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Relation of toxic dinoflagellate blooms to finfish in the southern Bay of Fundy and northwestern Gulf of St. Lawrence: a summary of recent studies and a rationale for investigations on fish larvae

by

Alan W. White
Fisheries and Environmental Sciences
Fisheries and Oceans Canada
Biological Station
St. Andrews, New Brunswick EOG 2XO

Introduction

Fish-killing toxic dinoflagellate blooms occur frequently in certain parts of the world. In the Gulf of Mexico blooms of Gymnodinium breve and Gonyaulax monilata cause extensive fish kills nearly annually, with severe economic impact on the Gulf fisheries. Blooms of Gymnodinium breve and Gyrodinium aureolum have been associated with fish kills in various locations around the United Kingdom.

In Canada toxic dinoflagellate blooms occur regularly along the west

and east coasts. East coast blooms, occurring during summer and fall, are caused by Gonyaulax excavata (tamarensis) and appear in the southern Bay of Fundy and in the area of the St. Lawrence River estuary.

Until recently toxic blooms in Canada were not recorded to be associated with fish kills. It was, in fact, generally assumed that the extent of the consequences of these blooms to Canadian fisheries resources was limited to the problem of paralytic shellfish poisoning (PSP), caused by accumulation of paralytic, dinoflagellate toxins by benthic filter-feeding molluscs. However, a herring kill in 1976 in the Bay of Fundy was linked to dinoflagellate toxins (White 1977). In this case the mortality probably resulted through a series of planktonic food chain events. This raises the question of the extent of the consequences to fish of Gonyaulax toxins in the plankton community and the possibility that other such planktonic food chain events may occur and go unnoticed. Nearly ten years ago Adams et al. (1968) considered the possible role of planktonic herbivores in transmitting dinoflagellate toxins when G. excavata was implicated in a kill of sand launce (Ammodytes sp.) off the east coast of the United Kingdom. Sand launce feed on copepods predominantly.

This report summarizes work on the herring kill and recent field work on the acquisition of Gonyaulax toxins by zooplankton, and discusses work in progress to determine if Gonyaulax toxins, transmitted by zooplankton, affect fish at the most sensitive (and least noticeable) stage, i.e. the larval stage. If so, then Gonyaulax blooms may have consequences for year-class strengths of certain fishes.

Herring Kill

On July 15, 1976 a sizeable kill of mature Atlantic herring (20-26 cm)

occurred off the east coast of Grand Manan Island in the Bay of Fundy during a bloom of G. excavata (White 1977). Examination of dead fish revealed no external or internal abnormalities. Stomachs contained pteropods (Limacina retroversa), algal remains, and paralytic toxins - about 21 µg of toxins per stomach. The evidence, although somewhat circumstantial, indicates the strong possibility that the kill was caused by Gonyaulax toxins and that the toxins had been transmitted to the herring via pteropods. A summary of the evidence is as follows:

1) Herring stomachs contained only pteropods, degraded algal material (verified by pigment analysis), and paralytic toxins.
2) The pteropods, in various stages of digestion, also contained degraded algal material.
3) Pteropods are mucilaginous filter-feeders and feed on phytoplankton.
4) The kill occurred during the peak of the Gonyaulax bloom when this organism strikingly dominated the phytoplankton.
5) Mature herring are not known to ingest phytoplankton directly.
6) Subsequent laboratory experiments confirmed the lethality of 21 µg of orally administered Gonyaulax toxin to herring (Table 1).

Although experimental fish were generally smaller (Table 1) than fish from the kill, a 21-µg dose is probably sufficient to have caused the kill of larger herring for the following reasons (White 1977). Experimental fish regurgitated substantial amounts of the toxin within the first few minutes of administration so that the actual effective dose was considerably less than 21 µg. Also, experiments were conducted during the winter when the water temperature was 2.5-3.0°C and one would expect increased toxin sensitivity at 11-12°C (temperature at time of the kill) when fish are metabolically more active.

