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The Impact of Toxic Algae on Scallop Culture and Fisheries

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ABSTRACT: Harmful algal blooms occur worldwide and their associated phycotoxins are accumulated by filter-feeding bivalve molluscs. Because only the adductor muscle of scallops has been traditionally marketed, scallops are not usually included in routine monitoring programs. A renewed interest in marketing both whole and "roe-on" scallops from various geographic regions along with intensified aquaculture ventures in areas prone to toxic blooms have provoked public health concerns regarding the safety of this resource.

Our studies have focused on the sequestering and biotransformation of phycotoxins in scallops. Our results, coupled with a review of historic data, indicate that (1) toxins are not distributed evenly throughout the scallop tissues—more toxin is usually concentrated in the mantle and digestive gland; (2) some scallop tissues, e.g., digestive glands and mantles, remain highly toxic throughout the year; (3) toxicity varies considerably (43.5% coefficient of variation) between individual animals collected in the same area; (4) no correlations could be made between toxicity levels in gonadal tissue and other tissues.

Scallop culture and commercial fisheries can thrive in areas prone to toxic algal blooms if only the adductor muscle is utilized. Safe marketing of roe-on scallops is feasible only under strict regulatory regimes. Marketing of mantles or whole scallops poses a high risk to public health and should be undertaken only after extensive monitoring. Scallop mariculturists should be acutely aware of the potential risks associated with phycotoxins. Furthermore, public health guidelines, with particular emphasis on toxin levels in individual tissues, is necessary if scallops are to be marketed whole or in conjunction with tissues other than adductor muscles.

KEY WORDS: Toxic algae, phycotoxins, scallops, fisheries, aquaculture, paralytic shellfish poisoning.

I. INTRODUCTION

Scallops are common inhabitants of most coastal ecosystems throughout the world, and major commercial fisheries and mariculture operations are dependent on this resource (see Hardy, 1991; Shumway, 1991 and references therein). Blooms of toxic

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and noxious microalgae are frequent episodic events in scallop-growing areas (see LoCicero, 1975; Taylor and Seliger, 1979; Anderson et al., 1985; Okaichi et al., 1987, 1989; Graneli et al., 1990; Hallegraeff, 1992). The cosmopolitan distribution of many toxic microalgal species has had a dramatic deleterious effect on shellfish utilization (reviewed by Shumway, 1990). Scallop culture and harvesting of wild stocks are frequently carried out in areas prone to blooms of highly toxic algal species, e.g., in coastal waters of Canada, Japan, U.S., and France (see Shumway, 1990, 1991 and Figure 1 for scallop species and geographical locations). While some microalgal species, such as the brown tide picoplankter *Aureococcus anophagefferens*, have had devastating consequences on the health of scallop populations (Cosper et al., 1987; see Table 1 for summary), the primary threat to industry and public health is the risk of human illnesses.

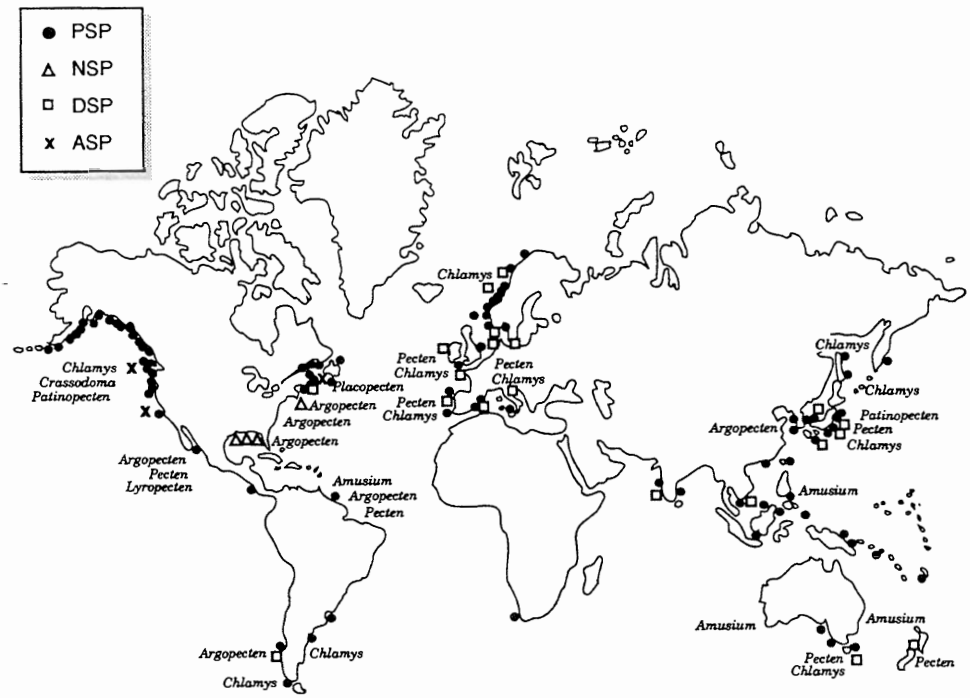

FIGURE 1. Known distribution of PSP, DSP, NSP, and ASP toxins in commercially exploited (fished or cultured) scallop genera.

Bivalve molluscs, including scallops, accumulate microalgal toxins in their tissues through filter-feeding. Scallops are opportunistic filter feeders that take advantage of both pelagic and benthic microorganisms as food sources (see Shumway et al., 1987; Bricelj and Shumway, 1991). These organisms are consumed and concentrated in the digestive gland; where toxic algal species are present, shellfish can be rendered unfit for human consumption. Filter-feeding shellfish can thereby serve as vectors of various seafood poisoning syndromes, such as paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning (DSP), and amnesic shellfish poisoning (ASP), in human consumers.

TABLE 1

A Summary of Toxic and Noxious Algal Species Associated with Scallops

Algal species	Shellfish species affected	Notes	Location	Ref.
Dinophysis acuminata *D. fortii*	*Chlamys nipponensis* *Patinopecten yessoensis* *Pecten albicans*	Toxic	Japan	Anraku (1984)
Alexandrium tamarense	*Chlamys opercularis* *Pecten maximus*	Highly toxic; not adversely affected	Northumberland, U.K.	Ayres and Cullum (1978); Ingham et al. (1968); Wood and Mason (1968)
A. tamarense	*Placopecten magellanicus*	Highly toxic	Gulf of Maine and eastern Canada; Bay of Fundy and St. Lawrence regions	Prakash (1963); Bourne (1965); Caddy and Chandler (1968); Prakash et al. (1971); Medcof(1972); Hurst (1975); Hartwell (1975); Hsu et al. (1979); Tufts (1979); Jamieson and Chandler (1983); Shumway et al. (1988); Gillis et al. (1991); Cembella and Shumway (1991)
A. tamarense	*Placopecten magellanicus*	Highly toxic	Georges Bank, Gulf of Maine	White et al. (1992a,b)
A. tamarense	*Chlamys nipponensis* *Patinopecten yessoensis*	Toxic	Japan	Oshima et al. (1982a)
A. tamarense	*Argopecten irradians*	Toxic	Massachusetts	Bicknell and Collins (1973)
A. tamarense	*Patinopecten yessoensis*	Toxic	Japan	Sekiguchi et al. (1989)
A. tamarense (GT429)	*Placopecten magellanicus*	Violent swimming activity; production of mucus	Laboratory	Shumway and Cucci (1987); Gainey and Shumway (1988a,b)

TABLE 1 (continued)

