Section 2

Bloom dynamics: Physiology, behavior, trophic effects

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What is a bloom? A commentary

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Abstract

There are at least eight different modes and mechanisms by which harmful phytoplankton species can cause mortality, physiological impairment, or other negative in situ effects. Their distinction from nonharmful phytoplankton taxa is clearly warranted. Increasing use of bloom descriptors such as "exceptional," "unusual," "nuisance," and subjective reference to specific occurrences as blooms reveal widespread confusion concerning: what is a bloom?, how is it to be defined?, what is harmful?, and what distinguishes a harmful bloom from other blooms? Such efforts have been influenced by comparison with perceived spring diatom bloom characteristics. Examples, selected from among harmful bloom events, are presented to support the view that the classical focus on the spring diatom bloom has significant conceptual and operational biases as to what constitutes a bloom. This and the inexact criteria used to define a bloom compromise research on both harmful algal blooms and phytoplankton blooms in general. The subjective, differing, and arbitrary criteria used to define blooms and their presumed ecological consequences need to be replaced by a quantitatively based, ecological classification of the various types of phytoplankton blooms. The issue of what constitutes a bloom is more than simply a biomass issue.

The term "harmful" algal blooms (HABs), referred to as "red tides" in the older literature, has been applied to a class of blooms increasingly thought to have unique properties (Smayda 1997). This emergent bloom phenomenon, rapidly expanding in global coastal waters (Anderson 1989; Smayda 1990; Hallegraeff 1993), has many puzzling aspects. Efforts to understand these and to develop mitigation strategies require clarification of *what is a harmful bloom?* Evaluation of this and the related issues of *what is a bloom?* and what is meant by "harmful" is needed to circumvent problems associated with application of traditional views as to what constitutes a bloom, e.g. it is a significant population increase, during which the bloom and subordinate species within the community have equivalent ecological and physiological valence, and such blooms intrinsically are beneficial to food-web processes. These characterizations, current paradigms of bloom regulation and dynamics, and conceptual, experimental, and modeling approaches to blooms are based primarily on a diatom template. This reflects the historical focus of marine phytoplankton ecologists on the annual, high biomass, diatom-dominated spring (upwelling) bloom, whereas harmful algal blooms are primarily flagellate events (Sournia 1995). "Miniblooms" of diatoms developing during other seasons in response to episodic, "new" nutrient pulses are usually ignored. Likewise, blooms of other phylogenetic groups traditionally have been dismissed as ephemeral, transient pulses or, as in the case of red tides, as anomalous rogue blooms of peripheral interest. Rarely are the frequencies and trophodynamic impacts of such blooms considered. The dogma which seems to have emerged is that the spring diatom bloom is the only one worthy of serious study, that it is the major bloom event driving marine trophodynamics, and that resolution of its dynamics will generally explain nutrient-regulated fertility and bloom dynamics.

This restricted focus and the spring diatom bloom bias have a practical explanation. Methodological constraints make it difficult to deal with the successions of bloom and subordinate species occurring during bloom events, and their variable ecophysiology, bloom magnitude potential, and suitability as prey. Inherently, particularly during blooms, these interrelated successional features have both causes and consequences. Quantitative measurements of bloom processes as a function of these parameters are not only methodologically difficult, but usually further constrained by the need for high population abundance. These analytical problems are classically accommodated by applying a reductionist, whole-community approach, with community size-fractionation a partial refinement (Malone 1980). Bloom abundance and fluctuations are then commonly expressed in terms of community biomass, with chlorophyll amount usually the index of abundance against which total-community rate processes, such as productivity, are often normalized. Of the bloom events in the sea, the high abundance and monotonous diatom flora of the annual spring (upwelling) bloom historically have best satisfied these analytical needs. This largely explains our biased understanding of blooms, based primarily on proximate, whole-community approaches, with neglect of the organismal approaches essential to elucidation of bloom events. Some phytoplankton ecologists, who typically focus on mass balance dynamics and primary production, have slowly come to recognize what organismally focused ecologists accept as a truism—the trophic consequences of blooms should vary with the bloom species. This is particularly evident to those who investigate HAB

events and who keenly recognize that traditional mass balance approaches to bloom events are inappropriate to investigation of harmful bloom dynamics.

Given the availability of newer techniques allowing investigation of species-specific dynamics, including molecular probes, and greater awareness of other bloom types, such as HAB events, the historical raison d'etre for virtually exclusive focus on spring (upwelling) diatom blooms is no longer tenable. In fact, the spring bloom-diatom bias, the reductionist, whole-community approach, and derived paradigms and experimental approaches are compromising research on both harmful algal blooms and phytoplankton blooms generally. In the case of HAB events, these blooms should no longer be viewed as periodic, rogue blooms of peripheral interest unworthy of serious scientific analyses. Rather, they provide a unique opportunity to evaluate entrenched views based on the spring bloom-diatom template (Smayda 1997). In my view, such blooms provide a scientific Rosetta Stone allowing deeper insights into the underlying principles and processes regulating phytoplankton growth in the sea and the blooms of diatoms vis a vis those of other phylogenetic groups.

