
MINI REVIEW

COMMUNITY AND ECOSYSTEM LEVEL CONSEQUENCES
OF CHEMICAL CUES IN THE PLANKTON

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Abstract—Aquatic organisms produce compounds that deter consumers, alter prey behavior, suppress or kill target and nontarget species, and dramatically affect food-web structure, community composition, and the rates and pathways of biogeochemical cycles. Toxins from marine and freshwater phytoplankton create health hazards for both aquatic and terrestrial species and can significantly affect human activities and the economic vitality of local communities. A reasonable case can be made that phytoplankton metabolites such as dimethyl sulfide (DMS) link interaction webs that span hundreds to thousands of kilometers and connect production from oceanic phytoplankton to desert cacti and coyotes via zooplankton, fishes, and sea birds. The possible role of DMS in global heat budgets expands this effect even further. The ecosystem-wide and potentially global consequences of aquatic chemical cues is an underappreciated topic that warrants additional attention.

Key Words—Aquatic chemical ecology, biotoxin, complex interactions, DMS, harmful algal bloom, phytoplankton, zooplankton.

INTRODUCTION

Chemical signals produced by aquatic organisms play important roles as deterrents of consumers, pathogens, and competitors and as cues used for foraging, reproduction, and assessing danger from a variety of natural enemies. Such direct

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effects are becoming well documented in a variety of aquatic systems (Hay, 1996; Tollrian and Harvell, 1999; McClintock and Baker, 2001). In this review, we emphasize that these same chemical cues commonly have indirect and cascading effects on the ecology and evolution of entire communities and ecosystems. These larger-scale spatial and temporal effects are less well studied and understood. As such, they offer considerable intellectual challenges to, and opportunities for, enhancing our appreciation for how chemical cues produce cascading impacts on species, communities, and ecosystems beyond the initial group of directly interacting organisms. Given our charge to focus on novel and less well-known topics for this mini-review, we chose to emphasize chemical cues among planktonic organisms. Direct effects of chemical cues in these systems is poorly known relative to those documented in benthic systems (Hay, 1996; McClintock and Baker, 2001). Additionally, studies of chemically mediated planktonic interactions have rarely progressed to the point of unambiguously identifying the metabolites responsible. However, it is clear that chemically mediated interactions are omnipresent among the plankton and that chemical cues commonly produce cascading effects far beyond the initial participants. We discuss examples of this below.

PHYTOPLANKTON TOXINS IN LAKES

Given the smaller size and contained nature of lakes and ponds relative to oceans, ecosystem level effects of chemically mediated interactions could be most apparent in these systems. Indeed, there are numerous cases in which toxins produced by pelagic freshwater cyanobacteria have been shown, or suspected, to produce strong effects on both aquatic organisms and nearby terrestrial species, including humans. Cyanobacterial poisoning of animals has been reported since the 1800s. Kills have included fishes, birds, dogs, livestock, and 50 renal dialysis patients in Brazil (Jochimsen et al., 1998; Chorus, 2001). In many cases, liver-targeting cyanobacterial microcystins are believed to have caused mortality. Microcystins are contained within cyanobacteria cells and are not commonly released into the water. However, cell lysis releases microcystins, and these can persist in freshwater systems for several weeks because their cyclic structure and unusual amino acid composition renders them resistant to degradation by many common bacterial proteases. Thus, animals can be affected via consumption of cells or via water-borne exposure to these released toxins. However, despite the copious literature on microcystins and closely related compounds, numerous other assays and chemical investigations suggest that cyanobacteria produce many as yet unidentified peptides, depsipeptides, lactones, alkaloids, and other metabolites with broad-reaching effects, some of which may be responsible for the ecological consequences attributed to microcystins (Chorus, 2001).