A Summary of Toxic and Noxious Algal Species Associated with Scallops

Algal species	Shellfish species affected	Notes	Location	Ref.
A. tamarense (MOG 835)	*Pecten maximus*	Toxic	Laboratory	Lassus et al.(1989)
A. tamarense	*Patinopecten yessoenis*	Toxic	Japan	Maruyama et al. (1983)
A. tamarense	*Chlamys nipponensis* *Chlamys nobilis*	Toxic	Japan	Anraku (1984) Oshima et al. (1982a)
Alexandrium sp.	*Patinopecten yessoensis*	Toxic	Japan	Nishihama (1980)
A. catenella	*Patinopecten yessoensis* *Chlamys nipponensis akazara*	Toxic	Japan	Noguchi et al.(1978; 1980a,b; 1984)
A. catenella	*Hinnites multirugosus* *Chlamys hastata*	Toxic; human illnesses	British Columbia, Canada	DFO (1987;1989)
A. catenella	*Hinnites multirugosus*	1 death from eating viscera	California	Sharpe (1981)
A. catenella	*Chlamys hastata* *Hinnites multirugosus* *Pecten caurinus* *Pecten sp.*	Toxic	Pacific Coast states of U.S.	Nishitani and Chew (1988)
A. catenella	*Chlamys patagonicus*	Toxic	Chile	Avaria (1979); Guzman and Campodimico (1978)

TABLE 1 (continued)

A Summary of Toxic and Noxious Algal Species Associated with Scallops

Algal species	Shellfish species affected	Notes	Location	Ref.
A. catenella	*Patinopecten yessoensis*			
Gymnodinium catenatum	*Equichlamys bifrons* *Mimachlamys asperrimus* *Pecten fumata*	Toxic	Tasmania	Hallegraeff and Summer (1986); Hallegraeff et al. (1989); Oshima et al. (1982a; 1987a,b)
G. catenatum	*Pecten albicans*	First report of toxicity by this species	Japan	Ikeda et al. (1989)
G. breve	*Argopecten irradians*	Scallop mortality; recruitment failure	North Carolina	Barris (1988); Tester and Fowler (1990); Summerson and Peterson (1990)
G. veneficum	*Pecten maximus*	Scallop mortality	Laboratory	Abbott and Ballantine (1957)
Gyrodinium aureolum	*Pecten maximus*	Mortalities in young scallops	France	Lassus and Berthome (1988)
Gy. aureolum	*Pecten maximus*	Numbers of larvae declined during bloom	Lough Hyne, Ireland	Minchin (1984)
Gy. cf. aureolum	*Pecten maximus*	High mortality in post-larvae and juveniles; reproduction and growth inhibited in adults	France	Erard-LeDenn et al. (1990)

TABLE 1 (continued)

A Summary of Toxic and Noxious Algal Species Associated with Scallops

Algal species	Shellfish species affected	Notes	Location	Ref.
Ceratium tripos	*Placopecten magellanicus*	Nontoxic; mass mortalities due to oxygen depletion	New York Bight	Mahoney and Steimle (1979)
Aureococcus anophagefferens	*Argopecten irradians*	Larval shell growth reduced and mortality increased	Laboratory	Gallagher et al. (1988)
Au. anophagefferens	*Argopecten irradians*	Mass mortalities	Long Island embayments NY; Narragansett Bay, RI; Barnegat Bay, NJ	Cosper et al. (1987); Tracey et al. (1988); Tracey (1985); Smayda and Fofonof (1989)
Au. anophagefferens	*Argopecten irradians*	76% reduction in adductor weights; recruitment failure of year class	Long Island, NY	Bricelj et al. (1987)
Rhizosolenia chunii	*Pecten alba*	Bitter taste rendered shellfish unmarketable for 7 months; digestive gland lesions and subsequent shellfish mortalities	Australia	Parry et al. (1989)
Not specified; probably *Alexandrium sp.*	*Patinopecten yessoensis* *Chlamys farreri*	Toxic	Korea	Jeon et al. (1988)
Not specified	*Chlamys nobilis*	Toxic	Japan	Nagashima et al. (1988)

Note: Alexandrium tamarense (=Gonyaulax tamarensis = Protogonyaulax tamarensis); A. catenella (=Gonyaulax catenella = Protogonyaulax catenella); Gymnodinium breve (=Ptychodiscus brevis).

II. PHYCOTOXINS AND TOXIC ALGAL SPECIES

The marine toxins of algal origin, collectively known as phycotoxins, are a diverse group of biologically active compounds with high acute toxicities in humans. Among the most potent toxins known to accumulate in scallops are the PSP toxins, consisting of the neurotoxin saxitoxin (STX) and at least 17 naturally occurring derivatives (Figure 2), with strong sodium channel-blocking activity that can cause muscular paralysis in vertebrates. In most temperate waters, the organisms responsible for PSP are toxigenic marine dinoflagellates of the genus *Alexandrium* (alternatively known as members of the *Protogonyaulax catenella/tamarensis* species complex, and formerly as *Gonyaulax* spp.) and *Gymnodinium catenatum*; in the tropics, *Pyrodinium bahamense* var. *compressum* is often implicated (Taylor, 1984; Shimizu, 1987). The DSP toxins, the polyether okadaic acid (OA) and dinophysistoxin (DTX) derivatives, as well as related yessotoxins and pectenotoxins found in scallops and certain other shellfish (Yasumoto et al., 1984), are primarily gastrointestinal toxins in humans, exerting a potent (though non-lethal) effect

R1	R2	R3	Carbamate Toxins	N-Sulfocarbamoyl Toxins	Decarbamoyl Toxins
H	H	H	STX	B1	dc-STX
OH	H	H	NEO	B2	dc-NEO
OH	H	OSO_3^-	GTX 1	C3	dc-GTX 1
H	H	OSO_3^-	GTX 2	C1	dc-GTX 2
H	OSO_3^-	H	GTX 3	C2	dc-GTX 3
OH	OSO_3^-	H	GTX 4	C4	dc-GTX 4

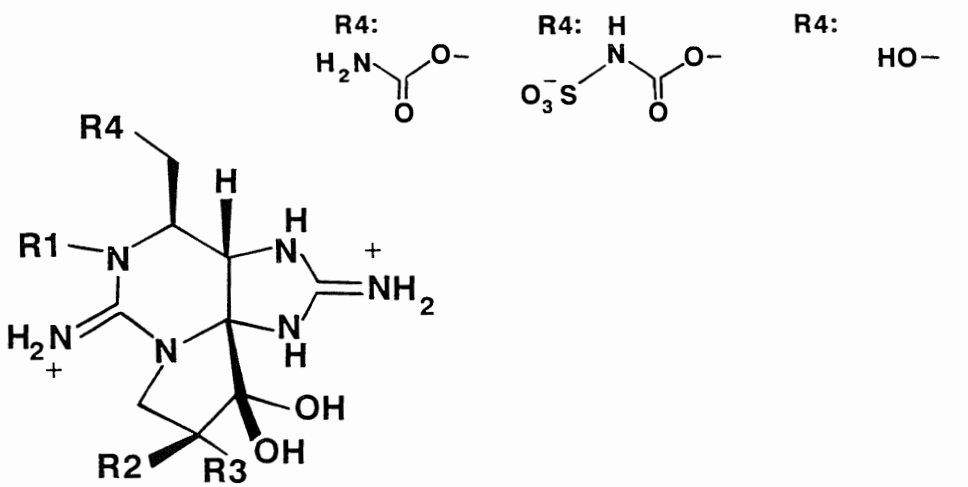

FIGURE 2. Structures of PSP toxins, including carbamate, N-sulfocarbamoyl, and decarbamoyl derivatives found in various species of bivalve molluscs. Saxitoxin = STX; neosaxitoxin = NEO; gonyautoxins 1,2,3,4 = GTX 1,2,3,4.

through phosphatase inhibition. The organisms usually implicated in DSP incidents include planktonic *Dinophysis* spp., and possibly a few epiphytic/benthic species of the dinoflagellate genus *Prorocentrum*. The neuroexcitory amino acid, domoic acid, produced by several pennate diatom species, most prominently by *Nitzschia pungens* f. *multiseries*, was responsible for cases of ASP in consumers of mussels from Atlantic Canada (Todd, 1990). Domoic acid also was found in digestive glands of sea scallops from the Bay of Fundy, albeit at lower levels than those associated with ASP caused by consumption of mussels (Gilgan et al., 1990). The accumulation of other phycotoxins in scallops, such as brevetoxins (the cause of neurotoxic shellfish poisoning, NSP) produced by *G. breve* (= *Ptychodiscus brevis*) is currently unknown, and no incidents of human intoxication by scallops have been confirmed.