What is a harmful species?

Three distinct modes of negative impact have led to the characterization of certain species as harmful, with the descriptors "toxic," "noxious," and "nuisance" used either synonymously or to characterize relative degree of impact. Phytoplankton species which cause fish poisoning (ciguatera) and shellfish-vectored poisoning of humans are clearly harmful (*see* Hallegraeff 1993). Public health concerns over bloom-related, shellfish-borne diseases affecting seafood safety are an emergent global concern. Species whose blooms induce die-offs of farmed fish and cultivated shellfish leading to aquacultural financial losses are also harmful. The magnitude of economic loss can be staggering. On a per bloom basis, it has exceeded $60 million (U.S.) per single episode in the case of shellfish culture (Shirota 1989) and, over an 18-yr period, farmed-fish losses in the Seto Inland Sea exceeded $100 million (Okaichi 1989; Shirota 1989). Commercial fisheries can also be impacted. Gelatinous secretion during an unusual bloom of the diatom *Coscinodiscus wailesii* in the North Sea was so great that the weight of the slime tore fishing nets and impaired the fishery (Boalch and Harbour 1977). Deflections of migratory routes (Savage 1930), emigrations (Lenanton et al. 1985), and other avoidance (Potts and Edwards 1987) reactions of commercially important fish during harmful blooms also occur.

With regard to ecological effects—the third major category of impact—field and experimental evidence reveals at least eight different modes and mechanisms by which harmful species cause mortality or physiologically impair target victims (Table 1). These fall into two general types: nonchemical effects which lead to starvation or cause harmful mechanical and physical damage, and chemical effects attributable to physical-chemical reactions, phycotoxins, or other metabolites. Starvation-impaired growth and fecundity resulting from nutritional and prey-size mismatches are well known experimental results. Excess prey density ($>250\times10^6$ cells liter^{-1}) of the brown tide species, *Aureococcus anophagefferens,* led to spawning failure and mortality of the mussel, *Mytilus edulis* (Tracey 1988). Mechanical damage leading to death of larval shellfish resulted from collisions ("bumping") with dinoflagellates at high bloom population densities (Ho and Zubkoff 1979). Lethal piercing of gill filaments of penned and feral populations of fish and crustaceans by the setae of chaetocerid diatoms also occurs (Horner et al. 1990; Rensel 1993; Yang and Albright 1992; Tester and Mahoney 1995). Respiratory failure, hemorrhaging, or bacterial infection result from such particle irritation. Extracellular polymer secretions of harmful species can induce mortality in three physical ways: as a viscosity barrier (Partensky et al. 1989; Potts and Edwards 1987), by forming a gelatinous barrier (Taylor et al. 1985), and through mucoid layer reduction (Endo et al. 1985). Gill clogging, elevated shear stress which tears gill filaments, and osmoregulatory failure lead to death.

Among the chemically harmful mechanisms, die-offs due to anoxia or hypoxia following blooms of large, relatively ungrazed species, such as *Ceratium,* are well known (Granéli et al. 1989; Mahoney and Steimle 1979). *Noctiluca* produces a remarkably simple ichthyotoxin: NH_4 (Okaichi and Nishio 1976). Phycotoxins, in contrast, have a much more elaborate chemical structure and diverse modes of inimical effects. Mortality can occur through direct ingestion (endotoxin) of the harmful species, upon exposure to secreted toxins (ex-

Table 1. Mortality modes and impact mechanisms of harmful species and their blooms.

1. Starvation	
	Nutritional mismatch
	Size mismatch
	Excess prey density
2. Mechanical	
	"Bumping"
	Particle irritation (*Chaetoceros* spp.)
3. Physical	
	Viscosity barrier (*Gyrodinium aureolum*)
	Gelatinous barrier (*Cerataulina pelagica*)
	Mucoid layer reduction (*Chattonella marina*)
4. Anoxia	
	Ceratium blooms
5. NH_4 toxicity	
	Noctiluca blooms
6. Phycotoxins	
	Direct vs. vectored toxicity
	Saxitoxin
	Brevetoxin
	Hemolysins
	Cytotoxins
7. Allelopathic	
8. Ambush predation	
	Pfiesteria piscicida
9. Unresolved	
	Aureococcus anophagefferens

otoxins), or from toxin vectoring through the food web, accompanied by conformational changes in the toxic principles and their potencies (Shimizu 1989; Yasumoto and Murata 1993). The impact may be on a directly targeted predator or lead to indiscriminate die-offs and physiological impairment during food-web vectoring. Specific toxins are water- or lipid-soluble and have cytolytic, hemolytic, or neurotoxic activity. Allelopathic substances are distinguished from phycotoxins in being secondary metabolites; both can co-occur within a given harmful species. Allelopathic substances tend to be directly targeted and may physiologically impair, stun, repel, induce avoidance reactions, and kill grazers. Examples of these diverse modes of impact were given by Smayda (1992). Information on the recently discovered, remarkable ambush predatory behavior of the harmful dinoflagellate, *Pfiesteria piscicida,* was presented by Burkholder and Glasgow (1995) and Lewitus et al. (1995).

