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Food Web and Ecosystem Impacts of Harmful Algae

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7.1 Introduction

This chapter considers food web and ecosystem-level impacts of major groups of harmful algae in U.S. and nearby waters, including high-biomass "blooms" (outbreaks) of nontoxic, allelopathic, or toxic algae that are visually noticeable and discolor the surrounding area (water or substrata); allelopathic algae in smaller populations whose bioactive substances have major impacts on other organisms; and low-biomass outbreaks of highly toxic microalgae can cause adverse effects on food webs at low cell concentrations without visible water discoloration. The cyanobacteria (blue-green algae) are included here, based on their major ecological role as primary producers (Burkholder, 2002, 2009 and references therein). Routes of exposure to harmful algae can be *direct*, via consumption of toxic algae, cell surface contact (sometimes causing physical damage), or exposure to algal toxins (e.g., in exudates or in the surrounding water), or *indirect* through related habitat degradation (e.g., oxygen deficits, shifts in community composition), transfer of toxins across trophic levels (bioaccumulation, biomagnification), or consumption of toxin-laced prey (e.g., Figures 7.1 and 7.2). Food web effects from exposure to harmful algae are also sustained by early life stages of grazers, with ramifications for recruitment and, therefore, future generations.

Harmful algal blooms (HAB) cause a "vast array" (Landsberg, 2002) of food web effects across multiple trophic levels. Surprisingly, such blooms have seldom been implicated in modifying trophic cascades, but there is increasing recognition that they do so through many mechanisms (Shumway *et al.*, 2003; Karjalainen *et al.*, 2007; Kvetik and Bretz, 2004, 2005; Branch, 2008; Casini *et al.*, 2008). They disrupt ecosystem function by decreasing biodiversity and altering energy flow within food webs, and they also cause low oxygen stress and/or destroy important habitats such as submersed vegetation meadows (below).

Research emphasis on impacts from harmful algae overwhelmingly has been anthropocentric. The published literature contains a wealth of information about effects on mammals (e.g., mice and rats as surrogates for humans) and modes of action of major classes of microalgal toxins, especially microcystins (MCs) from mostly freshwater cyanobacteria; saxitoxins (STXs), brevetoxins (BTXs), ciguatoxins, dinophysistoxins, and other toxins from marine dinoflagellates; and domoic acid (DA) from a small group of marine diatoms. Not until the studies by Shumway and colleagues in the early 1980s was there a focus on impacts of these toxins on invertebrates and other lower organisms (see Basti *et al.*, 2018 – Chapter 4). Moreover, nearly all of what is known about food web impacts from *toxigenic* (potentially toxic) microalgae is piecemeal, based on acute toxicity tests with one to a few strains (populations) of fauna or flora (other microalgae and macroalgae, aquatic vascular plants – Table 7.1).

These tests often have been conducted with purified toxins rather than with the toxic microalgae themselves. Use of purified toxins in such

Harmful Algal Blooms: A Compendium Desk Reference, First Edition. Edited by Sandra E. Shumway, JoAnn M. Burkholder, and Steve L. Morton.

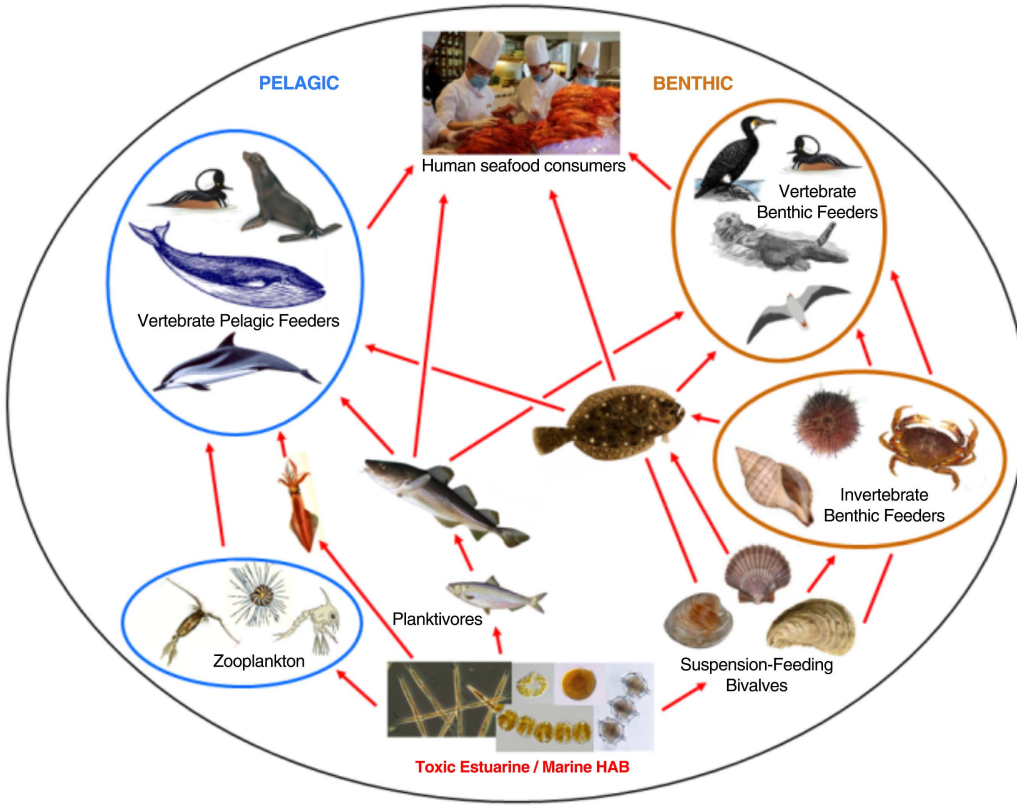

Figure 7.1 Simplified conceptual diagram showing how toxins from toxic algae can move through both pelagic and benthic communities of food webs, here depicting a north temperate marine coastal region. Note that arrows from the higher trophic levels in both pelagic and benthic routes can involve the same species, such as various ducks. Not shown are other complexities, such as when squid consume other squid, or squid eat fish, or invertebrate benthic feeders consume small flounder and small birds, etc.

research is important for verification purposes, but the data are difficult to extrapolate to the impacts of toxigenic algae in their natural setting (e.g., DeMott and Dhawale, 1995; Lürling and van der Grinten, 2003; Zurawell *et al.*, 2005; and see below). While there is ample evidence that *chronic/sublethal* effects of toxigenic microalgae are much more pervasive and ecologically important than acute impacts (Table 7.2), such effects generally are much more difficult to track or quantify (Burkholder, 1998). Mortality of aquatic fauna has been emphasized and (for cyanobacteria) of terrestrial animals of human interest such as livestock, based on acute exposures to selected purified toxins from harmful microalgae (Table 7.1).

Even less common than food web studies that include chronic/sublethal effects from harmful algae are *ecosystem-level analyses* of their impacts. High-biomass blooms can be more easily linked to ecosystem-level analysis than low-biomass outbreaks of some toxigenic algae, because high-biomass blooms often cause hypoxia or anoxia and some obvious, pervasive impacts across trophic levels (Figure 7.2). Thus, although it has been recognized that many HAB severely alter or degrade ecosystem function (Sunda *et al.*, 2006), the actual number of ecosystem-level analyses in the published literature remains very small (e.g., Landsberg *et al.*, 2009).

In this chapter, *food web impacts* are loosely defined as effects (mostly negative, but also sometimes positive) on organisms from one or more trophic levels. An overview is also provided of the present status of *ecosystem-level impacts*, considered as effects on the entire food web structure. Future directions are suggested to fill some major gaps in scientific understanding about influences of harmful algae on food webs and ecosystems from both ecological and economic perspectives.

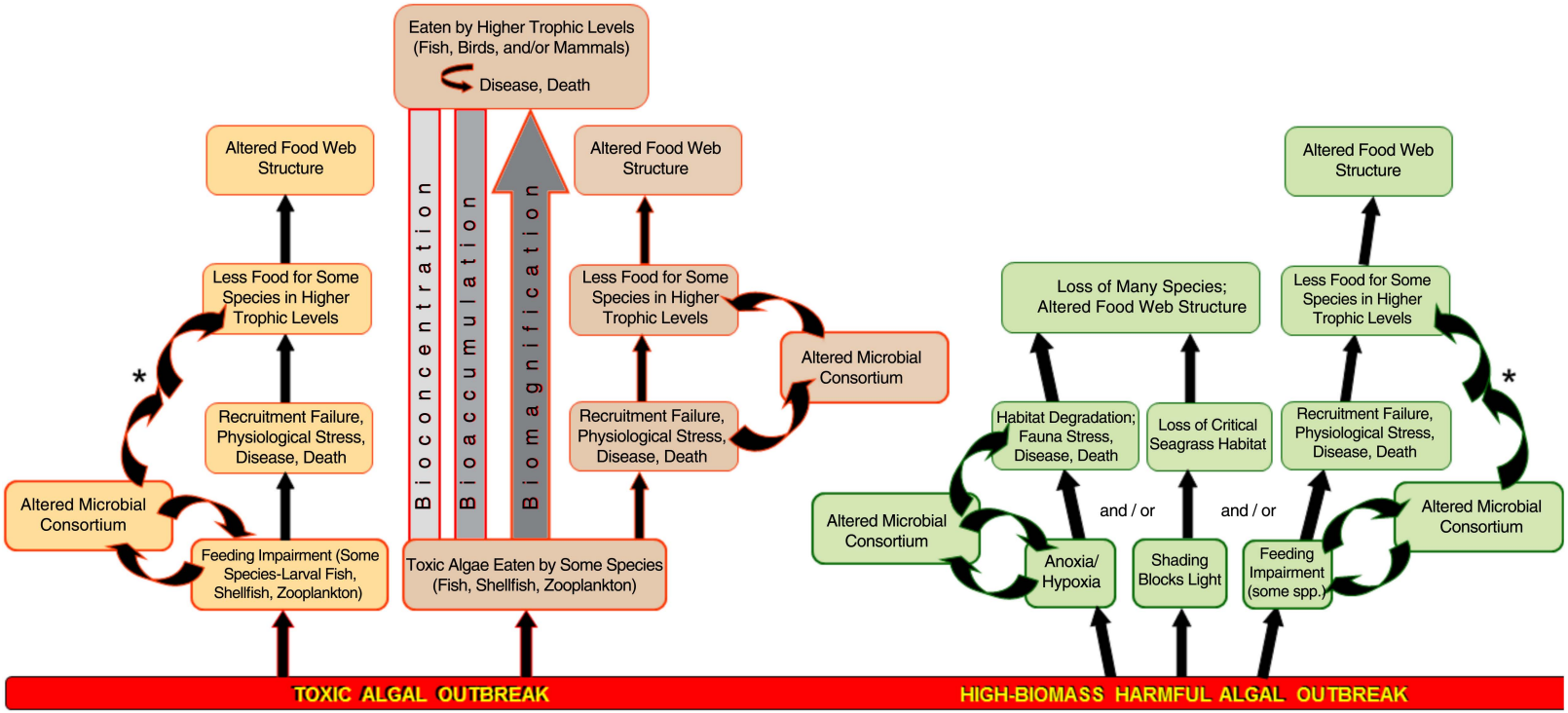

Figure 7.2 A conceptual model of food web effects and ecosystem-level changes from a toxic algal outbreak (bloom; left [gold boxes] and center [brown boxes], with toxin involvement or movement indicated by boxes and arrows, respectively, with red outlines) versus a high-biomass harmful algal outbreak (bloom; right, green boxes). Asterisk (*) indicates that multiple steps can be involved.

Left (gold boxes): Impacts that can occur, depending on the toxin(s), concentration(s), species, and life stage/physiological condition, when grazers avoid consuming the toxic algae or when feeding is inhibited.

Central (brown boxes): Impacts that can occur when toxic algae are eaten by grazers. Three processes (in shades of gray with red outline) involving the toxin(s) can occur depending on the organisms, their life stage/physiological condition, and the toxin congener(s)/concentration (from Gray, 2002; also see Doucette *et al.*, 2006b): *Bioconcentration* refers to uptake of the toxin directly from the water, resulting in a higher concentration of the toxin in the organism than in the water. *Bioaccumulation* is a process that causes an increased chemical concentration in an aquatic organism compared to the water, due to uptake by all exposure routes. *Biomagnification* is the process of transfer of the toxin from food to an organism, resulting in a higher concentration in the organism than its toxic food item. The overall result is a much higher concentration of the toxin as it moves up the food web to organisms at higher trophic levels. Note that one or more of these processes may be operable, depending on the toxin and the organisms.

Right (green boxes): Impacts that can occur from a high-biomass harmful algal bloom, here depicted as not involving toxin production.

In situations where toxic algae form high-biomass blooms, all of these pathways and impacts can occur. Not shown are all of the pathways involving toxic algae as mixotrophs consuming a wide array of other microbial prey (see Jeong *et al.*, 2010).

Table 7.1 Aquatic biota that have been killed by blooms of some selected harmful algae in U.S. and neighboring waters, emphasizing toxigenic microalgae but also including a few examples of otherwise-harmful microalgae and filamentous macroalgae as noted (Lapointe *et al.*, 2018 – Chapter 15). Note that the major algal group is indicated where the genus is first mentioned. This table is not meant to be complete but, rather, to provide examples of food web effects.

Taxon (taxa) and organisms killed
Cyanobacteria (blue-green algae, Cyanophyta)[a]
General: Cyanotoxins can bioaccumulate (Ettoumi *et al.*, 2011 and references therein).
Anabaena flos-aquae [F; T (strain-dependent), low DO]: MCs, anatoxins (also see impacts under *Microcystis aeruginosa*/other MC producers). From Schwimmer and Schwimmer (1968 and references therein), unless otherwise noted:
Mammals: Various wild animals – foxes, squirrels, mink, muskrat, skunks; domestic – cats, cows, dogs, horses, sheep, swine (also see Rose, 1953; Moore, 1977).
Birds: Wild birds – ducks, Franklin's gulls, coots, pheasants, hawks, herons, songbirds; domestic – chickens, ducks, turkeys (also see Rose, 1953; Moore, 1977).
Reptiles: Snakes.
Amphibians: Salamanders.
Fish: Buffalo fish, carp, black bullhead [low DO?]; perch (also see Rose, 1953; Hammer, 1968).
Aphanizomenon flos-aquae [F; T (strain-dependent), low DO] – Aphantoxins, CYL, STXs[b] (also see impacts under *Cylindrospermopsis raciborskii*, other CYL producers)
Mammals: Domestic cattle (Nelson, 1903, in Fitch *et al.*, 1934).
Amphibians: Tadpoles (Farrão-Filho and Kozlowski-Suzuki, 2011 and references therein).
Fish:
Massive fish kill following collapse of blooms in eutrophic lakes (Barica, 1978).
American eel, black bullhead, black crappie, bluegill sunfish, buffalo, carp, hog sucker, northern pike, perch, suckers, yellow pike (Mackenthum *et al.*, 1948).
Cylindrospermopsis raciborskii[c] (F; T [strain-dependent]: CYL, deoxy-CYL, STX, anatoxin; Zagatto *et al.*, 2012)
Mammals: Domestic cattle (Saker *et al.*, 1999).
Fish: Zebrafish larvae (Zagatto *et al.*, 2012).
Amphibians: Cane toad (White *et al.*, 2007).
Cnidarians: Larvae of the coral *Acropora surculosa* (Kuffner and Paul, 2004).
Arthropods: Brine shrimp (Metcalf *et al.*, 2002).
Zooplankton: *Daphnia magna* (Nogueira *et al.*, 2004).
Daphnia similis, Ceriodaphnia dubia (Zagatto *et al.*, 2012).
Lyngbya majuscula[c] [M, benthic macroalga; T (strain-dependent): aplysiatoxin, debromaplysiatoxin, kalkitoxin, LYNGTX; B = ~80 other biologically active chemicals (strain-dependent) – Wu *et al.*, 2000; Osborne *et al.*, 2001; Le Page *et al.*, 2005; Taylor *et al.*, 2014]
Fish: Goldfish (Wu *et al.*, 2000: toxin assays).
Arthropods:
Blue shrimp (raceway-reared); necrosis of the lining of the epithelium of the midgut, dorsal caecum, and hindgut, and hemolytic enteritis (Lightner, 1978; Osborne *et al.*, 2001).
Brine shrimp (Wu *et al.*, 2000: toxin assays).
Cnidarians: Larvae of staghorn coral, *Acropora surculosa*; more generally, scleractinian corals and gorgonians (Kuffner and Paul, 2004).
Seagrasses: Round-leaf seagrass, shoalgrass (Watkinson *et al.*, 2005; Tilling, 2007).
Red algae (Rhodophyta): Crustose coralline macroalgae (Kuffner and Paul, 2004 and references therein).
Microcystis aeruginosa (and various other ***Microcystis*** spp., and other MC producers.[b]) [Planktonic; can also be benthic; F, Br; T (strain-dependent) and low DO. MCs, cyanoginosins, cyanoviridin, LPSs, BMAA, anatoxin-a, unidentified volatile sulfur compounds (Fristachi and Sinclair 2008; Rastogi *et al.*, 2014 and references therein).] From Schwimmer and Schwimmer (1968), Codd (1995), and Codd *et al.* (1996) unless otherwise noted:

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Mammals: Southern sea otter (Miller *et al.*, 2010); domestic – cattle (Puschner *et al.*, 1998), dogs, horses, pigs, sheep.
Birds: Heron, snipe, ducks, geese, pheasants; domestic – chickens, ducks, geese, turkeys; waterfowl may be at higher risk because they feed on floating cyanobacterial scums (also see Ibelings and Havens, 2008).
Fish: Major fish kills; toxin-induced mortality of embryos of loach (Liu *et al.*, 2002), medaka (Jacquet *et al.*, 2004), and zebrafish (Oberemm *et al.*, 1999), and of juvenile carp (Osswald *et al.*, 2007).
Arthropoda: Crustacea other than zooplankton – Brine shrimp (Akin-Oriola and Lawton, 2006).
Zooplankton:
Microcrustaceans – *Daphnia ambigua* (Fulton and Paerl 1987), *D. galeata* (Rohrlack *et al.*, 1999), *D. magna* (Nizan, 1986), *D. parvula* (Fulton, 1988b), *Eucypris virens* (Stangenberg, 1968), *Monia macrocopa* (Yasuno and Sugaya, 1991), *M. micrura* (Liu *et al.*, 2006).
Rotifers – *Brachionus calyciflorus*: by consuming toxic cells, or by passive uptake of dissolved toxin from the surrounding medium (Starkweather and Kellar, 1987; Zhao *et al.*, 2014; but conflicting information depending on the *M. aeruginosa* strain and other factors). *Brachionus rubens* (Rothhaupt, 1991).
Benthic member(s) of the order Stigonematales (e.g., epiphyte of *Hydrilla*) [F; T: BMAA (Corbell *et al.*, 2014)][d]
Birds: Via avian vacuolar myelinopathy, mostly in bald eagles and American coots, from an uncharacterized cyanobacterial neurotoxin.[d]
Synechococcus (with ***Synechocystis* sp.** – Richardson, 2004) [T: BMAA (Cox *et al.*, 2005; also see Martins *et al.*, 2005); cultures produced substances with neurotoxic and hepatotoxic effects – Brand *et al.*, 2010; EDAB][e]
Crustaceans: Juvenile spiny lobsters (via loss of sponge habitat; Butler *et al.*, 1995).
Cnidarians: Reef-building corals on patch and bank reef ecosystems (e.g., *Montastrea annularis, Porites porites*; Tomascik and Sander, 1985, 1987).
Poriferans: Branch candle sponge, gray-purple sponge, loggerhead sponge, sheepswool sponge, stinker sponge, and vase sponge – implicated in mass mortalities (Tomascik and Sander, 1985, 1987; Lapointe and Clark, 1992; Butler *et al.*, 1995; Diersing, 2009 and references therein).
Trichodesmium spp. (e.g., *T. thiebautii*, *T. erythraeum*) [M, T (strain-dependent): ciguateratoxin-like activity and saxitoxins-like properties (neurotoxicity with paralytic symptoms) – Kerbrat *et al.*, 2010; trichamide, a cyclic peptide produced by senescing blooms, may have mild cytotoxicity and likely is used in anti-predation defense – Sudek *et al.*, 2006]
Zooplankton: *Macrosetella gracilis* (O'Neil and Roman, 1994), *Penaeus merguiensis* (Preston *et al.*, 1998).
Copepods: *Clausocalanus furcatus, Farranula gracilis* – Highly neurotoxic extracts from *T. thiebautii* blooms (but these species are not reported to consume *T. thiebautii* – Hawser *et al.*, 1992). *Note*: The natural grazers (*Macrosetella gracilis* and *Miracia efferata*) were not affected, suggesting that they may have developed resistance to the toxins.
Diatoms (Heterokontophyta – Bacillariophyceae)
Chaetoceros concavicornis (sometimes with ***C. convolutus***) [M: Str]
Fish:
Atlantic salmon (cultured in net pens). The primary mechanism appears to be reduction of gas exchange caused by mucus production when the gill epithelium is irritated by *Chaetoceros* setae wedged between the secondary lamellae.
Chinook salmon, Coho salmon, rainbow trout (Landsberg, 2002 and references therein).
Crustacea: Implicated in mortality of juvenile red king crabs, as *Chaetoceros* cells and setae were the dominant component of the debris in the mucus covering of damaged crab gills (Horner *et al.*, 1997 and references therein).
Toxic *Pseudo-nitzschia* complex [M; T (strain-dependent): DA, bioaccumulates]
Pseudo-nitzschia australis
Mammals: California sea lion, sea otter; multiple species of cetaceans and pinnipeds; minke whale; baleen whale, wherein DA was detected in the urine, gastric fluid, and feces. More than 560 otoliths from northern anchovies were identified from a whale's stomach, indicating recent feeding on that toxin vector prior to death (Scholin *et al.*, 2002; Fire *et al.*, 2010; Lewitus *et al.*, 2012 and references therein).
Birds: Brandt's cormorant, brown pelican, double-breasted cormorant, western gull (Fritz *et al.*, 1992; Work *et al.*, 1993).

(*continued*)

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Dinoflagellates (Dinophyta)
Akashiwo sanguinea[c] [M; B (strain-dependent); surfactant-like protein exudates, copious mucilage (Jessup *et al.*, 2009); T? (ROS hydrogen peroxide – Kim *et al.*, 1999); low DO – Mohamed and Al-Shehri, 2012; Badylak *et al.*, 2014a, 2014b and references therein]
General: Blooms have been the suspected cause of fish kills and marine mammal strandings (Badylak *et al.*, 2014a, 2014b).
Birds: Clark's grebe, common murre, northern fulmar, Pacific loon, red-throated loon, surf scoter, western grebe, white wing scoter; widespread seabird mortality (Landsberg, 2002 and references therein; Jessup *et al.*, 2009).
Fish:
Decomposition of a major bloom led to oxygen depletion and death of fish and shellfish (Horner *et al.*, 1997).
Anchovy, Atlantic bumper, Atlantic croaker, bluefish, Gulf menhaden, hardhead catfish, inland silverside, sand seatrout, silver perch, southern kingfish, southern stargazer, spadefish, spot, striped mullet, thread herring, threadfin (Harper and Guillen, 1989).
Molluscs:
Japanese littleneck clam, Olympia oyster, Pacific oyster – Larval stages (Shumway, 1990 and references therein).
Abalone – Larvae exposed to *A. sanguinea* blooms (Botes *et al.*, 2003).
Crustacea: Blue crab (Harper and Guillen, 1989).
Phytoplankton: *A. sanguinea* consumes microalgal prey such as *Alexandrium tamarense, Amphidinium carterae, Heterocapsa triquetra, Prorocentrum minimum*, and *Scrippsiella trochoidea* (dinoflagellates); *Isochrysis galbana* (haptophyte); *Rhodomonas salina* and a second unidentified taxon (cryptophytes); and *Heterosigma akashiwo* (raphidophycean; Jeong *et al.*, 2005a). Also consumes the cyanobacterium *Synechococcus* sp. (Jeong *et al.*, 2005b).
The ingestion rate of unicellular cyanobacteria by *A. akashiwo* was 62.9 ± 5.4 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 standard error [SE]; Jeong *et al.*, 2005b).
Toxic *Alexandrium* complex [M; T (strain-dependent): STX and derivatives, which can bioaccumulate (e.g., Doucette *et al.*, 2006a)]
Alexandrium catenella [T: STX and derivatives, ROSs – Dorantes-Aranda *et al.*, 2015]
Fish: Post-smolt Atlantic salmon (Aguilera *et al.*, 2016).
Molluscs: Blue mussel, California mussel, eastern oyster, European flat oyster, gaper clam, littleneck clam, Nuttall's cockle, Olympia oyster, Pacific oyster, rock scallop, spiny scallop, weathervane scallop (Shumway, 1990).
Phytoplankton: The ingestion rate of unicellular cyanobacteria by *A. catenella* was 29.5 ± 6.7 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE). The ingestion rate by *A. minutum* was much lower, 3.2 ± 2.2 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (Jeong *et al.*, 2005b).
Alexandrium fundyense [T – Bricelj *et al.*, 1990]
Fish:
Newly settled winter flounder, larval sheepshead minnow, and larval mummichug, after they consumed six or more toxin-contaminated copepod zooplankters (Samson *et al.*, 2008).
Shortnose sturgeon (mass mortality; high STX concentrations in stomach contents, liver, and gill tissues; Fire *et al.*, 2012).
Molluscs:
Eastern oyster, Pacific oyster: caused immunosuppression of hemocytes and increased hemocyte death (Hégaret *et al.*, 2007a).
Softshell clam: Depressed phagocytosis and adhesion (Hégaret *et al.*, 2007a).
Zooplankton: The copepod *Acartia hudsonica*, when *A. fundyense* was 25% or more of the diet by carbon content, and only 20–30% of males survived to adulthood. Males were more susceptible than females. Differential mortality and skewed sex ratios acted as feedback mechanisms that potentially can affect the population dynamics of grazers and toxic algal bloom development (Avery *et al.*, 2008).
Alexandrium monilatum[c] [T (strain-dependent): STXs, gonyautoxins (Schmidt and Loeblich, 1979), goniodomin A (Hsia *et al.*, 2005); bioaccumulate]
Fish: General (fish kills), crested blenny, jack species, needlefish, pinfish, sheepshead minnow, stippled clingfish, whip eel, whiting (Wardle *et al.*, 1975; Gunter, 1942; Connell and Cross, 1950; Howell, 1953; Gates and Wilson, 1960; Williams and Ingle, 1972).
Invertebrates (general): Ichthyoplankton, zooplankton (International Council for the Exploration of the Sea, 1999).