Humans who have ingested water exposed to cyanobacterial blooms have experienced abdominal pain, nausea, vomiting, diarrhea, headache, fever, muscular

pain, sore throat, dry cough, blistering of the mouth, atypical pneumonia, and elevated liver enzymes (Chorus, 2001). Illnesses in Europe, Australia, and the United States have been tracked to toxic cyanobacteria, but the role of the particular compounds causing these symptoms has not been clearly demonstrated. Given these symptoms, humans alter their behavior once cyanobacterial blooms are detected. This includes prohibition of human uses [drinking, swimming, water-contact activities—especially for those with compromised livers (e.g., alcoholics, those infected with hepatitis B)] and treatment of drinking water by filtration, chlorination, sieving, flocculation with chemicals, adjustment of pH, and ozonation.

Numerous investigations have also demonstrated the effects of cyanobacterial metabolites on aquatic organisms. Survival, growth, and time of hatching were modestly affected when fish embryos were exposed to ecologically realistic concentrations of dissolved microcystins; older fish exposed to microcystins exhibited altered levels of mobility and feeding and shifted activity between day- and nighttime patterns (Chorus, 2001). Given the dramatic trophic cascades that fish feeding produces in lakes (Carpenter et al., 1985), these altered behaviors may affect ecological patterns and processes throughout the entire body of water.

Some zooplankters (e.g., copepods) are discriminating feeders that select among phytoplankton particles based on characteristics including taste (Porter, 1977). In contrast, other zooplankters (e.g., *Daphnia*) feed more indiscriminately and do not appear to select based on flavor or other chemical signals (DeMott, 1986; DeMott et al., 1991). Because they are more willing to feed on the foods that are offered, these indiscriminate feeders offer unusual opportunities for assessing the consequences of consuming toxic prey. Lampert (1982) surveyed how the presence of the toxin-producing cyanobacterium *Microcystis aeruginosa* affected 13 species of cladocerans and demonstrated that the smaller species were less affected than the larger ones. He speculated that this differential tolerance could drive seasonal shifts in species composition within the zooplankton community. Differential effects of the toxic cyanobacteria *Anabaena affinis* on cladocerans and rotifers can also reverse competitive outcomes among zooplankters and may change the species composition and size structure of zooplankton communities (Gilbert, 1990; Kirk and Gilbert, 1992). An overview of recent investigations (Christoffersen, 1996) suggests that cyanobacteria: (1) affect both phytoplankton and aquatic macrophytes via allelopathic compounds, (2) disrupt linkages between phytoplankton production and zooplankton growth, and (3) shift rates and pathways of energy flow, nutrient cycling, and degradation from pelagic to benthic zones. Thus, phytoplankton metabolites appear to produce cascades throughout the food web, affecting not only community structure and species composition, but also fundamental pathways and rates of biogeochemical cycles.

As might be expected from the strong effects noted above, cyanobacteria that produce toxic compounds can exert strong selection on herbivores, altering their genotypes over relatively short periods of time. As an example, Hairston et al.

(2001) hatched eggs of *Daphnia galeata* that had been deposited in stratified lake sediments over a span of 35 years; the prevalence of planktonic cyanobacteria had increased substantially during this 35-year period. *Daphnia* representing genotypes from three distinct deposition times were fed either a palatable alga, or a mix of the palatable alga plus the toxic cyanobacterium *Microcystis aeruginosa*. Effects of the cyanobacterium on the performance of each genotype demonstrated that *Daphnia*'s ability to tolerate the cyanobacteria had increased within little more than a decade.

Phytoplankton may also respond to, or be chemically manipulated by, other phytoplankton. Keating (1977) monitored bloom sequence in a lake over a three-year period, then tested the effects of cell-free filtrates of dominant cyanobacteria on other phytoplankters that either preceded or followed the cyanobacterial bloom. Filtrates suppressed predecessors in five of eight trials (no effect in the other three) while enhancing successors in six of nine trials (no effect in the other three). This suggests that allelopathic agents may affect the sequence of blooms in lakes, but the involvement of particular compounds has not been demonstrated.