The general impression that inimical effects of diatoms, unlike flagellates, are relatively rare and restricted to mechanical and physical impacts (Table 1) needs comment. Recent findings implicate domoic acid produced by species of the pennate diatom *Pseudo-nitzschia* as the cause of amnesic shellfish poisoning of humans and die-offs of piscivorous birds (Bates et al. 1989; Hallegraeff 1993; Work et al. 1993). Poulet et al. (1994) have generalized from experimental evidence that diatoms have evolved an allelopathic, antipredation strategy by which they reduce copepod population levels by inhibiting their reproductive success. These unexpected antagonisms of diatoms may reflect broader based occurrences within this group awaiting discovery.

Despite convincing evidence that harmful phytoplankton species occur and bloom, knowledge of what defines a harmful species is qualitative. Such species are still being identified, and the modes and mechanisms of harmful effects, and ecophysiological divergence of harmful taxa from species considered to be nonharmful are still being described. Nonetheless, several patterns reveal themselves. Harmful taxa effects are not limited to the public health threats and aquacultural financial losses traditionally focused upon. Growing evidence suggests that negative in situ ecological effects are potentially much more significant and remarkably diverse. All trophic compartments of the marine food web are now known to be vulnerable to harmful blooms (*see* figure 13.5 of Smayda 1992). However, it is not a corollary that species which are harmful to humans because of their seafood toxins, or induce die-offs of fish enclosed in aquaculture pens preventing avoidance reactions, are also antagonistic in situ. There is thus reason for concern over increasing, indiscriminate use of the descriptors "exceptional," "unusual," "toxic," "nuisance," and "benign" applied to specific bloom events, and during which ecological disruption of "normal" dynamics is implied. Rarely is this based on ecological data. Quantitative ecological studies to establish the in situ impacts of harmful algal species and their blooms should therefore be a high priority of future research. Among the more relevant issues needing investigation are the extent to which such blooms impair or disrupt "equilibrium" trophodynamics and whether they fall within the variance in natural catastrophic events which shape this equilibrium. Such studies are also needed to assess the validity of the spring diatom bloom model as a general model of bloom dynamics in the sea.

What is a harmful bloom?

How abundant does a harmful species have to be to form a harmful bloom? Are its harmful effects density-dependent? Historically, blooms have been inferred to be significant population increases, without concern over bloom magnitude or impacts. Recognition that harmful species may be unique in their broad spectrum of antagonisms relative to blooms of other species, notably diatoms, has stimulated efforts to distinguish formally between such bloom types. While this has broadened insights into the nature of phytoplankton blooms, it has also revealed widespread confusion concerning "what is a bloom?" and how it is to be defined. This becomes even more problematic when efforts to define a bloom in terms of abundance are linked to a descriptor, such as "exceptional bloom." Tett (1987), for example, defines exceptional blooms as those with >100 mg Chl m^{-3}. Admittedly, blooms of this magnitude are noteworthy, particularly if maximal biomass levels of the annually recurrent blooms are usually far below this level. Attendees at the 1984 ICES Exceptional Plankton Blooms Meeting concluded that exceptional blooms should be defined in terms of the deviation from the "normal cycle of phytoplankton biomass" (Parker 1987). Richardson (1989) defined a bloom as an outburst resulting in "high biomass," stating also that "a red tide is an exceptional bloom." Reid et al. (1987) would apply the term exceptional to blooms, if accompanied by "deleterious side effects," and Richardson acknowledged the popular tendency to refer to toxic species' occurrences as blooms independent of their abundance levels. Cellular abundance levels have also been used to define red tide blooms. Kim et al. (1993) classify blooms as "red tides" (in Korean waters) when abundance of the causative species (those >30 μm) exceeds 1,000 cells ml^{-1}. This criterion results in ~200 diatom species being classified as red tide species, which represents about two-thirds (65%) of the total number of species classified as such (Sournia 1995).