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Molluscs:
Atlantic surfclam, auger snail, Brazil arc, calico crab, double moonshell, dwarf crab, eastern oyster, Florida coquina, Florida dogwinkle, gray augur, green mussel, green porcelain crab, hermit crab, hooked mussel, lettered olive, northern quahog (juveniles), shark eye, spotted porcelain crab, striped false limpet, stone crab, surfclam (Wardle *et al.*, 1975).
Larval eastern oyster, northern quahog (May *et al.*, 2010).
Veined rapa whelk (in association with a bloom of *A. monilatum*; eastern oysters and northern quahogs in the same flow-through system apparently were unaffected). External signs of stress included reduced ventilation, inability to attach to hard substrata, periodic pumping of the opercular plate, and increased mucus production over a 24- to 48-hour period prior to death. High concentrations of toxin goniodimum A were measured in bivalves attached to rapa whelk shells (Harding *et al.*, 2009).
Annelids: Polychaetes *Americonuphis magna* and *Nereis* sp. (Wardle *et al.*, 1975).
Crustacea:
Blue crab, calico crab, dwarf crab, Florida stone crab, green porcelain crab, saltwater porcelain crab, sand flea, speckled swimming crab, surf hermit, thinstripe hermit crab (Wardle *et al.*, 1975).
Daggerblade grass shrimp (*note*: not affected – depressed mud crab, ivory barnacle) (Sievers, 1969).
Cnidarians: Sea anemone *Bunodosoma cavernata* (Wardle *et al.*, 1975).
Echinoderms: Brittle star, six keyhole sand dollar, holothuroids (sea cucumbers) (Wardle *et al.*, 1975).
Alexandrium tamarense [T (strain-dependent) – major STXs: C2, gonyautoxin 4, neosaxitoxin and gonyautoxin 3 bioaccumulate (Shimada *et al.*, 2011 and references therein); also ROS hydrogen peroxide – Kim *et al.*, 1999]
Mammals: Humpback whale (Geraci *et al.*, 1989).
Birds: Black duck, black-legged kittiwake, common tern, other terns, gulls, shag, shorebirds, waterfowl (Shumway *et al.*, 2003 and references therein).
Fish: Larval and adult Atlantic herring; bluefish, larval capelin monkfish, sand lances (sand eels), skates, spiny dogfish (White, 1980; Smayda, 1991 and references therein).
Molluscs: Atlantic deep-sea scallop (juveniles – Lesser and Shumway, 1993), blue mussel (Desbiens *et al.*, 1989), softshell clam (juveniles – Lesser and Shumway, 1993).
Zooplankton:
Ciliates – *Favella ehrenbergii* (with toxic strain; Hansen, 1989); *Favella taraikaensis* and *Eutintinnus* sp. (with toxic strain; Fulco, 2007).
Heterotrophic dinoflagellates – Lysis of *Polykrikos kofoidii* occurred when fed (presumed toxic) *A. tamarense* (Cho and Matsuoka, 2000).
Phytoplankton:
A. tamarense consumes *Isochrysis galbana*, *Rhodomonas salina* and an unidentified cryptophyte taxon, *Heterosigma akashiwo*, *Amphidinium carterae*, and *Prorocentrum minimum* (Jeong *et al.*, 2005a).
The ingestion rate of unicellular cyanobacteria by *A. tamarense* was 13.7 ± 0.9 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).
Ceratium tripos (high-biomass bloom) [M; low DO]
Fish: Fish kills (anoxic conditions, hydrogen sulfide at lethal levels over large areas, e.g., 14,000 km^2); massive mortalities of finfish (Mahoney, 1978 and references therein; Mahoney and Steimle, 1979).
Molluscs: Ocean quahog, sea scallop, surfclam (Mahoney and Steimle, 1979; anoxic conditions were also associated with the bloom).
Crustacea: American lobster (Mahoney and Steimle, 1979).
Cochlodinium polykrikoides (or *Margalefidinium polykrikoides* – Gómez *et al.*, 2017) [Br, M; T (strain-dependent), but toxin(s) uncharacterized (Dorantes-Aranda *et al.*, 2010); toxicity to fish could be caused by non-hydrogen peroxide, highly reactive labile toxins such as other ROS-like chemicals: Tang and Gobler, 2009a; Kim *et al.*, 2002, but see Onoue and Nozawa, 1989 (zinc-bound paralytic shellfish poisoning toxins)].

(*continued*)

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed

Fish:

In bioassays, sheepshead minnows (1 week old), adult striped killifish and silverside. Moribund fish had epithelial proliferation of gills with focal areas of fusion of gill lamellae, suggesting impaired gill function (Gobler *et al.*, 2008; also see Tang and Gobler, 2009a).

Cyprinodon variegatus (age 1 week); adult *Fundulus majalis, Menidia menidia,* and *Fundulus heteroclitus* (Gobler *et al.*, 2008).

Molluscs:

Bay scallop, eastern oyster, and northern quahog larvae (Tang and Gobler, 2009b); eastern oyster larvae (Ho and Zubkoff, 1979: as *Cochlodinium heterolobatum*).

Juvenile bay scallop, eastern oyster (Gobler *et al.*, 2008).

Bay scallop larvae, eastern oyster: Moribund animals exhibited hyperplasia, hemorrhaging, and apoptosis in gill and digestive tissues, with gill inflammation specifically associated with areas containing *C. polykrikoides* cells (Gobler *et al.*, 2008).

Zooplankton: Female copepods (*Acartia tonsa*) at low *C. polykrikoides* densities; both male and female *A. tonsa* at bloom densities. Earlier nauplii stages were most susceptible (Jiang *et al.*, 2009).

Phytoplankton:

C. polykrikoides consumed an unidentified cryptophyte: The maximum ingestion rate was 9.4 cells grazer^{-1} day^{-1}, and the calculated grazing coefficients ranged from 0.001 to 0.745 hour^{-1}; up to 53% of cryptophyte populations were removed by *C. polykrikoides* in 1 hour. The data suggest that *C. polykrikoides* can have considerable grazing impact on cryptophytes (Jeong *et al.*, 2004a).

C. polykrikoides consumed small phytoplankton prey including *Isochrysis galbana*, *Rhodomonas salina*, *Heterosigma akashiwo*, and *Amphidinium carterae* (Jeong *et al.*, 2004a).

Various phytoplankton species: Allelopathic; cell lysis (Fistarol *et al.*, 2004; Ma *et al.*, 2009, 2011); distortion of cell shape, loss of motility, death (Tang and Gobler, 2010).

The ingestion rate of unicellular cyanobacteria by *C. polykrikoides* was 38.7 ± 1.1 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).

Bacteria: Maximum ingestion and clearance rates by *C. polykrikoides* were 17.4 bacteria and 1.0 nL algal cell^{-1} hour^{-1}, respectively (Seong *et al.*, 2006).

Gambierdiscus toxicus [M; T (strain-dependent): CTXs, bioaccumulate in fish – Lewis, 1992; Naar *et al.*, 2007]

Mammals: Hawaiian monk seal (CTXs in the liver, muscle, and brain of five dead stranded animals – Dechraoui *et al.*, 2011).

Fish: Medaka (experimental data); the dose response for lethal adverse effects was within the range of the CTX load found in adult reef fish (Edmunds *et al.*, 1999).

***Hematodinium perezi*, *Hematodinium* spp.** [F, Br, M; Par]

Arthropods: Crustacea – blue crab, Tanner crab (bitter crab disease or bitter crab syndrome: Horner *et al.*, 1997 and references therein). *Note*: Crabs are often the dominant prey in the diets of various invertebrates, fish, marine mammals.

Karenia brevis[c] [M; T (strain-dependent): BTXs, bioaccumulate (e.g., Naar *et al.*, 2007)]. From Landsberg (2002 and references therein), National Oceanic and Atmospheric Administration (2006), and Gannon *et al.* (2009) unless otherwise noted:

General: The most affected organisms are fish; it is unclear whether invertebrate mortalities are due to BTXs or to hypoxia resulting from the blooms, or both (Landsberg *et al.*, 2009).

Cascading effects, ecosystem level: During intense, protracted blooms (1971, 2005), entire benthic communities over thousands of km^2 (eastern Gulf of Mexico) were killed (Smith, 1975; Dupont *et al.*, 2010). Contributing factors – Unusually high surface water temperatures/strong thermoclines, isolated *K. brevis* populations at depth, fish and invertebrate kills from bloom toxicity and/or hypoxia, increased biochemical oxygen demand from decomposition of dead organisms, increased hydrogen sulfide levels, and decreased light availability at depth (Landsberg *et al.*, 2009).

Mammals:

Bottlenose dolphin, manatee (O'Shea *et al.*, 1991; Bossart *et al.*, 1998; Twiner *et al.*, 2012).

Coyotes, dogs near a bloom (Castle *et al.*, 2013).

Birds: Double-crested cormorant, ducks, frigate birds, gulls, lesser scaup, red-breasted merganser, terns, vultures.

Reptiles: Sea turtles (loggerhead, Kemp's ridley, and green. During two intense *K. brevis* red tides (Feb.–Dec. 2005, Aug.–Dec. 2006), sea turtle strandings were much higher in the affected areas during Jan. 2005–Dec. 2006 (174 in 2005, 144 in 2006) than the 12-year average (43 ± 23, mean ± 1 SE; Fauquier *et al.*, 2013).

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed

Fish (114 taxa): Atlantic bumper, Atlantic menhaden, Atlantic midshipman, Atlantic moonfish, Atlantic needlefish, Atlantic spadefish, balloonfish, bank cusk-eel, barracuda, bay anchovy, belted sandfish, black drum, black grouper, blackcheek tonguefish, black tip shark, bluerunner, bluestriped grunt, catfish, checkered puffer, cobia, cowfish, crevalle jack, damselfish, eel, gafftopsail catfish, gag, gar, goldspotted killifish, grass carp, gray angelfish, gray snapper, gray triggerfish, graysby, groupers, grunts, Gulf flounder, Gulf kingfish, Gulf menhaden, halfbeak, hardhead catfish, harvestfish, hogfish, inshore lizardfish, jacks, jack crevalle, jewfish, ladyfish, lancelet, leatherjacket, leopard searobin, lined sole, longnose batfish, longnose killifish, northern puffer, orange filefish, oyster toadfish, palespotted eel, planehead filefish, perch, pikeperch, pinfish, pompano, porgy, porcupine fish, puffer, purplemouth moray, queen triggerfish, rays, red drum, red grouper, red snapper, redfin needlefish, redfish, round scad, sailfish, sailors choice, sand perch, sand seatrout, scaled sardine, scamp, sharksucker, sheepshead, shiners, short bigeye, shortnose batfish, silver jenny, silver perch, silver trout, snook, sole, sooty eel, southern kingfish, southern seabass, southern stargazer, spadefish, Spanish mackerel, speckled worm eel, spinner shark, spiny boxfish (butterfish), spot, spotted moray (?), spotted seatrout (or speckled trout), striped burrfish, striped mullet, tarpon, thread herring, tidewater silverside, toadfish, tomtate, tripletail, trunkfish, Warsaw grouper, white grunt, yellowtail amberjack.

Chordata: Ascidians (sea squirts).

Invertebrates (general): Mortality of benthic invertebrates, from a combination of BTX neurotoxicity and anoxia due to oxygen depletion from decomposition of dead organisms and respiration of *K. brevis* blooms at night (Simon and Dauer, 1972).

Molluscs:

Bay scallop [mortalities and failed recruitment], bruised nassa, conch, dwarf surfclam (or coot clam), Florida coquina, minor jacknife, oysters, quahogs. In laboratory experiments, larval bay scallop, eastern oyster, northern quahog.

Eastern oyster and northern quahog – When larvae (age 3 days) were exposed to toxic *K. brevis* (10^3 cells mL^{-1}), survival was significantly less in lysed culture than whole culture (Leverone *et al.*, 2006).

Bay scallop, eastern oyster, and northern quahog – Mortality of older larvae (age 7 days) from exposure to 5000 cells mL^{-1} of toxic *K. brevis* (Leverone *et al.*, 2006).

Brachiopod: *Glottidia pyramidatum.*

Echinoderms: Sea urchins.

Poriferans: Sponges (Smith, 1975).

Annelids: *Onuphis eremita oculata*, common clam worm, and polychaetes (*Clymenella mucosa, Diopatra cuprea, Glycera americana, Glycera capitata, Laeonereis culveri, Scoloplos fragilis, Scoloplos rubra, Scolelepis squamata*).

Crustaceans (other than zooplankton) – Barnacles, blue crab, horseshoe crab, lady crab, pea crab, shrimps; amphipods (*Acanthohaustorius* sp.).

Cnidaria: Corals (Smith, 1975).

Phoronids: *Phoronis architecta.*

Zooplankton:

Microcrustaceans

Acartia tonsa – Lethargy, paralysis (Turner *et al.*, 1996).

Calanus pacificus – Reduced development, lethargy (Huntley *et al.*, 1986, 1987); rapid heart rate, paralysis, lethargy, regurgitation (Sykes and Huntley, 1987).

Phytoplankton:

Competitors *Asterionellopsis glacialis (diatom), Prorocentrum minimum*, and *Skeletonema costatum* (diatom), from exposure to extracts from *K. brevis* natural blooms; and these taxa as well as *Akashiwo* cf. *sanguinea* were killed from exposure to extracts from toxic culture (Prince *et al.*, 2008).

The ingestion rate of unicellular cyanobacteria by *K. brevis* was 5.0 ± 0.1 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b), and up to 84 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (Glibert *et al.*, 2009).

Karlodinium veneficum[c] [Br; T (strain-dependent): KTXs, linear polyketides that cause membrane permeabilization in other protists, and interact strongly with certain membrane sterols which form stable complexes with KTSs, increasing ionic permeability of affected membranes (Deeds and Place, 2006); also can act as ichthyotoxins; hemolysins, ROSs (Yamasaki *et al.*, 2004, Van Wagoner *et al.*, 2008 and references therein). Low DO can develop in association with large blooms.]

(*continued*)

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Fish:
Black drum, bluegill sunfish, common snook, grass carp, hardhead catfish, red drum, sheepshead, hybrid striped bass, striped mullet (Deeds *et al.*, 2002, Kempton *et al.*, 2008 and references therein).
Cod – Before death the juvenile fish became lethargic. Affected fish showed increased plasma osmolality; gill tissue showed severe pathological changes, with extensive separation of the respiratory epithelium from the underlying pillar cells (Nielsen, 1993).
Sheepshead minnow, zebrafish – In laboratory experiments, larvae and juveniles exposed to KTXs (Deeds *et al.*, 2006).
Molluscs:
Eastern oyster larvae (Glibert *et al.*, 2007; Stoecker *et al.*, 2008).
Common whelk, lamellibranch bivalve *Lasaea rubra*, and great scallop (Abbott and Balantine, 1957); juvenile Atlantic surfclam, bay scallop, blue mussel, European flat oyster, northern quahog (Lesser and Shumway, 1993).
Crustacea: Blue crab, lady crab, shrimps (Landsberg 2002 and references therein), barnacles (Günter *et al.*, 1948).
Zooplankton: Ciliates – *Favella ehrenbergii* (Hansen, 1995).
Phytoplankton: Consumes cryptophytes; abundant cryptophyte prey can trigger toxic blooms (Adolf *et al.*, 2008). KTXs react strongly with high concentrations of desmethyl sterols in some phytoplankton (e.g., the heterotrophic, herbivorous dinoflagellate *Oxyrrhis marina*), resulting in their death (Adolf *et al.*, 2007).
Lingulodinium polyedrum[c] [M; T (strain-dependent): STXs, yessotoxins; Paz *et al.*, 2004, 2008]. From Landsberg (2002 and references therein) unless otherwise noted:
Fish: Blindfish, California anchovy, dogfish, guitarfish, Haller's round ray, horn shark, red perch, smelt, stingray, thornback guitarfish. Other descriptors: dead bottom fauna including fish and shellfish; fish and shore fauna.
Holothurians: *Trachostoma arenata.*
Molluscs: Limpets, banded pheasant, barrel-bubble, beatic dwarf olive, blister glassy-bubble, California cone, California semele, California tagelus, California venus, Californian beanclam, frilled venus, Gould beanclam, kelp scallop, Mexican pyramidella, moonsnail, octopus, onyx slippersnail, Pacific eggcockle, purple dwarf olive, straight horsemussel, western mud nassa.
Crustacea: Brown rock crab, flat porcelain crab, morbid sand crab.
Phytoplankton:
Consumes microalgal prey such as *Isochrysis galbana*; *Heterosigma akashiwo*; *Rhodomonas salina* and an unidentified cryptophyte taxon; and dinoflagellates *Alexandrium tamarense, Amphidinium carterae, Heterocapsa triquetra, Prorocentrum minimum,* and *Scrippsiella trochoidea*. Its maximum specific growth rates on *P. minimum* or *S. trochoidea* were much higher than in the same light regime without prey. The data suggest that *L. polyedrum* can have a potentially significant grazing impact on some algal prey populations (Jeong *et al.*, 2005a).
The ingestion rate of unicellular cyanobacteria by *L. polyedrum* was 64.2 ± 2.2 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).
***Noctiluca* spp. (e.g., *N. miliaris, N. scintillans*)** [M; allelopathic/ammonia toxicity, NH_3/NH_4^+; Okaichi and Nishio, 1976]
General: Blooms have been linked to massive fish and invertebrate kills (Tirkoglu, 2013 and references therein).
Fish: These heterotrophic dinoflagellates can consume fish eggs and larvae (e.g., anchovy eggs: Hattori, 1962), and produce NH_3/NH_4^+ following deamination of ingested prey; may become progressively more toxic (10-fold range) with more food intake (Okaichi and Nishio, 1976).
Zooplankton: *N. miliaris* consumes copepod eggs of *Acartia clausi, A. tonsa, Calanus euxinus, Centropages hamatus,* and *Temora longicornis* (Kimor, 1979; Daan, 1987; Quevedo *et al.*, 1999; Nikishina *et al.*, 2011) and copepod nauplii (Dela-Cruz *et al.*, 2002), with resulting NH_3/NH_4^+ production as above.
Phytoplankton:
Species with an equivalent spherical diameter (ESD) > 10 µm were consumed as prey; species with an ESD < 5 µm did not support growth. Growth rates of *N. scintillans* fed toxigenic raphidophyceans *Chattonella antiqua* or *Heterosigma akashiwo* increased linearly with prey concentration, without an obvious threshold. This dinoflagellate is well adapted to eutrophic environments (Nakamura, 1998).
Noctiluca spp. may act as vectors of toxigenic microalgae (e.g., *Dinophysis, Pseudo-nitzschia*; Escalera *et al.*, 2007).

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed

In laboratory trials, *N. scintillans* consumed *Dunaliella marina* (small chlorophyte flagellate), *Rhodomonas baltica*, *Ditylum brightwelli* and *Thalassiosira weisflogii* (diatoms), and *Prorocentrum minimum*, *Heterocapsa triquetra* and *Prorocentrum micans* (dinoflagellates; Kiørboe and Titelman, 1998).

Lowest growth rates occurred when *N. scintillans* was given very small algal prey (haptophyte *Isochrysis galbana* and pelagiophycean *Aureoumbra lagunensis*). Moderate growth rates were supported by consumption of *Dunaliella tertiolecta* (small chlorophyte flagellate), and *Gyrodinium dorsum* and *Prorocentrum minimum*. Highest growth rates occurred when *N. scintillans* was given diatom (e.g., *Thalassiosira* sp.) and prasinophyte prey (Buskey, 1995).

N. miliaris also consumed toxic *Alexandrium minutum*; the maximum ingestion rate was 466 prey cells *N. scintillans* cell^{-1} day^{-1} (Frangópulos *et al.*, 2011).

Bacteria: *N. miliaris* consumed live cells of *Vibrio* sp. and *Serratia plymuthica* at a rate of 10^4 to 10^6 bacteria *N. scintillans* cell^{-1} hour^{-1} (Kirchner *et al.*, 1996).

Ostreopsis ovata [M, benthic; T (strain-dependent): palytoxin and analogs (Ciminiello *et al.*, 2010, 2011, 2012)]

Fish: European seabass juveniles (Fiamali *et al.*, 2011).

Crustacea: Brine shrimp (instars, stages 2–3), striped barnacle larvae (Faimali *et al.*, 2011).

Zooplankton: Crustaceans – *Tigriopus fulvus* nauplii (Faimali *et al.*, 2011).

Pfiesteria piscicida*, *Pfiesteria shumwayae [planktonic, some stages benthic; Br; T: PfTXs (strain-dependent); Burkholder *et al.*, 2005; Marshall *et al.*, 2006; Moeller *et al.*, 2007, Burkholder and Marshall 2012; can also kill via physical attack alone: Burkholder *et al.*, 2001]

Fish (death via toxin alone, or toxin along with physical attack): American eel, Atlantic croaker, Atlantic menhaden, channel catfish, hogchoker, hybrid tilapia, striped bass, sheepshead, southern flounder, spot, spotted sea trout, striped mullet, white perch (Burkholder and Glasgow 1997); Atlantic croaker and spot (Gordon *et al.*, 2002); cultured hybrid striped bass, tilapia (both species: Burkholder and Glasgow, 1997), hybrid tilapia (Gordon *et al.*, 2002), pikeperch (Moestrup *et al.*, 2014 – *Pfiesteria shumwayae*).

Molluscs (same mechanisms as for fish): Bay scallop (cultured adults, larvae), eastern oyster (cultured adults, larvae), green mussel (cultured adults), northern quahog (wild adults, cultured larvae – Springer *et al.*, 2002; Shumway *et al.*, 2006).

Crustacea: Blue crab (Burkholder and Glasgow, 1997).

Zooplankton: Ciliates – *Euplotes vannus, Euplotes woodruffi* (Lewitus *et al.*, 2006).

Phytoplankton: Toxigenic strains in the absence of live fish (TOX-B functional type), and NON-IND strains consume cryptophytes and other small microalgae (Burkholder and Marshall, 2012, and references therein).

Benthic *Prorocentrum* complex[f] [T (strain-dependent): DTXs, OA, diol esters of OA, 7-deoxy-OA, D8- and/or D-9 congeners of OA]. From Glibert *et al.* (2012 and references therein), unless otherwise noted:

Fish: European sea bass – Feeding *P. lima* complex cells to juveniles, either directly or by feeding them brine shrimp that had been consuming *P. lima*, led to fish death (Ajuzie, 2008).

Crustacea: Brine shrimp grazed continuously on *P. lima* complex cells until they died (Ajuzie, 2007).

Prorocentrum micans [M; T? (reported to produce hydrogen peroxide ROS – Kim *et al.*, 1999); low DO]

Fish: High-biomass blooms can deplete oxygen and cause major fish kills (Red-Tide, 2011; Smithsonian Institution, 2011).

Phytoplankton:

Allelochemicals from *P. micans* may kill other phytoplankton (Smithsonian Institution, 2011).

P. micans consumes *Isochrysis galbana*; *Rhodomonas salina* and an unidentified cryptophyte taxon; *Heterosigma akashiwo*; and *Amphidinium carterae, Prorocentrum minimum,* and *Heterocapsa triquetra* (Jeong *et al.*, 2005a).

The ingestion rate of unicellular cyanobacteria by *P. micans* was 35.4 ± 2.1 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).

Prorocentrum minimum [T (strain-dependent): venerupin shellfish poisoning; Grzebyk *et al.*, 1997); low DO]

Molluscs: Bay scallop, eastern oyster (adults and larvae; Wikfors 2005 and references therein).

Eastern oyster – When fed a diet consisting of only *P. minimum* (1.6×10^3 cells mL^{-1}; Luchenbach *et al.*, 1993).

(*continued*)

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed

Phytoplankton:

Consumes *Isochrysis galbana*, *Rhodomonas salina* and an unidentified cryptophyte taxon, *Heterosigma akashiwo*, and *Amphidinium carterae* (Jeong *et al.*, 2005a).

P. minimum is consumed by various mixotrophic and heterotrophic dinoflagellates (e.g., *Akashiwo sanguinea, Alexandrium tamarense, Gonyaulax polygramma, Gymnodinium catenatum, Gyrodinium resplendens, Heterocapsa triquetra, Lingulodinium polyedra, Polykrikoides kofoidii, Prorocentrum micans*) (Skovgaard, 2000; Burkholder *et al.*, 2008 and references therein).

The ingestion rate of unicellular cyanobacteria by *P. minimum* was 5.9 ± 1.2 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).

Bacteria: In stationary phase under nutrient-replete conditions, *P. minimum* engaged in bacterivory (Wikfors and Fernandez, 2013).

Scrippsiella trochoidea [T – but the toxin(s) have not been isolated or chemically characterized (see Tang and Gobler, 2012); low DO]

Molluscs: Eastern oyster and northern quahog larvae (at environmentally relevant densities, 10^4 cells mL^{-1}). Cultured *S. trochoidea* in later growth stages was more toxic than exponential growth stages. Mortality may have been caused in part via a physical/chemical mechanism such as clogging of larval feeding apparati by materials (e.g., lipids, extracellular polysaccharides, and/or cell debris) produced by *S. trochoidea* (Tang and Gobler, 2012).

Phytoplankton:

S. trochoidea consumes *Isochrysis galbana*; *Rhodomonas salina* and an unidentified cryptophyte taxon; *Heterosigma akashiwo*; and *Amphidinium carterae, Prorocentrum minimum,* and *Heterocapsa triquetra* (Jeong *et al.*, 2005a).

The ingestion rate of unicellular cyanobacteria by *S. trochoidea* was 7.1 ± 1.1 *Synechococcus* cells dinoflagellate^{-1} hour^{-1} (mean ± 1 SE; Jeong *et al.*, 2005b).

Bacteria: Maximum ingestion and clearance rates were 21.9 bacteria and 2.3 nL, respectively, algal cell^{-1} hour^{-1} (Seong *et al.*, 2006).

Haptophytes (Haptophyta – golden algae)

Prymnesium parvum[c] [Br; T (strain-dependent): many different toxins, e.g., hemolysins (lipopolysaccharides, a galactoglycerolipid, polene polyethers, polyketides such as prymnesins 1 and 2, cycloamines ["fast-acting ichthyotoxins"]), ROSs, dimethylsulfonio-propionate, polyunsaturated fatty acids, and fatty acid amides). Some of these toxins form micelles and require activation by various cofactors (Henrikson *et al.*, 2010; Manning and Claire, 2010; Bertin *et al.*, 2012a, 2012b and references therein). Also, various allelochemicals such as glycolipids, galactolipids, proteolipids, and lipid-carbohydrate complexes (Manning and Claire, 2010). Low DO can develop in association with large blooms. EDAB (Sunda *et al.*, 2006).]

General: Blooms of *P. parvum* have caused death of gill-breathing organisms such as fish (all species present), mussels, and larval amphibians (Burkholder, 2009 and references therein).

Fish:

Massive fish kills; primary uptake of toxins by gill filaments (Van Landeghem *et al.*, 2013 and references therein).