PHYTOPLANKTON TOXINS AND EFFECTS IN THE SEA

Phytoplankton dominate primary production in the world's oceans and are found in almost all marine habitats. Approximately 7% of phytoplankton species are known to form large-scale blooms, dramatically impacting marine communities. Of these, about one third have been documented to produce noxious compounds that kill marine organisms or that are subject to trophic transfer and bioconcentration in animals (Smayda, 1997). The tendency of phytoplankton to form blooms, coupled with their ability to be moved over hundreds of kilometers and to produce potent toxins, can result in massive wildlife kills and shutdowns of coastal tourism and fisheries, dramatically impacting local economies and the health and function of coastal ecosystems. The increased frequency and wider geographic distribution of toxic algal blooms (Anderson, 1989) have become major environmental and health concerns around the world (NRC, 1999). Many phytoplankton toxins are passed to higher trophic levels, eventually reaching fishes, marine mammals, and human consumers (Shumway, 1990). Animal kills worldwide have been attributed to phytoplankton compounds with diverse molecular structures including polycyclic polyketides, macrolides, isoprenoids, and amino acid-derived compounds produced by dinoflagellates, diatoms, and other microalgal taxa (Figure 1). Much remains to be learned about how blooms initiate, why populations of phytoplankton expand so rapidly, how they are affected by interactions with other organisms, and why blooms stop. The crucial ecological question of why many microalgal species produce potent neurotoxins remains unanswered, although these compounds clearly have dramatic consequences

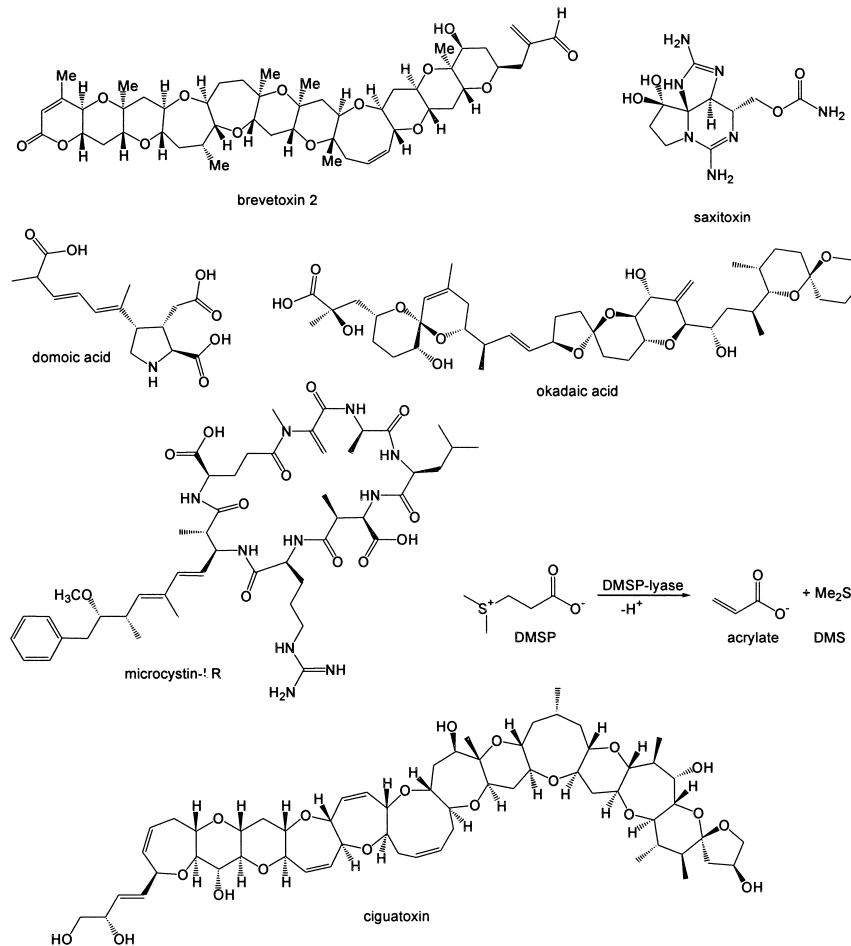


FIG. 1. Microalgal metabolites that affect food-web structure, community composition, human health and economy, and/or the rates and pathways of biogeochemical cycles.

on populations of many marine species, on community structure, and often on ecosystem function.

