

TOXIC DINOFLAGELLATE BLOOMS IN THE CAPE COD REGION OF MASSACHUSETTS

DONALD M. ANDERSON

Woods Hole Oceanographic Institution, Woods Hole, Massachusetts 02543

FRANCOIS M.M. MOREL

Department of Civil Engineering, Massachusetts Institute of Technology,
Cambridge, Massachusetts 02139

ABSTRACT

Recent research has linked the location, timing, and geographic spreading of toxic *Gonyaulax tamarensis* and *G. excavata* blooms on Cape Cod to the dormant overwintering cysts (hypnozygotes) of these species. In addition, variations in the trace metal speciation (and thus the toxicity) of coastal waters has been proposed as a second mechanism controlling the distribution of *G. tamarensis* populations. A model is presented here which synthesizes these and other recent developments and offers a coherent explanation of the historical pattern of shellfish toxicity in the region.

INTRODUCTION

In September 1972, Cape Cod was the southern terminus of a massive bloom of *Gonyaulax excavata*¹ that extended along the New England coast from Southern Maine. Prior to that date there had been no record of paralytic shellfish poisoning (PSP) in Massachusetts. This spread of toxicity to the Cape has been followed by a gradual dispersal of PSP to new locations within the region and by the persistent toxicity of one location (Mill Pond, Orleans) every year since 1972². Although the 1972 outbreak produced visible "red water" patches^{3,4}, all PSP episodes in the ensuing years have been marked only by the presence of toxic shellfish - not by discolored water. Further, the shellfish toxicity on Cape Cod has only been reported in estuarine or near-estuarine locations and not along the open coastline.

This historical pattern of shellfish toxicity is now more understandable since the discovery of overwintering cysts of *G. tamarensis* and *G. excavata*^{5,2} which had been postulated as seed populations for bloom initiation by Prakash⁶, Steidinger⁷ and Wall⁸ but not actually identified. Another constraint to the timing and location of toxic blooms was proposed by Anderson and Morel⁹ who demonstrated that copper could be toxic to *G. tamarensis* at cupric ion activities which are expected to occur in coastal waters. They suggested that variations in the

trace metal toxicity of coastal waters could thus influence the growth of the dinoflagellate, leaving other species unaffected. It is the purpose of this paper to incorporate these and other recent developments into a model that accounts for the temporal and spatial pattern of toxic dinoflagellate blooms in the Cape Cod region.

DISCUSSION

Figure 1 is a diagrammatic representation of the red tide phenomenon in the Cape Cod region - a "model" that may explain not only the timing, location, and dispersal of toxic outbreaks, but perhaps the longevity or viability of the blooms as well. Although this model is based on observations from a limited geographical area and presents toxic blooms (both spring and fall) as estuarine-generated phenomena, certain pathways are relevant to blooms originating in near-coastal waters as well as to toxic outbreaks of other cyst-forming dinoflagellate species in different climates.

Hypnozygotes are the key to the timing and localization of the initial phase of the outbreak. The early hypotheses of Prakash⁶, Steidinger⁷, and Wall⁸ concerning the potential role of dormant cysts in seeding toxic blooms have been well substantiated by several recent studies of the cysts of *G. tamarensis* and *G. excavata*^{5,2,10}. In particular, it is now established that the thick-walled elongate cyst (or hypnozygote¹¹) can successfully overwinter in the sediments^{5,2}, accumulating in topographical depressions and other sinks for fine particulate matter within estuaries. Excystment can be triggered by a temperature increase of only a few degrees¹⁰ after a requisite period of dormancy^{5,2}. The length of the dormancy period is determined by the length of the cyst, the ambient "conditioning" temperature, and the timing of the subsequent temperature change^{5,2,10}. Once initiated, germination generally continues through excystment, regardless of the chemical nature of the ambient seawater².

In contrast to the durable hypnozygote, the resulting motile cell is highly sensitive to adverse environmental parameters. As shown in Figure 1, motile cells shed their thecae and produce thin-walled non-motile cells in response to stress. These cells are only capable of limited periods of dormancy under natural conditions². They have been called temporary⁵, pellicle², or ecdysal cysts¹¹ by different authors. Although the formation of pellicle cysts has been induced in the laboratory through variations in light, temperature, salinity, nutrients and trace metals, the stresses were often unrealistic in terms of natural processes^{6,12,2,9}. For example, a sudden change from 16 to 5°C

yields a population with nearly equal numbers of motile cells and pellicle cysts, but this is clearly not a commonplace event. However, pellicle cysts do form rapidly in response to trace metal toxicity, and it is probable (based on laboratory experiments, chemical calculations and preliminary field data) that certain coastal waters are sufficiently toxic from a trace metal standpoint to force the *G. tamarensis* cells to form pellicle cysts, thus inhibiting their growth and the spread of toxicity^{9,10}. Since chelation of toxic metals can remove this inhibition, the presence of organic materials (from land drainage or local biological activity) might be a critical factor in the viability of the newly excysted populations. Land drainage has often been implicated as a critical factor in red tide development^{6,12}.