Massive fish kill affecting all fish species present in the area (blue catfish, bluegill sunfish, channel catfish, common carp, carpsucker, flathead catfish, freshwater drum, gar, largemouth bass, longnose gar, mosquitofish, Rio Grande darter, white bass, white crappie (James and de la Cruz, 1989).

Molluscs: *Corbicula fluminea* – Although this exotic/invasive species had previously been common in an affected river with densities as high as 1076 individuals m^{-2} (100 individuals ft^{-2}), no live animals were observed for several years following a massive fish kill (James and de la Cruz, 1989).

Zooplankton:

Copepods – *Eurytemora affinis*: Cell-free filtrates from toxic *P. parvum* cultures negatively affected survivorship (Sopanen *et al.*, 2008).

Ciliates – *Euplotes affinis* (Granéli and Johansson, 2003a).

Favella ehrenbergii, Eutintinnus pectinis, Metacyclis angulata, Strombidium conicum, and *Strombididinopsis* sp. (Rosetta and McManus, 2003).

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Phytoplankton:
Oxyrrhis marina – Consumed by *P. parvum* as prey, especially rounded and partly lysed cells (toxin effect; Tillmann, 2004).
Rhodomonas sp.: Consumed as prey (Fistarol *et al.*, 2003).
Rhodomonas cf. *baltica*, *Rhodomonas salina*, and *Thalassiosira weissflogii* (Granéli and Johansson, 2003; Barreiro *et al.*, 2005; Uronen *et al.*, 2007).
Heterocapsa rotundata (Skovgaard and Hansen, 2003).
Minidiscus trioculatus, *Thalassiosira* sp. (small diatoms, maximum cell dimension 5 µm): High interim grazing rates during the first eight hours (0.30 *M. trioculatus* and 0.74 *Thalassiosira* sp. *P. parvum* cell^{-1} hour^{-1}). When bacteria were added as potential prey, prey-switching from diatom cells to bacteria occurred in cultures with *M. trioculatus* (Martin-Cereceda *et al.*, 2003).
Bacteria: Bacterial grazing was similar with or without diatom prey (0.17 bacteria *P. parvum* cell^{-1} hour^{-1}) (Martin-Cereceda *et al.*, 2003).
Euglenoids (Euglenophyta) [F, Br, M; T: EUGL (Zimba *et al.*, 2010, 2017)]
Euglena sanguinea[g]
General: The toxin structure is similar to that of alkaloids produced in fire ant venom. This class of alkaloids has strong biological activity ranging from necrotoxicity and hemolysis to phytotoxicity, as well as insecticidal and antibiotic activities. Cultured euglenoids apparently produce EUGL regardless of growth phase, suggesting use of the toxin in defense against herbivores (Zimba *et al.*, 2010 and references therein). Toxic euglenoid blooms have only recently begun to be examined.
Fish: Blue tilapia, channel catfish, sheepshead (scientific naming information not included), and striped bass. Tilapia appeared to have euglenoid cells associated with gills, resulting in distressed breathing (indicated by surface porpoising; Zimba *et al.*, 2010).
Pelagophyceans (Heterokontophyta: brown tide algae)
Aureococcus anaophagefferens [T-Br (strain-dependent) – see Robbins *et al.*, 2010; note that the toxin(s) are not chemically characterized. Low DO can develop in association with large blooms. EDAB (Sunda *et al.*, 2006).]
Fish: Failure of bay anchovies to spawn (Smayda, 1991).
Molluscs: Bay scallop, blue mussel, northern quahog (Tracey 1988; Smayda and Villareal, 1989); bay scallop larvae: Gallager *et al.*, 1989; Gainey and Shumway, 1991).
Zooplankton: Failure of the cladoceran community to develop during a brown tide bloom (Smayda, 1991).
Seagrasses: *Zostera marina*, via light reduction – large-scale die-offs of eelgrass, a critical habitat species for shellfish, larval fish, and many other fauna (Dennison *et al.*, 1989).
Beneficial macroalgae: Macroalgal die-off (kelps, others) occurred during a brown tide (Dennison *et al.*, 1989; Smayda, 1991).
Aureoumbra lagunensis [B? T? (strain-dependent): e.g., Liu and Buskey 2000; or, from Buskey *et al.*, 1996, p. 43: "there is evidence that brown tide [*A. lagunensis*] may be directly toxic to some species of zooplankton at cell concentrations similar to those found in nature." Low DO can develop in association with large blooms. EDAB (Sunda *et al.*, 2006).]
Fish: Massive fish kills (Gobler *et al.*, 2013).
Molluscs: Bay scallop, blue mussel, northern quahog (Smayda *et al.*, 1991; Gobler *et al.*, 2013 and references therein).
Seagrasses: Reduced biomass and areal extent of seagrass meadows (Onuf, 1996); steady decline in the biomass of roots and rhizomes after several years of the brown tide bloom, and major reduction in the ratio of root+rhizome biomass to shoot biomass, indicating a loss in critically important food reserves (data of K. Dunton in Buskey *et al.*, 1996).
Raphidophyceans (Heterokontophyta)
Heterosigma akashiwo [Br, M; T (strain-dependent): ROSs (e.g., superoxide, hydrogen peroxide), BTX like compound(s), hemagglutinating and hemolysing compounds, and an uncharacterized polysaccharide–protein complex (Yamasaki *et al.*, 2009, Mohamed and Al-Shehri, 2012 and references therein unless otherwise noted). Low DO can also develop during/following large fish kills linked to *H. akashiwo*.]
Fish: Atlantic salmon (wild and cultured), Chinook salmon (including the endangered Wild White River spring chinook salmon), Coho salmon, chum, sockeye salmon, and rainbow trout (Rensel *et al.*, 2010 and references therein). Massive fish kills (Liu *et al.*, 2008 and references therein).

(*continued*)

Table 7.1 (*Continued*)

Taxon (taxa) and organisms killed
Note: Blooms occurred in coastal waters of British Columbia, Canada, every year beginning in the 1960s, and fish kills linked to *H. akashiwo* were reported in most years from 1986 through the following decade. In addition, fish kills linked to *H. akashiwo* were reported in Washington state waters during some years, with substantial economic losses (Horner *et al.*, 1997 and references therein). *Zooplankton*: Ciliates *Tintinnopsis tubulosoides, Favella* sp., and *Synchaeta cecilia* (Verity and Stoecker, 1982; Egloff, 1986). *Flavella* sp. and *Metacylis* sp., when *H. akashiwo* was the only prey offered; with mixed algal prey, however, toxic effects of *H. akashiwo* were not apparent, probably because of selective feeding on the nontoxic prey (Graham and Strom, 2010). *Bacteria*: The maximum ingestion rate and maximum clearance rate were 11.7 bacteria and 2.6 nL, respectively, algal cell^{-1} hour^{-1} (Seong *et al.*, 2006).

Note: Plankters are listed unless otherwise indicated. T-toxigenic, B-impacts known from other bioactive allelopathic compound(s); low DO-high-biomass blooms cause anoxia/hypoxia; Str-structural feature of the algal cell causes impacts, e.g., needle-like extensions; EDAB – ecosystem-disruptive algal bloom; Par, parasitic. F-freshwater, Br-brackish, M-marine. Toxins: BMAA, β-methylamino-L-alanine; BTXs, brevetoxins; CTXs, ciguatoxins (ciguateratoxins); CYL, cylindrospermopsin; DA, domoic acid; DTX, dinophysis toxin; EUGL, euglenophycin toxin; KTXs, karlotoxins; LPS, lipopolysaccharides; LYNGTX, lyngbyatoxin; MC, microcystin; TMCs, total microcystins; OA, okadaic acid; PfTXs, *Pfiesteria* spp. toxins; PTX, palytoxin and analogs; ROSs, reactive oxygen species; STXs, saxitoxins and derivatives. *Bacteria* refers to eubacteria. See Appendix 1 for scientific names if not included here. Taxonomy is based on AlgaeBase (http://algaebase.org), the World Register of Marine Species (WORM: http://marinespecies.org/), and recent science publications.

[a] Throughout this table for some taxa, the species listed is not the only harmful species in the genus. For example, toxic strains of *Anabaena circinalis* produce STXs (Llewellyn *et al.*, 2001); *Cochlodinium* cf. *fulvescens* has been linked to mortality of California mussels (Curtiss *et al.*, 2008), etc.

[b] Microcystins are known to be produced by some strains within the cyanobacteria *Microcystis* spp., *Anabaena* spp., *Planktothrix/Oscillatoria* (*P. agardhii, P. rubescens*), *Anabaenopsis* spp., *Nostoc rivulare,* and *Hapalosiphon* spp. (de Figueiredo *et al.*, 2004a). The species *Aphanizomenon flos-aquae* is also sometimes listed as a MC producer (e.g., de Figueiredo *et al.*, 2004a), but that information apparently resulted from a long-standing error in the literature wherein tested *Aphanizomenon flos-aquae* material had been contaminated with MC-producing, cryptic *Microcystis* spp. (W. Carmichael, pers. comm., 1999). For toxins of *Aphanizomenon flos-aquae*, see http://www.env.gov.bc.ca/wat/wq/reference/cyanophytes.html#m1.

[c] Engene *et al.* (2012) suggested renaming the cyanobacterium *Lyngbya majuscula* as *Moorea producens*, but the issue has not been resolved. Since *Lyngbya majuscula* is so widely used by resource managers and scientists, we have retained that name here. Among various former names used for the following species, the most common previous names are as follows:

Cyanobacteria – *Cylindrospermopsis raciborskii:* previous name *Anabaenopsis raciborski*i.

Dinoflagellates –
Akashiwo sanguinea: previous names *Gymnodinium sanguineum, G. splendens, G. nelsonii*;
Alexandrium monilatum: previous name *Gonyaulax monilata*;
Alexandrium tamarense: previous names *Alexandrium excavata, Alexandrium excavatum, Gonyaulax tamarensis*;
Cochlodinium polykrikoides: previous name *Cochlodinium heterolobatum*;
Karenia brevis: *Gymnodinium breve, Gymnodinium brevis, Ptychodiscus brevis*;
Karlodinium veneficum: previous names *Gymnodinium veneficum, Gymnodinium galatheanum, Gyrodinium galatheanum, Karlodinium micrum*;
Lingulodinium polyedrum: previous name *Gonyaulax polyedra*.

Haptophytes –
Prymnesium parvum: previous names *Prymnesium patelliferum, P. parvum* f. *patelliferum*.

Raphidophyceans –
Heterosigma akashiwo: previous names *Olisthodiscus luteus, Entomosigma akashiwo*.

[d] "Avian vacuolar myelinopathy (AVM) is a neurological disease that produces uncoordinated behavior in affected birds in wetland ecosystems of the southeastern U.S. Feeding and sentinel trials, field surveys, and genetic studies have implicated the introduced flowering plant species hydrilla (*Hydrilla verticillata*) and an associated epiphytic cyanobacterial species (Order Stigonematales) as a causal link to AVM. All morphotypes of cyanobacteria have been shown to produce the neurotoxic amino acid BMAA, including cyanobacteria of the Stigonematales that are epiphytic on hydrilla. If biomagnification of BMAA occurs in other ecosystems, as has been observed in the Guam ecosystem, then the consumption of fish (e.g., shad and herring) and waterfowl (e.g., Canada geese and mallards) from AVM-confirmed reservoirs in Arkansas, Texas, Georgia, North Carolina and South Carolina, U.S., could represent a significant human health risk" (Bidigare *et al.*, 2009, p. 71; also see Cox *et al.*, 2005; Lürling *et al.*, 2011; Holtcamp, 2012; Al-Sammak *et al.*, 2014). Importantly, BMAA has been found in nearly all cyanobacteria that have been assessed from terrestrial, freshwater, brackish, and marine environments (e.g., Cox *et al.*, 2005; Wilde *et al.*, 2005; Brand *et al.*, 2010; Lürling *et al.*, 2011; Al-Sammak *et al.*, 2014). Its structural isomer, 2,4-diaminobutyric acid (DABA), frequently co-occurs with BMAA (Banack *et al.*, 2010; Al-Sammak *et al.*, 2014).

[e] After reviewing the available information about *Synechococcus* blooms, Beardall (2008, p. 3) wrote, "While reports of toxicity in *Synechococcus* blooms are rare, this is largely because investigators have not been attuned to the possibility of this genus producing toxins." The toxicity of *Synechococcus* blooms likely has been underestimated.

[f] Examples of toxigenic benthic species: *Prorocentrum belizeanum*, *P. concavum*, *P. faustiae*, *P. hoffmannianum*, *P. lima* complex (likely several cryptic species), *P. maculosum*, and *P. rhathymum* (see Glibert and Burkholder 2018b, and references therein).

[g] The toxin EUGL is also produced by other euglenophytes including, thus far, *Euglena clavata*, *E. socialis*, *E. stellata*, *Euglenaria anabaena*, *Lepocinclus acus*, *Strombomonas borysteniensis*, and *Trachelomonas ellipsodalis* (Zimba *et al.*, 2017).

Table 7.2 Reported sublethal or chronic lethal effects of selected harmful algae on other organisms.[a]

Organism(s) impacted and reported effect(s)
Cyanobacteria
Anabaena flos-aquae (also see impacts under *Microcystis aeruginosa* and other MC producers)
Zooplankton:
Microcrustaceans
- *Daphnia pulex*: Lower fitness based on reduced filtering rate, smaller brood size, depressed survivorship, reduced rate of increase, and depressed net reproductive rate (Gilbert, 1994 and references therein).
- *Daphnia hyalina, D. pulicaria*: Feeding was inhibited (DeMott *et al.*, 1991).
- *Daphnia parvula, D. pulex*: Reduced feeding (Fulton, 1988a).
- *Diaptomus reighardi, Eurytemora affinis*: Avoided feeding (Fulton, 1988a).
- Rotifers: *Asplanchna girodi*, *Brachionus calyciflorus*, *Keratella cochlearis*, *Synchaeta pectinata* reduced fecundity, and reproduction was inhibited (Gilbert, 1994 and references therein).
Phytoplankton:
- *Chlamydomonas reinhardtii* (chlorophyte flagellate): Allelopathic; crude extracts decreased the number of motile cells, inhibited growth, and induced settling (Kearns and Hunter, 2000, 2001); and crude extracts or MC-LR increased the settling rate. Such effects would help to reduce inter-algal competition for resources such as water-column nutrients (Zurawell *et al.*, 2005 and references therein).
- *Thalassiosira weissflogii*, *Rhodomonas* sp., *Prymnesium parvum*: Allelopathic; inhibited growth (Suikkanen *et al.*, 2004).
Aphanizomenon flos-aquae
Fish:
- Common carp (three months old): Exhibited rapid opercular movement and abnormal swimming, suggested to have negative consequences on fish populations due to changes in reproductive and predator–prey interactions (from exposure to freeze-dried cells; Osswald *et al.*, 2007).
- Pacific herring: Severe reductions in spontaneous swimming behavior and touch response (Lefebvre *et al.*, 2005).
Zooplankton:
Microcrustaceans
- *Acartia bifilosa*: Reduced feeding and fecundity (Sellner *et al.*, 1996).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

- *Daphnia longispina, D. magna*: Impaired reproduction (de Figueiredo *et al.*, 2004b).
- *Daphnia carinata*: Reduced appendage beat rate (Haney *et al.*, 1995).
- *Diaptomus reighardi, Eurytemora affinis*: Avoided feeding (Fulton, 1988a).
- *Eurytemora affinis*: Reduced fecundity and feeding (Fulton, 1988a).

Phytoplankton:

- *Rhodomonas* sp.: Inhibited growth (Zanchett and Oliveira-Filho, 2013 and references therein; Suikkanen *et al.*, 2004).

Cylindrospermopsis raciborskii

General: Other bioactive compounds present in *C. raciborskii* extracts and cells along with CYL likely contribute to its toxicity (Seifert, 2007). Toxins CYL and deoxy-CYL concentrate and bioaccumulate in a range of aquatic flora and fauna (Seifert, 2007; Kinnear, 2010).[b]

Mammals: *C. raciborskii* was implicated in cattle mortality (Hawkins *et al.*, 1997).

Fish:

- General: "In every investigated ontogenic stage, reports of the biological effects of CYL on fish species are scarce" (Sotton *et al.*, 2015, p. 5). Nearly all available information is for exposure to CYL rather than exposure to toxic *C. raciborskii* (Sotton *et al.*, 2015). CYL has been shown to accumulate in various fish species (e.g., brown trout; Sotton *et al.*, 2015).
- Zebrafish: Exposure to STX-producing *C. raciborskii* increased the mean swimming distance covered and the mean swimming velocity (Ferrão-Filho *et al.*, 2007).
- Zebrafish: Embryos were malformed (e.g., lateral and ventral body curvature and edema; Zanchett and Oliveira-Filho, 2013).
- Tilapia: Adults exposed by immersion to environmentally relevant concentrations of CYL sustained damage especially to the liver and kidneys, but also to heart, intestine, and gill (Gutiérrez-Praena *et al.*, 2012; Puerto *et al.*, 2012).

Amphibians: Cane toads – Tadpoles exposed to whole toxic cell extracts survived, but decreased relative growth rates and time spent swimming. Exposure to live toxic cultures led to bioaccumulation of CYL (~19-fold) (White *et al.*, 2007).

Molluscs:

- CYL bioaccumulated in tested mussels (*Anodonta cygnea*; Metcalf *et al.*, 2004).[c]
- Malaysian trumpet snail: Number of hatchlings decreased when exposed to live toxic culture. In contrast, exposure to whole-cell extracts with extracellular CYL increased hatchling numbers, and adults were not affected. Since CYL is a protein synthesis inhibitor, it may be especially toxic to rapidly developing tissues such as in snail embryos (Kinnear *et al.*, 2007).
- Swan mussel: Exposure of adults to CYL-producing cultures of *C. raciborskii* for 16 days led to CYL accumulation (up to 2.52 mg g tissue dry wt^{-1}). Most toxin was in the hemolymph (~68%), viscera (~23%), and foot and gonad (~8%). CYL was not detected in gill or adductor muscle tissue. After 2 weeks of depuration, ~50% of the toxin still remained in the tissues. The animals appeared to be unaffected (Saker *et al.*, 2004).

Arthropods other than zooplankton: CYL bioaccumulated in crayfish, especially in the hepatopancreas. The animals appeared unaffected, without histological abnormalities (Saker and Eaglesham, 1999; Nogueira *et al.*, 2004; Saker *et al.*, 2004).

Zooplankton:

- The community was more diverse, with larger species, when *C. raciborskii* abundance was low or undetectable. As abundance of *C. raciborskii* increased, micrograzers such as rotifers increased and became dominant (Leonard and Paerl, 2005).
- Small trichomes of *C. raciborskii* tended to clog cladoceran filters, reducing food intake and decreasing daphnid body size (Hawkins and Lampert, 1989; Nogueira *et al.*, 2004).
- Zooplankton diversity increased during and after a *C. raciborskii* bloom; copepods and rotifers apparently were able to sever the long filaments and shorten them to edible size for small-bodied herbivorous cladocerans (Bouvy *et al.*, 2001).
- *Ceriodaphnia dubia:* Animals were immobilized (Zagatto *et al.*, 2012).
- Daphnids sustained high mortality and low fecundity. Toxic blooms may reduce grazing pressure, giving the toxic strains a survival advantage over nontoxic strains (Nogueira *et al.*, 2004).
- *Daphnia magna:* Both fitness (fecundity) and growth potential decreased during *C. raciborskii* blooms (Nogueira *et al.*, 2004).
- *Daphnia pulex:* Decreased swimming velocity, mean swimming distance covered, and mean swimming velocity (Ferrão-Filho *et al.*, 2007, 2008).
- *Daphnia pulex, Moina micrura:* Reproduction rates decreased (Zanchett and Oliveira-Filho, 2013, and references therein).
- *Daphnia similis*: Fitness decreased (Zagatto *et al.*, 2012).
- *Eudiaptomus gracile:* Feeding was depressed on a toxic strain relative to feeding on a nontoxic strain (Rangel *et al.*, 2016).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Phytoplankton:

- Decreased assemblage diversity (Dobberfuhl, 2003).
- *Microcystis aeruginosa* (cyanobacterium): Allelopathic; inhibited growth and MC-LR production, and promoted upregulation of alkaline phosphatase activity (Rzymski *et al.*, 2014).
- *Microcystis wesenbergii* and *Coelastrum sphaericum*, *Monoraphidium contortum* (chlorophytes): Allelopathic; inhibited photosynthesis (Figueredo *et al.*, 2007).

Bacteria: Total biomass and abundance of larger size classes increased post-bloom (Bouvy *et al.*, 2001).

Lyngbya majuscula

General:

- Significantly alters marine ecosystem dynamics. The many bioactive compounds known to be produced by toxic strains include tumor promoters, substances that enhance oncogene-induced cell transformations, and immunosuppressants. Three toxins are tumor promotors (Osborne *et al.*, 2001).
- Specific metabolites produced by *L. majuscula* act both as feeding attractants (e.g., to the long-tailed sea hare, a specialist herbivore) and as effective feeding deterrents to generalist fishes (Charpy *et al.*, 2012).

Birds: Altered foraging behavior (Estrella *et al.*, 2011).

Reptiles: Tumors developed in sea turtles that consumed *L. majuscula* (Arthur *et al.*, 2006). Toxins of *L. majuscula* are a suspected cause of the debilitating neoplastic disease of marine turtles known as fibropapillomatosis (Osborne *et al.*, 2001).

Fish:

- Altered feeding behavior and reduced biomass in the fish community; also, depressed species richness and reduced growth of juveniles, and decreased carrying capacity for fish (Gilby *et al.*, 2011; Hudon *et al.*, 2014).
- Parrotfish: Three bioactive substances produced by *L. majuscula* deterred feeding by juveniles (Thacker *et al.*, 1997; Kuffner and Paul, 2004; Kuffner *et al.*, 2006).

Invertebrates:

- Reduced recruitment (and survival – Table 7.1) of scleractinian corals and gorgonians, depressed species diversity, and decreased biomass (Kuffner and Paul, 2004, and references therein):
- Coral *Pocillopora damicornis*: The presence of *L. majuscula* significantly adversely affected recruitment. Larvae also exhibited avoidance behavior. *Note*: Certain species of crustose coralline red algae contain compounds that attract coral larvae and, thus, provide positive chemical cues for scleractinian settlement. *L. majuscula* mats have killed the crustose coralline algae beneath.

Seagrasses:

- Blooms decreased the habitat nursery capacity, and blooms have overgrown and smothered seagrass beds (Watkinson *et al.*, 2005; Tilling, 2007; Ng *et al.*, 2012).
- Adverse effects on shoalgrass occurred after declines in bloom biomass, indicating that *L. majuscula* can cause prolonged effects on shoalgrass production (Tilling, 2007).

Lyngbya wollei [T – aplysiatoxins, CYL, deoxy-CL, LYNGTX, STX analogs (e.g., decarbamoylsaxitoxin, decarbamoylgonyautoxin) (Camacho and Thacker, 2006; Seifert, 2007; Foss *et al.*, 2012)]

General: A diverse assemblage of invertebrate mesograzers lives on and within *L. wollei* mats. Trichomes of *L. wollei* are surrounded by a prominent extracellular polysaccharide sheath (~55% of the dry weight – apparently functions as a structural defense against herbivory; Camacho and Thacker, 2006).

Fish: Wetlands dominated by *L. wollei* have supported lower biomass of large fish, lower fish species richness, and more slowly growing juvenile perch (*Perca flavescens*) than macrophyte [vascular plant]-dominated wetlands (Hudon *et al.*, 2012).

Invertebrates:

- Apparently toxic to some amphipod species; eaten (but not preferred) by the amphipod *Hyalella azteca* (Camacho and Thacker, 2006).
- Acetylcholinesterase and glutathione-S-transferase (GST) activities were higher for amphipods (*Gammarus fasciatus*) within *L. wollei* mats, suggesting toxicity to the amphipods (Gélinas *et al.*, 2013).
- Wetlands dominated by *L. wollei* had lower invertebrate biomass than macrophyte-dominated wetlands (Hudon *et al.*, 2012).
- Grazer biomass was significantly less in areas with dominant *L. wollei*, apparently related to reduced food and habitat availability via declines in macrophytes and epiphytes (Lévesque *et al.*, 2012).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Habitat:

- There was an inverse relationship between abundance of *L. wollei* and abundance of macrophytes (e.g., freshwater eelgrass); blooms are considered symptomatic of ecosystem degradation (Hudon *et al.*, 2014). Dominance of *L. wollei* coincided with low macrophyte biomass, yielding a simplified, less productive ecosystem (Hudon *et al.*, 2014).
- *L. wollei* appears to proliferate when native primary producers are limited by unfavorable light or nutrient conditions, or physically removed (Evans *et al.*, 2007).
- Macrophytes suppressed benthic *L. wollei* mats (Doyle and Smart, 1998).

***Microcystis aeruginosa* (and various *Microcystis spp.* and *other MC producers*)**

General: Example, San Francisco Bay – Total microcystins [TMCs] were in all levels of the food web; higher TMC concentrations in striped bass than their prey suggested that MCs accumulated in biota of higher trophic levels. "This study suggests that even at low abundance, *Microcystis* may impact estuarine fish production through toxic and food web impacts at multiple trophic levels" (Lehman *et al.*, 2010, p. 229). MCs can bioaccumulate in many biota (White *et al.*, 2006 and references therein).

Mammals:

- The liver-to-body mass ratio increased due to liver hemorrhaging (Zurawell *et al.*, 2005).
- MCs bind specifically to hepatic cells, irreversibly inhibiting serine/threonine protein phosphatases PP1 and PP2 (important enzymes involved in tumor suppression), and causing disintegration of hepatocyte structure, apoptosis, liver necrosis, and internal hemorrhage. MC-LR may also bind to ATP synthetase, potentially leading to cell apoptosis. Some symptoms characteristic of MC poisoning include weakness, anorexia, gastroenteritis, vomiting, and diarrhea. Chronic exposure to MCs promotes liver cancer by inducing DNA damage. Other chronic effects such as increased liver weight, liver tissue damage, and kidney damage have been detected in mammals after treatment with low concentrations of MCs in drinking water (de Figueiredo *et al.*, 2004a – mostly based on mouse models).

Birds:

- Black-crowned night heron, mallard duck: High levels of MCs in gonad, egg yolk, and egg white, suggesting potential effects of MCs on waterbird embryos; also high MC content in spleens of both species (Ferrão-Filho and Kozlowsky-Suzuki, 2011).
- Piscivorous birds were considered to be at risk due to high MC levels in planktivorous smelt prey (but see Ibelings *et al.*, 2005).

Reptiles: European pond turtle, Mediterranean turtle – High MC content was found in liver; MCs also were detected in viscera and muscle tissues from fresh carcasses; and "liver crumbling" occurred during necropsy (Nasri *et al.*, 2008).

