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FATE AND CONSEQUENCES OF DINOFLAGELLATE TOXINS IN MARINE FOOD CHAINS

by

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ABSTRACT

Recent studies indicate that toxins from the marine dinoflagellate Gonyaulax excavata can be transferred through herbivorous zooplankton and cause kills of Atlantic herring. Accumulation of the toxins seems to be a fairly general capability among herbivorous zooplankters. The extent to which this mechanism of toxin transfer affects other species of fish, larval fish, and pertains to other toxic dinoflagellates remains to be determined.

RÉSUMÉ

Des études récentes indiquent que les toxines provenant du dinoflagellé marin Gonyaulax excavata peuvent être transmises par le zooplancton herbivore et peut causer la mort aux harengs atlantiques. Les zooplanctons herbivores semblent avoir une capacité générale d'accumuler les toxines. Il reste à déterminer la mesure des effets de la transmission des quantités de toxines à d'autres espèces de poissons, aux larves, et aussi concernant d'autres dinoflagellés toxiques.

INTRODUCTION

Gonyaulax excavata (Braarud) Balech, formerly G. tamarensis, is the toxic marine dinoflagellate responsible for annual blooms, red tides, and paralytic shellfish poisoning in eastern Canada and New England, and for occasional blooms in the United Kingdom and neighboring areas (Loeblich and Loeblich 1975). It is well known that benthic, filter-feeding molluscs can accumulate the dinoflagellate toxins, posing the danger of poisoning warm-blooded animals which ingest the contaminated shellfish.

Until recently, it was thought that the consequences of G. excavata blooms were limited to shellfish resources. In 1968, however, Adams et al. (1968) suggested that a kill of sand launce (Ammodytes sp.) off the east coast of the United Kingdom may have been caused by G. excavata toxins and speculated that the toxins may have been transmitted to the fish by planktonic herbivores. More substantial evidence implicating G. excavata toxins in fish kills appeared in 1976 when a herring kill in the Bay of

Fundy was traced to the toxins which, in this case, were probably transmitted by pteropods (White 1977). This event prompted further studies of the fate of G. excavata toxins in marine food chains and the consequences to marine organisms, especially finfish. Results of these recent studies (White 1977, 1979, 1980a, b) are briefly summarized and discussed here.

1976 HERRING KILL

On July 15, 1976, a kill of adult Atlantic herring (Clupea harengus harengus) occurred off the east coast of Grand Manan Island in the Bay of Fundy during the annual G. excavata bloom (White 1977). The only identifiable food items in the herring stomachs were pteropods (Limacina retroversa) in various stages of digestion and containing degraded algal material. G. excavata toxins were measured in the herring stomachs - about 21 µg per stomach - using the standard mouse bioassay (AOAC 1975). Subsequent laboratory tests showed that this amount of toxins injected orally kills herring rapidly, with fish displaying the same symptoms observed during the kill, i.e. loss of equilibrium, swimming on sides, and gasping for breath (Table 1).

The evidence, although largely circumstantial, indicates that the kill was caused by G. excavata toxins and suggests that the toxins were 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 when G. excavata strikingly dominated the phytoplankton community.

5) Mature herring are not known to ingest phytoplankton directly, at least not in substantial numbers.

6) Experiments confirmed the lethality of 21 µg of orally administered G. excavata toxins to herring.

ACCUMULATION OF TOXINS BY ZOOPLANKTON

The herring kill raised the question of whether planktonic herbivores can, in fact, acquire G. excavata toxins and whether this is a specific or general capability among this group of animals. Therefore, field studies were conducted during the 1977 and 1978 Gonyaulax blooms in the Bay of Fundy to determine if the toxins could be measured in zooplankton samples (White 1979). Bioassays revealed that G. excavata toxins could be measured in plankton material collected with 20, 64, 243, or 571 µm-mesh nets. The timing of the rise and fall in toxin content was generally similar for each fraction, roughly following the rise and fall of the G. excavata population, with maximum toxicity decreasing as the fraction size increased (Fig. 1).