III. REGULATORY, PUBLIC HEALTH, AND ECONOMIC ASPECTS

The effect of toxic algal blooms on scallop culture and fisheries is often underestimated or even ignored, particularly in North America, because traditionally only the solitary, large adductor muscle is consumed. The adductor muscle tissue is usually free of accumulated toxins, although exceptions to this rule are found occasionally (see later discussion). Scallops are normally shucked at sea, where shells and unwanted tissues are discarded immediately. These other tissues include the mantle (rings or rims), gonad (roe), digestive gland (hepatopancreas, liver), and gills, together comprising >80% of the total tissue weight (Schick et al., 1992; Figure 3). Historically, in North America, scallops have not been routinely included

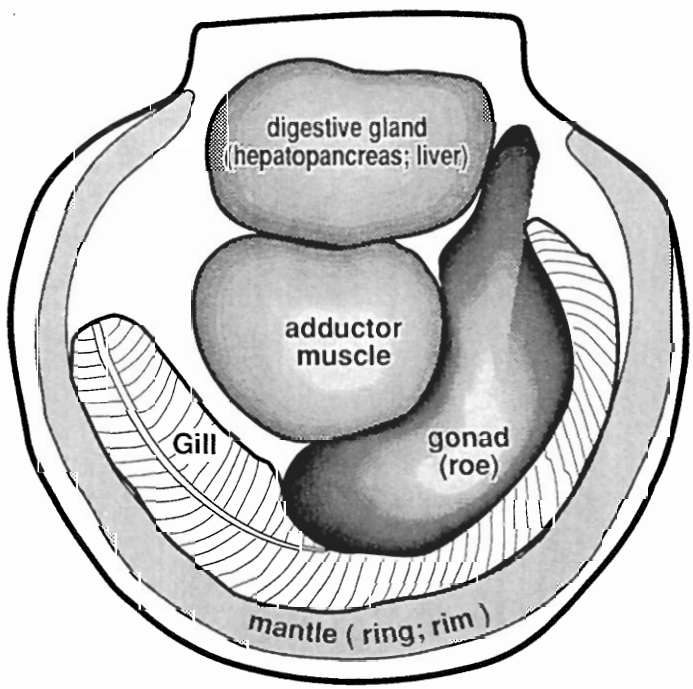

FIGURE 3. Diagrammatic representation of scallop organs.

in PSP-monitoring programs, and scallops have only recently been covered by regulations of the Interstate Shellfish Sanitation Conference (ISSC). Scallops are, nevertheless, subject to the inimical effects of toxic algal blooms (see Table 1). Figure 1 shows areas prone to toxic algal blooms and zones where scallops are either fished or cultured on a commercial scale. The overlap is obvious and, coupled with worldwide expansion of scallop culture and increased interest in marketing non-traditional scallop tissues (e.g., whole and "roe-on" scallops), a clear understanding of the potential problems and hazards posed to the scallop industry by toxic algae is required. In some markets, e.g., in Europe and in Australia, scallops are sold with the gonad attached (roe-on) and there has been a steady and continuing interest in fuller utilization of scallop tissues in other countries, particularly in the U.S. and Canada (Bourne and Read, 1965; Dewar et al., 1971).

The sea scallop *Placopecten magellanicus* and the Japanese scallop *Patinopecten yessoensis* support the two largest scallop fisheries worldwide. *P. magellanicus* is currently the focus of efforts to market roe-on product and whole animals, as is already done with *Pt. yessoensis*. Prior to the identification of PSP toxicity on Georges Bank (see White et al., 1992a,b), Bourne and Read (1965) advocated the marketing of scallop adductor muscles with gonads and mantles attached. Dewar et al. (1971) presented procedures and recipes for high-quality frozen and canned products and, based on the results of Japanese taste panels, indicated consumer acceptance. There is a renewed interest in marketing both whole and roe-on sea scallops from Canada and the northeastern U.S. (Gillis et al., 1991; Merrill, 1992), and whole pink scallops (*Chlamys hastata*) from the Pacific northwest (Nishitani and Chew, 1988). However, the presence of high levels of PSP toxins in scallops from Georges Bank (Sharifzadeh et al., 1991; White et al., 1992a), and the persistent PSP toxicity in the Pacific northwest (Nishitani and Chew, 1988; Shumway, 1990) have sparked new concerns with regard to consumer safety (Ahmed, 1991).

The idea that consumption of scallops is always safe should not be accepted unreservedly. Although scallops are not frequent causes of phycotoxic episodes, there have been illnesses due to PSP and DSP, and even deaths attributable to the former. In Japan, since 1977, DSP toxicity in scallops has resulted in several hundred illnesses (Nomata, personal communication). In North America, cases of PSP have been reported after consumption of whole sea scallops (Medcof et al., 1947; Washington Office of Public Health Laboratories and Epidemiology, 1978), as well as pink and spiny scallops (consumed whole) (Canadian Department of Fisheries and Oceans, 1989). In the Philippines, a 5 1/2-year-old boy died of PSP after eating scallops from Olotayan Island (Estudillo and Gonzales, 1984). One death attributed to PSP was recorded from Iwate prefecture, Japan, following consumption of *C. nipponensis* (Nomata, personal communication), and another fatality occurred in California after the purple-hinge rock scallop *Hinnites* was eaten (Sharpe, 1981).

In regions such as the Pacific and Atlantic coasts of North America and in Japan, where toxic blooms are regular events (Ogata et al., 1982; Gillis et al., 1991; Nishihama, 1980), the risk of PSP and DSP has had a depressive effect on the scallop industry. Japan has ceased supplying whole scallops to large markets, e.g., France, because of PSP toxicity (Merrill, 1992; see Figure 4). Careful monitoring in eastern Canadian waters resulted in the closure of the major portion of the Canadian sector of Georges Bank to roe-on fishing during 1989–1990, when PSP toxin levels