This herring kill has prompted many questions about the fate and consequences of dinoflagellate toxins in marine systems. Perhaps similar events affecting marine resources occur through a variety of planktonic food chain routes.

This past summer (1978) there was another fish kill due to Gonyaulax toxins. A kill of sand launce occurred off Monomoy (Cape Cod), Mass. (I.T. Nisbet, pers. comm.), again raising the likelihood of involvement of zooplankton intermediates. In this instance mortality of bluefish (Pomatomus saltatrix) also occurred, but the cause of the bluefish deaths is unknown.

Acquisition of Gonyaulax Toxins by Zooplankton

A study of the extent to which Gonyaulax toxins occur in zooplankton was initiated following the herring kill. During the summers of 1977 and 1978 plankton tows were made weekly with 20-, 64-, 243-, and 571-µm mesh nets to determine the degree of toxin acquisition in various sized plankton fractions during the Gonyaulax bloom (White 1979). Tows were made with 1-m nets in the Bay of Fundy off Head Harbour, New Brunswick. Toxin measurements were made on tow contents using the mouse bioassay.

Paralytic toxins were measured in all fractions. The timing of the rise and fall in toxicity was generally similar for each fraction although the maximum toxicity decreased as the fraction size increased (Fig. 1). The major components of the tows are listed in Table 2. The 20-µm fraction was dominated by Gonyaulax (which is 30-40 µm across), the 64-µm fraction by the tintinnid (Favella sp.) which was feeding on Gonyaulax, the 243-µm fraction by cladocerans, and the 571-µm fraction by copepods. There was some

contamination of the 64-μm samples with free Gonyaulax cells, but very few or no free cells were present in the 243- and 571-μm samples. Therefore, the toxin measured in these larger fractions probably represented toxin acquired by the zooplankters. This is also supported by the retention of toxin in these larger fractions beyond the time of the Gonyaulax peak (Fig. 1). The secondary peak in toxin content of these larger fractions in mid-October (Fig. 1) may be apparent only, perhaps caused by sampling of different zooplankton populations on different dates. In any event, the data show toxin in the 243- and 571-μm samples even when no Gonyaulax remained in the water.

Maximum toxin concentration in 1977 for the 20-, 64-, 243-, and 571-μm fractions were 690, 91, 59, and 8 μg toxin per gram wet plankton, respectively. The levels in the larger fractions are significant in that they were comparable to the maximum toxin concentration for local shellfish during this period, 6-54 μg toxin per gram meat.

This study shows not only that Gonyaulax toxins can be taken up by zooplankton but also that they may be taken up to ecologically significant levels. Considering that the lethal oral dose of Gonyaulax toxin to herring is 20 μg or less, then in 1977 only about 0.3 g (wet weight) of the 243-μm zooplankton or about 2 g of the 571-μm zooplankton contained a potentially lethal dose for a herring.

Effects of Gonyaulax Toxin on Larval Fish?

The preceeding has demonstrated that Gonyaulax toxins can accumulate in zooplankton and that under certain conditions mortality of finfish may result. An obvious question now arises concerning the potential effects of toxin-containing zooplankters on larval stages of fish. One could

hypothesize the effects to be quite dramatic given the small size of larvae, coupled with their necessity of feeding upon zooplankton. Hypothetical mass mortalities of larvae during blooms would go unnoticed, but could affect year-class strengths of certain fishes which have larval stages overlapping temporally and spatially with Gonyaulax blooms. In other words, obvious mortalities of adult fish may represent only the "tip of the iceberg." Gonyaulax-related events of much more consequence to fish populations may perhaps occur during the larval stages.

At this point such effects on fish larvae are entirely speculative. However, I believe the previously mentioned work lends sufficient plausibility to the idea to make it worth investigating.