The 20-µm fraction was dominated by G. excavata, the 64-µm fraction by tintinnids (Favella sp.) which were seen to contain Gonyaulax, the 243-µm fraction by cladocerans (Podon polyphemoides and Evadne nordmanni), and the 571-µm fraction by copepods (Calanus finmarchicus and Centropages typicus). 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 toxins measured in these larger fractions represent toxins acquired by the zooplankters.

This study strongly suggests not only that G. excavata toxins can be acquired by zooplankton, but also that they may be acquired to ecologically significant levels. Considering that the lethal oral dose of the toxins to herring is 21 µg or less, and that maximum toxicities in 1977 in the 243- and 571-µm fractions were 59 and 8 µg toxins per gram wet plankton (Fig. 1), then in 1977 only about 0.3 g (wet weight) of the 243-µm zooplankton material or about 2 g of the 571-µm material contained a potentially lethal dose of toxins for a herring. Finally, the data suggest some degree of retention of the toxins in the zooplankters for a period of several weeks past the peak of the Gonyaulax bloom (Fig. 1).

Upon completion of these field studies, a laboratory study was conducted to investigate the accumulation of G. excavata toxins by zooplankton in more detail and to confirm, or deny, whether toxin uptake actually occurs. During the spring of 1979 freshly collected zooplankton from Passamaquoddy Bay (Bay of Fundy) were allowed to feed on cultures of G. excavata and the removal of dinoflagellates and toxin content of the animals were measured at intervals (White 1980a). In repeated experiments, the copepod Acartia clausii and barnacle nauplii (Balanus sp.) rapidly grazed on G. excavata and rapidly acquired its toxins, reaching maximum levels of toxin content within 6 h of initiation of feeding. Furthermore, the toxins were measurable in the animals for at least several days beyond the time at which their guts became clear of particulate material, indicating toxin retention. Results for toxin uptake and retention by Acartia clausii are shown in Fig. 2. Results of other experiments on A. clausii and on barnacle nauplii were similar except that the toxins were gradually eliminated from the animals, althouth they were retained at 20-50% of maximum levels for up to 7 days. In summary, this study confirms that G. excavata can be acquired by herbivorous zooplankters, and can be retained, or stored, to some degree (White 1980a).

1979 HERRING KILLS

In July 1979 two more herring kills occurred in the Bay of Fundy during the G. excavata bloom (White 1980b). Herring displayed the same symptoms as in the 1976 kill, and again G. excavata toxins were measured in the herring stomachs. Interestingly, in these cases, the cladoceran Evadne nordmanni appeared to be the vector of G. excavata toxins. It overwhelmingly dominated the zooplankton community during this period, and bioassays measured G. excavata toxins in E. nordmanni samples (18 µg/g wet animals). Furthermore, E. nordmanni was nearly the only item, besides degraded algal material and G. excavata toxins, found in stomachs of herring from the kills.

DISCUSSION

All the evidence from these fish kill events, and field and laboratory studies, points to the conclusion that fish kills, at least of herring and sand launce, can be caused by G. excavata toxins being transferred through herbivorous zooplankton and that the acquisition of G. excavata toxins is a fairly general capability among planktonic herbivores.

Several of the most important questions that arise concern: 1) the sensitivity of other fishes to the toxins, 2) the effects of toxin-containing zooplankton on larval fish, 3) the consequences of the toxins on marine resources through undetermined food chain routes, 4) whether toxin transfer through zooplankton occurs during blooms of other toxic dinoflagellates.

Regarding the sensitivity of other fishes, experiments are in progress which show that the sensitivities of winter flounder, cod, pollock, and Atlantic salmon to G. excavata toxins are about the same as for herring (White, unpublished). The oral LD50 value is about 650 µg/kg body weight. The intraperitoneal LD50 value is about 5-10 µg/kg, which is similar to that for warm-blooded animals. Therefore, the potential for kills of other species of fish during G. excavata blooms seems to exist.

