrhea and emaciation and 'soiling' from diarrhea; the cloacal region and feathers surrounding it were soiled with a gray-green excretory product. Stomachs and intestines of necropsied birds were empty and some contained large amounts of blue mussels. While no actual toxins were detected in the birds, algal toxins were suspected as causing nerve damage that led to excessive pecking and subsequent damage to bills and also for birds being in a generally weakened condition leading to frostbite. It is interesting to note that *G. aureolum* is only known to produce a hemolysin, thus it is unclear how the birds may have died form neurological involvement. It is possible that a toxin has yet to be identified or there are other chronic effects as yet unknown. Despite the lack of demonstrated cause and effect of algal toxins and deaths, it seems that toxic algae caused the massive deaths.

An excellent detailed account of intoxication and death in terns and other birds is given by Nisbet (1983). This is a particularly important study because detailed data were available on the bird colonies prior to the toxic blooms thus allowing a quantitative assessment of the impacts of the bloom. He was consequently able to document differential effects upon age- and sex-classes, reporting that mortality was greatest in 3-year-old birds and that almost all terns that died were females in pre-laying condition. He further noted that other birds vomited and survived. It is interesting to note also that, although the PSP toxin was present in local shellfish for about 3 weeks, tern mortality was limited to a few hours on 2 days. Given that the principal food of common terns at this colony is the sand lance (*A. americanus*) (Nisbet, 1983), it is most unlikely that the terns were feeding on toxic shellfish and that sand lance was the primary toxin vector. Shellfish are known to harbor toxins for weeks to months (Shumway, 1990); however, no data are available for the retention time of toxins in the sand lance. We suggest two explanations for the limited time period during which the birds were affected. First, it is highly possible that the birds developed an aversion response to the toxic fish. Nisbet noted many piles of vomited sand lance and estimated that they had been vomited within 20–30 min of ingestion. He suggested that more birds vomited than were killed. It is also possible that, in fish not killed by the toxins, that the toxins were rapidly egested and that only birds feeding on fish during the initial bloom period would be impacted.

Gannets (2) which had recently migrated from Canada along the eastern coast of the US were found dead along coastal Carteret Country, North Carolina in 1992 and tissues analyzed for PSP toxins which were confirmed in both cases. Tissues were also analyzed for presence of brevetoxins which were not detected (Fish and Wildlife Service, Ecological Services, Raleigh, NC, unpublished).

Fritz et al. (1992), Work et al. (1993a,b), and Ochoa et al. (1996) have provided detailed accounts of the impacts of domoic acid on seabirds in California and Mexico, respectively. In both cases, the birds killed (predominantly cormorants and pelicans), were piscivorus and naive. In 1991, large numbers of Brandt's cormorants and brown pelicans were found dead in Santa Cruz, California. Hemorrhages and necrosis of the skeletal muscle was consistently present and domoic acid was positively identified in stomach contents of sick and dead birds as well as in their prey, northern anchovies. Dying birds displayed classic signs of distress associated with the central nervous system. A detailed description of the epidemiology is given in Work et al. (1993a,b).

In New Zealand in the summer of 1992/1993, marine biotoxins reached crisis levels. There was no precedent for outbreaks of algal biotoxins in New Zealand and ASP, PSP, NSP, DSP were recorded simultaneously. In addition to human illnesses and toxic shellfish that could be positively linked to the algal toxins, there were several reports of seabird kills during this period. These represented above average levels of deaths of little blue penguins, red bill gulls, shags, petrels, gulls, spotted shags, sooty shearwaters and spotted shags (Jasperse, 1993). While these bird deaths could not be positively linked with the algal

toxins, the presence of algal blooms in the general vicinity of at least some of the deaths certainly is suspicious. Again, it would be interesting to study any archived bird carcasses.