Fish:

- In various species, MCs have modified immunological and blood indices, causing increased activities of ALT (alanine amino-transferase), AST (aspartate aminotransferase), and LDH (lactate dehydrogenase). There has been damage to the liver, kidneys, heart, digestive tract, gills, spleen, and skin (Malbrouck and Kestemont, 2006 and references therein). Respiration and behavior also have been adversely impacted (Wiegand and Pflugmacher, 2005 and references therein). Younger stages have been more adversely affected than adults, and the toxins have been rapidly cleared from fish tissues (Malbrouck and Kestemont, 2006; Ibelings and Havens, 2008 and references therein). Oral and immersion exposure is slow to induce adverse effects, so acute toxic episodes are rare (Zurawell *et al.*, 2005).
- Increased MC production has occurred when *M. aeruginosa* is exposed to certain fish; apparently, blooms can respond to a chemical signal related to feeding, even when fish are not vigorously consuming the cyanobacteria (Jang *et al.*, 2004).
- Histopathology of liver tissue (striped bass, Mississippi silverside) suggests that fish health was adversely affected by tumor-promoting substances, especially in areas where TMC concentrations were elevated (Zurawell *et al.*, 2005).
- Lipopolysaccharides present on the cell surface of cyanobacteria such as *Microcystis* can stimulate drinking in fish, which would increase the potential for toxin exposure and promote osmoregulatory imbalance (Ferrão-Filho and Kozlowsky-Suzuki, 2011 and references therein).
- Atlantic salmon, Chinook salmon, steelhead trout: MCs have been linked to "net-pen liver disease;" histopathological changes have included diffuse necrosis and hepatic megalocytosis (Zurawell *et al.*, 2005 and references therein).
- Brown trout: Exposure to lysed *Microcystis* cells indicated an energy allocation from growth processes toward stress responses (Bury *et al.*, 1996b).
- Carp: MC-LR blocks gill activity: Ion pumps (e.g., Na^+-K^+, Na^+, HCO_3^-, and Ca^{+2}-ATPases) in gill tissue were directly inhibited by MC-LR. This toxin also blocks the hydrolysis of phosphorylated protein and inhibits the aspartic dephosphorylation step of the sodium pump enzymes (Gaete *et al.*, 1994; Zambrano and Canelo, 1996).
- Carp: Embryotoxic effects have included delayed hatching, low number of hatched embryos, suppressed embryonic development, disturbance of air bladder filling, significant inhibition of GSTs; missing eye pigmentation at 48 hours post-fertilization; an incomplete filling of air bladder after 120 hours of exposure to MCs; and atrophy of hepatocytes. In addition, gills exhibited pinpoint necrosis, epithelial ballooning, folded lamellar tips, exfoliation of the lamellar

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

epithelium, elevated aspartate aminotransferase activity, and elevated serum bilirubin concentrations (Chorus and Bartram, 1999).

- Carp: In field studies during toxic *M. aeruginosa* blooms, fish consumed the toxic cells and appeared healthy but showed signs of hepatocyte atrophy; and 37% of the collected fish had gills displaying folded lamellar tips with epithelial ballooning and localized necrosis (Zurawell *et al.*, 2005 and references therein).
- Loach: Exhibited abnormalities such as pericardial edema and tubular heart, bradycardia, homeostatis, poor yolk resumption, small head, curved body and tail, and abnormal hatching. Overall, exposure to crude MC extracts led to gross malformations in embryo development, such that progression to the larval stage ceased (Zurawell *et al.*, 2005 and references therein).
- Medaka: Sustained hepatobiliary damage (hepatobiliary hypertrophy, hepatic hemorrhage, and necrosis at late development stages in embryos; Jacquet *et al.*, 2004).
- Roach: When fed sublethal cell densities of *Microcystis* cells, fish were unable to digest and lyse enough cells to release a harmful amount of toxin. Culture experiments with roach feces revealed that most *Microcystis* cells were not digested, and *Microcystis* grew exponentially after passing through the gut (Kamjunke *et al.*, 2002).
- Silver carp, tilapia: In laboratory studies, fish selectively consumed nontoxic strains *of M. aeruginosa*. Ingestion decreased as the proportion of toxic cells increased; grazing response decreased linearly as the proportion of toxic cells increased above 25% (Keshavanath *et al.*, 1994). Fish exposed to nontoxic strains had higher opercular beat rates (which effectively maintain the flow of water and suspended food particles over the gills; Beveridge *et al.*, 1993; Keshavanath *et al.*, 1994).
- Silver carp (80-day sub-chronic experiment): Fish fed *Microcystis viridis* that was collected from a eutrophic pond had measurable MC-RR in their blood (highest levels), liver, and muscle. In contrast, most MC-LR was in the intestines, and MC-LR was not detected in blood or muscle. The date suggest that silver carp have a mechanism to degrade MC-LR actively and to inhibit MC-LR transport across the intestines (Xie *et al.*, 2004).
- Silver carp, goldfish: MCs accumulated in the liver (up to 150 ng g dry wt^{-1}; Chen *et al.*, 2009).
- Tilapia (*Oreochromis mossambicus*): *Microcystis* cell lysate inhibited ion pumps (e.g., Ca^{+2}) in the gills more effectively than exposure to pure MC-LR, due to fatty acids present in the cells which interacted with the membranes of gill epithelial cells. These fatty acids inhibited the p-nitrophenol phosphatase activity of the gill basolateral membrane (Bury *et al.*, 1996a, 1998).
- Tilapia (*Oreochromis mossambicus*): Chronic exposure to low MC-LR levels (0.5 μg L^{-1}) reduced growth in larval fish, perhaps by increasing energy demands as required by detoxication processes (Zurawell *et al.*, 2005 and references therein).
- Zebrafish: Reduced growth and reduced weight by 25%. Environmentally relevant concentrations of MC-LR (0.5 to 50 μg L^{-1}) influenced the diurnal rhythm, leading to impaired food uptake and depressed spawning success (Baganz *et al.*, 1998).
- Zebrafish: Vitellogenin genes were highly upregulated in fish exposed to *Microcystis* but not to MC, suggestive of potential endocrine disrupting effects of *Microcystis* blooms. There was a significant decrease in the percentage of adults that spawned when exposed to *Microcystis*, but fecundity and larval survival were not affected (Rogers, 2010).

Molluscs:

- *Anodonta simpsonina* (freshwater clam): This species, an important food source of muskrats, bioaccumulated high amounts of MCs. The clams were suggested as a route of toxicity for muskrats and their predators (Prepas *et al.*, 1997).
- Asian clam (exotic/invasive): Cytotoxic effects occurred from exposure to toxic strains of *M. aeruginosa* (Martins *et al.*, 2009).
- Blue mussel: After being fed toxic *M. aeruginosa* for 3 days, MCs bioaccumulated (Williams *et al.*, 1997).
- Freshwater clam: Bioaccumulated MCs from phytoplankton containing MC-LR (~0 to 8.3 μg L^{-1} as cellular toxin); after ~12 days, the animals contained from ~24 to 527 ng g^{-1} MC-LR equiv. MC-LR concentrations were usually higher in the visceral mass than in gill and muscle tissues. After clams were placed in toxin-free water, MC-LR equiv. levels in tissues rapidly decreased for 6 days (by ~70%), but remaining levels were relatively stable for the following 15 days (Prepas *et al.*, 1997).
- Great pond snail: Sustained a marked decrease in egg production. Snails accumulated MC-LR; the amount accumulated depended on the toxin content and *Microcystis* abundance in the phytoplankton assemblage (Zurawell *et al.*, 1999).
- Mediterranean mussel: Accumulated MCs when fed *M. aeruginosa*, especially in the digestive tract (muscle, gill, and foot had very little toxin). When transferred to nontoxic phytoplankton food, depuration occurred with a 50% decrease within 2 days, but MCs were still detectable after 2 weeks and increased via consumption of feces (Vasconcelos, 1995; Amorim and Vasconcelos, 1999).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
- Swan mussel: Accumulated high amounts of *Oscillatoria agardhii*, mainly into the hepatopancreas. The mussels apparently were not harmed, but MCs extracted from them were toxic to mice, suggesting that the MCs were not metabolized (Eriksson *et al.*, 1989).
- Zebra mussel: Decreased food intake, filtration, absorption, and fecal loss, and showed significantly lower net energy balance in growth (Juhel *et al.*, 2006). Pseudofeces production was higher in the presence of toxic than nontoxic *Microcystis* strains, but pseudofeces contained more living cells of co-consumed nontoxic green algae than of the cyanobacterium (Pires and Van Donk, 2002). After exposure to *Microcystis* crude extracts, zebra mussels increased GST and glutathione peroxidase activities, accompanied by short-term depletion and oxidation of the glutathione pool (Peuthert and Wiegand, 2004). In a food web study, MCs were found in ~90% of all zebra mussel samples, indicating transfer of MCs within the food web (Zurawell *et al.*, 2005 and references therein).
- Zebra mussel (exotic/invasive, Hudson River): Preferentially ingested *Microcystis* (Baker *et al.*, 1998).
- Zebra mussel (exotic/invasive, Lake Erie): Given credit for promoting toxic blooms, and also reject live *Microcystis* cells in their pseudofeces (Vanderploeg *et al.*, 2001).
- Zebra mussel: Biotransformed MC-LR by conjugation to a glutathione/MC-LR conjugate, apparently the first step in detoxication (also found in the macrophyte coontail, the crustacean zooplankter *Daphnia magna*, and zebrafish; Pflugmacher *et al.*, 1998).
Note: Freshwater and marine mussels and clams mostly have been found to be relatively resistant to MCs and other cyanotoxins (Wiegand and Pflugmacher, 2005).
Crustacea other than zooplankton:
- Brine shrimp: Exposure to MC-LR increased GST activity of the detoxification system and conjugation of the toxin to GST (de Figueiredo *et al.*, 2004a).
- Red swamp crayfish: Accumulated MCs in the intestine and hepatopancreas (de Figueiredo *et al.*, 2004a).
- Signal crayfish: Accumulated MCs both during a benthic *Oscillatoria* bloom and in laboratory experiments. Harmful effects were not detected (Liras *et al.*, 1998).
- Southwestern Atlantic burrowing crab: Sustained physiological impacts from exposure to extracts of *M. aeruginosa*, including inhibition of Na^+ and K^+ ATPase, increased GST activities, and enhanced oxygen radical scavenging capacity (Vinagre *et al.*, 2002).
Note: In general, crayfish grow well on toxic *Microcystis*; MCs accumulate in intestine and hepatopancreas, but not in muscle (Vasconcelos *et al.*, 2001).
Zooplankton
Microcrustaceans
- Responses are highly species- and strain-specific. MCs have accumulated in natural zooplankton communities (Ferrão-Filho *et al.*, 2002 and references therein).
- Declines in biomass and altered species composition of zooplankton communities have occurred during blooms, attributed to difficulty in filtering large colonies, indigestibility, poor nutritional quality, and/or toxicity. Large-bodied cladocerans (e.g., larger *Daphnia* spp.) seem more susceptible than smaller species (e.g., *Ceriodaphnia reticulata, Bosmina longirostris*), perhaps because their feeding behavior may limit their ability to avoid the toxic cyanobacteria. Unlike cladocerans, copepods appear to feed size-selectively on colonial cyanobacteria, and to avoid toxic strains (Zurawell *et al.*, 2005 and references therein).
- MCs significantly decreased beat rates of thoracic legs, mandibles, foregut, and second antennae. Stimulation of gut muscles led to permanent contraction of the midgut (interfered with digestion, nutrient assimilation, and uptake of ions; caused exhaustion, loss of cell-to-cell contact within the digestive epithelium, and inhibition of protein phosphatases. In a food web study, MCs were found in 80% of all zooplankton samples. Thus, transfer of MCs within the food web occurred, as well as temporary feeding inhibition in *Daphnia* (Zurawell *et al.*, 2005 and references therein).
- Highest *M. aeruginosa* abundance coincided with a low ratio of cladocerans-to-calanoid copepods (Lehman *et al.*, 2010).
- *Acartia tonsa*: Reduced fecundity (Schmidt and Jónasdóttir, 1997).
- *Bosmina longirostris*: Reduced feeding (Jiang *et al.*, 2013; but there was conflicting information depending on the cyanobacterial strain and the zooplankton strain).
- *Bosmina longirostris, Ceriodaphnia quadrangula, Daphnia ambigua, Diaptomus reighardi, Simocephalus serratulus*: Reduced feeding (Fulton and Paerl, 1987).
- *Ceriodaphnia quadrangula*: Reduced feeding (Watanabe *et al.*, 1997).
- *Cyclops vicinus*: Avoided contact with *Microcystis* colonies (Watanabe *et al.*, 1997).
- *Daphnia ambigua*: Reduced feeding (Zhang *et al.*, 2009). Abundance decreased during blooms of toxic *M. aeruginosa* in laboratory experiments (Fulton and Paerl, 1987).
- *Daphnia galeata*: Feeding was inhibited (Rohrlack *et al.*, 2001). Toxic *Microcystis* cells in the midgut caused the epithelium to lose cohesion. Loss of cell-to-cell contact may facilitate MC uptake into the blood (Rohrlack *et al.*, 2005).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
- *Daphnia hyalina, D. pulcaria*: Feeding was inhibited (De Mott *et al.*, 1991; Rohrlack *et al.*, 2001).
- *Daphnia longispina*: Reduced growth and clutch size (Reinikainen *et al.*, 1999; Hietala *et al.*, 1995).
- *Daphnia magna, Moina macrocopa*: Avoided feeding (Yasuno and Sugaya, 1991).
- *Daphnia magna*: Reduced grazing activity (Łotocka, 2001).
- *Daphnia pulex*: Reduced feeding (DeMott, 1999); also exhibited reduced growth, depressed reproduction rate and clutch size (DeMott *et al.*, 1991; Reinikainen *et al.*, 1999); formed ephippia (molted carapaces enclosing one or more fertilized eggs, resistant to harsh conditions; Zurawell *et al.*, 2005 and references therein). Effects differed depending on the zooplankton clone (Hietala *et al.*, 1997). Outcompeted *Bosmina longirostris* in the absence of *M. aeruginosa*, but the competitive outcome was reversed when *M. aeruginosa* was added, and competitive reversal was more pronounced both when more *M. aeruginosa* was added and when the temperature was increased from 20 to 28 °C (Jiang *et al.*, 2014).
- *Daphnia pulicaria*: Feeding was inhibited (Rohrlack *et al.*, 2001).
- *Diaphanosoma*: Did not consume *Microcystis* (Watanabe *et al.*, 1997).
- *Diaptomus reighardi* (and other small copepods): Increased abundance during blooms of toxic *M. aeruginosa* (laboratory experiments; Fulton and Paerl, 1988).
Rotifers
- *Brachionus rubens*: Reduced ingestion rates (Rothhaupt, 1991).
Macrophytes:
- Duckweed (*Spirodela oligorrhiza*): MC-LR levels as low as 10 μg L^{-1} inhibited growth and reduced plant chlorophyll *a* and chlorophyll *b* content. MC-LR also caused a reduction in the number and mass of fronds, and plants concentrated the toxin (Romanowska-Duda and Tarczynska, 2002; de Figueiredo *et al.*, 2004a).
- Coontail: MC-LR at environmentally relevant concentrations (0.1 to 5 μg L^{-1}) decreased the chlorophyll *a*-to-chlorophyll *b* ratio (considered to be a stress reaction that reduces photosynthetic efficiency; de Figueiredo *et al.*, 2004a; Wiegand and Pflugmacher, 2005 and references therein).
- Coontail: Exposure to MC-LR led to enhanced formation of hydrogen peroxide, in turn causing elevation of anti-oxidative enzymes. Elevation of superoxide dismutase (SOD), glutathione peroxidase, and ascorbate peroxidase indirectly indicated formation of ROS and ongoing detoxification in the plants (Ou *et al.*, 2005; Wiegand and Pflugmacher, 2005 and references therein).
- Duckweeds (*Lemna minor, Wolffia arrhiza, Spirodela oligorrhiza*): Reduced growth in the presence of small quantities of MC-LR, usually 5 μg L^{-1} or less (Mitrovic *et al.*, 2005; Saqrane *et al.*, 2007; de Figueiredo *et al.*, 2004a; Wiegand and Pflugmacher, 2005 and references therein).
- Duckweed (*Lemna japonica*): Reciprocal allelopathic responses occurred between axenically cultured *L. japonica* and two toxic strains of *M. aeruginosa*. Exposure to toxic *M. aeruginosa* inhibited *L. japonica* growth, whereas exposure to axenic duckweed increased MC production and also inhibited growth of the cyanobacteria (Jang *et al.*, 2007).
- Elodea, coontail, Eurasian watermilfoil: After 24 hours of exposure to 0.5 μg MC-LR L^{-1}, photosynthesis decreased by 50–90% relative to pre-treatment values, and there was an overall reduction in growth (de Figueiredo *et al.*, 2004a; Wiegand and Pflugmacher, 2005; Zurawell *et al.*, 2005 and references therein).
- Phragmites: After 24 hours of exposure to 0.5 μg MC-LR L^{-1}, photosynthesis decreased by 10%. MC-LR was absorbed especially by the stems and rhizomes, which had elevated soluble GSTs (de Figueiredo *et al.*, 2004a and references therein).
- Java moss: Bioaccumulated high levels of MC-LR (de Figueiredo *et al.*, 2004a, and references therein).
Filamentous Macroalgae:
- *Cladophora fracta*: After 24 hours of exposure to 0.5 μg MC-LR L^{-1}, photosynthesis decreased by 10% (de Figueiredo *et al.*, 2004a; Wiegand and Pflugmacher, 2005 and references therein).
Phytoplankton:
- *Chlorella vulgaris*, *Oocystis marssonii* (chlorophytes), and *Microcystis wesenbergii*: Allelopathic; *M. aeruginosa* inhibited growth (Zak and Kosakowska, 2014; Dunker *et al.*, 2013; and Yang *et al.*, 2014, respectively).
- *Chlamydomonas neglecta* (chlorophyte flagellate): The allelochemical kasumigamide, produced by *M. aeruginosa*, inhibited growth and flagellar movement (Ishida and Murakami, 2000).
- *Chlamydomonas reinhardtii*: Was paralyzed by MC-LR, and settlement to the bottom of the culture vessels was enhanced (Engelke *et al.*, 2003).
- *Nephroselmis olivacea* (chlorophyte flagellate): Reproduction was inhibited by MC-LR (Christoffersen, 1996).
- *Nostoc muscorum, Anabaena* sp. (cyanobacteria): Growth and photosynthesis were inhibited, and cell lysis increased, after exposure to MC-LR for 6 days (Singh *et al.*, 2001).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

- Addition of nontoxic *Planktothrix agardhii* enhanced MC production by *M. aeruginosa* (Sukenik *et al.*, 2002).
- *Peridinium gatunense* (dinoflagellate): Allelopathic; growth and photosynthesis were depressed by *Microcystis* sp. (Sukenik *et al.*, 2002).

Synechococcus elongatus (with *Synechocystis* sp. – Richardson, 2004)

Zooplankton: Macrozooplankton in *Synechococcus* blooms switched to micrograzers as their food resource. This change, along with increased egg production and high hatching success rates, apparently enabled them to maintain their populations (described as a food web effect; Vargo *et al.*, 1996).

Invertebrates: Longstanding blooms in south Florida (Keys, Florida Bay – Lapointe *et al.*, 1994; Butler *et al.*, 1995) have caused widespread loss of spiny lobsters and multiple sponge species (food web effect; see Table 7.1).

Seagrasses: Longstanding blooms in south Florida have exacerbated loss of seagrasses (turtlegrass, shoalgrass, and manateegrass) via light reduction (Phlips *et al.*, 1995, 1999; Hall *et al.*, 1999). "Algal blooms [largely *Synechococcus*] in Florida Bay have had a number of negative impacts on the ecosystem, such as . . . increased light attenuation which has reduced the distribution of seagrass beds" (Berry *et al.*, 2015, p. 362).

***Trichodesmium* spp.**

Zooplankton:

- *Acartia tonsa*: Reduced fecundity; also exhibited lethargy and paralysis (Guo and Tester, 1994).
- *Clausocalanus furcatus*: Reduced feeding (Hawser *et al.*, 1992).
- *Farranula gracilis*: Did not feed (Hawser *et al.*, 1992).
- *Labidocera* sp.: Did not feed (O'Neil and Roman, 1994).
- *Macrosetella gracilis*: Were lethargic (O'Neil and Roman, 1994).
- *Penaeus merguiensis*: Starved (Preston *et al.*, 1998).
- *Temora turbinata*: Reduced feeding (O'Neil and Roman, 1994).
- *Tigriopus californicus*: Did not feed (O'Neil and Roman, 1994).

Diatoms

Toxic *Pseudo-nitzschia* complex, e.g., *P. australis, P. delicatissima* (Lefebvre and Robertson, 2010; Prince *et al.*, 2013)

General: DA is a polar, water-soluble amino acid (rapidly depurated from the digestive tract, within 2–3 days) that interacts with glutamate receptors in the central nervous system, leading to overstimulation of excitable tissues, neurotoxicity, and neuronal cell death (Iverson *et al.*, 1990; Tryphonas *et al.*, 1990; Lefebvre *et al.*, 2007). A short food chain is required to transfer DA efficiently through the food web, such as large immediate consumers with a direct link between toxic *Pseudo-nitzschia* and large predators. A wide array of pelagic fauna as well as some benthic fauna have been found to contain DA (Bargu *et al.*, 2011), including zooplankton, shellfish, crustaceans, worms, marine mammals, and birds. DA has also been measured in sediments. These data demonstrate the stable transfer of DA through the marine pelagic food web and to the benthos (Trainer *et al.*, 2012). Low-level exposure (that is, less than doses that do not cause obvious symptoms) may result in different whole-body responses than high-level exposure. Low-level exposure to DA over several weeks has made zebrafish more sensitive to subsequent exposures. Chronic, low-level (apparently asymptomatic) exposure to DA has caused an immune response and production of a DA-specific antibody in serum (Landsberg *et al.*, 2014 and references therein).

Mammals:

- California sea lion: A chronic neurological syndrome was characterized by epilepsy and abnormal behavior long after the initial exposure, due to lasting damage in the central nervous system and progressive, cumulative effects from seizure propagation (Goldstein *et al.*, 2008). Reproductive failure occurred with increased abortion rates and premature live births (Goldstein *et al.*, 2009), as well as degenerative cardiomyopathy from chronic exposure to DA (Zabka *et al.*, 2009).
- California sea lion, sea otter: Degenerative cardiomyopathy occurred from chronic exposure to DA (Kreuder *et al.*, 2005; Zabka *et al.*, 2009).

Fish:

- In general, toxicity to fish from DA has not been documented, but fish can be important vectors of DA in the food web (Lefebvre *et al.*, 2012). Plankton grazers (e.g., West Coast-Pacific sardine, northern anchovy, jack smelt; Gulf (Gulf Coast) menhaden) can contain high levels of DA. Example: the viscera of northern anchovies associated with mass mortality of California sea lions contained 220 μg DA L^{-1}; Lewitus *et al.*, 2012 and references therein). Non-planktivorous fish such as chub mackerel and jack mackerel have contained detectable levels of DA during *Pseudo-nitzschia* blooms (Busse *et al.*, 2006; Del Rio *et al.*, 2010).
- Diverse benthic fish taxa can also accumulate high levels of DA in association with blooms, partly because as the blooms wane, toxic "marine snow" settles out with dying *Pseudo-nitzschia* cells (Parsons and Dortch, 2002; Bates and Trainer, 2006; Sekula-Wood *et al.*, 2009). Examples: Pacific sanddabs accumulated DA to levels 25-fold greater than the federal regulatory limit for seafood (Kvitek *et al.*, 2008); and eight species of commercially valuable benthic fish species (soles, turbots, halibut) contained detectable levels of DA even during non-bloom periods (Vigilant and Silver, 2007).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Molluscs: Pacific oyster: Reduced the number of hemocytes (*P. multiseries* – Jones *et al.*, 1995a); shell valves closed (Jones *et al.*, 1995b).

Other Invertebrates aside from Zooplankton: Diverse filter-feeding bivalve species accumulated elevated concentrations of DA when in the vicinity of blooms (*P. australis*; predatory Pacific sanddabs contained up to 514 μg DA L^{-1}) (Goldberg, 2003).

Zooplankton: Reduced feeding and reduced fecundity were sustained by the rotifer *Brachionus plicatilis* (*P. multiseries*; Whyte *et al.*, 1996).

Phytoplankton:

- *Akashiwo sanguinea*: Allelopathic; inhibited growth (*P. multiseries*; Xu *et al.*, 2015).
- *Chattonella marina* (toxigenic raphidophycean), *Rhodomonas salina*: Allelopathic; inhibited growth (*P. pungens*; Xu *et al.*, 2015).

Dinoflagellates

Akashiwo sanguinea

Birds: Clark's grebe, Pacific loon, red-throated loon, surf scoter, western grebe – Developed subtle, gross, nonspecific lesions. Affected birds also had slimy yellow-green material on their feathers, which were saturated with water, and they were severely hypothermic (Jessup *et al.*, 2009). Live-stranded birds responded well to rinsing, rehydration, warming, and nutritional supplements using standard treatment protocols for rehabilitating birds oiled with petroleum products (Jessup *et al.*, 2009; Gaydos, 2012).

Zooplankton: Natural assemblage – Total abundance of herbivorous zooplankton was lower within a layer of *A. sanguinea* than in any other depth interval between 0 and 40 m; 96% of all herbivores sampled belonged to zooplankton groups that exhibited patterns of avoidance behavior as indicated by their vertical distributions (Fíedler, 1982).

Microcrustaceans:

- *Acartia tonsa*: Reduced feeding and egg hatching (Fíedler, 1982; Turner *et al.*, 1998).
- *Calanus pacificus*: Reduced feeding (Fíedler, 1982).
- *Paracalanus parvus*: Reduced feeding (Fíedler, 1982).
- *Centropages hamatus*: Survival of nauplii was low when fed *A. sanguinea* in comparison to survival when feeding on benign algal controls (Murray and Marcus, 2002).

Phytoplankton: *A. sanguinea* consumes various microalgal prey (see Table 7.1).

Toxic *Alexandrium* complex: *A. catenella, A. fundyense, A. minutum* (and various other species)

Fish:

- Newly settled winter flounder, larval sheepshead minnow, larval mummichog: Sublethal exposure via consumption of toxin-contaminated copepod prey caused reduced swimming performance. Larval sheepshead minnows also exhibited reduced prey capture and reduced predator avoidance (*A. fundyense*). "Adverse effects on prey capture or predator avoidance may reduce larval survival and facilitate the transmission of STXs through the food web" (Samson *et al.*, 2008, p. 168).
- Cultured post-smolt Atlantic salmon exposed to *A. catenella*: Exhibited convulsions; significant increase in blood levels of sodium, potassium, and chloride; and, in gill tissue, congestion, telangiectasia, blanching epithelia, cell hypertrophy, and lamellar hyperplasia, suggesting disruption of osmoregulatory capacity (Aguilera *et al.*, 2016).