From a population point of view, the most serious effect of G. excavata toxins on fish may perhaps occur during their early life history stages. It is conceivable that mass mortalities of fish larvae could result from ingesting zooplankton containing G. excavata toxins. Such kills may 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, mortalities of adult fish may represent only the "tip of the iceberg." Gonyaulax-related events of much more consequence to fish populations may occur during the larval stages. At present, such events involving larval fish are speculative. However, preliminary results do show an increased mortality of Atlantic herring larvae when fed toxin-containing copepods versus toxin-free copepods (White, unpublished).

Some transfer routes through which G. excavata toxins may affect fisheries resources are shown in Fig. 3. One can imagine many additional possibilities. Although progress is being made in understanding the fate and consequences of dinoflagellate toxins in marine systems, many facets of this general problem remain to be explored.

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Table 1. Effect of a 21-μg oral dose of G. 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 1977, with permission).

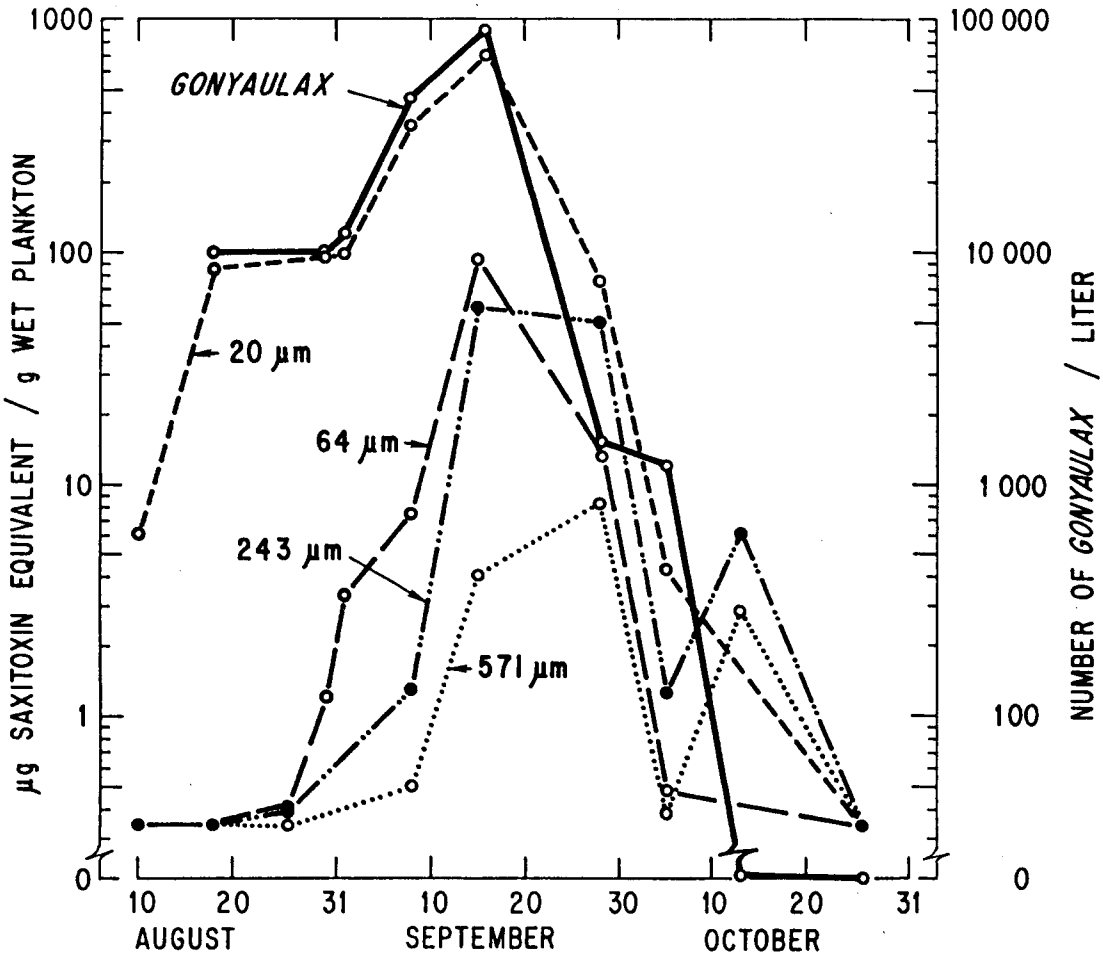

Fig. 1. Number of G. excavata in Bay of Fundy surface water during the 1977 bloom and content of G. excavata toxins in plankton material collected with nets of various mesh size. Results in 1978 were similar (from White 1979, with permission).