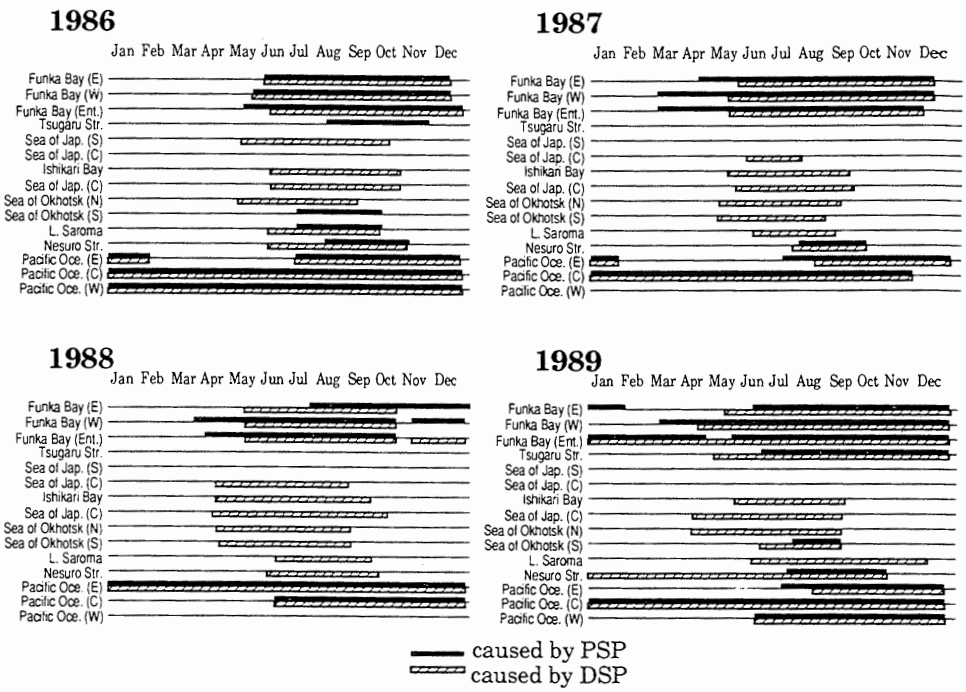

FIGURE 4. Shipment holdback of fresh scallops (*Patinopecten yessoensis*) in Hokkaido, Japan as a result of PSP and DSP toxins. (Modified from Y. Nishihama 1987-1990; after Nomata, personal communication.)

exceeded the regulatory limit (80 µg STXeq/100 g shellfish tissue; note: STXeq = saxitoxin equivalents) (Gillis et al., 1991). In the U.S., efforts are currently underway by the National Marine Fisheries Service (NMFS), the Food and Drug Administration (FDA), and other concerned agencies to develop a protocol for certification of roe-on or whole scallops (*Placopecten*) harvested in northeast federal waters west of 71°W longitude (Figure 5). In Canada, the Inspection Branch of the Department of Fisheries and Oceans is pursuing a similar objective. Until the issue of residual toxicity is resolved satisfactorily by regulatory programs, the exploitation of off-shore scallop resources will remain severely restricted.

IV. VARIABILITY IN TOXICITY AND TOXIN COMPOSITION

Little information is available regarding the uptake and detoxification kinetics of phycotoxins in scallops other than for PSP toxins. It is known, however, that domoic acid is strongly retained by the digestive gland of Atlantic sea scallops; specimens kept in a flow-through seawater system failed to detoxify completely even after 4 months at 8 to 13°C (Gilgan et al., 1990). Work is anticipated on the uptake and elimination of domoic acid by bay scallops *Argopecten irradians* (Scarratt, 1991), but these experiments have not been completed. As a consequence, the following discussion is restricted to the variation observed in PSP toxicity. Although the fundamental accumulation mechanisms are undoubtedly similar, the rates of accumulation and release of highly water-soluble toxins, such as PSP toxins and domoic

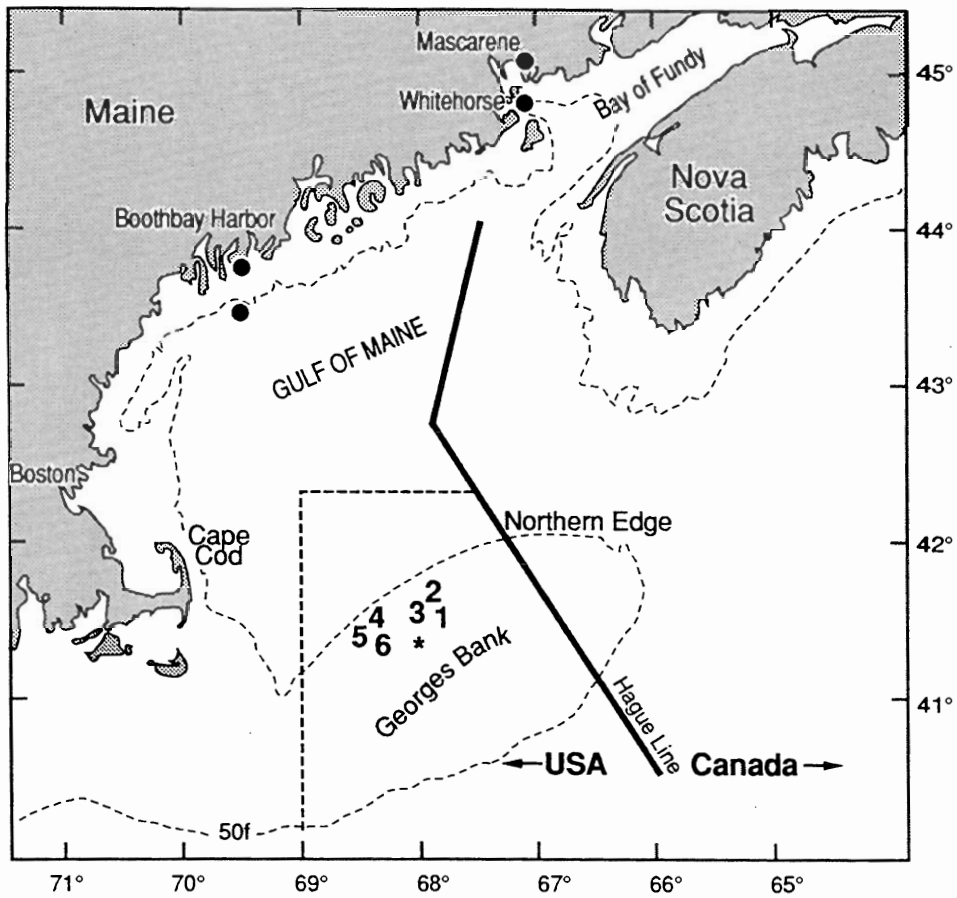

FIGURE 5. The Georges Bank region of the Gulf of Maine in the northwestern Atlantic. The heavy dashed line bounds the American sector of Georges Bank, which is closed to shellfish harvesting due to PSP toxicity in excess of regulatory limits. Also shown are Mascarene, Whitehorse, and the Northern Edge (see Figure 7). Numbers indicate sampling stations.

acid, would be expected to be rather different from those of lipophilic compounds, including DSP toxins and brevetoxins.

A. SPATIO-TEMPORAL VARIATION

Toxicity of scallops does not always coincide with observed toxic blooms determined by cell counts from the water column; this may be due in part to slow detoxification kinetics (discussed later) compared to rapidly depurating species such as the blue mussel, *Mytilus edulis*. In fact, maximal PSP toxicity in scallops usually occurs following a significant lag phase after peak toxic cell densities in the water column are reached. Maximum toxicity in *Pt. yessoensis* cultured populations in Japan, as determined by AOAC mouse bioassays for PSP toxins (AOAC, 1984), did not occur until approximately 1 week after the peak abundance of *A. tamarense*

(= *Protogonyaulax tamarensis*) cells was observed (Kodama et al., 1982a,b; Kodama and Ogata, 1988). In one instance, Ogata et al. (1982) noted an increase in PSP toxicity in scallops, even after *A. tamarense* cells had disappeared completely from the water column (see also Bourne, 1965). Nishihama (1980) monitored PSP toxicity by mouse bioassay in *Pt. yessoensis* populations at two depths (10 and 25 m) and found that the toxicity of scallops at 10 m depth increased rapidly at the end of May, and began to decrease suddenly at the beginning of June. The toxicity of scallops at 25 m reached maximum levels at the end of June, and then declined rapidly through mid-July. The changes in scallop toxicity at both depths corresponded with seasonal and vertical abundance of *Alexandrium* sp. In both groups of scallops, toxicity continued to decrease and had almost disappeared by the end of January. Rates of accumulation and detoxification did not appear to be affected by depth (Figure 6).