Microalgal blooms that involve transfer of algal metabolites to fish or filter-feeding shellfish, that cause animal mortality, and/or that reduce coastal water quality are referred to as harmful algal blooms (HABs) because of their negative economic and human health consequences. In the United States, conservative estimates of direct and indirect costs due to HABs averaged \$49 million per year for the period 1987–1992, including public health, commercial fishery,

recreation/tourism, and monitoring/management costs [reported in year 2000 US dollars (Anderson et al., 2000)]. Ciguatera poisoning, the most prevalent human illness caused by ingestion of HAB toxins, sickens 50,000 people in tropical countries each year, and the reported numbers are believed to represent only 20–40% of the actual cases (NRC, 1999). Ciguatera toxins such as ciguatoxins, maitotoxin, and gambieric acids are produced by benthic dinoflagellates such as *Gambierdiscus toxicus* that grow epiphytically on seaweeds and on coral rubble. The toxins are bioconcentrated through the food web as herbivorous fishes and invertebrates feed on seaweeds and small turf communities harboring the dinoflagellates. Toxins consumed by the herbivores are then transferred, via predation, to larger fishes such as moray eels and barracuda. During the process of bioconcentration, the parent metabolites may be altered to become more toxic, generating greater risk for humans consuming top carnivores from reefs (Yasumoto and Murata, 1993). Many species of tropical fishes accumulate these toxins without showing signs of physiological stress; however, when these fish are consumed by humans, ciguatera toxins activate voltage-gated sodium channels, causing gastrointestinal and respiratory distress, followed by neurological problems, bone and joint pain, and chronic fatigue that can last for months or years. Because the distribution of ciguateric dinoflagellates is geographically patchy but generally persists over time, fishermen and local residents often know which reefs harbor contaminated fishes.

There has been a strong association between large-scale animal kills and blooms of several pelagic microalgal species, such as *Karenia brevis* (ex *Gymnodinium breve*), which produces brevetoxins. Immunohistochemical and pathological data have supported the role of brevetoxins in the death of animals such as manatees (Bossart et al., 1998), and strong circumstantial evidence points to brevetoxins as the cause of death for dolphins, pelicans, and various fishes and invertebrates during several blooms over the last century (Gunter et al., 1948; Steidinger et al., 1973).

Toxins produced by HABs can fundamentally affect entire marine ecosystems, can impact locations hundreds or thousands of kilometers from where blooms initially form, and can even affect human activities and terrestrial ecosystems. As an example, in 1987–1988, satellite images showed a bloom of *Karenia brevis* initiating along the Gulf coast of southern Florida, being transported northward by the Gulf Stream, and being delivered to inshore North Carolina by a loop current from the Gulf Stream. *K. brevis* had never before been seen so far north of the tropics. This unanticipated bloom had a variety of deleterious effects including: (1) massive scallop mortality in coastal North Carolina, (2) closure of shellfish harvesting, (3) respiratory distress in humans and pets along coastal beaches with 48 documented cases of human illness attributed to brevetoxicosis, and (4) reduced coastal tourism and recreation by humans; costs to coastal communities were estimated at \$24 million due to loss of the shellfish harvest alone (Tester and Fowler, 1990).

The effects of HABs on coastal resources and on charismatic megafauna such as whales, dolphins, and manatees is relatively well documented compared to their potential effects on less obvious systems such as detrital food webs and soft-sediment communities (e.g., Gunter et al., 1948; Kvitek, 1993; Bossart et al., 1998). When toxic blooms senesce, do their natural products affect detrital pathways or biogeochemical cycles and rates of processing? The direct effects of compounds are unknown, but faunal changes do occur in soft-bottom communities beneath toxic algal blooms. When Olsgard (1993) monitored soft-bottom communities at depths of 69–184 m and described changes in fauna over a five-year period that contained a bloom of the toxic flagellate *Chrysochromulina polylepis*, he found a bloom-associated depression in numbers of both species and individuals; this effect persisted for nearly two years following the bloom. Although such contrasts confound the effects of phytoplankton toxins with the potential effects of increased detrital biomass and decreased light/oxygen, they suggest the potential for substantial chemically mediated effects on detritivore communities.