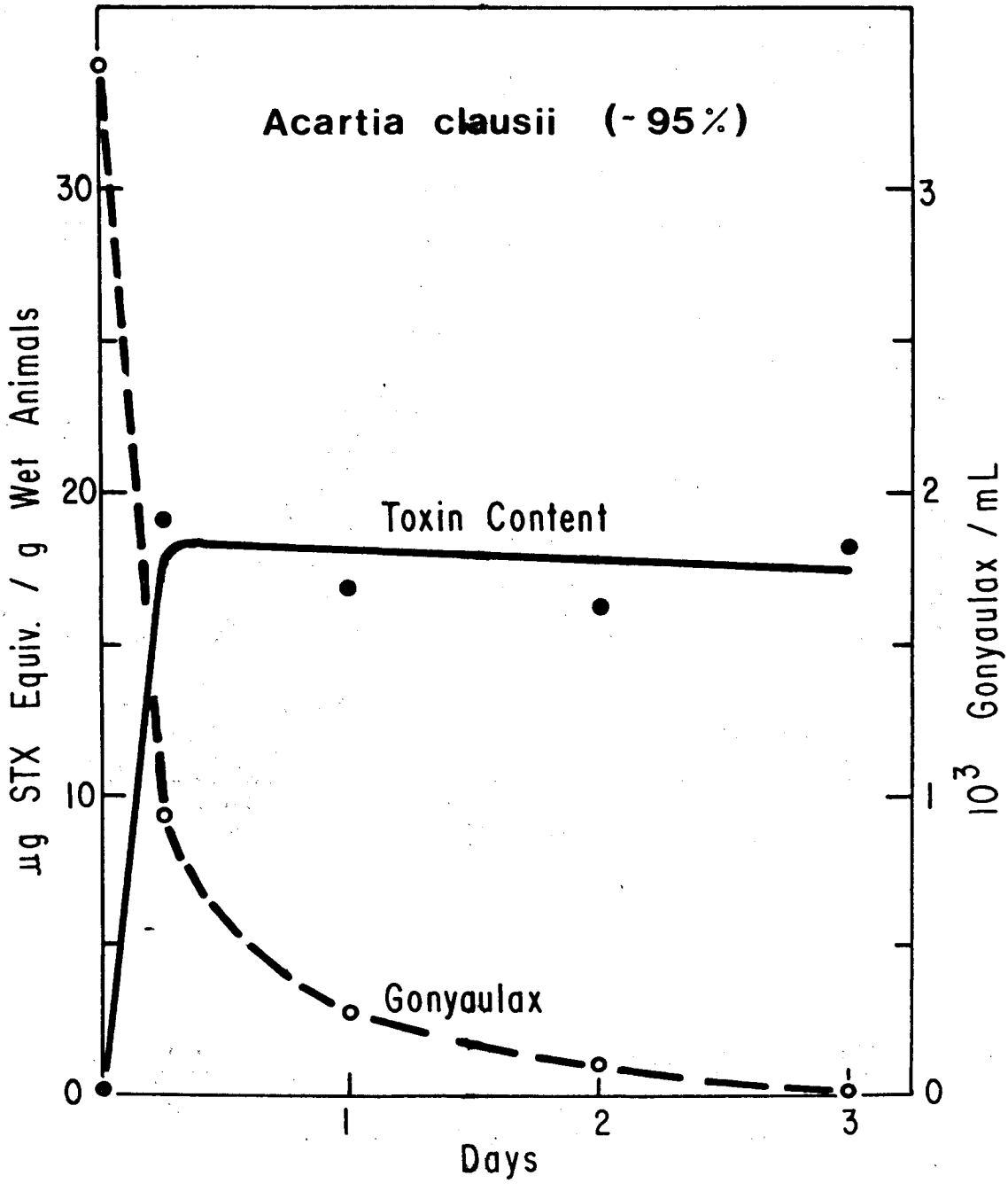

Fig. 2. Accumulation of G. excavata toxins by the copepod Acartia clausii while feeding on the toxic dinoflagellate (from White 1980a, with permission).

Fate of Gonyaulax Toxins in Marine Food Chains