Molluscs:

- Eastern oyster: The adductor muscle was paralyzed from exposure to bloom densities of *A. fundyense*, but there was no significant effect on hemocyte numbers, morphology, or functions. These findings are consistent with known interference of STXs with sodium channel function in neural tissues (Hégaret *et al.*, 2007a).
- Pacific oyster: Reduced pumping and increased pseudofaeces production (*A. catenella*; Dupuy and Sparks, 1967); reduced clearance rate (*A. fundyense*; Lassus *et al.*, 1996); reduced clearance rate with inhibited shell valve activity, and reduced rate of biodeposition (*A. minutum*; Lassus *et al.*, 1999). STXs bioaccumulated, with no significant effect on hemocytes (Hégaret *et al.*, 2007a).

Zooplankton:

- *Acartia clausi*: Decreased fecundity (*A. minutum*; Guisande *et al.*, 2002); delayed development (*A. minutum*; Frangópulos *et al.*, 2000).
- *Acartia tonsa*: Inhibited grazing (*A. fundyense*; Marcoval *et al.*, 2013).
- *Acartia tonsa*: Waterborne chemicals produced by *A. tonsa* caused a 2.5-fold increase in STX production by *A. minutum* in nitrate-rich medium (but not in low nitrate treatments) relative to control cultures without the grazers, and further grazing was inhibited (Selander *et al.*, 2006). The magnitude of grazer-induced STX production was directly proportional to the degree of N availability (Selander *et al.*, 2006; Bergkvist *et al.*, 2008).
- *Acartia tonsa, Eurytemora herdmanii*: Reduced feeding, and STXs bioaccumulated (*A. fundyense*; Teegarden and Cembella, 1996).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

- *Centropages typicus*: Waterborne chemicals from this grazer caused a 20-fold increase in STXs production (similar to the dinoflagellate response to *A. tonsa*, but much more so), versus negligible change in toxin production when exposed to the grazer *Pseudocalanus* sp. (*A. minutum*; Bergkvist *et al.*, 2008).
- *Euterpina acutifrons*: Greatly reduced nauplii activity (*A. minutum*; Bagøien *et al.*, 1996).

Phytoplankton:

- *Thalassiosira* cf. *gravida*: Allelopathic; culture filtrate inhibited growth and nutrient utilization (*A. fundyense;* Lyczkowski and Karp-Boss, 2014).
- *Chaetoceros neogracile* (diatom): Allelopathic; inhibited photosynthesis and decreased cell size (*A. minutum*; Lelong *et al.*, 2011).

Alexandrium monilatum

Molluscs:

- Hooked mussel: Inhibited byssus production (Sievers, 1969).
- Eastern oyster: Shell valves closed (no filtration), shell valve gape decreased, and clearance rate was depressed (May *et al.*, 2010).
- Green mussel: Shell valve gape decreased, and clearance rate was depressed (May *et al.*, 2010).
- Northern quahog: Shell valve gape decreased, and clearance rate was depressed (May *et al.*, 2010).

Alexandrium tamarense

Mammals:

- North Atlantic right whale: Trophic transfer of STXs, accumulated from *A. tamarensis* via zooplankton prey, was suggested to be a factor contributing to failure of a population of this endangered mammal to recover (Doucette *et al.*, 2006a).
- Sea otter: Foraging behavior (food preference, foraging efficiency, and distribution) of this keystone marine predator was altered by toxic *A. tamarense*. Butter clams are preferred prey of sea otters in the southeastern Alaska area. Otters foraged on butter clams in areas of intermediate prey toxicity (200–500 µg STX eq. 100 g^{-1}), but discarded the most toxic body parts. At highly toxic sites (prey toxicity > 500 µg STX eq. 100 g^{-1}), sea otters avoided butter clams and other large, abundant but toxic bivalve mollusc prey, and consumed smaller and/or less abundant, nontoxic prey (Kvitek and Bretz, 2004). Earlier laboratory experiments with analogous findings guided the field research (Kvitek *et al.*, 1991).

Birds:

- Common eider: Avoided toxic blue mussels as prey under field conditions. In laboratory experiments, eider were offered toxic versus nontoxic mussel meats and refused the toxic meats. If force-fed toxic mussel meat, the food was regurgitated almost immediately. "This selective behavior could have long-term implications for the nutrition of the ducks. While ducks would normally choose large mussels low on the shore . . . the presence of red tide in Maine appears to drive the ducks higher up the shore, where they must settle for smaller, less toxic mussels or cease feeding altogether. . . . The ratio of shell to meat is higher, forcing ducks to be less effective predators. In some areas the eiders switch their prey to sea urchins" (Shumway *et al.*, 2003, p. 12, and references therein).
- Shag: Lost equilibrium and staggered, and many vomited sand eel (sand lances) prey that had accumulated STX (Wood and Mason 1968).
- Black oystercatcher: Dropped or rejected mussel meat (levels > 1500 µg 100 g^{-1}) during a bloom of toxic STX dinoflagellates, but did not engage in that behavior when exposed to nontoxic prey. Birds also switched to nontoxic prey and only partly consumed mussel prey with high levels of STXs (Shumway *et al.*, 2003 and references therein).
- Glaucous-winged gull: Initially regurgitated toxic butter clams within 5 minutes of ingestion, whereas nontoxic butter clams were not regurgitated. In experiments, gulls previously conditioned with toxic butter clams refused to eat either toxic or nontoxic butter clams, but ate other bivalve mollusc species. In the field, gulls at a highly toxic site consumed significantly fewer butter clams than at a nontoxic site. Gulls foraging at a toxic site discarded the siphons (major site of toxin storage) of both toxic and nontoxic butter clams, but did not discard the siphons of other bivalve molluscs that were eaten. In contrast, gulls feeding at a nontoxic site did not discard the siphons from butter clams (Shumway *et al.*, 2003 and references therein).
- Cormorants: Recovered birds that were banded and released during *K. brevis* outbreaks were readmitted to the clinic with the same cerebellar ataxa noted during their first admittance within 5 or more days after release, suggesting either no learned response to the presence of toxins in their food source, or a role of aerosol-borne toxins (which they could not avoid; Shumway *et al.*, 2003 and references therein).

Fish:

- Atlantic herring, American pollock, winter flounder, Atlantic salmon, cod: When experimentally dosed, fish exhibited loss of equilibrium (sideways or upside-down swimming), immobilization, and shallow arrhythmic breathing (White, 1981b).
- Larvae of many fish species directly depend on dinoflagellates as food items for the first week of feeding or slightly longer. Experiments based on use of toxic cells as a food source indicated that "the quality, density, and patchiness of prey, and larval behavioral feeding mechanisms, are significant to successful larval feeding process and growth" (Smayda, 1991, p. 284). Larval fish can serve as vectors for biomagnification and food web transfer of the toxins (Smayda, 1991 and references therein).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

- Neurotoxic effects and behavior modification occurred following ingestion of toxic cells (Robineau *et al.*, 1991a, 1991b).
- Atlantic cod, Atlantic herring, Atlantic mackerel, capelin, red sea bream, winter flounder: Larvae that consumed toxic cells exhibited signs of paralysis (swam erratically and sank); red sea bream lost equilibrium and swam on their side, upside down, and/or in circles prior to paralysis. "Such behavioral dysfunction poses a fundamental constraint on searching behavior of fish larvae for food via chemical, acoustical, and/or tactile stimuli" (Smayda, 1991, p. 284, and references therein). Adult fish exhibited mouth gaping (White, 1977).

Mussels:

- Atlantic surfclam: Reduced clearance rate (Lesser and Shumway, 1993).
- Bay scallop: Reduced clearance rate (Lesser and Shumway, 1993).
- Blue mussel: Exhibited shell valve closure, increased mucus production (Shumway and Cucci, 1987); reduced clearance rates (Lesser and Shumway, 1993); inhibited byssus production (Shumway *et al.*, 1987); and altered heart rate (Gainey and Shumway, 1988).
- Butter clam: Were larger and more abundant in high-toxicity areas, and were avoided by sea otters despite the fact that butter clams are usually the otters' preferred prey (explained above), lending support to the premise that STX toxicity provides a refuge for the butter clams from sea otter predation (Kvitek and Bretz, 2004). STXs appear to function as an effective chemical defense for butter clams in areas of high *A. tamarense* toxicity (Kvitek *et al.*, 1991; also see *Mammals* above).
- Farrer's scallop: Inhibited egg hatching and reduced larval survival (Yan *et al.*, 2001).
- Northern quahog: Exhibited shell valve closure (Shumway and Cucci, 1987) and reduced clearance rate (Lesser and Shumway, 1993).
- Pacific oyster: Reduced clearance rate (Lassus *et al.*, 1996).
- Ribbed mussel: Exhibited shell valve closure, reduced clearance rate, and increased mucus production (Shumway and Cucci, 1987); inhibited byssus production (Shumway *et al.*, 1987; note that the northern horsemussel was unaffected).
- Sea scallop: Exhibited shell valve closure, increased mucus production, and violent swimming activity (Shumway and Cucci, 1987); reduced oxygen consumption (Shumway *et al.*, 1985).
- Softshell clam: Exhibited shell valve closure (Shumway and Cucci, 1987), reduced clearance rate (Shumway and Cucci, 1987; Bricelj *et al.*, 1996), impaired burrowing response (Bricelj *et al.*, 1996), and decreased heart rate (Gainey and Shumway, 1988).

Zooplankton: Can accumulate and retain STXs and cause fish kills (White, 1981a).

Microcrustaceans:

- *Acartia hudsonica, Pseudocalanus* sp.: Depressed feeding and paralysis occurred with increasing levels of *A. tamarense* cellular toxicity (Ives, 1985, 1987).
- *Acartia tonsa, Eurytemora herdmanii*: Decreased feeding, and toxins bioaccumulated (Teegarden and Cembella, 1996).
- *Calanus finmarchicus*: Avoided feeding (Turriff *et al.*, 1995).
- *Calanus helgolandicus*: STX production in *A. tamarense* was positively correlated with both presence of the grazers and waterborne cues from them. This zooplankton species has high grazing impact on *A. tamarense*, whereas two other species tested (*Acartia clausi* and *Oithona similis*, with generally low grazing impact on *A. tamarense*) did not stimulate STX production (Wohirab *et al.*, 2010).
- *Calanus helgolandicus, Temora longicornis*: Reduced fecundity (Gill and Harris, 1987).
- *Calanus pacificus*: Depressed feeding (Huntley *et al.*, 1986).
- *Centropages hamatus*: Depressed feeding and fecundity (Turner *et al.*, 1998).

Ciliates:

- *Euplotes affinis*: Inhibited feeding (Johansson, 2000).
- *Favella ehrenbergii*: Reversed ciliary motion and caused abnormal backward swimming (Hansen, 1989).

Heliozoans: *Heterophrys marina* – Reduced growth (Tobiesen, 1991).

Phytoplankton: *A. tamarense* consumes various microalgal prey (see Table 7.1).

Cochlodinium polykrikoides (*Margalefidinium polykrikoides*)

Fish:

- Sheepshead minnow (age 1 week), Atlantic silverside (adults), striped killifish (adults): Gill function was impaired based on epithelial proliferation, with focal areas of fusion of gill lamellae (Gobler *et al.*, 2008).
- Spotted rose snapper: Liver catalase activity and lipid peroxidation decreased when exposed to toxic *C. polykrikoides* as a short-term effect. Fish developed an abnormal mucus secretion on the gills that was directly related to the dinoflagellate cell densities (2 to 4×10^3 cells mL^{-1}). The data suggested that oxidative stress contributes to the ichthyotoxic effect (Dorantes-Aranda *et al.*, 2010).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Molluscs:

- Bay scallop: Growth rates of juvenile survivors were significantly depressed. Gills exhibited hyperplasia; gill tissues near *C. polykrikoides* cells showed inflammation; both gills and the digestive tract hemorrhaged; and there were signs of starvation (Gobler *et al.*, 2008).
- Eastern oyster: Gills of juveniles hemorrhaged and apoptosis occurred, and digestive glands showed severe hemorrhaging and squamation (Gobler *et al.*, 2008).
- Eastern oyster: Larvae were deformed (Ho and Zubkoff, 1979).
- Pacific oyster: Metamorphosis of larvae slowed during blooms (Matsuyama *et al.*, 2001), and calcium uptake was depressed (Ho and Zubkoff, 1979).
- Mediterranean mussel: First feeding by larvae was delayed 12 days in comparison to feeding on control benign algae (Jeong *et al.*, 2004b).

Zooplankton:

- *Acartia tonsa*: Inhibited grazing, including significant reduction in ingestion rates; and impaired reproduction, with depressed egg production rates and smaller egg size when adult females were fed bloom densities of *C. polykrikoides* (Jiang *et al.*, 2009).
- *Acartia omorii*: Egg viability rapidly decreased when adults were fed *C. polykrikoides* (Shin *et al.*, 2003)

Phytoplankton: High grazing rates on cryptophytes (*Rhodomonas salina* and a second unidentified cryptophyte species) have supported significantly more growth than when in the same light regime without prey (maximum specific growth rates 0.324 day^{-1} versus 0.166 day^{-1}, respectively). *C. polykrikoides* has also consumed other small microalgae such as *Isochrysis galbana*, *Heterosigma akashiwo*, and *Amphidinium carterae*. The data suggest that *C. polykrikoides* can substantially affect some algal populations (Jeong *et al.*, 2004a). In addition, *C. polykrikoides* has consumed the cyanobacterium *Synechococcus* sp. (Jeong *et al.*, 2005b).

Bacteria: *C. polykrikoides* consumes bacteria (Seong *et al.*, 2006), and has shown resistance to algicidal bacteria (e.g., *Alteromonas, Pseudoalteromonas*; Imai and Kimura, 2008).

***Gambierdiscus toxicus*, other CTX producers**

General: CTXs accumulate in fish muscle tissue. "The potential adverse effects of carrying a high CTX body burden on the population dynamics of reef fishes are unknown" (Richlen *et al.*, 2012, p. 42).

Mammals: Hawaiian monk seal: Survey of free-ranging animals revealed CTXs in body tissues (liver, brain, and muscle) and blood (Dechraoui *et al.*, 2011).

Fish:

- Bluehead wrasse: Fish fed toxic *Gambierdiscus* cell pellets exhibited altered feeding behavior, erratic swimming, loss of equilibrium/orientation, respiratory distress, and inability to avoid capture, suggesting enhanced susceptibility to predation, which would also increase the rate of toxin concentration and bioaccumulation in coral reef food webs (Davin and Kohler, 1986).
- Coral reef fish: When exposed to a bloom of toxic *Gambierdiscus* in a zoo, several reef fish exhibited unusual "spinning" and "figure eight" swimming behaviors (Goodlet *et al.*, 1994).
- Coney, schoolmaster, mahogany snapper, largemouth bass (fed a ciguatoxic great barracuda as ether-soluble extracts or as ground flesh): Signs of intoxication included skin color variations, inactivity, loss of equilibrium, erratic swimming, jerky feeding movements, and loss of orientation. These signs were usually observed within 24 hours after consuming the toxic tissues, and were evident for up to 76 days. Largemouth bass fed freeze-dried toxic cells of *G. toxicus* exhibited similar signs (Davin *et al.*, 1988).
- Exposure adversely affected fish development during embryonic and larval stages, suggesting the potential for population-level impacts (Richlen *et al.*, 2012, and references therein). CTXs accumulated at highest concentrations in the viscera, including the gonads (Vernoux *et al.*, 1985), and also at relatively high concentrations in the eggs (Colman *et al.*, 2004). Lipophilic toxins in the ovaries were mobilized with fat stores during oogenesis and became available to developing embryos (Ungerer and Thomas, 1996).
- Medaka: CTXs were micro-injected into egg yolk of embryos so as to cause exposure over the course of yolk sac absorption. Exposure elicited dose-dependent hyperkinesis and tachycardia in embryos and, at highest toxin levels, prevented hatching. Larval fish that hatched had spinal curvature and symptoms of intoxication, exacerbated with increasing CTX levels (Edmunds *et al.*, 1999). Developing embryos exposed to purified CTX and toxic barracuda extracts sustained cardiac effects and spinal deformities (Colman *et al.*, 2004).

Karenia brevis

General:

- BTXs bioaccumulate; for example, various fish (e.g., menhaden, pigfish, pinfish, spot, striped mullet) can accumulate BTXs at sufficient levels to cause disease and death of seabird and mammal consumers both during *K. brevis* outbreaks and long afterward (Flewelling *et al.*, 2005; Fire *et al.*, 2008; van Deventer *et al.*, 2012). It can take several

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
days to several weeks for BTXs to be completely depurated, based on laboratory experiments with mammals and fish (Cattet and Geraci, 1993; Hinton and Ramsdell, 2008). Toxins can cause hyperexcitability of affected neurons, in turn negatively influencing control and homeostasis of the target effector organs enervated by these neurons and leading to organ and locomotor dysfunction (Franz and LeClaire, 1989). - Adverse effects of *K. brevis* BTXs have occurred throughout the food web, in both pelagic areas and benthic habitats (Simon and Dauer, 1972, Roberts *et al.*, 1979, Summerson and Peterson, 1990) where BTX was documented (Mendoza *et al.*, 2008). - High toxic cell densities have suppressed clearance rates in benthic suspension-feeding invertebrates, creating a positive feedback for bloom formation. High BTX concentrations have accumulated in the tissues of benthic suspension-feeding invertebrates, which have then been transferred to higher-level consumers (Echevarria *et al.*, 2012). *Mammals*: - Manatee: Toxins have destroyed immune system functioning (Walsh *et al.*, 2015). BTXs and metabolites have persisted in seagrass beds long after blooms subsided and, thus, may have continued to affect grazing manatees (Flewelling *et al.*, 2005). - Bottlenose dolphin: Piscivorous bottlenose dolphins in a region of the west Florida coast that frequently sustains *K. brevis* outbreaks consumed several species of fish which can be BTX vectors (Flewelling *et al.*, 2005; Fire *et al.*, 2008). - Domestic dog: Signs of brevetoxicosis have occurred in areas near blooms; urine samples tested positive for BTXs and signs included heavy salivation, seizures, paralysis, and temporary blindness, but the dogs recovered in one to several weeks (Landsberg *et al.*, 2009 and references therein). - Coyote: Similar signs of brevetoxicosis have occurred as those described above for dogs, in areas near blooms (Castle *et al.*, 2013). *Birds*: At fish kills, sick cormorants, brown pelicans, and seagulls were observed. During prolonged blooms, most assessed piscivorous seabirds tested positive for BTXs and exhibited clinical signs of brevetoxicosis including severe cerebellar ataxis indicated by incoordination, hypermetric gait, inability to stand, slumping of the head, reluctance to fly, seizures, shaking, nasal and oral discharges, tachycardia, labored breathing, depressed reflexes, impaired motor control, atrophied musculature, dehydration, and disorientation (Landsberg *et al.*, 2009 and references therein). Many piscivorous seabirds taken to wildlife rehabilitation centers during a severe bloom tested positive for BTXs and exhibited impaired motor functioning, disorientation, and seizures (Fauquier *et al.*, 2013a), indicating that adverse impacts of BTXs have been vectored through the food web (van Deventer *et al.*, 2012). *Reptiles*: - Sea turtles: Exhibited clinical signs of neurointoxication, and tested positive for BTXs. Some of their stomach contents were finfish remains that were the likely toxin vectors (Fauquier *et al.*, 2013b). - Sea turtles: Exposure to BTXs resulted in swimming in circles, lack of coordination, head bobbing, muscle twitching, and odd, abrupt movements. In more extreme cases, animals exhibited extreme lethargy or coma (Foote *et al.*, 1998; Manire *et al.*, 2013). Sea turtles appeared to have slower BTX clearance rates than mammals (Landsberg *et al.*, 2009). *Fish*: - Impaired swimming behavior; fish also exhibited defecation, regurgitation, fin paralysis, and loss of equilibrium (Landsberg *et al.*, 2009 and references therein). - Killifish: Showed decreased schooling and shoaling behaviors (Salierno, 2005). - Goldfish: Sublethal BTX concentrations caused locomotor dysfunction and temporary hearing loss based on auditory evoked potentials (Lu and Tomchik, 2002). - Silver perch, spotted seatrout: Fish chorusing was significantly higher during years without *K. brevis* blooms than in years with blooms, suggested to be linked to ichthyotoxic effects of BTXs (Indeck *et al.*, 2015). - Sand seatrout: Exhibited altered spatial distribution of spawning aggregations (Walters *et al.*, 2013). *Molluscs*: - Bay scallop: Recruitment failed (Summerson and Peterson, 1990). - Banded tulip, crown conch, lettered olive: Lost muscle control (Roberts *et al.*, 1979). - Eastern oyster, northern quahog: Larval development was protracted by toxic *K. brevis* (10^3 cells mL^{-1}; Leverone *et al.*, 2006). *Zooplankton*: - *Acartia tonsa*: Sustained lethargy and paralysis. - *Calanus pacificus*: Avoided ingestion of *K. brevis* cells, or regurgitated the cells; exhibited rapid heart rate, lethargy, and paralysis (Huntley *et al.*, 1986; Sykes and Huntley, 1987).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Other invertebrates:

- Adverse effects have been documented on the abundance and distribution of a wide range of benthic fauna (e.g., brachiopods, echinoderms, gastropods, and polychaetes; Smith, 1975).
- Bryozoan *Bugula neritina*, sponge *Halicona tubifera*: Clearance rates on benign *Rhodomonas* sp. significantly decreased when *K. brevis* was present. Animals accumulated high levels of BTXs after 4 hours of exposure to *K. brevis*. When they were transferred to filtered seawater, BTX concentrations in their tissues decreased by ~80% (Echevarria *et al.*, 2012).
- Corals (see Appendix A): Apparently bleached during an extended toxic bloom, but recovered fairly quickly (Dupont and Coy, 2008).

Phytoplankton:

- Growth was inhibited from exposure to waterborne compounds from *K. brevis* (exudate from a natural bloom – uncharacterized allelochemical(s), 500–1000 Da, with aromatic functional groups) in co-occurring (competitor) species *Amphora* sp., *Asterionellopsis glacialis* (diatoms), and *Prorocentrum minimum*. These three taxa and a fourth, *Akashiwo* cf. *sanguinea*, were also growth-inhibited when exposed to exudate from a toxic culture of *K. brevis*. The exudates suppressed photosynthetic efficiency and damaged cell membranes of competing phytoplankton, but had no effect on competitor esterase activity and did not limit competitor access to iron (Prince *et al.*, 2008).
- In laboratory experiments, multiple lipophilic substances from *K. brevis* inhibited growth of *Akashiwo sanguinea*, *Asterionellopsis glacialis, Prorocentrum minimum,* and *Skeletonema grethe* (diatom). The two dinoflagellate competitors reacted to only 1 of 6 lipophilic substances, whereas the two diatom taxa reacted to 3 or 4 lipophilic substances. Thus, species varied in susceptibility; in addition, early-stage (lag phase) *S. grethae* cells were more susceptible to allelopathic effects than later growth stages (Poulson *et al.*, 2010).
- In a microcosm experiment with natural plankton assemblages, extracellular extracts from *K. brevis* (2 strains) inhibited growth of some diatom taxa but had no effect on others, suggesting that in a natural setting the importance of *K. brevis* allelochemicals may not be as great as predicted by laboratory studies (Poulson *et al.*, 2010).
- *K. brevis* exhibited high grazing rates on the cyanobacterium *Synechococcus* sp. (Jeong *et al.*, 2005b; Glibert *et al.*, 2009).
- *Amphora* sp., *Asterionellopsis glacialis, Rhizosolenia* sp., *Skeletonema costatum*, and *Thalassiosira pseudonana* (diatoms); and *Prorocentrum mexicanum* (toxigenic dinoflagellate): Allelopathic; inhibited growth (Kubanek *et al.*, 2005; Prince *et al.*, 2008, 2010; Poulson *et al.*, 2010; and Poulson-Ellestad *et al.*, 2014).

Karlodinium veneficum

Fish: In laboratory experiments with juvenile sheepshead minnows and zebra fish, gills showed epithelial necrosis and shortening or loss of secondary lamellae, or clubbing and bridging between secondary lamellae; and extensive cellular hypertrophy and lysis of epithelial and chloride cells (Deeds *et al.*, 2006).

Molluscs:

- Blue mussel: Reduced growth (Nielsen and Strømgren, 1991), reduced clearance rates, and reduced growth in juveniles (Glibert *et al.*, 2007 and references therein). Exposure to a toxic strain also caused increased infiltration of the percentage of phagocytic hemocytes into the digestive gland, and increased hemocyte production of ROSs (Galimany *et al.*, 2008a).
- Eastern oyster: When exposed to *K. veneficum* (10^4 cells in stationary phase mL^{-1}), adults developed mantle and gill lesions. Larvae (age 2 weeks) sustained a severe reduction in motility. Larvae (age several days) became deformed after embryos from freshly spawned animals were immediately exposed to *K. veneficum* (Glibert *et al.*, 2007).
- Eastern oyster, Suminoe oyster (former candidate species for introduction to Chesapeake Bay): Growth rates of spat (age 3–14 days) were severely depressed and organ development apparently was reduced when the spat were fed a toxic strain, in comparison to animals fed nontoxic *K. veneficum*. Effects were worse for Suminoe oyster spat. When exposed to bloom densities for 6 hours daily for 5 days, clearance rates of older juveniles (length 1–2 cm) were severely depressed for both species, again with worse effects for Suminoe oyster (Brownlee *et al.*, 2008).
- Great scallop: Inhibited feeding in post-larvae (Lassus and Berthome, 1988).
- Suminoe oyster: Exhibited severe reduction in motility of larvae; and larvae became deformed after embryos from freshly spawned animals were immediately exposed to toxic *K. veneficum* (10^4 cells mL^{-1}, stationary phase; Glibert *et al.*, 2007).