The concentration of toxins in scallops is known to vary both seasonally and according to geographical location. Jamieson and Chandler (1983) noted that PSP toxicity peaked in Bay of Fundy scallops during fall and winter, when no toxic blooms were evident. This toxicity variation is undoubtedly due to a combination of factors, including the timing, persistence, and magnitude of toxic blooms; the specific toxicity per cell and toxin composition of the contaminating organism; environmental effects on scallop metabolism; and perhaps genotypic differences among scallop populations. Problems associated with monitoring scallops for PSP toxicity have been further exacerbated by high variability in toxicity among individual specimens from the same location (Whitefleet-Smith et al., 1985; Gillis et al., 1991; Beitler, 1991; Cembella et al., 1993; White et al., 1992b and references therein).

B. ANATOMICAL DISTRIBUTION OF TOXINS

The distribution of PSP toxins among tissues of bivalve molluscs is rather variable and has been found to be species-specific for a number of species, including the softshell clam, *Mya arenaria,* (Martin et al., 1990), sea scallop, *Placopecten*

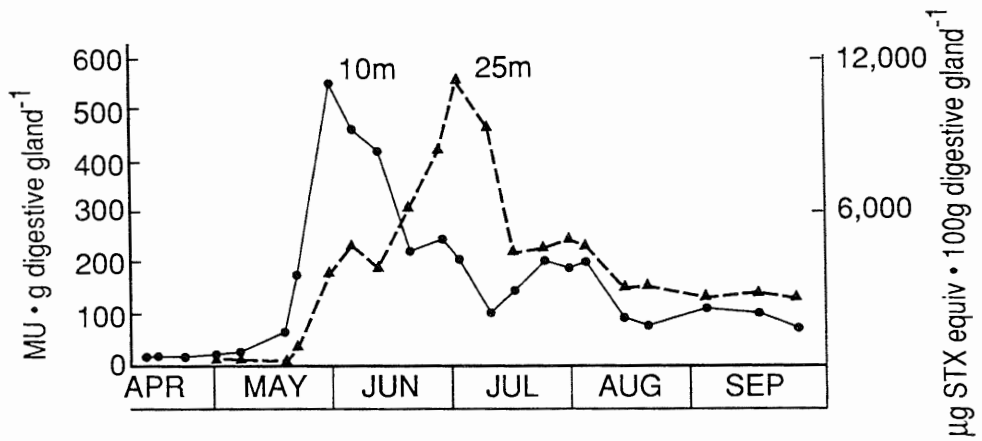

FIGURE 6. Change in the PSP toxicity of scallops (*Patinopecten yessoensis*) suspended at 10 and 25 m depth off Sawara, Japan, between April and September, 1979. (After Nishihama et al., 1980.)

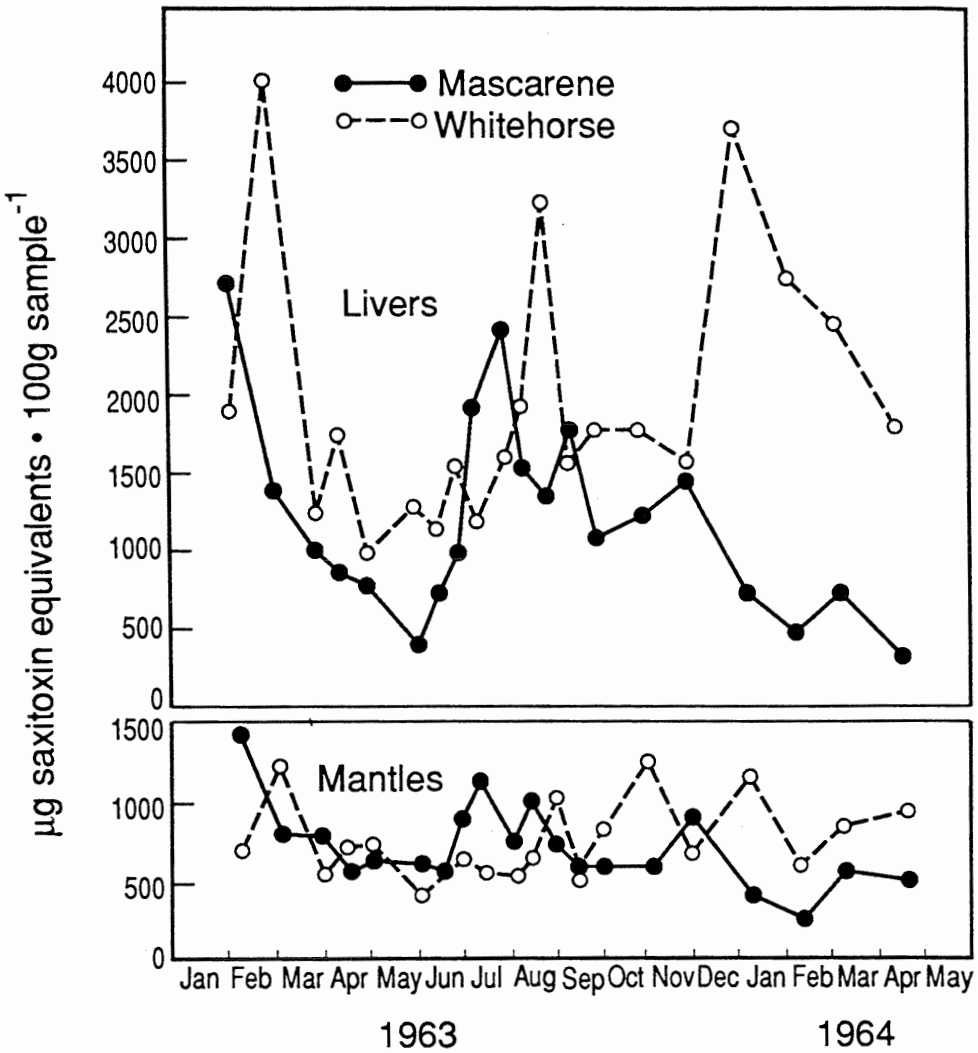

FIGURE 7. PSP toxicity in sea scallop (*Placopecten magellanicus*) livers (hepatopancreases) and mantles from the Whitehorse and Mascarene beds, as determined by mouse bioassay. See Figure 5 for location. (After Bourne, 1965.)

magellanicus, surfclam, *Spisula solidissima* (Shumway, unpublished), as well as the Alaskan butter clam, *Saxidomus giganteus*, and the Pacific oysters, *Crassostrea gigas* and *Pinctada* sp. (Maclean, 1975; Beitler and Liston, 1990). Clear seasonal changes in the anatomical distribution of PSP toxins have not been demonstrated from mouse bioassay data (Figure 7).

Most available data on PSP toxin distribution among scallop tissues are for the sea scallop and Japanese scallop (Table 2). The digestive gland is usually the most toxic tissue; levels in excess of 45,000 µg STXeq/100 g tissue have been recorded from *P. magellanicus* (Watson-Wright et al., 1989) and over 130,000 µg STXeq/100 g was found in *Pt. yessoensis* from Japan (Noguchi et al., 1984). Bourne (1965) carried

TABLE 2
Levels of Paralytic Shellfish Toxins (μg STXeq/100 g Tissue) Recorded in Scallop Species from Various Geographical Locations