Diatoms belonging to the genus *Pseudonitzschia* also have cascading effects on coastal food webs. On both the east and west coasts of North America, these diatoms have been shown to produce domoic acid, a neuroexcitatory amino acid that enters the food web through shellfish such as mussels (Bates et al., 1989) and filter-feeding fishes such as anchovies (Scholin et al., 2000). Humans, sea lions, cormorants, and other consumers of toxic fish or shellfish have all been killed by domoic acid poisoning in episodes that have only been documented in the last 15 years. Domoic acid kills mice at concentrations similar to those found in contaminated seafoods, and the toxin was isolated from the body fluids of animals that died after consuming these foods, providing strong support for toxin-induced mortality (Bates et al., 1989; Scholin et al., 2000).

When neurotoxic dinoflagellates such as *Alexandrium* spp. bloom in coastal waters, they are consumed by filter-feeding animals such as butter clams. These clams are relatively unaffected by consuming the toxins because their neurons are insensitive to saxitoxin and other closely related sodium channel-blocking toxins produced by *Alexandrium* spp. (Kvitek, 1993). Saxitoxins are concentrated in clam siphons where small fish can detect them during “siphon-nipping” predatory behavior, leading the fish to avoid toxic clams. Siphon-nipping behavior by fishes may have selected for the sequestration of toxins in these more exposed regions of the clams. When clam toxicity levels are high, sea gulls and sea otters reduce their overall feeding on clams and reject clam siphons. Thus, the microalgal toxins are transferred from primary producers to clams, where they act as a chemical defense against the next trophic level, deterring both aquatic (otters and fishes) and terrestrial (birds) vertebrate predators. Because otters avoid toxic shellfish and suffer physiological distress if they mistakenly feed on them, the dinoflagellate blooms that occur primarily in protected bays may historically have restricted otters from these bloom areas and limited the feeding activities of these keystone

species to outer coasts where clams have lower concentrations of dinoflagellate-derived toxins (Kvitek, 1993). Because otter feeding has dramatic cascading effects on nearshore ecosystems, turning unproductive urchin barrens into productive and species-rich kelp forests (Estes and Duggins, 1995), restriction of otters due to microalgal toxins has the potential to fundamentally alter the distribution, abundance, and function of entire coastal ecosystems (Duggins et al., 1989, Kvitek, 1993). Consequently, these phytoplankton metabolites have cascading effects on community structure and on temporal and spatial ecosystem dynamics involving seaweeds, invertebrates, fishes, and keystone predators such as otters.

Additionally, recent work by Bricelj et al. (2000) has shown that some bivalves show differential susceptibility to the accumulation of dinoflagellate toxins and to their negative physiological effects. In experiments in which softshell clams (*Mya arenaria*) from two populations were exposed to toxic *Alexandrium tamarensis*, clams from the population with no previous history of exposure to *Alexandrium* showed higher mortality, lower respiration, impaired burrowing capacity, reduced food clearance rates, and a 10-fold lower toxin accumulation, relative to clams from a historically exposed location. Failure of clams to burrow could have negative consequences for toxin-susceptible individuals, because exposed bivalves are more vulnerable to predatory birds and crustaceans. Thus, exposure to occasional blooms of toxic *Alexandrium* may lead to increases in the number of toxin-resistant individuals in a population, due to differences in fitness between resistant and susceptible bivalves. Ongoing changes in the frequency and geographic distribution of toxic microalgae may result in further selection for tolerance of toxins among filter-feeding organisms whose populations show variance in their degree of resistance to toxins. This could produce a gradual increase in the levels of toxins contained in bivalves that humans harvest. The mechanism(s) by which some bivalves minimize the effects of phytoplankton toxins are as yet unknown, although the possibility of resistance-conferring mutations in mollusk sodium channels is the subject of current investigation (V. M. Bricelj, personal communication).