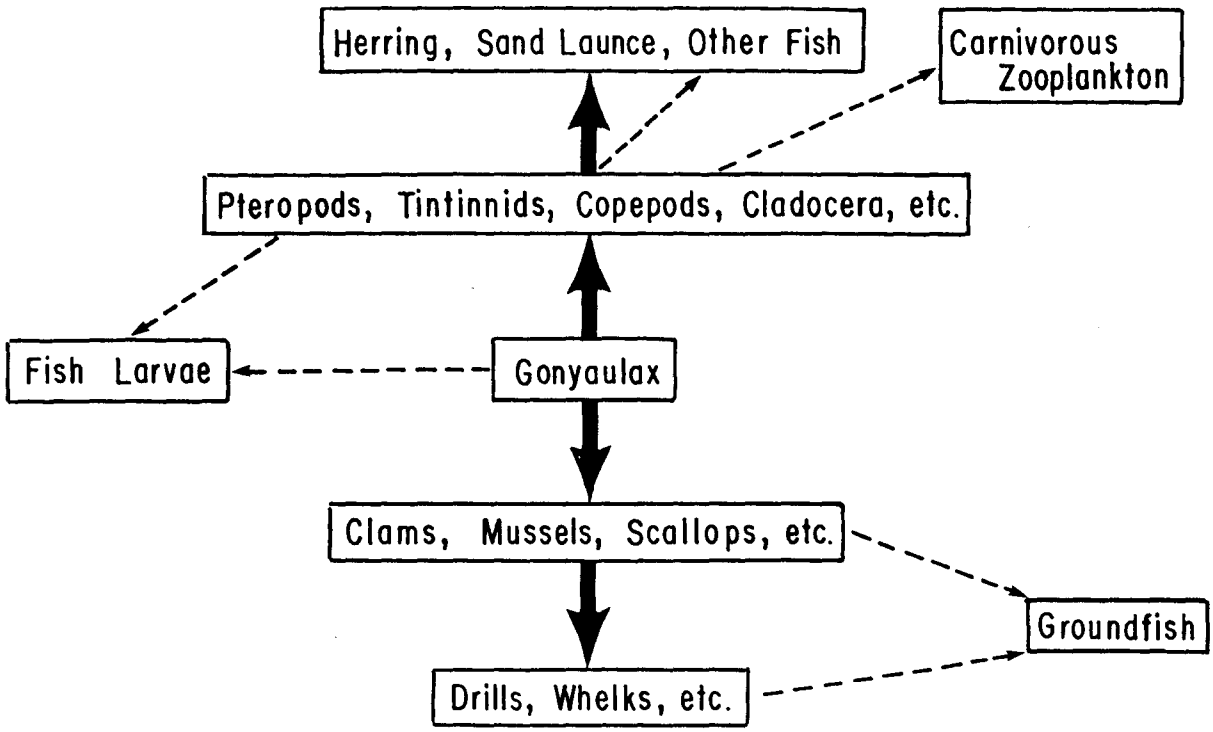

Fig. 3. Some routes of transmission of G. excavata toxins in the marine food web. Solid arrows represent known routes. Broken arrows represent possible routes.