Species	Tissue	Toxin Level (μg STXeq/100 g)	Location	Ref.
Chlamys nipponensis akazura	Adductor	80	Ofunato Bay, Japan	Noguchi et al. (1978)
	Gonad (ovary)	640		
	Mid-gut gland	4,000		
Chlamys opercularis	Whole	256	Flamborough Head, U.K.	Ingham et al. (1968)
Chlamys rubida	Adductor	56	Washington	Anonymous (1987)
*Crassadoma gigantea** (=*Hinnites multirogosus*)	Adductor muscle	130	British Columbia, Canada	DFO (1989)
	Viscera+	2,500		
	Whole body	1,200		
	Adductor muscle	229	Washington	DFO (1989)
	Viscera+	2,036		
	Whole body	295		
	Adductor muscle	2,000	California	Anonymous (1980); Sharpe (1981)
	Viscera+	26,000		
	Whole body	13,593		
Patinopecten caurinus	Adductor	58	Alaska	Anonymous (1987)
Patinopecten yessoensis	Adductor	400	Ofunato Bay, Japan	Noguchi et al. (1978)
	Gonad (ovary)	900		
	Mid-gut gland	16,000		
	Adductor	40	Funka Bay, Japan	Noguchi et al. (1980a,b)
	Digestive gland	2,040		
	Other	220		

TABLE 2 (continued)

Levels of Paralytic Shellfish Toxins (μg STXeq/100 g Tissue) Recorded in Scallop Species from Various Geographical Locations

Species	Tissue	Toxin Level (μg STXeq/100 g)	Location	Ref.
Patinopecten yessoenis	Digestive gland	20,000	Ofunato Bay, Japan	Sekiguchi et al. (1989)
	Digestive gland	8,400	Ofunato Bay, Japan	Kodama et al. (1990)
	Digestive gland	6,000	Kawauchi Bay, Japan	Ogata et al. (1982)
	Digestive gland	15,000	Funka Bay, Japan	Nishihama (1980)
	Digestive gland Adductor Muscle	130,000–220,000 60–260	Japan	Noguchi et al. (1984)
	Hepatopancreas	34,000	Ofunato Bay, Japan	Oshima et al. (1982a)
	Digestive gland Rectum Foot Gonad Mantle Gill Adductor muscle	42,000-70,000 4,200–12,400 3,200–4,600 2,200–3,200 1,500–2,200 1,420–2,200 320–860[a]	Ofunato Bay, Japan	Maruyama et al. (1983)
	Digestive gland	15,000	Funka Bay, Japan	Nishihama (1980)
Pecten maximus	Whole	1,568	Farne Bank, U.K.	Ingham et al. (1968)
Pecten maximus	Whole(?)	2,700	Laboratory	Lassus et al. (1989)

TABLE 2 (continued)

Levels of Paralytic Shellfish Toxins (μg STXeq/100 g Tissue) Recorded in Scallop Species from Various Geographical Locations

Species	Tissue	Toxin Level (μg STXeq/100 g)	Location	Ref.
Pecten grandis (=*Placopecten magellanicus*)	Whole	1,520	Off Lepreau Basin, New Brunswick, Canada	Medcof et al. (1947)
	Digestive gland	8,000		
	Gill	560		
	Adductor muscle	<40		
	Gonad	190		
	Other	680		
Placopecten magellanicus	Hepatopancreas	1,440	Canadian Georges Bank	Gillis et al. (1991)
	Gonad	44		
	Adductor muscle	<40	Bay of Fundy, Canada	Hsu et al. (1979)
	Gonad	2,400		
	Hepatopancreas	50,000		
	Gill	570		
	Rims	4,500		
Placopecten magellanicus	Adductor	<40*	Maine	Shumway et al. (1988; unpublished)
	Gonad	420*		
	Digestive gland	4,180*		
	Mantle	2,830*		
Placopecten magellanicus	Whole+	3,888	Georges Bank (Loran 13365–43777)	White et al. (1992a,b) Shumway (unpublished)
	Adductor	183*		
	Whole (minus adductor)	14,775*		
Placopecten magellanicus	Hepatopancreas	45,000*	Bay of Fundy, Canada	Watson-Wright et al. (1989)
	Gonad	1,700*		
	Adductor muscle	Undetectable		
	Gills	≈250*		
	Rims	≈4,700*		

TABLE 2 (continued)

Levels of Paralytic Shellfish Toxins (μg STXeq/100 g Tissue) Recorded in Scallop Species from Various Geographical Locations

Species	Tissue	Toxin Level (μg STXeq/100 g)	Location	Ref.
Placopecten magellanicus	Whole	2,200*	Bay of Fundy, Digby, Canada	Jamieson and Chandler (1983)
	Digestive gland	150,000		
	Gonad	184–286*		
	Adductor	60		
	Gill	100–600*		
	Digestive gland	140*	Northern Edge, Georges Bank	Jamieson and Chandler (1983)
	All other tissues	<32		
	Adductor	120*	Mascarene, Nova Scotia Canada	Jamieson and Chandler (1983)
	Digestive gland	25,000		
Placopecten magellanicus	Liver[b]	36-66	Northern Edge, Georges Bank	Bourne (1965)
	Gonad	43*	Bay of Fundy, Canada	Bourne (1965)
	Liver	4,000*		
	Mantle	1,440*		
	Adductor muscle	<32*		
	Gill	<32*		

*Note: A conversion factor of 1 MU ≅0.20 μg STXeq has been used to standardize data sets. One mouse unit (MU) is defined as the amount of saxitoxin required to kill a standard (~20 g) mouse within 15 min. * = maximum reported values; + = whole body minus adductor.*

[a] *Probably leached from other tissues; scallops were frozen whole for several months prior to dissection and analysis.*

[b] *Stomach and digestive diverticulum.*

out a comprehensive study of PSP toxicity in *P. magellanicus* in the Bay of Fundy by monitoring toxicity of various tissues over a 15-month period using the AOAC mouse bioassay (Figure 7). He also reported low PSP toxin levels (36 to 62 μg STXeq/100 g tissue) in scallop livers (stomach and digestive diverticulum) from the Northern Edge region of Georges Bank (Figure 5) during 1961 and 1962. In almost all cases, scallop livers were more toxic than mantles, although both tissues retained detectable levels of PSP toxins throughout the study period. Although these low levels would not pose a threat to public health, they clearly indicate that PSP toxicity in this region is not a new phenomenon.

Toxicity of scallop gills is generally much lower than digestive gland and mantle tissues. Of 121 analyses of *P. magellanicus* gills from the Gulf of Maine, only 3 samples contained measurable PSP toxins (maximum recorded value—73 μg STXeq/100 g; Shumway, unpublished); in Bourne's (1965) previous study from the Fundy region, gills were always toxin free. Recently, Watson-Wright et al. (1989) reported PSP toxicity in only 36% of *P. magellanicus* gill samples from the Bay of Fundy. Other authors have reported varying PSP toxicity levels in gill tissue, as determined by bioassay (see Table 2).

Levels of PSP toxicity in scallop roe are usually below the regulatory limit for human consumption accepted in North America and many other countries (80 μg STXeq/100 g). In the Bay of Fundy study (Bourne, 1965), maximum toxicity levels in gonads (38 μg STXeq/100 g) were typically at or below the mouse bioassay detection limit (ca. 30 to 42 μg STXeq/100 g, depending upon the strain of mouse used). However, Watson-Wright et al. (1989) later reported detectable PSP in 69% of scallop gonadal samples (n = 41) from the Bay of Fundy, as determined by mouse bioassay. Bourne (1965) suggested that PSP toxicity registered in the roe was most likely contributed by that part of the intestine that loops through the gonad. As a consequence, particularly where extremely high PSP toxicity is found in scallop livers, PSP toxin levels in excess of the regulatory limit have been found in gonadal tissue. In exceptional cases, high toxin levels (e.g., 1300 μg STX/100 g in *P. magellanicus* gonads from Mascarene, New Brunswick) have been recorded (Microbiology Division, Department of Health and Welfare, Canada, Black's Harbour, New Brunswick, Canada). Neither a seasonal study of scallop toxicity in the Bay of Fundy, based on mouse bioassay data (Watson-Wright et al., 1989), nor a comprehensive analysis of PSP toxin composition of inshore and offshore scallop populations from the Gulf of Maine using high-performance liquid chromatography (HPLC) (Cembella et al., 1992) revealed significant correlations between the toxin load in gonads and any other tissue.