Although phytoplankton metabolites can enter food webs via shellfish and filter-feeding fishes, phytoplankton face the greatest threat of herbivory from grazing zooplankton including ciliates, copepods, rotifers, tintinnids, and invertebrate larvae (Turner and Tester, 1997). The greatest opportunity for chemically mediated interactions may therefore occur between phytoplankton and zooplankton. Nevertheless, toxicity of phytoplankton compounds has generally been determined against vertebrates rather than zooplankton, with effects on zooplankton being uncertain. Zooplankters sometimes prefer, or perform better on, nontoxic versus toxic species or clones (e.g., Huntley et al., 1986; Teegarden, 1999). However, correlations such as these can be confounded by other microalgal characteristics, including differences in morphology, nutritional value, and chemicals other than known neurotoxins. Purified phytoplankton compounds added to seawater containing copepods have occasionally been found to negatively affect zooplanktonic grazers (e.g.,

Miralto et al., 1999), although it is unlikely that this method adequately represents the consumption of compounds as part of a natural diet. Rigorously documenting how zooplankters are affected following consumption of particular compounds may demand further technological developments. Despite uncertainty regarding the effect of phytoplankton metabolites on zooplankton behavior and physiology, a handful of studies have shown that some zooplankton (e.g., copepods) do accumulate phytoplankton metabolites such as brevetoxins (Tester et al., 2000). Thus, zooplankton provide another route whereby phytoplankton toxins can enter the food web and reach higher consumers such as fishes, whales, and humans. If zooplankters avoid feeding on phytoplankters due to their production of specific compounds or selectively graze on nontoxic phytoplankton (favoring the proliferation of toxic phytoplankton), then phytoplankton compounds may play a crucial role in the initiation and persistence of large-scale harmful algal blooms.

Additionally, toxic microalgae may proliferate due to the allelopathic effects of their excreted metabolites on competing microalgae. For example, in cross-culturing experiments, medium preconditioned by the benthic dinoflagellate *Prorocentrum lima* reduced growth of diatoms (Windust et al., 1996) and other epiphytic dinoflagellates (Sugg and VanDolah, 1999). Windust et al. (1996, 1997) reported that okadaic acid, a product of *P. lima* that inhibits protein phosphatases, depressed diatom growth, and they proposed that less toxic sulfated and diol esters are rapidly biotransformed to okadaic acid upon release from *P. lima* cells and/or upon contact with competing cells. *P. lima* was found to be considerably less sensitive to okadaic acid than were other microalgae. In contrast, Sugg and VanDolah (1999) showed that although protein phosphatase activity of competing dinoflagellates was inhibited by *P. lima* exudates and by pure okadaic acid, reduced growth of competitors was associated with a nonokadaic acid fraction.

PREY RESPONSES TO CHEMICAL CUES FROM CONSUMERS

Many marine and freshwater zooplankton undergo diel vertical migration (DVM), usually living at darker depths during the day and feeding in surface waters at night when their visually foraging predators are less active. This behavior is cued by chemical detection of zooplankton predators (De Meester et al., 1999). DVM apparently lessens zooplankton susceptibility to consumers, but the (as yet unknown) compounds mediating this behavior also affect biogeochemical processes systemwide. As an example, in marine systems, about 15–50% of all metazoan zooplankton biomass above 500 m moves to near the surface at night and migrates to depths of 300–600 m during the day (Longhurst and Harrison, 1988). Given the permanent density discontinuity at 40–200 m in tropical and subtropical oceans, this migration can transport carbon and nutrients below the pycnocline, mediate the alkalinity of surface waters, and influence CO₂ uptake at the surface. As zooplankters migrate from the photic zone to below the pycnocline, assimilated

carbon and nutrients move with them. Some of this stays in the deeper waters due to respiration, excretion, and predation by deep-water consumers. It is estimated that the flux of respired carbon due to DVM equals 13–58% of computed particulate sinking flux across the pycnocline and adds 5–20% to current estimates of global sinking flux of organic carbon across the pycnocline (Longhurst et al., 1990).