There has been one recent study focusing on these concerns. Mills and Klein-Macphee (1979) examined the direct effects of G. excavata on winter flounder larvae (Pseudopleuronectes americanus). This fish spawns in February, March, and April in coastal New England waters, with eggs requiring about 2 wk to hatch. Larvae remain planktonic for 5 or 6 wk so that they might be expected to encounter G. excavata blooms which appear there in April and May. Although these authors report a slight increase in larval mortality in the presence of Gonyaulax, they have missed the point that zooplankton intermediates which acquire the toxin may well be the key to understanding the real effects of Gonyaulax on larval fish.

Presently we are trying to answer some of these questions. Experiments in the laboratory show us that we can intoxicate live copepods from plankton tows by exposure to cultures of G. excavata. Uptake and persistence of the toxins in copepods is being examined quantitatively. The toxin-containing copepods will be used in feeding experiments with fish larvae. We are also exploring the methodology of rearing larval fish in the laboratory in order

to have a supply of healthy larvae for such tests.

So far there are many more questions than answers concerning the fate of Gonyaulax toxins in planktonic marine food chains in general and the consequences to finfish resources in particular.

References

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Table 1. Effect of Gonyaulax excavata toxins on herring.[a]

Herring weight (g)	Total length (cm)	Time after treatment until paralysis[b] (min)	Time after treatment until death (min)
10.6	12.8	12	110[c]
11.1	13.1	10	75[c]
11.1	12.8	11	60
11.4	13.1	8	80
11.6	12.7	11	43
11.6	13.4	11	60
11.7	13.0	9	42
12.0	13.1	9	58
12.2	13.1	15	110[c]
12.3	13.4	10	54
12.4	13.2	13	98[c]
12.3	14.2	20	-[d]
12.7	13.1	19	140
12.9	13.7	15	-[d]
13.5	13.6	7	37[c]
13.5	13.6	9	75[c]
13.8	14.1	12	-[d]
14.5	14.1	16	-[d]
14.6	14.1	16	150[c]
14.6	13.7	20	-[d]
15.0	13.7	7	51
15.6	14.0	15	100[c]
15.6	14.8	11	105[c]
10.6	14.5	10	51
17.3	14.7	14	420-1500[c]
17.9	14.8	14	49
18.2	15.2	loss of equilibrium only	-[d]
20.4	15.0	18	120[c]

[a]All controls survived for 48 h and showed no abnormal behavior.
[b]Time at which fish was immobilized and lay on its side at bottom of tank.
[c]Fish died with mouth gaping widely.
[d]Fish normal 48 h after treatment.

From White, A. W. 1977. J. Fish. Res. Board Can. 34: 2421.

Table 2. Composition of plankton samples. Major components of plankton fractions at times of maximum toxicity.

20 µm	64 µm
Gonyaulax excavata[a]	*Favella* sp.[a]
Favella sp.[c]	*Peridinium* sp.[c]
Coscinodiscus asteromphalus[c]	*Acartia clausi*[c]
Rhizosolenia delicatula[c]	*nauplii*[c]
Peridinium sp.[d]	*Gonyaulax excavata*[d]
Nitzschia seriata[d]	*Ceratium longipes*[d]
Ceratium longipes[d]	*Coscinodiscus asteromphalus*[d]
243 µm	**571 µm**
Podon polyphemoides[a]	*Centropages typicus*[a]
Evadne nordmanni[a]	*Calanus finmarchicus*[a]
Acartia clausi[b]	*Centropages hamatus*[b]
Eurytemora americana[b]	*Acartia clausi*[c]
Temora longicornis[c]	*Temora longicornis*[c]
Temora stylifera[c]	*Temora stylifera*[c]
Pseudocalanus minutus[c]	*Evadne nordmanni*[c]

[a]very abundant, [b]abundant, [c]common, [d]some.

From White, A. W. 1979. Taylor and Seliger, eds. Elsevier North Holland.