Gill and Darby (1993) reported the deaths of approximately 150 adult, yellow-eyed penguins over a short period during the summer of 1989–1990 in New Zealand. Given that the total mainland population of breeding pairs was only 240, this number represented a significant die-off. The authors state in their abstract that the toxins of dinoflagellates were ruled out as possible poisons; however, in their discussion they cite a personal communication from Jillett of unusual "red tides" observed in the surrounding waters in the summer of 1989–1990 and noted that local fishermen also reported "red tides" at the time the mortalities occurred. They further noted that it is possible that domoic acid or similar toxins may not have been detected by the methods employed in their study, i.e. they did not entirely rule out the possible role of algal toxins in the penguin deaths. It is interesting to note that the 1989–1990 time period is just prior to the beginnings of several outbreaks of various species of toxic algae in coastal New Zealand waters (Jasperse, 1993) and above average recorded bird deaths. These algal toxins were previously unknown to that region, and it seems a strong possibility that the penguins, naive to any algal toxins, succumbed to one or more diatom (domoic acid) or dinoflagellate (brevetoxin, saxitoxin) toxins. It would be both interesting and useful to test any archived samples using current, more sophisticated and sensitive methods of toxin detection.

Black-legged kittiwakes (diet almost exclusively fish) have suffered high mortalities from toxic algae. Coulson and Strowger (1999) presented an extraordinary data set comprising 45 years of continuous monitoring of the mortality rates of adult black-legged kittiwakes (*Rissa tridactyla*) at colonies near the mouth of the River Tyne in northeast UK. In 1997 and 1998, elevated mortality rates from PSP toxins reached 39 and 58%, respectively. Birds were found dead at sea, 6–8 km offshore. During the summers of 1996 and 1997, about 2000 dead kittiwakes were washing ashore but only when prevailing winds were onshore, i.e. many more birds were probably lost at sea. In this case, only kittiwakes were affected and perhaps because the feeding ranges of other species were near-shore and the highest toxin levels were offshore. Coulson and Strowger (1999) estimated that about 13,000 birds were lost from the colonies in those 2 years. It is interesting to note that in addition to the PSP toxins, a positive test for diarrhetic shellfish poison was noted. Algae known to cause DSP have been collected at an offshore sampling station only a few km from the site where the kittiwake mortality occurred (Coulson and Strowger, 1999). While it is unlikely that the DSP toxins were directly responsible for bird deaths, there is every reason to believe that their presence negatively impacted the birds' well-being and their presence clearly indicates yet another threat to the bird colonies. It is particularly sad in this instance to note that the outbreaks of toxic algae appear to be related to the dumping of human sewage and subsequent increased algal concentrations.

Coulson and Strowger (1999, p. 7) suggested that PSP poison was unlikely to have been the cause of the death in the 1996/1997 mortality, stating that mussels on the coast were not toxic. It is possible, however, that food sources offshore could be toxic and it seems to be quite possible that low levels of toxins could have been present and accumulated over time. The symptoms are indicative of PSP poisoning. The authors also noted presence of DSP toxins, known to cause severe gastroenteritis and diarrhea in humans. The first cases of DSP in humans in the UK were reported in June of 1997 but the source of the mussels consumed was not noted. It is also possible that these DSP toxins caused hemorrhaging, hence blood on vents or even that other, toxins, e.g. ASP were present. During this same time period, Krokowski (personal communication) noted high mortalities of kittiwakes off the northeast coast of UK as reported through routine and reactive monitoring as carried out by the Environment Agency of England and Wales (Environment Agency, 2000a,b). Birds died rapidly with signs of hemorrhaging from the gut and in considerable distress. He indicated that historical records indicated similar mortalities. High levels of DSP toxins (okadaic acid and dinophysis 1 were detected in kittiwake livers and high levels of *Dinophysis* sp. cells were noted offshore. DSP was believed to be the cause, directly or indirectly, through weakened individuals.