If environmental conditions are favorable, the first divisions of newly-excysted cells occur rapidly as the large (presumably diploid) germlings simply divide into two cells, each approximately half the original volume². It is unknown at this time how dense the initial excystment "inoculum" must be relative to the eventual bloom concentration, (i.e., how many cysts actually germinate prior to a bloom) but it is evident that certain localized areas within salt ponds and estuaries on Cape Cod have dense accumulations of cysts (e.g., Mill Pond, Perch Pond) and that these are the sites that first become toxic². Adjacent water bodies where the cyst populations are much smaller (or non-existent) become toxic at a later time, perhaps through advection of motile cells¹⁰.

During the critical period of population growth, the specific hydrodynamic characteristics of the water body can influence the cell densities directly through tidal advection and storm processes. As the population increases at one location, the cells can either be gradually dispersed into surrounding waters or they can accumulate to high densities without significant advection losses¹⁰. In the former case, the chemical suitability of the receiving waters is again important to the viability of the advected cells. Since lower density estuarine water masses can remain relatively intact over the more saline coastal waters (in the absence of mixing), continued growth is possible until nutrients are exhausted or until mixing produces an adverse chemical environment. This will either result in pellicle cyst formation and the probable destruction of the dispersed population, or, through a poorly understood mechanism (possibly nitrogen depletion¹¹), in the formation of new hypnozygotes that accumulate in locations where they are available to seed future blooms. The actual formation of hypnozygotes during a *G. tamarensis* bloom on Cape Cod has recently been

verified (D.M. Anderson, in prep.). It is also possible that a major tidal or storm event could flush many well-established localized motile populations from estuaries into coastal waters to produce the type of widespread shellfish toxicity seen in 1972 after the passage of hurricane Carrie. In this manner, hypnozygotes can function not only as a seed population for annually recurrent blooms but also as a dispersal mechanism whereby toxicity can spread to, and be "anchored" in, previously unaffected areas^{7,8,5,2}. This geographical spreading is a major characteristic of the New England, eastern Canadian red tide phenomenon.

Anderson and Morel¹⁰ demonstrated that hypnozygotes formed during a spring bloom and conditioned to summer water temperatures (20-22°C) remained dormant until the temperature dropped in the fall, resulting in a new excystment/encystment cycle. As seen in the diagram, this fall sequence of events differs from the spring only in the initiation or excystment stage. Figure 1 depicts the alternation of spring and fall blooms that is thus possible in a temperate zone estuary.

The model illustrates that although the mechanisms for bloom initiation are now understood, the precise timing of such outbreaks (i.e., when shellfish first become toxic) will vary with the water body. Not only is the rate at which cysts are warmed (or cooled) important, but the hydrodynamic characteristics of cell accumulation and dispersal, and the chemical characteristics of the receiving waters are critical as well. The model provides a plausible explanation for both localized episodes of estuarine shellfish toxicity and more widespread coastal events. The latter could alternatively be explained by a minor modification of the processes in Figure 1, whereby cyst "reservoirs" in near-shore topographic depressions serve as seed beds, and a suitable combination of upwelling, temperature change, and favorable environmental chemistry allow the necessarily low density initial population to flourish in the dynamic coastal environment^{5,2}. This sequence of events, though plausible, is intuitively less satisfying than the estuarine-generated hypothesis for coastal blooms in the Cape Cod region, especially in light of the consistent lack of shellfish toxicity at open coastline areas.

ACKNOWLEDGEMENTS

We thank D. Wall and S.W. Chisholm for helpful comments and criticisms. This research was supported in part by the Office of Sea Grant in the National Oceanic and Atmospheric Administration, United States Department of Commerce, through grant number 04-7-158-44079, in part by National Science Foundation grant number 77-09000, and in part by

International Copper Research Association Project Number 252.

REFERENCES

1. Loeblich, L.A. and Loeblich, A.R. III (1975) in: V.R. LoCicero (ed.), Toxic Dinoflagellate Blooms, Proc. intl. conf. (1st) Mass. Sci. and Technol. Fndn.
2. Anderson, D.M. and Wall, D. (1978) J. Phycol. 14, 224-234.
3. Yentsch, C.M., Cole, E.J., Salvaggio, M.G. (1975) in: V.R. LoCicero (ed.), Toxic Dinoflagellate Blooms, Proc. intl. conf. (1st) Mass. Sci. and Technol. Fndn.
4. Mulligan, H.F. (1975) in: V.R. LoCicero (ed.), Toxic Dinoflagellate Blooms, Proc. intl. conf. (1st), Mass.Sci. and Technol. Fndn.
5. Dale, B. (1977) Sarsia 63, 29-34.
6. Prakash, A. (1967) J. Fish. Res. Bd. Can. 24, 1589-1606.
7. Steidinger, K.A. (1975) in: V.R. LoCicero (ed.), Toxic Dinoflagellate Blooms, Proc. intl. conf. (1st) Mass. Sci. and Techno. Fndn.
8. Wall, D. (1975) in: V.R. LoCicero (ed.), Toxic Dinoflagellate Blooms, Proc. intl. conf. (1st) Mass. Sci. and Technol. Fndn.
9. Anderson, D.M. and Morel, F.M.M. (1978) Limnol. Oceanogr. 23, 283-295.
10. Anderson, D.M. and Morel, F.M.M. Estuarine and Coastal Marine Science (in press).
11. Turpin, D.H., Dobel, P.E.R., and Taylor, F.J.R. (1978) J. Phycol. 14. 235 - 238.
12. Prakash, A. (1975) Environ. Lett. 9, 121-128.