Zooplankton:

- KTXs depressed grazing capabilities of microzooplankton and copepods (Adolf *et al.*, 2007).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

- Microcrustaceans: *Acartia tonsa* females – Exhibited significantly higher clearance and ingestion rates when fed a nontoxic strain than when given a toxic strain or a mixed diet of toxic + nontoxic strains, suggesting that toxic strains can deter grazing by potential predators (Waggett *et al.*, 2008).
- Rotifers: *Brachionus plicatilis* – Exposure to field-equivalent densities of *K. veneficum* caused a reduction in all life history parameters measured, compared to parameters for control cultures with benign algal prey. Parameters included lifetime egg production, net reproductive rate, finite rate of increase, and intrinsic rate of population increase (Lin *et al.*, 2016).
- Ciliates: *Storeatula major* – KTXs served as a "predation instrument" to immobilize the ciliate prey prior to ingestion (Sheng *et al.*, 2010, 2082). Mixing ciliates with toxic *K. veneficum* caused prey immobilization at rates consistent with KTX potency and dosage. Ciliates that were able to continue swimming but decreased velocity. Swimming velocity of the toxic cells also slowed and they decreased vertical migration, likely to remain near the prey. In contrast, nontoxic cells did not alter their swimming and did not affect ciliate behavior. Separate exposure of ciliates to KTXs also caused ciliate immobilization at rates consistent with potency (Sheng *et al.*, 2010).
- Dinoflagellates: *Oxyrrhis marina* – Consumed mildly toxic *K. veneficum* as prey (Johnson *et al.*, 2003), but see Table 7.1 – the prey became the predator as the KTXs inhibited *O. marina*.

Phytoplankton: Abundant cryptophyte prey can trigger toxic blooms (Adolf *et al.*, 2008), and the prey population can be significantly affected. KTXs aid in prey capture and are used to stun cryptophytes prior to ingestion (Sheng *et al.*, 2010).

Lingulodinium polyedrum

Molluscs: Mediterranean mussel – First feeding by larvae was delayed 8 days in comparison to feeding on control benign algae (cells likely too large; Jeong *et al.*, 2004b).

Phytoplankton: *L. polyedrum* consumes various microalgal prey (see Table 7.1).

***Ostreopsis* spp. (*O. heptagona, O. labens, O. lenticularis, O. mascarenensis, O. siamensis, O. ovata*)** [T – palytoxin, PTX and analogs; ostreocin D; mascarenotoxins – Rhodes, 2011]

Arthropods: Impaired larval development in brine shrimp was thought to result from osmoregulatory dysfunction (Faimali *et al.*, 2011).

Echinoderms: Inhibited sperm motility in sea urchins (Morton *et al.*, 1982). Exposure also caused folding of spines and loss of spines after exposure for 3–4 days (*O. siamensis*), with recovery after 4 months (Shears and Ross, 2010).

Pfiesteria piscicida

Fish: Neurotoxic symptoms and behavior modification in juveniles and adults have occurred in the presence of actively toxic strains. Fish swam erratically, sank, became immobile, and then showed signs of panic, struggled to the water surface, and sank back down. They gulped air at the water surface and appeared to be suffocating, and also were lethargic and showed signs of narcosis (Burkholder and Glasgow, 1997 and references therein).

Molluscs:

- Bay scallop: Reduced ability to close shell valves, and reduced feeding (Springer *et al.*, 2002).
- Eastern oyster: Reduced swimming activity and reduced feeding when given actively toxic *P. piscicida*. Both juveniles and adults displayed significantly more feeding on nontoxic than on actively toxic *P. piscicida* (Springer *et al.*, 2002).

Zooplankton:

- "Semi-natural" microzooplankton assemblage (had been in the laboratory for 1 day to 2 weeks) – During 6-hour incubations, the zooplankton assemblage ingested both toxic and nontoxic *P. piscicida* (tested separately), but assemblage grazing coefficients were significantly lower when fed an actively toxic culture (Stoecker *et al.*, 2002).
- Microcrustaceans: *Acartia tonsa* – In short-term experiments there was no apparent effect on survival, but animals exhibited erratic behavior when fed an actively toxic strain of *P. piscicida*, alone or mixed with nontoxic algae, in comparison to behavior when fed only nontoxic algae (Mallin *et al.*, 1995).

Ciliates:

- *Euplotes vannus, Euplotes woodruffi*: Rapidly grazed nontoxic cultures, but showed no apparent grazing on actively toxic culture (Lewitus *et al.*, 2006).
- *Eutintinnus* sp., strobilidids (length < 20 μm): Abundance declined when exposed to actively toxic *P. piscicida* culture (Stoecker *et al.*, 2002). *Note*: Some ciliates showed comparable grazing activity when fed toxic or nontoxic *P. piscicida*.
- Dinoflagellates: *Oxyrrhis* sp., *Gyrodinium* spp. showed comparable grazing activity when fed toxic or nontoxic *P. piscicida* (Stoecker *et al.*, 2002).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
Pfiesteria shumwayae
Fish:
- Have shown the same neurotoxic symptoms and behavior modification as for *P. piscicida* in the presence of actively toxic strains (Gordon *et al.*, 2002; Burkholder *et al.*, 2005).
- *P. shumwayae* consumed a sterile fish cell line (Parrow *et al.*, 2005) and in those monoxenic cultures, the dinoflagellates produced a small amount of PfTXs (Burkholder *et al.*, 2005).
Molluscs: Eastern oyster juveniles and adults reduced feeding when exposed to actively toxic culture (Shumway *et al.*, 2006).
Prorocentrum micans
Molluscs: *Mytilus galloprovincialis* – First feeding by larvae was delayed 8 days in comparison to feeding on control benign algae (Jeong *et al.*, 2004b).
Zooplankton: Microcrustacean *Centropages hamatus* – Eggs were not produced by adult females fed *P. micans* throughout their life history. In contrast, there was substantial egg production by control animals fed benign algae (Murry and Marcus, 2002).
Phytoplankton:
- *Asterionella japonica, Chaetoceros lauderi*: Inhibited pigment synthesis (Gauthier *et al.*, 1978).
- *Chaetoceros didymus, Skeletonema costatum*: Reduced population growth (Uchida, 1977).
- *Karenia mikimotoi, Skeletonema costatum*: Suppressed population growth (Xiaoqing *et al.*, 2011).
- *Karenia mikimotoi, Skeletonema costatum*: Tested strains of both species were strongly inhibited by *P. micans.* At size:density ratios of 1:1 or 1:10 of *P. micans: S. costatum*, *P. micans* outcompeted *S. costatum*. At a size:density ratio of 1:0.1 of *P. micans*: *K. mikimoitoi*, growth of *K. mikimoitoi* was significantly depressed. Enriched filtrates of *P. micans* exerted similar effects as the presence of *P. micans* cells without direct cell contact, suggesting an allelopathic effect of *P. micans* on the competing species (Ji *et al.*, 2011).
- *P. micans* consumes various microalgal prey (see Table 7.1).
Benthic *Prorocentrum* complex
Fish:
- Limited data suggest that fish in contact with toxic *P. lima* complex cells cease feeding and die from chronic exposure (Ajuzie, 2008).
- European sea bass: When juveniles were fed toxic *P. lima* complex cells, they exhibited stress-related behavior such as hyperactivities (jumps, fast left-right turns, surface swims), poor feeding reflexes, and cessation of feeding in juvenile fish that were exposed to either cell-free medium or whole-cell cultures. Adverse effects from ingesting *P. lima* complex cells along with a commercial fish diet did not manifest for 3 weeks; then fish ceased feeding and became progressively less active. Cultures of this *P. lima* complex strain exuded a strong, repugnant odor. Fish initially rejected clumps of the cells by spitting them out. Affected fish developed swollen gills with lifted epithelium, vacuolated tips, and ruptured lamellae. Secreted mucus "overwhelmingly covered" the respiratory epithelium of primary and secondary gill lamellae, causing the aorta blood to become hypoxic. The livers of affected fish were swollen and congested, with parachymal necrosis and erosion (Ajuzie, 2008).
Molluscs:
- Bay scallop: In laboratory experiments, within 24 hours of exposure, adults bioaccumulated DSP toxin concentrations to levels exceeding commonly accepted regulatory levels. Most toxins were in the viscera (76%) and gonadal tissue (12%). During depuration, rapid release of DSP toxins from scallops indicated that the toxins were poorly bound to all tissues except viscera. There was no scallop death, and feeding inhibition was not observed for juveniles or adults over a 2-week exposure, but absorption efficiency of organic matter was significantly lower when scallops were fed *P. lima* (complex) in comparison to nontoxic diatom *Thalassiosira weisflogii* (Bauder *et al.*, 2001).
- Intact *P. lima* complex cells found in scallop fecal ribbons vegetatively reproduced after gut passage and emergence from the fecal ribbons (Bauder and Cembella, 2000).
- Pacific oyster: Expression of six stress genes depended on *P. lima* (complex) cell density and exposure duration (Romero-Geraldo and Hernandez-Saavedra, 2014).
Zooplankton: Microcrustacean *Paracalanus parvus* reduced feeding (Huntley *et al.*, 1986).
Phytoplankton: Exudates from a clone of the *P. lima* complex inhibited growth of toxigenic dinoflagellates *Coolia monotis*, *Gambierdiscus toxicus*, and *Ostreopsis lenticularis*. In contrast, the clone maintained similar growth rates in response to exudates from the other dinoflagellate species, although it appeared to enter stationary phase earlier than without the exudates (Glibert *et al.*, 2012 and references therein).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
Prorocentrum minimum
Molluscs:
- Bay scallop: Poor growth occurred as well as intestinal pathology (necrosis or absorptive and some basil cells of the digestive diverticula). Hemocytes accumulated throughout the open vascular system, consistent with tissue damage and/or effects of a chemical toxin; and there were systemic immune responses (Hégaret and Wikfors, 2005a; Heil *et al.*, 2005; Wikfors, 2005).
- Bay scallop: Exposure to a toxic strain of *P. minimum* in either growth or senescent phases decreased the degree of shell opening, the amount of biodeposits produced, motility, and byssal-thread attachment. These effects were more severe as *P. minimum* neared senescence. Pathological effects included derangements of scallop digestive tubules and the adductor muscles, and abnormal hemocyte distributions, which were also more severe with senescent *P. minimum* (Li *et al.*, 2012a).
- Blue mussel: As an apparent immune response, mussels responded to *P. minimum* exposure via diapedesis of hemocytes into the intestine. Circulating hemocytes retained hematological and functional properties. Bacteria greatly increased in the intestines of mussels exposed to *P. minimum*; hemocytes appeared to be either overwhelmed by the large number of bacteria, or engaged in an encapsulating response to *P. minimum* cells. When hemocytes reached the intestinal lumina, they underwent apoptosis (Galimany *et al.*, 2008b).
- Eastern oyster: Mixed findings have been reported depending on the *P. minimum* strain and physiological status –
-- *P. minimum* (10^4 cells mL^{-1}) positively affected spat growth (Brownlee *et al.*, 2008).
-- Larvae had poorer survival and lower settling success with *P. minimum* (3×10^3 cells mL^{-1}) in their diet (Wikfors and Smolowitz, 1995).
-- Larvae did not metamorphose and exhibited various developmental and histopathological abnormalities, with transient digestive gland and systemic pathologies (digestive gland epithelial cells accumulated undigested food vacuoles). Juveniles sustained systemic immune responses and poor assimilation of consumed cells. Adult growth was depressed, and *P. minimum* cells were rejected as pseudofeces (Hégaret and Wikfors, 2005b; Wikfors, 2005 and references therein).
-- At high *P. minimum* densities (10^4 cells mL^{-1}), spawning did not occur; also, there was histological damage and reduced growth of larvae and juveniles (Luckenbach *et al.*, 1993). In stages of growth decline, *P. minimum* appeared to be more toxic than when rapidly growing (Wikfors, 2005 and references therein).
- Mediterranean mussel: First feeding by larvae was delayed 4 days in comparison to first feeding on control benign algae (Jeong *et al.*, 2004b).
- Northern quahog: *P. minimum* cells were rejected as pseudofaeces, and reduced feeding and growth were apparent (Wikfors, 2005; Glibert *et al.*, 2012 and references therein).
- Suminoe oyster: Severe reduction in motility occurred after exposure of larvae (age 2 weeks) to a toxic strain of *P. minimum* (Glibert *et al.*, 2007).
Zooplankton:
- *P. minimum* is consumed by various copepods, ciliates, and nanoflagellates (Glibert *et al.*, 2012 and references therein).
- Microcrustacean *Acartia tonsa*: Consumed *P. minimum* with high ingestion rates, but egg production was depressed unless prey were augmented with the benign diatom *Thalassiosira weisflogii*. The *P. minimum* strain apparently was not toxic but also was not nutritionally sufficient (Dam and Colin, 2005).
- Ciliate *Favella ehrenbergii*: *P. minimum* was a poor food source, and was avoided in cultures (Stoecker *et al.*, 1981).
Phytoplankton (also see Table 7.1): *P. minimum* inhibited growth of *P. micans* (Heil *et al.*, 2005).
- *Heterosigma akashiwo*, *Karlodinium veneficum*: Dominance when in mixed culture with *P. minimum* depended on the (molar) N:P ratio. Near or above Redfield proportions (16 or 25), *P. minimum* was dominant, whereas at a N:P ratio of 5, *P. minimum* and *K. veneficum* were co-dominants (Handy *et al.*, 2008).
- *K. veneficum*: *P. minimum* outcompeted *K. veneficum* under all treatments with various N:P ratios using different forms of N, and several light levels. When *Rhodomonas* prey were added, growth of *P. minimum* was inhibited relative to that of *K. veneficum*; when *Synechococcus* prey were added, the cyanobacteria became dominant. Thus, while allelopathy could be important in competitive outcomes involving *P. minimum*, the competitive advantage of *P. minimum* was overcome when *K. veneficum* was given suitable prey, or when a faster-growing species was added as prey (Li *et al.*, 2012b; Glibert *et al.*, 2012).
Bacteria: *P. minimum* consumes bacteria (see Table 7.1).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
Pyrodinium bahamense* var. *compressum* and var. *bahamense [M; STX; toxins bioaccumulate in finfish and shellfish (Usup *et al.*, 2012 and references therein)]
Fish: Southern, checkered, and bandtail puffer fish bioaccumulated STXs from *P. bahamense* (in epidermis, muscle viscera, and ovary tissues) at lethal concentrations for human consumers (Landsberg *et al.*, 2006).
Zooplankton: During a major bloom of *P. bahamense*, numerical abundances of microcrustaceans *Oithona colcarva* and *Acartia tonsa* declined, whereas the abundance of tunicate *Oikopleura dioica* mimicked the pattern of *P. bahamense* var. *bahamense* abundance (Badylak and Phlips, 2008).
Phytoplankton: Picoplankton and diatom cell densities decreased during a major bloom of *P. bahamense* var. *bahamense* (Badylak and Phlips, 2008).
Scrippsiella trochoidea
Molluscs:
- Mediterranean mussel: First feeding by larvae was delayed 12 days in comparison to first feeding on control benign algae (Jeong *et al.*, 2004b).
- Pacific oyster: Clearance rates substantially declined when fed *S. trochoidea* (Bardouil *et al.*, 1993).
Zooplankton (Crustaceans):
- *Calanus helgolandicus*: Reduced feeding (Gill and Harris, 1987).
- *Calanus pacificus*: Females failed to maintain gut fullness when fed *S. trochoidea* (Sykes and Huntley, 1987), and fecundity decreased (Huntley *et al.*, 1986).
- *Centropages hamatus*: Eggs were not produced by adult females when fed *S. trochoidea* throughout their life history. In contrast, there was substantial egg production by control animals fed benign algae (Murray and Marcus, 2002).
Phytoplankton: *S. trochoidea* consumes various microalgal prey (see Table 7.1).
Euglenophytes
Euglena sanguinea
Phytoplankton: Growth of *Gomphonema parvulum* (diatom), *Oocystis polymorpha, Scenedesmus dimorphus* (chlorophytes), and *Microcystis aeruginosa and Planktothrix* sp. was inhibited by < 30 mg EUGL L^{-1} (Zimba *et al.*, 2010).
Haptophytes
Prymnesium parvum
General:
- Massive blooms have occurred in low-salinity coastal and inland waters, or freshwaters with high conductivity. The multiple toxins of *P. parvum* have cytotoxic, ichthyotoxic, neurotoxic, allelopathic, grazer deterrent, and antibacterial activities (Edvardsen and Imai, 2006). They commonly destroy the selective permeability of cell membranes and disrupt ion regulation in gills (Burkholder, 2009 and references therein).
- The toxins may function as defense compounds to prevent herbivory; some research suggests that they have allelopathic roles, but this has not been verified using purified toxins (Manning and La Claire, 2010).
Fish:
- Affected fish typically bleed from the gills and may develop a heavy mucus layer. They often swim slowly, lie on the bottom, gather nearshore or near a fresh source of water, or actively leap onto shore (Burkholder, 2009 and references therein).
- Long-term negative impacts on fish populations: In reservoirs repeatedly affected by *P. parvum* blooms, populations of white bass, white crappie, largemouth bass, bluegill sunfish, carpsucker, freshwater drum, channel catfish, flathead catfish, and blue catfish sustained declines in relative abundance, size structure, or both. The extent of the effect varied depending on the reservoir system and fish species (Van Landeghem *et al.*, 2013).
Zooplankton:
- Crustaceans:
-- *Acartia bifflosa, Eurytemora affinis*: Became inactive, without the zooplankton actually consuming toxic cells, and reproductive success decreased (Sopanen *et al.*, 2006).
-- *Acartia clausi*: Neither feeding nor reproduction occurred (Nejstgaard and Solberg, 1996).
-- *Daphnia magna*: Reduced survival and reproduction (laboratory and field studies; Ureña-Boeck, 2008).
-- *Eurytemora affinis*: Cell-free filtrates negatively affected survivorship (Sopanen *et al.*, 2008).
- Heliozoans: *Heterophrys marina*: Reduced growth (Tobiesen, 1991; Fisterol *et al.*, 2003).
- Ciliates: Cell-free filtrate from a *P. parvum* culture completely suppressed ciliates in a natural plankton community; no ciliates were present at the end of the 6- to 8-day experiments (Fisterol *et al.*, 2003).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Phytoplankton:

- Natural plankton community: Cell-free filtrate from a *P. parvum* culture affected the entire phytoplankton assemblage. Growth of cyanobacteria and dinoflagellates was depressed, and growth of diatoms was completely inhibited so that no live diatom cells were left at the end of the 6- to 8-day experiments (Fisterol *et al.*, 2003).
- *Rhodomonas* cf. *baltica*, *Rhodomonas salina*, *Thalassiosira weissflogii*: Allelopathic; inhibited growth (Granéli and Johansson, 2003; Barreiro *et al.*, 2005; Uronen *et al.*, 2007).
- *Heterocapsa rotundata*: Allelopathic; inhibited motility (Skovgaard and Hansen, 2003).

Bacteria:

- Natural plankton community: Cell-free filtrate from a *P. parvum* culture reduced bacterial production (Fisterol *et al.*, 2003).
- *P. parvum* grazed bacteria during a bloom (3.4–5.8 bacteria cell^{-1} hour^{-1} depending on the depth of the water column; Nygaard and Tobiesen, 1993).

Pelagophyceans

Aureococcus anophagefferens

General: Massive blooms (~4×10^6 cells mL^{-1}); "Impaired physiological processes and trophic transfer as well as trophic dysfunction can accompany [brown tides] in natural communities" (Smayda, 1991, p. 278, and references therein).

Fish: Anchovy – Reduced fecundity; failed to spawn in the next annual cycle following a major bloom (Castro and Cowen, 1989).

Annelids: Polychaete *Streblospio benedicti* – Reduced growth and swimming velocity (Ward *et al.*, 2000).

Molluscs:

- General: Severe detrimental impacts were sustained by suspension-feeding bivalves (bay scallops, blue mussels in particular; Bricelj and Lonsdale, 1997).
- Bay scallop: Decreased feeding (larvae and adults) and feeding efficiency (Tracey, 1988; Bricelj and Kuenstner, 1989); reduced fecundity and larval shell growth (Gallager *et al.*, 1989); reduced adductor weight; and recruitment failed (Bricelj *et al.*, 1987). Mass mortality and recruitment failure resulted in the collapse of an economically valuable scallop industry on eastern Long Island (Bricelj and Kuenster, 1989; Sunda *et al.*, 2006 and references therein).
- Blue mussel: Decreased feeding (larvae and adults) and feeding efficiency, and reduced fecundity; filtering ceased, leading to starvation; reproductive failure occurred (Tracey, 1988; Bricelj and Kuenster, 1989); and inhibited ciliary activity (Gainey and Shumway, 1991). Impacts were strain-dependent (Bricelj *et al.*, 2001), as with other harmful algae.
- Carpet shell clam: Hemocyte function and viability were damaged (Prado-Alvarez *et al.*, 2013).
- Eastern oyster: Reduced clearance rates and size of larvae (Gobler *et al.*, 2013); inhibited gill ciliary activity (Gainey and Shumway, 1991).
- Northern quahog: Reduced growth (juveniles and adults) (Greenfield and Lonsdale, 1997; Greenfield *et al.*, 2004; Padilla *et al.*, 2006), reduced clearance rates (Gobler *et al.*, 2013), decreased feeding efficiency (Tracey, 1988), and inhibited gill ciliary activity (Gainey and Shumway, 1991). There may be subtle adverse, chronic effects on shellfish even when *A. anophagefferens* is present at background cell densities (Greenfield *et al.*, 2004).
- When fed bloom concentrations, larvae developed faster but growth was reduced. Larvae that were fed slowly growing or near-stationary phase cultures exhibited reduced growth and slower development. These impacts may reflect the poor nutritional quality of *A. anophagefferens*, "which could have a lasting legacy through ontogeny" (Padilla *et al.*, 2006, p. 736).
- European flat oyster: Inhibited gill ciliary activity (Gainey and Shumway, 1991).

Zooplankton:

- Microcrustaceans: *Acartia tonsa* – Reduced feeding, growth, fecundity development, and nauplii survival (Durbin and Durbin, 1989; Bricelj and Lonsdale, 1997; Marcoval *et al.*, 2013). Nauplii development was delayed (Smith *et al.*, 2008).
- Ciliates: Populations of ciliated protozoans declined following a major bloom (Marcoval *et al.*, 2013). Net growth rate was negatively correlated with brown tide abundance (field study; Lonsdale *et al.*, 1996).
- *Strombidium* sp.: Population growth did not occur when *Strombidium* was fed only *A. anophagefferens* (Smith *et al.*, 2008; but see Caron *et al.*, 2004). The data indicate that impacts were strain-dependent, as for other harmful algae.
- Dinoflagellates: *Noctiluca scintillans* – Reduced feeding and fecundity (Buskey and Hyatt, 1995).

Aureoumbra lagunensis

General: Massive continuous blooms have occurred for up to eight years in shallow saline lagoons (Buskey *et al.*, 2001).

Fish: Significantly reduced egg hatch rates of red drum and spotted seatrout; and reduced feeding of spotted seatrout larvae occurred at 5–7 days post-hatch (data of J. Holt in Buskey *et al.*, 1996).

(*continued*)

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)

Benthic Invertebrates:

- Blooms have been associated with a substantial decrease in biomass and diversity of benthic invertebrates (Smayda, 1991; Buskey *et al.*, 1996, and references therein).
- Polychaete larvae (*Streblospio benedicti*): Reduced growth rates and swimming speeds (Ward *et al.*, 2000).

Zooplankton:

- Blooms have been linked to large decreases in grazing activity, growth, and egg release rates of mesozooplankton such as *Acartia tonsa*, and decreases in the abundance and grazing rates of microzooplankton (Buskey *et al.*, 1996).
- *Acartia tonsa* reduced fecundity (poor food resource – Buskey and Stockwell 1993); and nauplii exhibited depressed development and lower development and survival of nauplii (Buskey and Hyatt, 1995).
- Reduced abundance of microzooplankton (Buskey and Stockwell, 1993; Buskey *et al.*, 1997).
- There was no growth of the ciliate *Stromibinopsis* sp., the heterotrophic dinoflagellate *Noctiluca scintillans,* or the rotifer *Brachionus plicatilus* when fed this brown tide species (Buskey *et al.*, 1996).

Raphidophyceans

Heterosigma akashiwo

General: "*H. akashiwo* is a remarkably broad-spectrum antagonist against microzooplankton, copepods, benthic larvae, fish, and a poor food source for many benthic invertebrates. . . . Ichthyotoxic flagellates ([such as] *H. akashiwo*) can also be allelopathic against copepods" (Smayda, 1997, p. 1147).

Fish:

- ROS production induced hypersecretion of mucus in fish gills, epithelial lifting, cell necrosis, and alteration of chloride balance (Basti *et al.*, 2016 and references therein).

Molluscs:

- Eastern oysters: Closed their shells partially or totally when exposed to toxic *H. akashiwo* (Hégaret *et al.*, 2007b).
- Exposure to laboratory cultures or blooms significantly increased hepatopancreas lysosomal destabilization rates, which continued to increase even after 7 days of recovery in clean seawater. The data suggest that *H. akashiwo* toxins or other metabolites continued to damage the hepatopancreas, and that even short-term exposures to high cell densities of *H. akashiwo* could have long-term adverse physiological effects. Oyster health may be compromised in areas with repeated *H. akashiwo* blooms (Keppler *et al.*, 2005).

Zooplankton:

- Microzooplankton: When there was high abundance of *H. akashiwo* (40–99% of the phytoplankton assemblage) along with other, larger toxigenic raphidophyceans (*Chattonella subsalsa, Chattonella* cf. *verruculosa*, *Fibrocapsa japonica*), microzooplankton grazed *H. akashiwo* (grazing rates 0.88 to 1.88 day^{-1}, depending on the specific zooplankter and the site) but not the other raphidophyceans. Grazing pressure on *H. akashiwo* may have afforded a competitive advantage for the other raphidophyceans, which were too large to be consumed at high rates by the microzooplankton (Demir *et al.*, 2008).
- Crustaceans:
  - -- *Acartia hudsonica*: Reduced feeding (Tomas and Deacon, 1981).
  - -- *Acartia omorii*: Rejected feed and reduced fecundity (Uye and Takamatsu, 1990).
  - -- *Acartia tonsa*: Reduced feeding (Tomas and Deacon, 1981).
  - -- *Pseudodiaptomus marinus*: Rejected feed, and reduced both fecundity and survival (Uye and Takamatsu, 1990).
- Ciliates:
  - -- Preferential feeding on other prey reduced impacts of *H. akashiwo* toxicity on microzooplankton. "Avoidance of *H. akashiwo* by a major group of grazers would promote bloom formation by reducing *H. akashiwo* mortality and focusing community grazing pressure on potential competitor species" (Graham and Strom, 2010, p. 111).
  - -- *Tintinnopsis tubulosoides, Favella* sp., *Synchaeta cecilia*: Reduced growth (Verity and Stoecker, 1982; Egloff, 1986).
  - -- Ciliates were unable to sustain growth when fed a monoculture of *H. akashiwo*, regardless of *H. akashiwo* cell density (Chang *et al.*, 1990; Black *et al.*, 1991).
- Rotifers:
  - -- *Brachionus plicatilis*: Reduced feeding when exposed to toxic *H. akashiwo*; and the population decreased while *H. akashiwo* grew comparably as controls without rotifers (Xie *et al.*, 2008).
  - -- *Synchaeta cecilia*: Did not consume *H. akashiwo. H. akashiwo* inhibited feeding on other, acceptable algal food at densities as low as 50 cells mL^{-1}, and decreased reproduction at densities > 10^3 cells mL^{-1} (Egloff, 1986).