Kreuder et al. (2002) reported on cormorants (*P. auritus*) along the Florida Gulf Coast that had been

admitted to the Clinic for the Rehabilitation of Wildlife with consistent presentation of neurologic clinical signs over an extended time period. They compared the timing of bird admittances with outbreak-specific clinical signs (severe cerebellar ataxia) to blooms of the brevetoxin-producing marine alga, *Karenia brevis* (formerly *G. breve*) from 1995 to 1999. A total of 360 birds all presented with the same signs of ataxia and they were able to demonstrate a significant ($P < 0.05$) relationship between admittance of cormorants and concurrent presence of *K. brevis* in local waters. Necropsy of cormorants admitted from 1995 to 1999 failed to reveal a specific cause of death; however, through the use of histochemical staining techniques, brevetoxin uptake was demonstrated in each of the four birds admitted in 1997. Brevetoxin was found in the spleen and lung in all cormorants examined, demonstrating that inhalation is another route of exposure in this species. While the authors are careful to point out that this without experimental trials, a cause and effect relationship has not been established, but there seems little doubt that the etiology noted is a direct result of exposure to brevetoxins.

During the first weeks of October 2000 and again in the first part of December 2000, a large number of penguins and other birds died and washed up along the shore of Chubut, Argentina. Over 100 km of beach were walked from the Peninsula Valdes (42°28′S, 63°21′W) to Punta Ninfas (42°57′S, 2′S, 64°20′W) and a total of 1042 dead birds were found (Quintana et al., 2001). They included Magellanic penguins (*Spheniscus magellanicus*), South American terns (*Sterna hirundinacea*), Imperial Cormorants (*Phalacrocorax albivener*), and Great Grebes (*Podiceps major*). A second wave of mortality occurred from 28 November to 6 December. Again, 100 km of beach were checked from Punta Norte (42°4′S, 63°46′W) south of Punta Tombo (44°2′, 65°11′W). Mortality seemed higher than the first event but the same coastline was not walked. Mainly in the Golfo Nueveo, Quintana et al. (2001) found 3399 dead penguins. One of us (P.D.B.) performed a necropsy on a fresh dead penguin on the 18 December at Punta Tombo. The stomach was empty, the lining appeared as if it had been burned, blood vessels were at the surface and inflamed. Nine penguins with satellite transmitters last seen at their nest between 25 October and 13 December failed to return. All these birds were healthy when they departed and the last known position for these birds was over 150 km from land. These satellite tagged birds had chicks and never returned to the colony suggesting they probably died.

A large squid that had been dropped on the ground when a chick was unable to swallow in mid-December was cut up and fed to the chick. The chick lost muscle control, was unable to hold its neck upright and the neck became tonic with the head thrown backwards. The symptoms disappeared after a day of feeding the chick other food. Small filter-feeding fish such as anchovy and invertebrates like squid are an important part of the diet of many seabirds including penguins (Forero et al., 2002) and extensive blooms of *Alexandrium tamarense* and *A. catenella* have been recorded from these regions. The general theory is that these blooms originate offshore and are only sporadically advected inshore reaching intertidal shellfish populations, e.g. a good example of toxic coastal shellfish not always being a reliable indicator of toxic activities in offshore areas. These blooms are now experienced annually in this region (Carreto and Benavides, 1993; Carreto et al., 1986, 1998). Given the timing of the penguin deaths and the fact that they were known to be healthy birds, the symptoms found upon necropsy, the direct response to being fed parts of a squid, and the known incidence of toxic algae during this time period, it is highly likely that the penguins were victims of PSP. Moreover, the reaction of the chick to parts of one large squid it was fed suggests that many of the seabirds potential prey had high toxin levels. Invertebrates like squid are likely to tolerate higher toxins than are vertebrates and can thus be rendered as potentially lethal vectors of algal toxins.

The most recent reports from the Falklands Islands implicate, but have not yet confirmed, the deaths of large numbers of Gentoo Penguins (*Pygoscelis papua*) due to algal toxins. This is especially alarming, as this is the largest flock in the world (∼115,000 birds) and represents 40% of the global population of this species.