Although it has long been conventional wisdom that scallop adductor muscles tend to remain free of PSP toxins (Medcof et al., 1947; Bourne, 1965; Watson-Wright et al., 1989; Shumway, 1990), there is recent evidence for several scallop species indicating that adductor muscles may occasionally contain not only measurable toxicity by mouse bioassay, but also levels exceeding the regulatory limit (Table 2). In general, when surrounding viscera show no detectable toxicity, neither do adductor muscles. Adductor muscle scores are always lower than those of corresponding toxic viscera, even in the presence of extremely high toxicity in other tissues. For example, in *Pt. yessoensis*, when digestive gland toxicities of 130,000 to 220,000 μgSTXeq/100 g (= 6500 to 11,000 MU/g; MU = mouse unit) were

determined, adductor muscle toxicity was only 60 to 260 µg STXeq/100 g (= 3 to 13 MU/g) (Noguchi et al., 1984). Similarly, weathervane scallops, *Pecten caurinus*, from Alaska revealed PSP toxin levels in the adductor muscle below the regulatory limit, while other tissues contained up to 12,000 µgSTXeq/100 g (Alaska Department of Environmental Conservation, 1981–1987). Furthermore, Gillis et al. (1991) reported no detectable PSP toxicity in *P. magellanicus* adductor muscles, even when toxin levels in associated digestive glands reached 1440 µgSTXeq/100 g. In contrast, Shumway (unpublished, see Table 2) found 183 µgSTXeq/100 g in *P. magellanicus* adductor muscles, whereas surrounding visceral toxicity was 14,775 µgSTXeq/100 g. Obviously, it is impossible to estimate reliably the PSP toxicity of scallop adductor muscles based on extrapolation from the toxicity of surrounding viscera (see Beitler, 1991, for discussion; Watson-Wright et al., 1989; Cembella et al., 1993). No assumptions regarding the toxicity of individual scallop tissues should be made based on any such correlations.

Preliminary studies on the uptake, sequestering, and biotransformation of PSP toxins by *P. magellanicus* from the Gulf of Maine in 1988–89 as determined by the fluorescence HPLC method (Sullivan and Wekell, 1986; Cembella et al., 1987; Cembella and Shumway, 1991) supported previous findings (Fix-Wichmann et al., 1981; Shimizu and Yoshioka, 1981) that the PSP toxin profile in scallops may differ considerably from the toxic dinoflagellate (*Alexandrium tamarense*). Further analysis revealed substantial variation in the relative amounts of high toxicity carbamate derivatives (GTX1–4, NEO, and STX) vs. lower potency N-sulfocarbamoyl toxins (C1–4, B1, B2) among various tissues, as well as between inshore and offshore populations (Cembella et al., 1993, Figure 8a–d). There was, however, no apparent systematic seasonal trend in the proportions of these major toxin components. The PSP toxin profile in gonads (when toxin was present) was consistently dominated by the high toxicity carbamate analogs GTX2/GTX3, along with traces of NEO and N-sulfocarbamoyl toxins C1/C2 (Figure 9).

Oshima (1991) has advanced a classification for filter-feeding bivalves according to their profiles of accumulated PSP toxins. In this scheme, mussels and several clam species are shown to reflect the approximate toxin composition of the dinoflagellate responsible for their toxicity in all tissues, except for epimerization of the 11-hydroxysulfate epimers (e.g., GTX1/GTX4, GTX3/GTX2) and some reduction in the relative concentration of the N-sulfocarbamoyl toxins, particularly C1/C2 (Figure 2). In contrast, both the anatomical distribution and toxin profile in the various tissues of *Pt. yessoensis* may change during long-term detoxification. Over 6 months, a decrease in GTX1,4 and an increase in GTX2,3 occurred in the mantles, whereas in the kidney a decrease in GTX1,4 was accompanied by an increase in NEO and STX. Putative biotransformation in the mantle tissue was consistent with reduction of the N-1 hydroxyl moiety, while loss of the hydroxysulfate group at C-11 would explain the toxin profile in the kidney. Biotransformation of toxins within scallop tissues from less toxic sulfocarbamoyl derivatives to carbamate analogs also may account for some increase in toxicity observed over time in *P. magellanicus* (Hsu et al., 1979; Shimizu and Yoshioka, 1981). Nagashima et al. (1988) also suggested complex catabolism of PSP toxins in *Chlamys nobilis*. Lassus et al. (1989) reported that in *Pecten maximus* certain derivatives, specifically GTX3 and C1/C2 (epiGTX8/GTX8), dominated in the early stages of detoxification,

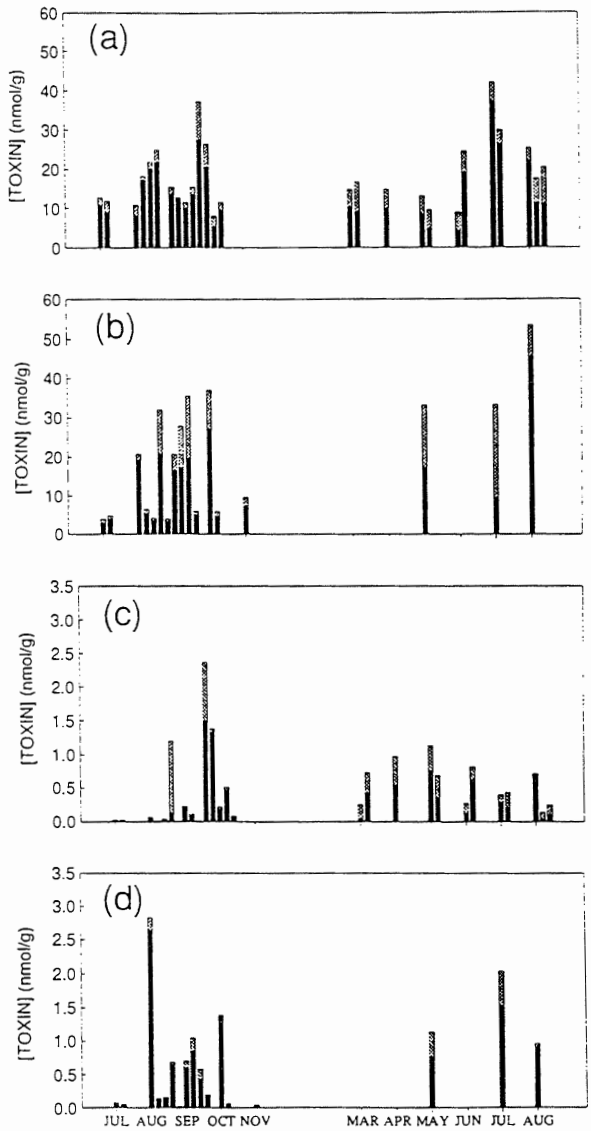

FIGURE 8. Relative PSP toxin composition, determined by HPLC-FD and expressed as total carbamate and N-sulfocarbamoyl toxins, in digestive glands [(a) inshore; (b) offshore] and gonads [(c) inshore; (d) offshore] of *Placopecten magellanicus* from the Gulf of Maine in 1988–89.