Different criteria clearly are being used to define "what is a bloom?" and its classification as harmful vs. benign. For nontoxic species, biomass is the criterion most often used to establish bloom status. For harmful species, mere presence or measurable toxin levels are increasingly defined as a bloom occurrence. These subjective, differing, and arbitrary criteria used to define and distinguish "what is a harmful bloom?" from other blooms ignore that blooms themselves are a trophodynamic process. Blooms, as a phenomenon, collectively have properties other than biomass, numerical population density, and the potential of the bloom species to inflict harm. These features are also relevant to their definitions, occurrences, and in situ trophodynamic consequences. The processes influencing species occurrences, whether harmful or not, may differ significantly from those regulating their blooms. And, at any given time or spatial location, as an inherent aspect of species succession, one or more species are in a state of bloom even though they may not achieve high biomass or high population density. Max-

imal, potential bloom levels differ intrinsically among species (Agusti et al. 1987), with variations in environmental carrying capacity further influencing bloom magnitude. A species does not have to achieve high biomass or high population densities to be in a bloom state nor intrinsically be capable of attaining high abundance. It may be in bloom, for example, even at a maximal population level of only 10^4 cells liter^{-1} (or lower) as a result of active growth from its lower level of 10^2 cells liter^{-1} (or less). (This approximates a net growth response of *seven* generations, assuming no population losses!) A bloom may occur as an isolated community event or co-occur with other species, some of which may then reach population maxima several orders of magnitude higher. In oligotrophic seas, a seasonal chlorophyll maximum of 4 mg m^{-3} developing from a baseline level of 0.2 mg m^{-3}, for example, would represent a bloom, even though this maximum may not be much higher than background levels in a temperate coastal system where the bloom maximum may reach 30 mg m^{-3}. What constitutes a bloom, therefore, has regional, seasonal, and species-specific aspects; it is not simply a biomass issue. Use of a specific biomass (chlorophyll) level as a criterion of bloom definition, whether generally or for a given species, is fraught with problems (Smayda 1997).

The salient criterion to use in defining whether a "harmful" species is in bloom and the distinctive feature of such blooms lie not in the level of abundance, but whether its occurrence has harmful consequences. Harmful bloom occurrences clearly reveal that pegging the definition of blooms generally to abundance levels, whether as biomass or numerical cell abundance, is inappropriate. This is easily shown. Maximal chlorophyll levels during a brown tide outbreak of the 2-μm *A. anophagefferens* in Narragansett Bay fell within the range of annual winter–spring bloom maxima (Smayda and Villareal 1989). The significant feature of this bloom was not its biomass, but its maximal population abundance which reached 2×10^9 cells liter^{-1}. At 250×10^6 cells liter^{-1}, the mussel, *M. edulis,* stopped feeding, starved, and died-off (Tracey 1988). In situ irradiance was also attenuated by the bloom leading to macroalgal die-off (Smayda and Villarcal 1989). An even greater disruption of trophodynamics accompanied the high population ($\sim10^8$ cells liter^{-1}), low biomass bloom of the nanophytoplanktonic *Chrysochromulina polylepis* (~10 μm) in Scandinavian coastal waters (Dahl et al. 1989). Now consider the other extreme (e.g. low biomass and cellular abundance): oysters filter-feeding on the dinoflagellate *Dinophysis* present at population densities of $<10^3$ cells liter^{-1} (i.e. six orders of magnitude lower than the Narragansett and Scandinavian blooms!) accumulated diarrhetic shellfish toxins which led to human poisoning (Belin 1993). This would be a "harmful bloom," applying common parlance, but from a traditional biological oceanographic and ecosystem perspective, it would be neither harmful nor a bloom! At yet another level, ingestion of only 6–11 cells of toxic *Alexandrium tamarense* was lethal to first-feeding sea bream (*Pagrus major*) larvae (White et al. 1989), and only one cell of a more toxic strain was lethal to capelin (*Mallotus villolus*) and herring (*Clupea harengus harengus*) larvae (Gosselin et al. 1989).

Harmful effects may therefore accompany very high or very low biomass bloom events, occur at very low or very high cellular abundances, be due to very small or very large species' size, and the same species may be simultaneously inimical to several trophic components or have multiple effects within a given one—and for different reasons. The presence of a toxic species does not necessarily lead to a deleterious impact; such effects require a threshold population density level, above which it becomes inimical. The population level at which this occurs need not be accompanied by seawater discoloration, contrary to anecdotal perceptions of red tides. Given these considerations, formal definitions of what makes a species or its blooms harmful, toxic, noxious, a nuisance, benign, exceptional, and(or) unusual are neither practical nor helpful, and the application of such descriptors to in situ processes should be discontinued until, or unless, quantitative data in support of such distinctions are available. However, the basic distinction between harmful and nonharmful blooms is not only warranted, it is an important first step toward classification of the various types of phytoplankton blooms which undoubtedly occur. It seems very unlikely that the spring diatom bloom model is generally representative. Research on harmful algal blooms should contribute significantly to the quantification of both the common and divergent properties of the different bloom events occurring in the sea, beyond resolving many of the puzzling features of this particular class of blooms.

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