Now it seems that penguins are vulnerable not only in their natural habitat, but in captivity. Naar et al. (2002) reported that during the summer of 2000, four king penguins (*Aptenodytes forsteri*) from a zoo in Kentucky (USA) died. Subsequent investigation

identified toxins (domoic acid, 20 ppm; and PSP toxins, 30–35 MU/100 g) in the guts of anchovies fed to the penguins. The fish were caught in Monterrey Bay, California (USA).

Landsberg (personal communication) reported a lesser scaup mortality (~20 birds) in Dead Lake along the west coast of Florida (USA) in the winter of 2002. Concentrations of brevetoxins in liver and lungs and high concentrations in intestinal tract were confirmed.

There are very few records of laboratory studies on birds and algal toxins. Gochfeld and Burger (1998) reported on the apparent paralytic shellfish poisoning of captive herring gulls fed commercial scallops. The authors fed diced adductor muscle from scallops purchased at a local grocery store to herring gull chicks. While the chicks were obviously severely impacted and exhibited a characteristic acute syndrome, no algal toxins (tested for brevetoxins, domoic acid, and saxitoxins) were found. It is possible, but unlikely, that algal toxins would be the source of distress in this instance as adductor muscles rarely contain toxins, even when the surrounding tissues contain very high levels (Shumway and Cembella, 1993). It is also possible that the chicks were highly sensitive and that levels of toxins present were below the levels of detection. Not being able to identify the original source of the scallops, however, makes it impossible to determine which toxin(s) might have been implicated or which species of scallops were involved.

3. Symptoms

It is evident that seabirds exhibit a wide range of sensitivities to algal toxins and symptoms vary depending upon the species of bird and the algal toxin involved. Some of these symptoms are summarized in Table 2 from Coulson et al. (1968b).

Several authors have noted no toxin in specific bird tissues (e.g. livers, muscles, hearts, see Table 3) but this is not surprising. Death appears to be rapid in most species and toxins are most likely not metabolized. The majority of bird deaths appear due to consumption of planktivorous fish by birds that consume their prey whole, i.e. no opportunity for 'tasting' or testing by naive birds. Inshore/coastal birds appear more likely to have developed conditioned aversions to algal toxins. Low doses of PSP and ASP toxins cause loss of motor coordination which leads to impaired swimming, flying, foraging which can lead to starvation. Higher doses will impair respiration and cause immediate death. Kreuder et al. (2002) noted severe cerebellar ataxis in all cormorants admitted to their clinic (360) and presumably exposed to brevetoxins. They reported that the ataxia was characterized by a "broad-based stance, truncal incoordination, hypermetric gait and intention tremors of the head", and that approximately half of the ataxic cormorants also exhibited vertical nystagmus (involuntary rapid movement of the eyeball) and that the ataxia lasted for 2–4 days.

PSP causes loss of motor coordination followed by paralysis and it is likely that birds unable to feed effectively when first poisoned and die from starvation (Hockey and Cooper, 1980). Black oystercatchers banded and weighed prior to the outbreaks lost approximately 36% of their body mass prior to death. Coulson et al. (1968a,b) also noted that shags (*P. aristotelis*) lost 16% of their mass after exposure to PSP toxins and also attributed it to starvation.

Evidence of vomiting by adult shags was also reported by Coulson et al. (1968b). Adult shags, unlike cormorants, only rarely regurgitate undigested food. Female terns died in the process of egg laying due to malfunction of the oviduct and implicated as the cause of death in 38 out of 44 females (Coulson et al., 1968b).

In birds that have consumed and not regurgitated toxic prey, symptoms appear similar, even in the chickens examined by McKernan and Scheffer (1942), where the entire length of intestine was inflamed, blood vessels enlarged, and sharply outlined. Inflammation followed by death was experimentally produced in chickens by feeding them offal from razor clams. McKernan and Scheffer reported the same condition in unidentified adult gull species and three white-winged scoters also had inflamed intestines.