Available nitrogen often limits production in the photic zone, and DVM further lowers available nitrogen. Crude calculations indicate that exudation of nitrogen from zooplankton performing DVM may move 21% of the total nitrogen pool below the photic zone (Longhurst and Harrison, 1988). Thus, when zooplankton detect chemical evidence of nearby consumers and initiate diel vertical migrations, their behavior moves significant quantities of both carbon and nitrogen from the photic zone to below the pycnocline, affecting both biogeochemical fluxes and the structure of marine food webs. Given the smaller and more contained nature of lakes versus oceans, these chemically cued processes might produce even stronger effects in stratified freshwater systems. However, because lakes are more prone to climatic disturbance, the stratification of nutrients could be “reset” more frequently.

Just as zooplankton chemically sense their consumers and undertake DVM, some phytoplankton chemically sense zooplankton grazers and respond in adaptive ways. Solitary cells of phytoplankton such as *Scenedesmus acutus* often form colonies that are more resistant to grazing if they are exposed to water conditioned by feeding *Daphnia* (Van Donk et al., 1999). Rather than inducing increased resistance, dinoflagellates use chemical indicators of zooplankton presence to determine when to excyst and emerge from the sediment “seed bank” into the water column. When Hansson (1996) incubated sediment cores in containers with no zooplankton, zooplankton in a cage so that they could not access the emerging phytoplankton, or dead zooplankton (presenting the chemical signals from *Daphnia*, but not their feeding action), a sixfold greater emergence was found in the absence of zooplankton than in either other treatment. In field assays, both *Peridinium wisconsinense* and *P. pusillum* emerged in greater numbers from sediments in lakes with small zooplankton populations than ones with larger populations. Additionally, like zooplankton, some species of mobile phytoplankton also undergo diel vertical migration, often moving to deep waters when their consumers move to shallow waters (Jones, 1993). Thus, (as yet unknown) chemical cues from zooplankters appear to be perceived by microalgae, producing effects on seasonality, succession, and composition of phytoplankton communities (Rengefors et al., 1998).

CHEMICALLY CUED CASCADES OVER THOUSANDS OF KILOMETERS?

Microalgal metabolites that function as defenses against ciliate or zooplankton consumers have the potential to create cascades of interactions that span

hundreds or thousands of kilometers and involve species and interactions that are far removed from the initial participants. One of the most interesting examples of this is the cascading effect that phytoplankton-generated dimethylsulfide (DMS) and acrylate appear to have on ciliates, zooplankton, fishes, sea birds and, potentially, desert plants, desert animals, and global weather patterns.

Many grazers selectively avoid, or less heavily utilize, phytoplankton strains that have high potential to cleave dimethylsulfoniopropionate (DMSP) enzymatically to DMS and acrylate when ingested by grazers (Figure 1) (see review by Wolfe, 2000). This potential appears to be regulated more by levels of the enzyme DMSP lyase than by levels of DMSP. The biochemical nature of the variance in DMSP lyase is not known, but consumers of ciliates and dinoflagellates avoid strains with high ability to create DMS and acrylate from DMSP, relative to strains with lesser ability to do this. DMS should rapidly diffuse from consumers, potentially providing a cue for other organisms. Acrylate sterilizes consumer guts and cascades through food chains from common phytoplankton such as *Phaeocystis*, to dominant zooplankton like euphausiids, and finally to higher level consumers such as penguins, with antimicrobial effects in the guts of both the primary and secondary consumers (Sieburth, 1960).

In areas of high productivity, significant quantities of DMS are released into surface waters, held in benthic pore waters beneath areas of high productivity, and released into the atmosphere (Andreae, 1985). There are numerous examples of atmospheric DMS being elevated three- to sixfold over background levels near productive areas such as polar frontal zones. Thus, DMS is often associated with areas of high productivity, areas where planktivores are feeding, areas of increased krill density, and areas where fishing success by birds is likely to be greater.