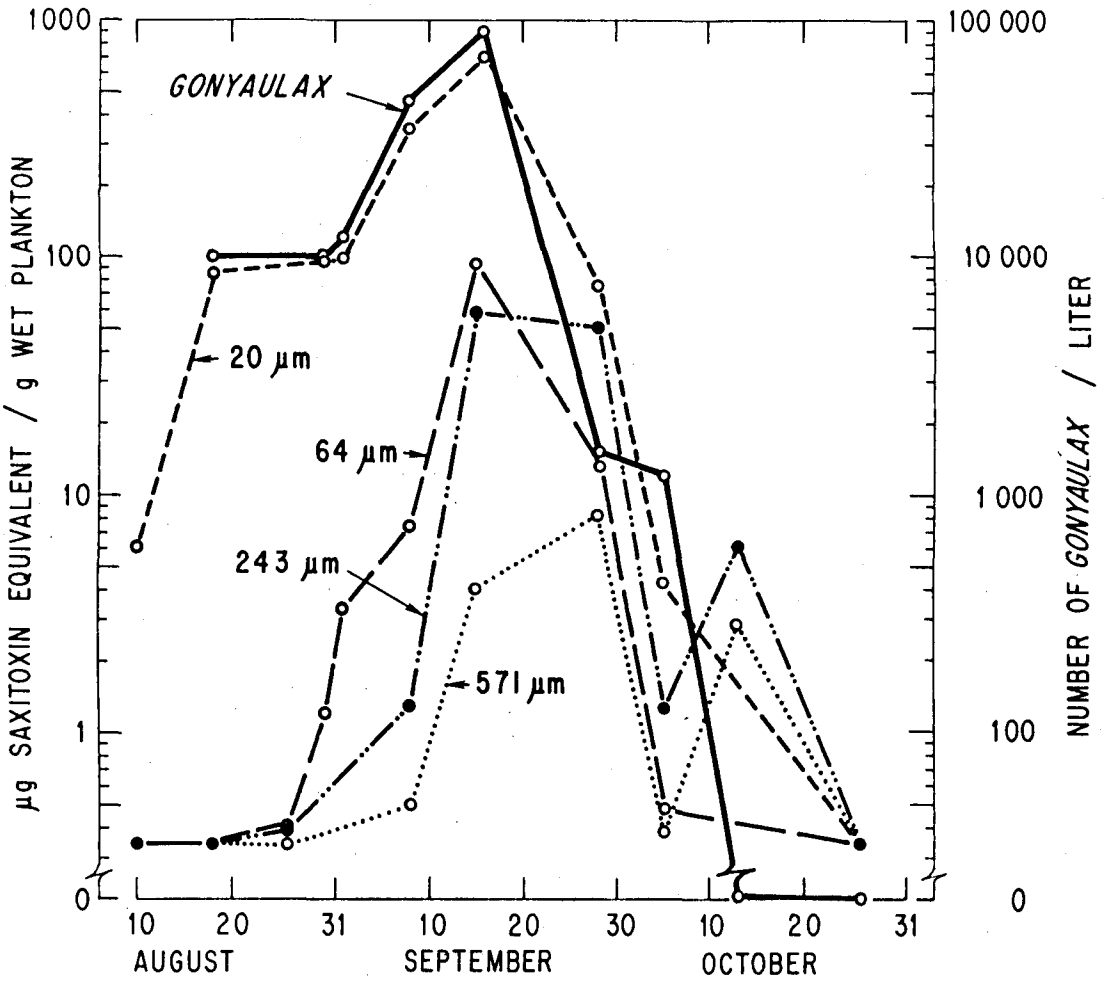

Fig. 1. Number of G. excavata in surface water off Head Harbour, N.B. in 1977 and content of paralytic toxins in surface plankton fractions. Sensitivity of bioassay is 0.34 µg toxins/g plankton.

From White, A. W. 1979. Taylor and Seliger, eds. Elsevier North Holland.