The same extensive inflammation of the alimentary canal, excessive defecation, and often hemorrhages at the base of the brain and elsewhere have been reported in birds exposed to saxitoxins (Coulson et al., 1968a,b). Kittiwakes that had died on land had blood on the feathers around the vent, also indicative of hemorrhaging (Coulson and Strowger, 1999).

A reassessment of historical data on bird kills coupled with records of HAB species/blooms might explain some of the bird kills.

4. Learning to avoid algal toxins

There is some evidence, both anecdotal and experimental, that birds in areas prone to toxic algae have developed a learned behavior to avoid consumption of deadly algal toxins. Naive birds, i.e. those that have not previously encountered blooms, may be at greatest risk. Also, those that eat food 'whole' rather than 'tasting' may be at greater risk.

The presence of HABs may influence both temporal and spatial patterns of predation pressure. Birds may avoid entire geographic regions or prey populations harboring algal toxins and thus it is likely that birds may play a major role in shaping benthic community structure. For example, blue mussel, *Mytilus edulis*, is the most important food item for the common eider (*Somateria mollissima*) (Leopold et al., 1996; Bustnes, 1998) and mussel farms are regularly plagued by the presence of these ducks (see Milne and Galbraith, 1986). These ducks are a sensitive indicator of toxic shellfish in some regions; Eider ducks have been shown to select mussels with the highest meat to shell ratio, i.e. minimize the shell intake (Bustnes, 1998; Bustnes and Erikstad, 1990). They demonstrated that the length of mussels eaten varies between areas and seasons, with ducks reducing the amount of indigestible shell by selecting mussels of different lengths. Mussels higher up the shore tend to be smaller than their sub-tidal counterparts. Coulson et al. (1968a) suggested that since the highest levels of toxins were found in mussels (*M. edulis*) and that very few eider (*S. mollissima*) died, that this species has some means of reducing the toxic effect of the poison. Subsequent observations seem to indicate that the ability of the ducks to sense toxins and avoid consumption of toxic mussels shields them from effects of the toxins.

In Maine waters where blooms of *A. tamarense* are an annual event leading to toxic shellfish, it has long been known that Eider ducks avoid toxic mussels under field conditions (Hurst, personal communication; personal observation). The ducks' position in the field in the upper estuary is an indication to field samplers that shellfish (mussels) are probably toxic. In laboratory studies, Eider ducks were offered toxic versus non-toxic mussel meats and refused the toxic mussels. Eider ducks that were force-fed toxic mussel meat regurgitated the food almost immediately (Hurst, unpublished). This selective behavior could have long-term implications for the nutrition of the ducks. While ducks would normally choose large mussels low on the shore (Bustnes, 1998), the presence of red tide in Maine appears to drive the ducks higher up the shore where they must settle for smaller, less toxic mussels or cease feeding altogether. Mussels at these higher levels may be less toxic, but the ratio of shell to meat is higher, forcing ducks to be less effective predators. In some areas the eiders switch their prey to sea urchins.

In other examples of seabird aversion of algal toxins as a learned behavior, black oystercatchers in California, USA, feed primarily on bivalves, often mussels, *Mytilus californianus*. It was noted by Falxa (personal communication) that during a 1989 bloom of PSP (levels in mussels >1500 μg/100 g) oystercatchers dropped or rejected mussel meat, a behavior he had not noted previously when birds were exposed to non-toxic prey. He also noted prey switching and partial consumption of mussel prey following detection of high levels of paralytic shellfish toxins in mussels (Faxla, 1992). He noted no mortality among banded or unbanded study birds during the bloom. Given that PSP-laden shellfish are a regular occurrence in California waters (State of California Department of Health Services Marine Biotoxin Monitoring Program Technical Reports Series), it seems to be that local oystercatchers have developed an aversion response not present in the naive South African oystercatchers where heavy mortalities were noted (Hockey and Cooper, 1980; Horstmann, personal communication). Oyster catchers may have an added advantage in that their bills are known to contain sensory nerves (Gill, 1995; Goss-Custard, 1996) which might detect neurotoxins prior to ingestion.