Table 7.2 (*Continued*)

Organism(s) impacted and reported effect(s)
Phytoplankton:
- *Chaetoceros muelleri, Skeletonema costatum* (diatoms): Allelopathic; inhibited growth (Yamasaki *et al.*, 2007).
- *Skeletonema costatum* (competitor): Was inhibited by high concentrations of *H. akashiwo*-conditioned medium, but was stimulated by low concentrations. *H. akashiwo* may have achieved dominance by producing large amounts of an ectocrine (tannoid?) that inhibited *S. costatum* at high concentrations, but was stimulatory at low concentrations (Pratt, 1966).
- *Skeletonema costatum* (competitor): Allelopathic; an uncharacterized polysaccharide-protein complex from *H. akashiwo* inhibited growth (Yamasaki *et al.*, 2009).
- *Skeletonema costatum* (reversal of effects): Allelopathic against *H. akashiwo*; inhibited growth (Yamasaki *et al.*, 2007, 2009, 2012).
Bacteria:
- Consumes bacteria (see Table 7.1).
- Bacterial strain BBB25 significantly promoted growth of *H. akashiwo* and two other toxigenic raphidophycean species, as well as nontoxic algae including two diatoms, a cryptophyte, and a chlorophyte. This strain is a gram-positive, rod-shaped, spore-forming bacterium, closely related to *Bacillus*. The data demonstrate the potential for bacteria to influence *H. akashiwo* bloom formation (Liu *et al.*, 2008).

Note: T- toxigenic, and toxic effects of outbreaks [blooms]; B-other bioactive allelopathic compound(s); low DO-blooms commonly cause anoxia/hypoxia; Str-structural feature of the algal cell causes impacts, e.g., needle-like extensions; F-freshwater, Br-brackish, M-marine. Taxonomy basis, acronyms, and other terms are defined in Table 7.1. See Appendix A for scientific names if not included here. Information on habitats (fresh to marine), and on toxins (T) and other bioactive substances (B) is given for taxa not included in Table 7.1.

[a] The general phytoplankton group is indicated for each taxon when the genus name is first mentioned, unless the group for the genus was given in Table 7.1.

[b] Although there has been some question as to whether CYL from *Cylindrospermopsis raciborskii* bioaccumulates, bioaccumulation is now generally accepted (see http://nas.er.usgs.gov/queries/GreatLakes/FactSheet.aspx?SpeciesID=2651 and http://www.glerl.noaa.gov/res/HABs_and_Hypoxia/cylindro_factsheet.html from the U.S. Geological Survey [USGS] and the National Oceanic and Atmospheric Administration [NOAA], respectively; and Kinnear, 2010 – but see Sotton *et al.* 2015).

[c] After 2 weeks of accumulation, CYL distribution was as follows: haemolymph, 68.1%; viscera, 23.3%; foot and gonad, 7.7%; and mantle, 0.9%. CYL was not detected in gills or adductor muscle. Following 2 weeks of depuration, ~50% of the toxin still remained in the tissues (Metcalf *et al.*, 2004).

7.2 Approaches, Pitfalls, Progress, and Goals

Harmful algal toxins have been shown experimentally to be cytotoxic, genotoxic, mutagenic, teratogenic, pathogenic, and/or immunosuppressive, but there are many uncertainties about how these toxins actually operate in natural exposures at environmentally relevant field concentrations. Most research on the impacts of harmful algae in food webs consists of correlative fieldwork, or controlled experiments testing effects of a purified algal toxin or cell extracts on a representative species from one trophic level. The field approach is limited in interpretative power due to potentially confounding factors, while the laboratory experimental approach has limitations because of the highly artificial nature of the setting.

In general when biological effects from exposure to purified dissolved toxins are compared to effects from whole-cell extracts, the effects of whole-cell extracts can be much worse (Palikova *et al.*, 1998; Oberemm *et al.*, 1999), perhaps due to synergistic effects among multiple toxins and/or cofactors that are present (Burkholder and Glibert, 2006; Burkholder, 2009 and references therein). Thus, caution has been recommended in interpretations extrapolated from purified toxin tests to natural settings. In addition to how the toxin is administered, the exposure route is especially important in controlling organismal responses. In general, toxin exposure via the surrounding medium results in much less effect, or no mortality, than the same lethal dose applied orally (e.g., Tencalla *et al.*, 1994).

As a few of many examples of this phenomenon, toxic substances from ingested algae (intact cells)

are adsorbed through the zooplankton gut, whereas the animals typically are exposed to toxins experimentally via the water column and must absorb them through the carapace (DeMott and Dhawale, 1995). Toxins are often injected into mouse or rat models intraperitoneally (IP) or intracoelomically (IC), but such approaches do not simulate how terrestrial or aquatic fauna are exposed to toxins (e.g., STXs) under natural field conditions (Lefebvre *et al.*, 2007). Recent work has indicated that orally dosed Coho salmon, for example, do not exhibit behavioral changes such as those displayed by IC-dosed fish (Lefebvre *et al.*, 2007). Experiments to simulate more natural conditions are challenging because phytoplankton assemblages, including mostly monospecific blooms, commonly show high spatial/temporal variation in biomass, species composition, and the ratio of toxic to nontoxic strains (Zurawell *et al.*, 2005; Gobler *et al.*, 2016 and references therein). The lack of knowledge, even for acute levels of harmful algal toxins, is still so gaping that even the well-funded research area on Florida red tides involving *Karenia brevis* was described as lacking controlled experiments to assess the level of BTXs that is lethal to ecologically and commercially valuable species in the food web (Landsberg *et al.*, 2009).

Erroneous conclusions have resulted from use of one strain to generalize about toxicity of an entire species or genus (see Burkholder and Glibert, 2006; Burkholder and Marshall, 2012 and references therein; and see Glibert and Burkholder, 2018 – Chapter 1). In addition to variable toxicity, other strain-specific characteristics can dramatically alter results and conclusions. For example, differences in ingestibility of toxic *Microcystis* strains by zooplankton can influence the overall herbivore ingestion rate of cells and colonies, and the amount of toxin consumed (Rohrlack *et al.*, 1999). Many toxic algae harbor multiple toxins within a single cell, the proportion of which changes depending on the season and other environmental factors (Burkholder and Glibert, 2006 and references therein). The occurrence and proportion of other toxins in a strain at a given time can influence the outcome of an experiment focusing on only one type of toxin. For example, *Microcystis* strains differ in toxic oligopeptide content (Weckesser *et al.*, 1996), which can influence toxic effects attributed to MCs (Rohrlack *et al.*, 1999). Compounding intraspecific variation in harmful algae are differences among target populations such as zooplankton. Clonal differences have been shown in the sensitivity of a given zooplankton species to toxic algae (Zurawell *et al.*, 2005 and references therein). Some researchers do not verify that a strain is actually toxic, or use conditions that depress toxic activity, prior to conducting "toxicity experiments" using that strain(!) (see discussion in Burkholder and Marshall, 2012 and references therein). This problem can result in erroneous information about fundamental traits such as toxicity. Use of a strain that was verified to be toxic months or years ago is insufficient, considering that toxicity is commonly diminished or lost over time in culture (Burkholder *et al.*, 2005 and references therein).

Over the past few decades, bioassays have progressed from those that detected bioactivity with little concern about ecological relevance, to ecologically relevant tests about possible functions of phytoplankton toxins (Ianora *et al.*, 2011a and references therein). A major impediment in conducting such studies is that often the natural concentrations of a toxic substance and, thus, the concentration range that should be tested, are not known. In addition, concentrations of toxic substances from a given algal species can vary both within a region and geographically (Selander *et al.*, 2006). As an example of progress, Buttino *et al.* (2008) examined use of liposomes as a delivery system for assessment of toxic effects on copepods. Liposomes within the same size range as the food ingested by copepods were prepared and encapsulated with decadienal to assess the effects of polyunsaturated aldehydes (PUAs) on reproduction of two copepod species. Exposure via liposomes reduced egg hatching success and female survival, with concomitant appearance of apoptosis in both embryos and female tissues, and the concentrations of decadienal that induced blockage of cell divisions were tenfold lower than those used in classical feeding experiments (e.g., Ianora *et al.*, 2004). Use of liposomes for delivery of toxins in feeding trials is promising as a more realistic way of delivering a known quantity of toxin (Caldwell *et al.*, 2004). In further advancement, technologies are being developed to co-encapsulate nutritional substances such as amino acids and fatty acids together with the test substance to simulate algal cells more closely (Ianora *et al.*, 2011a).

Beyond improvements in fundamental experimental approaches, significant milestones in understanding food web- and ecosystem-level impacts of HAB will require much more information about chronic/sublethal effects, such as variations in toxin mixtures and concentrations during and after blooms; the presence and extent of *lag*

effects from exposure to blooms that cause disease or death; *indirect effects* of blooms in adversely affecting biota; *cascading effects*; and long-term impacts of repeated exposures on populations at different trophic levels. A major challenge in this research is that trophic transfer of algal toxins in food webs is complex – it can involve many biota at various trophic levels, affecting both pelagic and benthic habitats, and may differentially affect organisms in different life stages. Thus, it has been described as "far more complicated than originally conceived," even for longstanding areas of research such as BTXs in Florida red tides (Landsberg *et al.*, 2009, p. 598). Progress is beginning to be made in the area of toxin transfer through multiple levels in food webs. Lethal lag effects to fauna from higher trophic levels, for example, recently were demonstrated months after *K. brevis* bloom exposures, and significant mortalities from *K. brevis* blooms were revealed from cascading ("domino") effects (Landsberg *et al.*, 2009). Such information is still well beyond reach in the present status of research about most harmful algae, but it can be considered as a future goal.

7.3 High-Biomass Algal Blooms

There are hundreds of published examples of high-biomass, nontoxic microalgal and macroalgal blooms, some described throughout this compendium. Various species of microalgae and macroalgae respond to nutrient pollution (cultural eutrophication) by forming high-biomass blooms (Glibert and Burkholder, 2018a – Chapter 1; and Chapter 15). The most common in freshwaters are planktonic cyanobacteria outbreaks (e.g., *Microcystis aeruginosa* and various other toxic taxa, visible in satellite imagery as covering most of Lake Erie seasonally – Burkholder *et al.*, 2018; filamentous benthic macroalgal cyanobacteria blooms – e.g., *Lyngbya wollei*; and filamentous green macroalgal blooms – Chlorophyta; e.g., *Cladophora glomerata*). Brackish inland habitats have been affected by filamentous green macroalgal blooms (*Ulva* spp., now including species within the genus formerly known as *Enteromorpha* Hayden *et al.*, 2003). Estuarine and coastal marine habitats are characterized by much more diverse microalgal taxa such as dinoflagellates, haptophytes, and diatoms and pelagiophyceans (Dinophyta, Haptophyta, and Heterokontophyta, respectively; Graham *et al.*, 2016) (Burkholder, 1998). They also sustain often-massive, high-biomass blooms of a wide array of filamentous and thallose macroalgae, mostly various greens (e.g., *Ulva*, *Codium*), reds (Rhodophyta – e.g., *Laurencia* spp., *Gracilaria* spp.), and browns (Phaeophyceae, Heterokontophyta – e.g., *Sargassum muticum*, *Pilayella littoralis*) that can cause regime shifts whereby the entire ecosystem is disrupted and readjusts at an altered, more degraded stable state (see Chapter 15, and below). Algae mostly form high-biomass blooms in shallow, poorly flushed embayments and lagoons, but some, such as the microalgal haptophyte *Emiliana huxleyi*, can also be highly productive in open-ocean waters. This species can form massive (10^4 to 10^5 km^2), nearly monospecific seasonal blooms that are visible in satellite imagery, and it is estimated to produce nearly half of the Earth's atmospheric oxygen (Seyedsayamdost *et al.*, 2011a, 2011b and references therein).

High nutrient regimes, especially enrichment with inorganic nitrogen (N) and phosphorus (P), can select for noxious or "weedy," rapidly growing species that can tolerate associated adverse changes in environmental conditions. The excessive inputs of nutrients and high-biomass production can shift aquatic ecosystems out of balance within a season or over longer periods (Burkholder and Glibert, 2013 and references therein). Due to photosynthesis, the water commonly becomes supersaturated with dissolved oxygen (DO). *Supersaturation* develops from algal blooms when the temperature changes too rapidly to allow the oxygen being produced by abundant algae to equilibrate with the overlying air (YSI Environmental, Inc., 2009).

During the night, in contrast, the high respiration rate of the excessive biomass leads to *hypoxic or anoxic* conditions (low DO or no DO, respectively; Junk, 1973; Lyons *et al.*, 2014). The DO "sag" is usually most extreme for aquatic life just before dawn. Low-oxygen stress is described as "one of the most important consequences of high anthropogenic nutrient loadings" because it decreases the amount and suitability of habitat for many beneficial organisms (Breitburg *et al.*, 2003, p. 280). Low DO commonly reduces the abundance and distribution of fish and invertebrates (Kramer, 1987; Diaz and Rosenberg, 1995; Breitburg, 2002 and references therein), with an overall impact of completely altering trophic pathways within food webs – that is, a shift in the fundamental functions and energy dynamics of the food web (Breitburg *et al.*, 1999; Diaz,

2001). Unlike many animal species that cannot withstand repeated periods of hypoxia without severe physiological stress and death (Burkholder and Glibert, 2013), many algae can survive such conditions if alleviated during daylight hours (Lewin, 1962; Wetzel, 2001). The extreme changes over a diel cycle, from supersaturation to hypoxia/anoxia, result in large diel variations or "swings" that have also been shown to be detrimental to sensitive aquatic life (Breitburg, 2002; Morgan *et al.*, 2006; Izagirre *et al.*, 2007; Wilcock *et al.*, 2010). These diel DO swings are accompanied by diel variation in pH, with daytime increases from photosynthetic CO_2 consumption (see Chapter 1).

Degradation of high-biomass algal blooms can also deplete DO (Valiela *et al.*, 1992; Duarte, 1995) and lead to multiple-species kills (Zingone and Enevoldsen, 2000; Dodds, 2006). Most microbial decomposers use oxygen to decompose the remains of dead organisms and release dissolved nutrients back into the system. Increased respiration from stimulation of bacteria, fungi, and protozoans can result in, or contribute to, increased diel DO flux (Dodds, 2006). Algal bloom-dominated systems additionally sustain inputs of large amounts of labile organic matter when the algae die and decompose periodically due to self-shading, other stressors, and seasonal growth patterns (e.g., Havens *et al.*, 2001 and references therein). The algae generally release labile (readily biologically available) nutrients rapidly during decomposition, which promote additional outbreaks when conditions become favorable (Buchsbaum *et al.*, 1991; Havens *et al.*, 2001; Gao *et al.*, 2013).

Thus, high-biomass algal blooms stimulated by nutrient pollution typically cause major hypoxia/anoxia due to their respiration, death, and decomposition (Chorus and Bartram, 1990; Valiela *et al.*, 1992, 1997; Sfriso and Marcomini, 1997; Howarth *et al.*, 2001; Teichberg *et al.*, 2010 and references therein). Many adverse effects have been documented from overgrowth of both microalgae and macroalgae, and their subsequent death and decay:

- Noxious algal overgrowth (micro- or macro-) imparts substantial biotic turbidity, decreasing or virtually eliminating the light needed by beneficial benthic flora (Valiela *et al.*, 1997; Hauxwell *et al.*, 2001). If the growth blankets the water surface, it can block oxygen diffusion into the water from the overlying air and exacerbate low-oxygen stress for aquatic life in the water below. The noxious algae can form thick, slimy masses over beneficial plants, leading to their death (Havens *et al.*, 2001). Loss of beneficial plants such as freshwater eelgrass or seagrass meadows translates, in turn, into disappearance of the critical habitat they provided for fish and other aquatic organisms (Lembi, 2003; Burkholder *et al.*, 2007). Habitat quality becomes compromised, as the algal overgrowth can otherwise alter bottom habitat so that beneficial fauna can no longer use it for spawning and recruitment (Wennhage and Pihl, 1994; Pihl *et al.*, 1995; Thomsen *et al.*, 2006).
- Massive dead/dying algal biomass sinks down to the bottom of the aquatic system at the end of a bloom, depleting the oxygen in the lower water column via respiration and decomposition processes. Living filamentous algal biomass, for example, can increase sulfide concentrations by creating hypoxic/anoxic areas at night from respiration, leading to loss of beneficial seagrass (Holmer and Nielsen, 2007). Dying and rotting algal masses emit strong, foul odors from hydrogen sulfide (H_2S; e.g., Pryor *et al.*, 2007; Green, 2011 and references therein). As Bagarinao (1992, p. 22) wrote, "sulfide is more than just a disagreeable odor from a stagnant marsh; it is a serious menace to all aerobic organisms" because it is toxic to many biota. High sulfide concentrations in the water column have been implicated in mass mortalities of fish and other aquatic life (Shumway *et al.*, 1983; Bagarinao, 1992 and references therein). Sulfide acts as a neurotoxin in mammals: it inhibits cytochrome c oxidase and oxidative phosphorylation, leading to histotoxic hypoxia and loss of energy; it inhibits many other enzymes, causing various metabolic impairments; and it generates reactive radicals and alters membrane permeability, causing edema and organ-specific dysfunction (Bagarinao, 1992; Reiffenstein *et al.*, 1992 and references therein).
- Hypoxia and anoxia promote increased solubility of toxic metals and their release from bottom sediments into the overlying water (Stumm and Morgan, 1996; Mitch and Gosselink, 2007), where beneficial aquatic life can be more easily exposed to them.
- Nutrient transformation processes are dramatically altered by low-oxygen conditions. For example, the capacity of sediments to bind and hold phosphorus (P) is greatly reduced under hypoxia/anoxia (Wetzel, 2001), so that much more P is released to the overlying water

to fuel more blooms. These feedbacks create persistent internal P loading even if external nutrient loads are reduced (Glibert *et al.*, 2011 and references therein). In addition, denitrification, and loss of nitrogen (N) from aquatic systems as N_2 (g), cannot occur in anoxic areas, and decomposition of organic remains becomes much slower (Wetzel, 2001).

- Many invertebrate species (D'Avanzo and Kremer, 1994; Diaz and Rosenberg, 1995; Gray *et al.*, 2002), as well as fish species and their young (Diaz and Rosenberg, 1995; Breitburg, 2002), are killed by hypoxia;
- Fish and macroinvertebrate foraging efficiencies are impaired, and prey abundance is reduced or otherwise adversely altered due to declines in available DO (Pihl *et al.*, 1991, 1995; Osterling and Pihl, 2001; Gray *et al.*, 2002);
- Foraging by some bird species declines because algal mats and scums are avoided (Cabral *et al.*, 1999; Green *et al.*, 2013);
- Fish recruitment and growth are reduced (Wennhage and Pihl, 1994, Shimps *et al.*, 2005) and fish are physiologically stressed, becoming prone to disease (Gray *et al.*, 2002; Stouder and McMullin, 2006 and references therein);
- Decaying blooms provide a substantial source of organic carbon and nutrients to the microbial loop (Karjalainen *et al.*, 2007); and
- Species diversity declines: Subsequent major, adverse changes occur in the trophic structure of invertebrates, birds, and fishes (Raffaelli *et al.*, 1989, 1991; Bolam *et al.*, 2000; Diaz, 2001). The habitat becomes unsuitable for many fish and benthic fauna, which move out of the area or die and exacerbate the hypoxic/anoxic conditions through their decomposition. Over time, the ecosystem becomes characterized by high abundance of relatively few, pollution-tolerant species, including various exotic/invasive taxa, while many beneficial species are lost (Burkholder and Glibert, 2013; Lyons *et al.*, 2014 and references therein). The loss of biodiversity has cascading effects on many ecological processes, such as bioturbation, nutrient generation, and invasion resistance (Solan *et al.*, 2004; Ieno *et al.*, 2006; Stachowicz *et al.*, 2007; Viaroli *et al.*, 2008).

Attempts at ecosystem-level restoration can be impeded by high-biomass algal blooms through diverse mechanisms. For example, blooms of the toxigenic dinoflagellate *Prorocentrum minimum* overlap the period of oyster spawning (Glibert *et al.*, 2007), and can reduce survival of early life history stages of oysters and decrease recruitment. The dense blooms can also decrease survival of beneficial submersed aquatic vegetation through lower light availability that, in turn, affects habitat suitability for many species (Glibert *et al.*, 2011). Thus, impaired physiological processes, impaired trophic transfer, and trophic dysfunction have occurred even in the absence of mortality for some biota.

High-biomass noxious algal blooms can severely alter or reduce ecosystem function by changing the energy flow within food webs. They tend to recur in affected ecosystems, such as blooms of various planktonic, toxigenic cyanobacteria (lower Great Lakes; e.g., Gobler *et al.*, 2016 and references therein); the toxigenic haptophyte *Prymnesium parvum* (certain rivers of Texas, U.S.; Van Landeghem *et al.*, 2013); the toxigenic dinoflagellates *Karlodinium veneficum* (Chesapeake Bay, U.S.; Li *et al.*, 2015) and *Karenia brevis* (west Florida coast, U.S.; Landsberg *et al.*, 2009); the nontoxic, benthic, filamentous freshwater macroalgae *Lyngbya wollei* (cyanobacterium; various freshwaters as described by Bridgeman and Penamon, 2010 and references therein) and *Cladophora glomerata* (filamentous green macroalga; lower Great Lakes – Higgins *et al.*, 2008); and the thallose marine macroalgae *Gracilaria tikvahiae* (e.g., Hawaiian Islands; Anonymous, 2001), *Codium fragile* ssp. *tomentosoides* (e.g., New England coastal waters; Provan *et al.*, 2007), and *Sargassum muticum* (e.g., northwestern U.S.; Britton-Simmons, 2004 and references therein). Blooms can remain or recur in a given environment for days to years to decades depending on the species and conditions (e.g., Valiela *et al.*, 1997; Buskey *et al.*, 2001; Landsberg *et al.*, 2009; Van Landeghem *et al.*, 2013).

In extreme cases, *regime shifts* can occur which involve high-biomass blooms in ecosystems that have sustained high nutrient pollution (also see Chapter 15). A regime shift is an abrupt shift in the biota of an ecosystem in response to a physical/chemical driver (Collie *et al.*, 2004; deYoung *et al.*, 2008; Kraberg *et al.*, 2011). Ecosystems can have more than one state with a self-stabilizing mechanism; a shift between states does not occur frequently and is not readily reversible (Genkai-Kato, 2011). Most often in regime shifts, abrupt changes in ecosystem structure are determined by the responses of biota such as algae to abiotic stressors (Collie *et al.*, 2004; Viaroli *et al.*, 2008). Increased intensity of the stressor or perturbation can induce persistent, dramatic changes in the abundance of one or more components of the community, leading to a major change in dominance, energy

pathways, and overall trophic structure (Viaroli *et al.*, 2008; Hershner, 2011). Basically, the ecosystem crosses an "ecological threshold" that causes an abrupt state shift which is difficult to reverse (Carpenter *et al.*, 1999).

Early research on regime shifts involved lakes driven by major nutrient pollution into chronic, high-biomass toxic cyanobacteria blooms (Scheffer *et al.*, 1993; Carpenter *et al.*, 1999 and references therein). Regime shifts in estuarine/marine coastal lagoons and embayments commonly involve high-biomass blooms of macroalgae (Valiella *et al.*, 1997; Viaroli *et al.*, 2008; Osman *et al.*, 2010). Benthic macroalgae often replace rooted vascular plants (macrophytes; Burkholder *et al.*, 2007; Hastings, 2013 and references therein). Shallow macrophyte-free aquatic ecosystems can also undergo regime shifts to undesirable, high-biomass blooms of benthic and/or floating macroalgae (Genkai-Kato *et al.*, 2012). Macroalgal blooms pervasively and fundamentally alter estuarine ecosystems (Valiella *et al.*, 1997). They dominate DO profiles in the water column of shallow estuaries, and thereby strongly control the biogeochemistry of the sediments and benthic community structure (Hauxwell *et al.*, 2001; Havens *et al.*, 2001; Viaroli *et al.*, 2008). In addition, macroalgal blooms tend to last, and can remain in an environment for years to decades (Valiella *et al.*, 1997).

7.4 Emerging Recognition of the Roles of Allelochemicals

Chemical ecology is the study of the production and interaction of bioactive molecules affecting organism behavior and function (Ianora *et al.*, 2011a). Harmful algae are prominent in this field. Algal allelochemicals other than characterized toxins are considered here because the available evidence, although piecemeal and generally sparse, indicates that they are important in aquatic chemical ecology and food web functioning. Although there are literally hundreds of algal *allelochemicals*, with exception of microalgal toxins (below), they are often overlooked in considerations about the ecology and impacts of harmful algae. Because of methodological impediments (e.g., purification of the substances involved, and use of allelochemicals at environmentally relevant concentrations and environmentally relevant routes of exposure), there historically was a controversy about the ecological relevance of allelopathy (Inderjit and Duke, 2003). Nevertheless, many ecological interactions in aquatic ecosystems have been shown to be mediated by secondary metabolites. As examples, they can deter or accelerate feeding by predators, act as settlement cues for larvae, prevent fouling by epiphytes, serve as pheromones in mate-searching behavior, assist in nutrient acquisition, and influence many other functions (Van Alstyne and Paul, 1989; Leão *et al.*, 2009 and references therein).

General groupings of algal secondary metabolites include terpenoids, tannins, phloroglucinol, phenolics (brominated phenols and polyphenolics), fatty acids (simple fatty acids and derivatives), highly oxygenated polyketides, polyethers, unusual amino acids, peptides, alkaloids, various reactive oxygen species (ROSs), and others (Van Alstyne and Paul, 1989; Cabrita *et al.*, 2010 and references therein). The reactive groups in these substances are often aldehydes, acetate, alcohols, or halogens (Cabrita *et al.*, 2010 and references therein). Other, less frequently occurring substances include acrylic acid, lanosol, and laurinterol (Stein and Borden, 1984). In Antarctic marine waters, for example, the bloom-forming haptophyte *Phaeocystis pouchetii* produces acrylic acid, which can comprise up to 8% of its dry weight (Sieburth, 1961). The sodium salt is an allelochemical that inhibits both gram-positive and gram-negative bacteria, with potential effects suggested at higher trophic levels (Sieburth, 1961). Overall, the hundreds of known algal bioactive substances are extremely diverse both structurally and (based on more limited knowledge) functionally, but they share one trait in common: they have no known role in the primary metabolism and other critical metabolic processes of the organisms that produce them (Van Alstyne and Paul, 1989).