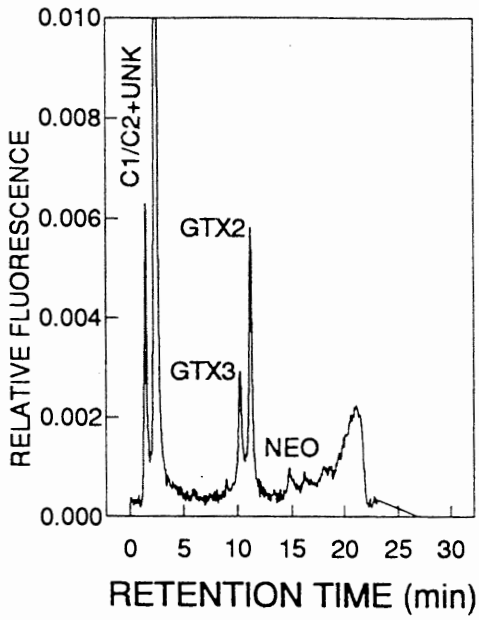

FIGURE 9. HPLC-FD chromatogram of the PSP toxin composition in *Placopecten magellanicus* gonads collected in August, 1988 from an inshore station (20 m depth) in the Gulf of Maine. UNK = unknown (nontoxic) artifacts that coelute with the N-sulfocarbamoyl toxins C1/C2.

whereas GTX2 was predominant during the subsequent slow decontamination phase. In addition to metabolic conversions, PSP toxins in scallops may be mobilized and transferred among tissue compartments, before ultimately being lost. Inoguchi et al. (1990) suggested that PSP toxins accumulated in the digestive gland of *Pt. yessoensis* may move to the mantle, after which they can be secreted or leaked into the surrounding seawater.

V. DETOXIFICATION KINETICS

Detailed knowledge of PSP toxin elimination kinetics is limited to three species of scallops: *Pt. yessoensis, P. magellanicus,* and *Chlamys nipponensis akazara.* The available data suggest that among filter-feeding bivalve molluscs, scallops can be classified among species that retain toxin for a long time. Toxin retention in *P. magellanicus* for periods ranging from several months to 2 years has been reported (Medcof et al., 1947; Jamieson and Chandler, 1983; Shumway et al., 1988; Shumway, unpublished). Certain tissues of *P. magellanicus*, particularly the digestive gland and mantles, can remain toxic throughout the year (Bourne, 1965; Shumway et al., 1988; unpublished). Similar results also are known for the pink scallop *C. hastata* (Nishitani and Chew, 1988).

There are several hypotheses to explain the chronically high PSP toxin levels in scallops, even throughout the winter when *Alexandrium* blooms are apparently

absent from the upper water column: (1) low basal metabolic rates and reduced filtration activity, especially in colder waters with reduced food supply, could result in low rates of toxin catabolism and elimination; (2) toxin conversion to more toxic derivatives could produce an increase in net toxicity, even as total toxin body burden is decreasing; (3) cryptic subsurface blooms of toxic vegetative cells could cause recontamination; (4) ingestion of fecal pellets and temporary cyst stages during the senescent phase of toxic blooms may contribute to toxicity; and (5) toxic benthic resting cysts that accumulate and over-winter in the sediments could be ingested by scallops during feeding activity. Bourne (1965) rejected the metabolic hypothesis of prolonged toxin retention, based on observations of two temporally distinct PSP toxin maxima in scallop tissues. He pioneered the suggestion that the scallops must have been actively feeding on toxic organisms, perhaps benthic *A. tamarense* (= *Gonyaulax tamarensis)* cysts. The benthic cyst contamination hypothesis as an explanation for apparent slow detoxification rates was popular for many years (Jamieson and Chandler, 1983; Tufts, 1979; Yentsch and Incze, 1982), and gained credence with evidence that naturally occurring benthic cysts could be an order of magnitude more toxic than vegetative cells of cultured isolates from the same region, according to mouse bioassays. Early work compared cultured dinoflagellate cells and cyst-containing sediment extracts (Dale et al., 1978). More recent work has shown, however, that cultured *Alexandrium* cells are usually less toxic that their counterparts from natural environments (Cembella et al., 1988). Detailed analysis of the toxin composition and toxicity of various life cycle stages of *Alexandrium* by mouse assay (Oshima et al., 1982b) and by HPLC (Cembella et al., 1990; Oshima et al., 1992) has indicated that the difference in toxicity between resting cysts and vegetative cells is less dramatic than originally reported. Furthermore, the number of toxic benthic cysts is probably not substantial enough to account for recorded levels of toxicity. Anderson (1984) calculated that it would require consumption of as many as 100 million cysts to achieve the toxin levels recorded in deep-water sea scallops from the Gulf of Maine—equivalent to the ingestion of all cysts in the top centimeter of a square meter of sediment by each individual scallop.

The PSP toxin levels in *Pt. yessoensis* digestive glands (initial toxicity 34,000 µg STXeq/100 g (=1700 MU/g tissue) decreased to 4000 µg STXeq/100 g (=200 MU/g) during the first 3 days of depuration, then rose inexplicably to 10,400 µg STXeq/100 g (= 520 MU/g) and remained >2000 µg STXeq/100 g (=100 MU/g) even after being held in outdoor tanks for 5 months) (Oshima et al., 1982a). A similar biphasic detoxification of scallops has been reported for *Pecten maximus* collected from Farne Bank, U.K., where PSP toxicity decreased from 1568 to 416 µg STXeq/100 g (7840 to 2080 MU/100 g) during the first 2 weeks, and then slowly declined to 76 µg STXeq/100 g (382 MU/100 g) after 9 weeks (Ingham et al., 1968). Lassus et al. (1989) also found evidence of biphasic detoxification kinetics in *Pecten maximus*: rapid toxin loss of the same order of magnitude as the accumulation rate was followed by a slow detoxification phase, with levels remaining above 80 µg STXeq/100 g, even after 45 days.

Cooking can reduce PSP toxin levels considerably (Medcof et al., 1947; McFarren et al., 1960) and canning is sometimes used to reduce scallop toxicity to acceptable levels (Noguchi et al., 1980a,b). As a means of reducing toxicity, canning is usually effective only at relatively low toxicities, although Noguchi demonstrated that

canning might be feasible for scallops with as much as 8000 µg STXeq/ 100 g (= 400 MU/g) tissue.

Freezing does not substantially reduce PSP toxin levels in the short term, although extended storage of whole tissues inevitably leads to some loss of toxicity after several months at –20°C. Moreover, freezing whole scallops can result in migration of toxins among tissues, e.g., from digestive glands into adductor muscles, rendering them unsafe for human consumption (Noguchi et al., 1984; Shumway, unpublished). Toxin also can be leached from attached gonads into the accompanying adductor muscle during shipping (Bruce and Delaney, 1972).

Further work on the PSP detoxification kinetics of a variety of commercially important scallop species is obviously required. The effects of age, reproductive status, genetic factors, and environmental variables, including ambient water temperature and salinity, on toxin accumulation and loss rates remain to be determined. Specific physiological mechanisms and enzymatic pathways involved in the catabolism of PSP toxins also are largely unknown. Completion of such studies should provide mitigating strategies of great benefit to scallop aquaculture and the safe exploitation of natural stocks.

VI. CONCLUSIONS

- Successful scallop culture and commercial fisheries can thrive in areas prone to toxic algal blooms if only the adductor muscle is utilized. When tissues other than the adductor muscle (e.g., gonad, mantle) are to be harvested, only through careful site selection and comprehensive monitoring can optimal utilization of scallop resources be realized and economic losses kept to a minimum .
- Establishment of public health guidelines with particular emphasis on toxin levels in individual tissues is necessary if scallops are to be marketed whole or in conjunction with tissues other than adductor muscles.
- Scallop mariculturists should be acutely aware of the potential risks associated with phycotoxins and the consequences of marketing various scallop products exposed to toxic algal blooms.
- Safe marketing of roe-on scallops is feasible only under a strict regulatory regime for monitoring phycotoxins.
- Marketing of whole scallops poses a high risk to public health and the economic success of such an industry is questionable, given the long toxin retention times and high toxicity levels often found in scallop populations.

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