In the Bay of Fundy, St. Lawrence Estuary area, and Gulf of Maine, toxic *Alexandrium* (*Gonyaulax*) blooms have been persistent for hundreds of years if not longer, yet there are no reports of mass mortalities of seabirds. It seems to be that these birds have also been conditioned to avoid toxic prey. During serious outbreaks in Maine in 1972 and 1980 gulls stopped eating surfclams (later found to be toxic) that were

washed ashore (Hurst, personal communication). The same is true for other geographic areas plagued by regular outbreaks of toxic algae. Kvitek (1993) suggested most top-level predators must have developed the means to detect and avoid toxic prey or greater numbers of mass mortalities of these top-level predators would be observed. This still leaves naive populations of seabirds at great risk in the face of new blooms species.

Kvitek (1991, 1993) has carried out the most detailed experimental studies on the interactions of birds and toxic prey. He demonstrated that wild, naive, free-ranging glaucous-winged gulls (*Larus glaucescens*) initially regurgitated toxic butter clams (*Saxidomus giganteus*) within 5 min of ingestion and that non-toxic butter clams were never regurgitated. Gulls previously conditioned with toxic butter clams refused to eat either toxic or non-toxic butter clams but ate other bivalve molluscs. In a field situation, gulls at a highly toxic site took significantly fewer butter clams than at a non-toxic site. He attributed this to conditioned aversions developed early in life and once conditioned, the gulls avoided all *Saxidomus*, regardless of toxicity, but not other clams. Further, he showed that gulls foraging at a known toxic site discarded the siphons (the site of toxin storage in this species of bivalve) of both toxic and non-toxic butter clams but never those of other bivalves. Gulls feeding at a non-toxic site never discarded the siphons of the butter clams. This not only demonstrates an extraordinary capability for learned aversion behavior in gulls, it goes a long way toward explaining why more seabirds are not killed by recurrent HABs.

It is very interesting to note that Kreuder et al. (2002) reported that recovered cormorants that were banded and released during red-tide events were readmitted to the clinic with the same cerebellar ataxia noted during their first admittance as soon as 5 days after release, suggesting no learned response to the presence of toxins in their food source or a larger role for the aerosol-borne toxins in these birds, i.e. they could not avoid the toxins.

Those birds that have the opportunity to taste, e.g. gulls, ducks, and oyster catchers that drop or break their prey, and regurgitate quickly are obviously at a greater advantage with respect to avoiding toxic algae through learned behavior than those that swallow their prey whole, e.g. cormorants and shags.

5. Seabird conservation

Seabird mortality is an ecosystem problem and HABs may present a real concern with regard to seabird conservation, especially in areas where new HABs occur and naive seabirds are at risk. Novel blooms in regions of naive populations of birds could have disastrous effects on populations, especially in areas with island populations. Most seabirds have low recruitment rates and hence recovery of populations may be slow (Lack, 1968; Hockey and Cooper, 1980).

Endangered species of marine birds are potentially at risk from the continued global spread of HABs. A heightened awareness of the potential impacts of HABs on birds coupled with a comprehensive data base can provide conservation managers with the information necessary to protect targeted bird colonies during HAB outbreaks. Compilation and maintenance of data regarding seabirds and HABs, even if only anecdotal, by a central collection point such as the data bases maintained for marine mammals and sea turtles could prove a valuable tool in the protection of marine birds.

Spitzer (1995) noted that species in southeastern ocean waters, such as the endangered Bermuda petrel (*Pterodroma cahow*) and the black-capped petrel (*Pterodroma hasitata*) might be highly vulnerable to unprecedented blooms and the same may be true for other birds such as Florida pelicans, loons and gannets. Spitzer (personal communication) has recently recorded large and unexplained die-offs of loons in Long Island (New York, USA) waters and Henvoldsen (unpublished) has reported bird deaths in Europe (summer 2002) as a possible results of DSP toxins.