There is mounting evidence that certain species of sea birds with strong olfactory capabilities use DMS as a foraging cue and that the use of an area by these birds sets up a cascade of increased use by other birds that do not have olfactory systems that are as highly developed. Nevitt (2000) provides a convincing overview of this topic. Certain tube-nosed seabirds (procellariiformes) have among the largest olfactory bulbs of any birds and forage over hundreds and even thousands of kilometers of featureless ocean searching for patchily distributed foods such as krill, squid, and fish. Field tests have shown that these birds are attracted to fishy odors (cod liver oil, tuna oil, fish homogenate) and that a specific subset of the smaller and more cryptic species that have especially strong olfactory capabilities are attracted to DMS dispersed in either water or air (Nevitt et al., 1995). For many organisms that migrate over long distances and use odors as cues, the notion of homing to odors produced hundreds or thousands of kilometers away seems unlikely due to turbulence disrupting odor plume gradients and due to dilution dropping signals below detection limits. Nevitt notes that DMS signals from damaged phytoplankton can persist for hours to days and suggests that birds use DMS associated with upwelling areas, seamounts, or other productivity

discontinuities, as olfactory guideposts to aid navigation to general fishing areas. Once in productive areas, they may use more localized searching strategies.

A second strategy for these birds is to fly directly from the colony to feeding grounds that are hundreds or thousands of kilometers away. To do this, procellariiformes may rely on spatial memory, experience, olfactory guideposts, or other navigational cues associated with persistent olfactory signals along bathymetric features. Numerous species of albatross and the white-chinned petrels seem to use this strategy, flying directly to distant feeding grounds without foraging along the way. These feeding grounds are not constant, but can fluctuate in space and time; therefore, birds may start area-restricted foraging only after changes in olfactory cues indicate a productive fishing area (Nevitt, 2000).

On research cruises where DMS and bird densities were recorded, blue petrels and prions were significantly associated with areas where DMS levels were highest (Nevitt, 2000). DMS is a predictor of krill grazing, and petrel and prion diets are composed predominantly of crustaceans, including krill, amphipods, and copepods. Surveys of areas with high, medium, and low DMS levels indicated that prions were the primary users of high DMS level sites. At medium- and low-concentration sites, prions were present, but there were also approximately 15 additional species of birds. This pattern suggests that high DMS sites may be new patches that erode over time. It seems possible that prions use superior olfaction to find sites first and specialize on these until competing birds, or predatory birds, become common. Later-arriving species seem to be less sensitive to DMS signals and may visually cue on foraging prions to find more productive sites. This is consistent with findings that smaller, more cryptic species such as prions and storm-petrels respond strongly to DMS-scented slicks or aerosols, whereas larger, more visible species do not (Nevitt et al., 1995). This process may link local production of phytoplankton chemical cues to abundant zooplankton and fishes, and then to birds and terrestrial systems that are hundreds of kilometers distant. As seabirds move marine biomass and nutrients to nesting sites on distant terrestrial habitats, they are providing nutrient and energy subsidies to these terrestrial systems. Such marine inputs can fundamentally alter resource-starved terrestrial systems by producing demonstrable, and often dramatic, increases in the growth rates or densities of organisms as diverse as desert cacti, coyotes, beetles, and spiders (Polis and Hurd, 1996; Rose and Polis, 1998; Sanchez-Pinero and Polis, 2000). The DMS affecting this transfer of energy and nutrients can even be argued to produce broader-scale effects, in that atmospheric oxidation products of DMS are important in the formation of aerosols in the lower atmosphere, affecting cloud formation, altering the earth's albedo, and impacting global heat balance (Charlson et al., 1987).

The cross-ecosystem effects of aquatic chemical cues that we describe here should not be unique to the plankton. Such interactions probably occur in numerous systems, but are simply underinvestigated. Leaves falling from trees into

forest streams are one of the primary energy sources for these streams, but little is known about how specific metabolites contained in these terrestrial leaves affect aquatic systems in terms of rates of degradation, use by shredding insects, or stream food webs and biogeochemical cycles in general. Similarly, when large masses of chemically defended seaweeds are torn from tropical reefs and deposited on deep sand or mud plains, do the metabolites in these tissues affect microbial processes, biogeochemical cycles, or the species normally using these sedimentary environments? Chemical signals directed at target organisms in one community may commonly affect nontarget species in other systems—this possibility seems worthy of further attention by experimentalists.

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