There are also potential problems associated with fishing vessel discard and offal. Finfish and shellfish discards constitute a major portion of the diet of many species of seabirds (Garthe et al., 1996) and in some areas, e.g. the North Sea, it has been estimated that discards could sustain ~5.9 million individual. Furness and Hislop (1981) showed that even when their preferred prey, lesser sand eels, were abundant, Great Skuas breeding in Shetland got 70% of their diet from discards.

The likelihood of encountering paralytic shellfish toxins in scallop viscera is extremely high in most of coastal Maine. Scallops are known to sequester paralytic shellfish toxins for extended periods of

time (months–years) and predominantly in the tissues discarded after harvest of the adductor muscles (Shumway and Cembella, 1993; Cembella et al., 1993) thus the use of scallops from this region as an added food source for any animals is a dangerous proposition. Seabirds feeding on this material would be in grave danger and, since death would most likely occur at sea, mortalities would go unnoticed. Dehydrated scallop viscera as a component of poultry rations was suggested (Blamberg and O'Meara, 1973). The authors reported chick mortalities and symptoms that closely resembled paralytic shellfish poisoning; however, they also demonstrated a thiamine deficiency that manifests itself with similar symptoms, thus it is not possible to determine which factor was responsible for the chick deaths. Medcof (1985) reported death of domestic hens after they were fed scallop shuckings.

In many regions, scallops are shucked at sea and the discarded materials can contain extraordinarily high levels of algal toxins throughout most of the year (see Shumway and Cembella, 1993). Seabirds feeding on this material would be in grave danger and, since death would most likely occur at sea, mortalities would go unnoticed. Thus, discards and offal may play a previously unrecognized and significant role in seabird ecology and trophic interactions. Finally, cells and cysts of HAB species may be spread by ballast water and be introduced to new areas; there is also increased incidence of aquaculture, and increased detection of cells/toxins with new methods, new techniques, and lower sensitivities.

6. Conclusions/recommendations

The full impact of HABs on marine birds is likely underestimated and the need for interdisciplinary research is obvious. While the impacts of the toxins themselves may not always be lethal, they may render the birds more vulnerable to other stressors in their environment and lead to mortalities. This would seem to be especially true of migratory species that have spent their energy reserves and arrive emaciated at toxic shellfish beds or encounter schools of toxic fish. In this condition, even a small dose of toxin would probably render the birds incapable of feeding and lead to starvation.

It is probable that more 'wrecks' of coastal species are recorded as only a small proportion of offshore 'wrecks' come ashore. Rarely is the size of the wreck any indication of the total mortality of birds at sea. Often the observed mortalities represent only a percentage of the total bird deaths (Work et al., 1993a,b; Ochoa et al., 1996). Work et al. estimated that total mortality for the duration of the epidemic (30 days) was probably at least twice their carcass count. McKernan and Scheffer noted that fishermen reported large numbers of dead birds floating 10–20 miles off the coast (murres and shearwaters); Coulson et al. (1968a,b) suggested that many of the dead shags sank or were eaten and were not recorded and that the corpses found along the shoreline may have represented only a fraction of the total mortality.

Shags and cormorants seem to appear in the records more often than other species, undoubtedly a function of their feeding habits (consumption of whole prey with no sampling or testing), choice of food items (sand eels), and their coastal habitat (more visible), makes them especially vulnerable to algal toxins and mortalities are more likely to be noticed.

With the increased incidence of HABs coupled with an increased global awareness regarding their potential impacts on bird populations, it is highly likely that more incidences of bird kills resulting from HABs will be confirmed. Environmental education programs are needed to train scientists and the public to recognize and help watch for the potential for seabird health impacts from HABs.

It would be both interesting and useful to test any archived samples of birds using current, more sophisticated and sensitive methods of toxin detection.

Finally, there needs to be a central repository for seabird kill data. Possibilities include the National Wildlife Health Laboratory in Madison, Wisconsin, or the ICES Working Group on Seabird Ecology (ICES, Copenhagen, Denmark). A standardized form for data collection/submission should be developed and distributed.

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