Algal bioactive metabolites can have one highly specific function or multiple, simultaneous functions, including roles in chemical defense (antipredator, antibacterial) and/or cell-to-cell signaling (e.g., PUAs of diatoms) (Ianora *et al.*, 2011a and references therein). In describing inducible responses of marine bloom-forming diatoms, haptophytes, and dinoflagellates to grazers, high species-specific variation has been found in the impacts from these inducible chemical defenses,

> ranging from severe physical incapacitation and/or death to no apparent physiological response, depending on predator susceptibility and detoxification capability. Most bioactive compounds are present in very low concentrations, in both the producing organism and the surrounding aqueous medium. . . .

> Bioactivity may be subject to synergistic interactions with other natural and anthropogenic environmental toxicants. Most, if not all phycotoxins are classic secondary metabolites, but many other bioactive metabolites are simple molecules derived from primary metabolism (e.g. PUAs in [benthic and planktonic] diatoms, dimethylsulfoniopropionate (DMSP) in prymnesiophytes [haptophytes]. Producing cells do not seem to suffer physiological impact due to their synthesis. . . . Understanding chemical ecological responses to environmental triggers and chemically mediated species interactions [eventually] will help define crucial chemical and molecular processes that help maintain biodiversity and ecosystem functionality. (Ianora *et al.*, 2011a, p. 1616)

In most cases, it has not yet been possible to identify the specific compounds involved because of their extremely low effective concentrations. Moreover, extrapolation from allelopathic effects in cultures to natural habitats must ensure that the population densities of the organisms and the concentrations of the allelopathic substances are representative of the natural situation, which can be a considerable challenge; and that the possible effect of bacteria and other contaminating microbes has been eliminated. Thus, verification requires axenic cultures, which usually are not maintained in research on harmful algae (Burkholder *et al.*, 2005 and references therein).

7.4.1 Microalgae

Food web impacts of bloom-forming algae fundamentally begin at the microscopic level, but the chemical ecology of the species involved is only beginning to be understood (Cabrita *et al.*, 2010; Ianora *et al.*, 2011a). It involves the production of allelochemicals, which are natural products produced by one species that elicit physiological or behavioral response(s) in another species (Dicke and Sabelis, 1988). These substances generally are secreted secondary metabolites (i.e., not involved in major metabolic pathways) with growth-inhibiting properties, although some are simple molecules derived from primary metabolism (e.g., PUAs and DMSP) (Cabrita *et al.*, 2010). The most potent allelochemicals are the toxins produced by harmful algae, some of which have been chemically characterized. They are the emphasis of most research to date in this subject area, and their impacts are mostly considered in Section 7.5. Many other, as-yet-uncharacterized allelochemicals are also produced by harmful algae (Legrand *et al.*, 2003; Cabrita *et al.*, 2010). Based on a sparse knowledge base (e.g., Granéli *et al.*, 2008), these substances are important in food web interactions because they contribute to growth, reproduction, and chemical defense. The latter function, involving herbivore–prey interactions, is the most commonly invoked but extremely variable depending on the species involved. In the category of inducible algal chemical defenses, high species-specific variation is known in allelochemical effects on grazers, ranging from disease and/or death to no apparent physiological response, depending on predator susceptibility (Ianora *et al.*, 2011a).

Allelopathic properties have been reported for many phytoplankton species, but few studies have obtained data indicating that allelopathic substances at field-relevant concentrations are used by some harmful algae to achieve dominance in natural habitats. In early work, Keating (1977, 1978) isolated seven species of eukaryotic algae and tested them for their effect on cyanobacteria species that were dominant in earlier or later blooms. Culture filtrates (unialgal or axenic) of each tested species had an inhibitory or neutral effect on the growth of species immediately preceding it in the bloom sequence, but also had a stimulatory or neutral effect on species that immediately followed it in the bloom sequence. Similar results were obtained using lake water filtrates obtained when the various species were dominant. Keating (1978) hypothesized that these algal substances are important in phytoplankton assemblage succession. Also in early research, Mason *et al.* (1982) reported that the filamentous freshwater cyanobacterium *Scytonema hofmanni* produces secondary metabolites that inhibit growth of other cyanobacteria. The antibiotic, a halogenated bioactive substance called cyanobacterin ($C_{23}H_{23}O_6Cl$), was isolated and characterized. It has a low molecular weight (430 daltons) and contains γ-lactone and a chlorinated aromatic nucleus. While it inhibited growth of many cyanobacteria, it only minimally affected tested eubacteria and protozoans.

Since that early work, cyanobacteria have been found to produce many bioactive substances (e.g., Borowitzka, 2016 and references therein). Exudates from *Microcystis aeruginosa* were reported to have much higher estrogenic potency than exudates from other tested species of cyanobacteria and green algae (Sychrová *et al.*, 2012). Cyanobacteria can produce dioxins (Haglund *et al.*, 2007), and they are a natural source of polybrominated diphenyl ethers (PBDEs) as well

(Malmvärn *et al.*, 2005), which are known from other sources used by humans as flame retardants (de Wit, 2002). These substances can act as endocrine disruptors, suppress the immune system, and be neurotoxic (Legler, 2008), and they can adversely affect shellfish, finfish, and wildlife (Darnerud, 2003). They are also widespread contaminants in marine mammals (Weijs *et al.*, 2009; Desforges *et al.*, 2012).

Various marine and freshwater diatoms as well as certain other algae, long considered as benign, and some harmful algae such as the haptophyte *Phaeocystis pouchetti*, produce PUAs (Ianora *et al.*, 2011a and references therein). For example, the highly reactive PUA, decadienal, appears to be involved in grazer defense (Miralto *et al.*, 1999; Ianora *et al.*, 2004), other allelopathy (Ribalet *et al.*, 2007), cell-to-cell signaling (Vardi *et al.*, 2006), antibacterial activity (Ribalet *et al.*, 2008; Balestra *et al.*, 2011), and onset of bloom termination (Vardi *et al.*, 2006; Vidoudez and Pohnert, 2008; d'Ippolito *et al.*, 2009; Vidoudez *et al.*, 2011). Laboratory and field observations have indicated that copepod species feeding exclusively on diatoms, or in diatom-dominated blooms, can be heavily compromised, with only a small percentage of their eggs hatching compared to ~90% hatching in post-bloom conditions (Miralto *et al.*, 1999 and references therein). Three aldehydes (all decatrienals) isolated from the predominant diatoms in the blooms (*Skeletonema costatum*, *Thalassiosira rotula*, and *Pseudo-nitzschia delicatissima* [the latter species, also toxigenic]) were found to be responsible for the poor hatching activity. These substances arrested embryonic development in both copepod and sea urchin bioassays (e.g., Miralto *et al.*, 1999). Some PUAs have teratogenic activity, which causes structural deformities in larval stages of organisms exposed to them during gestation, such as fetal growth, retardation, embryo and fetal mortality, and functional impairment due to malformed limbs or organs. As noted by Ianora *et al.* (2011a), these insidious effects would reduce predators' and grazers' overall fitness, which would help to facilitate bloom development by harmful algal species. The PUAs can also adversely affect the growth and physiological performance of other phytoplankton (Ribalet *et al.*, 2007). Another allelochemical, DMSP, can adversely affect some grazers and is especially common among marine flagellates including haptophytes such as the high-biomass bloom former *Emiliania huxleyi*, and dinoflagellates such as *Alexandrium*, *Amphidinium*, *Gonyaulax*, and *Gymnodinium* (Wolfe, 2000; Steinke *et al.*, 2006). This substance was shown to stimulate search behavior for algal prey in the copepod *Temora longicornis* (Steinke *et al.*, 2006). It apparently acts as a chemical cue, indicating inferior or weakened algal prey (Ianora *et al.*, 2011a).

The diatom *Pseudo-nitzschia delicatissima* is renowned for its production of the potent neurotoxin domoic acid (see Section 7.5), but it also can produce hydroxyl-fatty acid, epoxy-fatty acid, and oxoacid allelochemicals that cause reduced hatching, teratogenic effects, growth inhibition, and/or anti-mitotic apoptosis in copepods (e.g., Miralto *et al.*, 1999, described above, and Ianora *et al.*, 2011b). These substances can reduce grazer overall fitness through induced abortions, birth defects, and reduced larval survivorship (Ianora *et al.*, 2011a). Uncharacterized lytic compounds produced by the toxigenic dinoflagellate *Alexandrium tamarense* have caused cell membrane lysis in the microalga *Rhodomonas baltica* (Ma *et al.*, 2009). These lytic substances have been described as large molecules (> 5 kilodaltons), stable over broad temperature and pH ranges, and refractory to bacterial degradation (Ma *et al.*, 2009). Their lytic activity targets both competitor phytoplankton and grazers (Tillmann and Hansen, 2009).

Recent work by Seyedsayamdost *et al.* (2011a, 2011b) exemplifies the progress needed to strengthen understanding about the potentially widespread importance of algal-produced chemicals other than established toxins in food web interactions. The focus was the dynamic relationship between *E. huxleyi* and its bacterial symbiont, *Phaeobacter gallaeciensis*. The bacterium is a member of the roseobacter clade of α-proteobacteria, a large group that comprises up to ~25% of all marine coastal bacteria (Seyedsayamdost *et al.*, 2011b). The symbiosis is mutualistic when the alga is healthy; the bacterial population apparently promotes algal growth by synthesizing and secreting antibiotics and growth stimulants (auxins; Seyedsayamdost *et al.*, 2011a). When *E. huxleyi* senesces, however, it produces a lignin breakdown product, p-coumeric acid (pCA). In what has been described as "Jekyll-and-Hyde" chemistry (Seyedsayamdost *et al.*, 2011a), the bacterium responds to pCA by switching to become an opportunistic pathogen that produces roseobacticides (novel bacterial troponoids) which cause cell lysis and death of *E. huxleyi* (Figure 7.3). Assays with various other estuarine/marine microalgae (haptophyte *Isochrysis* sp., prasinophyte *Tetraselmis suecica*, diatom *Chaetoceros muelleri*, and cryptophyte *Rhodomonas salina*) have shown that the roseobacticides also have specific and

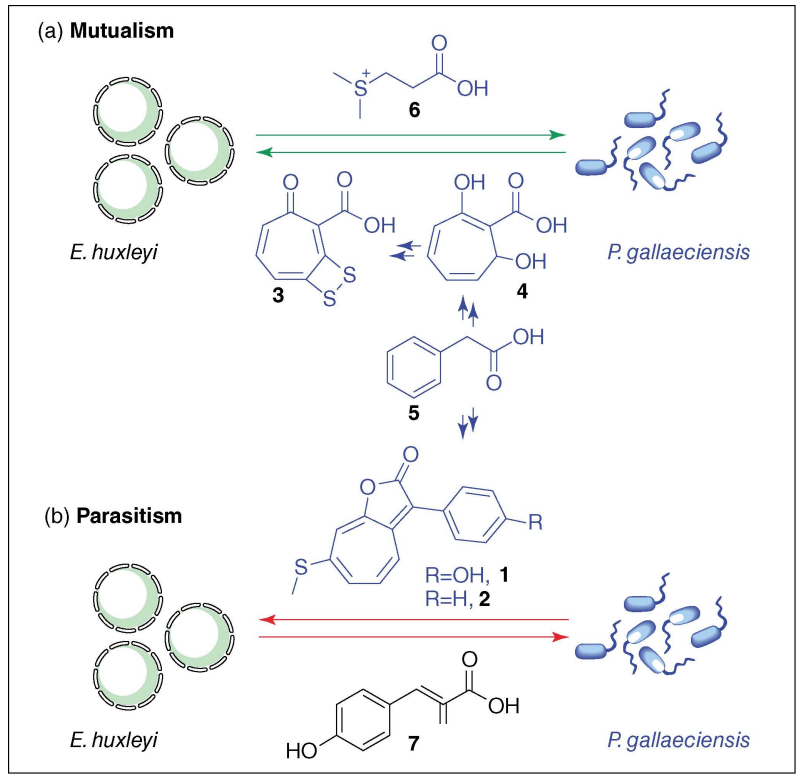

Figure 7.3 Proposed conceptual model from Seyedsayamdost *et al.* (2011a, 2011b) for the dynamic interaction between *P. gallaeciensis* B107 and *Emiliania huxleyi*. The two phases of the interaction are shown by green (mutualistic phase) and red (parasitic phase) arrows. Compounds produced by *P. gallaeciensis* BS107 and *E. huxleyi* are shown in blue and black, respectively.
(a) *Mutualistic phase* of the symbiosis. Under these conditions, the healthy algal host provides DMSP (**6**); DMSP attracts roseobacter (and other) bacteria, which use DMSP as a carbon and sulfur source, and as an attachment surface. Note that the bacteria metabolize DMSP to volatile DMS, which is converted in the atmosphere to condensation nuclei for water droplets. The bacterial symbiont provides the antibiotic tropodithietic acid (TDA, **3**), its precursor (**4**), and the plant growth promoter phenylacetic acid (**5**).
(b) *Parasitic phase* of the symbiosis. When the algal host senesces, it releases p-coumaric acid (pCA, **7**, a lignin component), which elicits the production of the anti-algal roseobacticides (**1, 2**), likely derived from **5**. Roseobacticides **1** and **2** contain a 1-oxaazulan-2-one core, and they can adversely affect marine phytoplankton with nanomolar potency. The roseobacticides arise from variable substituents at the C3 and C7 positions of the roseobacticide core, suggesting that they are produced via modifications and combinations of aromatic amino acids. Note that **5** is likely a precursor to metabolites that are health-promoting in the mutualistic phase (A) and toxic in the parasitic phase (B). Thus, **5** may be a critical player in the switch from mutualism to parasitism. *Source*: Reprinted with permission of the *Journal of the American Chemical Society*.

potent (nanomolar-level) algicidal activity against those algae, suggesting that the mechanism may be common among marine microalgae and roseobacteria (Seyedsayamdost *et al.*, 2011a). Terrestrial plant-associated bacteria respond to lignin components that are released into the surrounding soil when the plants senesce (Schaefer *et al.*, 2008). Considering that point, and the fact that lignin component substances have been found in green, red, and brown macroalgae, Seyedsayamdost *et al.* (2011b) suggested that a similar response may be widespread in marine macroalgal-bacterial interactions.

7.4.2 Thalloid Macroalgae

Allelopathy in thalloid estuarine/marine macroalgae is well known but poorly understood from an ecological perspective except for relatively few compounds, largely because many of the biologically active substances that are produced appear to have

multiple, interactive ecological roles which mostly remain to be elucidated (see review by Van Alstyne and Paul, 1989; and see Chapter 15). Nevertheless, some of the most fascinating information about the roles of allelochemicals has been published about thalloid macroalgae (e.g., Hay, 2009; Van Alstyne *et al.*, 2015 and references therein).

Various brown macroalgae release an array of phlorotannins that have been shown to inhibit growth of epiphytic bacteria and invertebrates (e.g., Sieburth and Conover, 1965), and anti-grazing polyphenolics that are induced by herbivores (e.g., *Fucus distichus*; Van Alstyne, 1988). Perhaps the most "extreme" allelochemical known among brown algae is concentrated sulfuric acid, produced by several species of *Desmarestia* within specially constructed, subcellular vacuoles (Meeuse, 1956; Eppley and Bovell, 1958). If these mostly subtidal species are exposed to the air during low tide, the acid is released, killing both the macroalga and other organisms in the surrounding area (O'Clair and Lindstrom, 2000). Sulfuric acid-laden *Desmarestia* is avoided by grazers, thus serving an important ecological role. For example, sulfuric acid in *Desmarestia* has inhibited grazing by the sea urchin *Strongylocentrotus droebachiensis* (Pelletreau and Muller-Parker, 2002).

Many halogenated (bromine, iodine) aliphatic haloketones and brominated phenols are produced by red macroalgae, with antimicrobial and anti-herbivore functions (Paul *et al.*, 2006), as well as more complex monoterpenes, sesquiterpenes, and diterpenes (up to 5% of the thallus dry mass; Hay and Fenical, 1988). For example, *Laurencia* spp. produce elatol, which can act as a cytotoxin, ichthyotoxin, insecticide, and herbivore deterrent (Hay and Fenical, 1988 and references therein). Species within this genus are known high-biomass bloom formers in response to anthropogenic nutrient over-enrichment (Lapointe *et al.*, 2002). In general, however, as noted by Cabrita *et al.* (2010, p. 2301), "The ecological role of marine algal halogenated metabolites has somehow been overlooked."

Insights about the roles of other macroalgal allelochemicals in food webs have been derived from experiments wherein at least one of the substances involved has been chemically characterized. These experiments indicate that *macroalgal allelochemicals affect marine food webs in many far-reaching ways* (Duffy and Hay, 1990; Hay, 2009; Van Alstyne *et al.*, 2015 and references therein). Species of ulvoid green algae (Chlorophyta, Ulvophyceae), for instance, have been a research focus for decades. Many of them produce allelochemicals with effects that have included reducing barnacle densities in tidepools; causing mortality of larval crabs, oysters, and juvenile abalones; inhibiting growth of planktonic microalgae and other benthic macroalgae; and reducing epiphytism by bacteria, algae, and invertebrates (Van Alstyne *et al.*, 2015 and references therein).

An example is provided here from recent research on the green macroalga *Ulvaria obscura* (e.g., Van Alstyne *et al.*, 2006, 2008, 2011, 2014, 2015). This species is abundant in subtidal "green tide" blooms, especially in urbanized areas of the northwestern U.S. The blooms can adversely affect marine communities, fisheries, and aquaculture. They can result in fragmented seagrass meadows, produce noxious odors, and release allelochemicals that detrimentally affect other algae and invertebrate larvae (Nelson and Lee, 2001; Nelson *et al.*, 2003; Van Alstyne *et al.*, 2011, 2015 and references therein). Among the metabolites produced by *U. obscura* are dopamine (averaging 4.4% of the thallus dry mass in some studies), quinones resulting from dopamine oxidation in seawater, ROSs (van Hees and Van Alstyne, 2013), and dimethyl sulfide (Van Alstyne and Houser, 2003). The dopamine and quinones have reduced growth and germination rates of other marine macroalgae, increased mortality rates of crab zoeae, and depressed feeding by sea urchins (*Strongylocentrotus droebachiensis*), snails (*Littorina sitkana*), and isopods (*Idotea wosnesenskii*) (Van Alstyne *et al.*, 2015 and references therein). Various herbivores avoid or minimize *U. obscura*, which may contribute to its ability to form persistent blooms (Van Alstyne *et al.*, 2006). Ulvoid algae tested thus far also produce DMSP (Van Alstyne *et al.*, 2007; Van Alstyne, 2008), which inhibits growth of epiphytic bacteria (Saha *et al.*, 2014) and can have other allelopathic effects as mentioned.

Recent research has indicated that competition among marine macroalgae can induce allelopathy while also suppressing growth and anti-herbivore defense. After eight days of competition with the coral *Porites cylindrica*, the red macroalga *Galaxaura filamentosa* (described as chemically rich) induced allelochemical release and became nearly twice as damaging to the coral, while also decreasing in growth and increasing in palatability to herbivores (likely due to reduced chemical defenses; Rasher and Hay, 2014). Under the same conditions, the brown macroalga *Sargassum polycystum* did not induce allelopathy, and maintained the same level of growth and palatability. The authors (Rasher and Hay, 2014, p. 1) described their observations on *G. filamentosa* as "the first

demonstration of induced allelopathy in a seaweed, or of competitors reducing seaweed chemical defences against herbivores." They concluded that the nuanced, complex chemical ecology of coral-seaweed-herbivore interactions underscores the need to consider more ecological complexity in studies of chemical defense.

Although some of the earliest research on allelopathy in thalloid macroalgae with substance identification was conducted on freshwater/brackish species, much less is known about allelopathy in those habitats. In classic work by Anthoni *et al.* (1980) and Wium-Andersen *et al.* (1982), the streptophyte *Chara* (Streptophyta, Charales) was shown to produce several low-molecular-weight sulfur compounds with important ecological roles as scent markers, insecticides, and inducers of feeding behavior. This macroalga can be a noxious benthic bloom former in some freshwaters of the western U.S. (Lembi, 2003 and references therein). Two sulfur-containing allelopathic compounds, 4-methylthio-1,2-ditholane and 5-methylthio-1,2,3-trithaine, were isolated and characterized from both freshwater and estuarine charaleans. The purified substances inhibited microalgal photosynthesis (of the benthic, co-occurring diatom *Nitzschia palea*) at a 3 micromolar (μM) concentration. The data suggested that these substances from *Chara* can reduce growth of epiphytic algae, which may explain why *Chara* is seldom found with epiphytes (Wium-Andersen *et al.*, 1982).

7.4.3 Filamentous Mat-Forming Macroalgae

Filamentous marine cyanobacteria were first evaluated for natural products (bioactive secondary metabolites) about 40 years ago, and they have become well known as rich sources of a wide array of bioactive substances (Tan, 2007; Tidgewell *et al.*, 2010). These substances have antibacterial, antifungal, antiviral, anticancer, antiplasmodial, algicidic, antiplatelet aggregation, and immumosuppressive properties (Ramamurthy *et al.*, 2014 and references therein). Allelochemicals from freshwater cyanobacteria are also well known (Chorus and Bartram, 1999), although not as intensively examined as those from marine species.

The cyanobacterial genus *Lyngbya* consists of species that are "prolific producers of secondary metabolites, primarily lipopeptides, cyclic peptides, and depsipeptides" (Sharp *et al.*, 2009, p. 2879). Foremost among them is *Lyngbya majuscula*, which occurs worldwide in tropical and subtropical environments (Tidgewell *et al.*, 2010). This species negatively affects coral larvae recruitment (Kuffner and Paul, 2004) and inhibits potential herbivores using chemical defenses (Paul *et al.*, 2005). More than 180 bioactive substances have been reported from *L. majuscula*, including curacin A (anticancer – Chang *et al.*, 2004) and jamaicamides (neurotoxic – Edwards *et al.*, 2004). Although the number of bioactive substances from *L. majuscula* likely has been overestimated due to contamination by co-occurring microbes (Jones *et al.*, 2011), even so, many bioactive secondary metabolites from this species have been confirmed (Tidgewell *et al.*, 2010). Most have been identified in efforts to find substances with pharmacological or other beneficial health applications (Dixit and Suseela, 2013 and references therein). Their roles in the survival and ecology of *L. majuscula* are poorly understood. Many of the bioactive substances from this cyanobacterium and other mat-forming species in fresh as well as marine waters (e.g., *Oscillatoria* spp., *Phormidium* spp., and freshwater *Lyngbya wollei*) have diverse chemical structures and often include bactericidal and algicidal activities (Priyadarshani and Rath, 2012). These activities are believed to afford an advantage for the cyanobacteria in competition for resources against co-occurring algal and bacterial populations.

Little is known about allelochemical production by other filamentous macroalgae. The noxious, widespread chlorophyte, *Cladophora glomerata* (see Chapter 15), produces various fatty acids (e.g., antibacterial steroids), polyphenols (antioxidants), terpenoids (regenerative), and other bioactive substances, but the ecological roles of these substances are poorly understood (Fabrowska *et al.*, 2015).

7.5 Toxigenic Algae in Aquatic Food Webs

Toxins are regarded as the "ultimate" in bioactive substance potency, and have been best expressed in selected microalgae including species of cyanobacteria, dinoflagellates, haptophytes, diatoms, and raphidophyceans. The term *toxin* (Greek: τοξικόν *toxikon*, first used by organic chemist Ludwig Brieger in the late 1800s) is loosely defined as a poisonous substance produced within living cells or organisms. Toxins can be small molecules (e.g., peptides or proteins) or much larger substances, which can cause disease and/or death upon contact or from absorption by other cells

or organisms. These substances vary greatly in potency, making their distinction from other allelochemicals a "gray area," not well defined. Some researchers use the term when there is clear biological activity toward organisms not found in the same habitat as the toxin producer (Leflaive and Ten-Hage, 2007; Zimba *et al.*, 2010).

The effects of chemically characterized, partially characterized, and uncharacterized (putative) toxins of harmful algae are considered here. Although much information is available on toxigenic algae and their impacts on various organisms (examples are given in Tables 7.1 and 7.2; see also reviews by Shumway, 1990; Landsberg, 2002; and see Chapter 4, and Broadwater *et al.*, 2018 – Chapter 5), few publications have attempted to consider their effects at the level of food webs or ecosystems. Those works are highlighted below. The term toxigenic is best applied to these algae because (1) within a given species known to be capable of producing toxin(s), strains commonly range from benign (with no toxin production), to producers of very small amounts of toxin, to highly toxic strains; and (2) toxic strains often do not consistently express toxicity – they may only express toxicity under certain environmental conditions (Burkholder *et al.*, 2005; Burkholder and Glibert, 2006 and references therein). These characteristics have led to differences and apparent contradictions in the published literature (Ibelings and Havens, 2008). Emphasis here is on findings from research with known toxic strains. Tables 7.1 and 7.2 reflect the highly variable knowledge base, depending on the species; for example, there are well over 100 published studies on effects of the cyanobacterium *Microcystis aeruginosa* and of the dinoflagellate *Karenia brevis* on other biota, but relatively few studies on impacts of most toxigenic raphidophyceans.

Many effects from toxic algae have been described. Their sublethal and chronic impacts – while often subtle or insidious and, therefore, much more challenging to detect and characterize under realistic conditions – are considered more important overall influences on affected populations than obvious, more easily detected, acute effects (Landsberg, 2002; Shumway *et al.*, 2003; Karjalainen *et al.*, 2007). Toxic strains of harmful algae adversely affected species within every trophic level of aquatic ecosystems, ranging from other phytoplankton, benthic algae, and seagrasses to apex predators including carnivorous fish and aquatic birds and mammals (also humans – which, although not aquatic and so not considered here, are surely top predators).

Some interesting generalizations emerge from the studies synthesized in Tables 7.1 and 7.2. Algae produce some of the most potent toxins known (e.g., Oshima *et al.*, 1989). Among the most common of their effects are:

- Damage of the liver or hepatopancreas, kidneys, nervous system, and/or gills, the latter often involving osmoregulatory dysfunction. Some algal toxins cause increased ion permeability and inhibit ATPase activity of sodium and potassium pumps in gills; e.g., Ulitzer and Shilo, 1966; Zambrano and Canelo, 1996);
- Reduced or inhibited growth;
- Reproductive impairment (reduced fitness) – lower fecundity, reduced spawning success, reduced embryo development and survival, depressed larval survival and settlement, deformed young, and lower recruitment; and
- Reduced or inhibited grazing, including avoidance of the toxic algae (but note: a complicating factor in zooplankton studies is that nontoxic cyanobacteria, which are generally considered low-quality food, can induce similar effects as toxic cyanobacteria; Laurén-Määttä *et al.*, 1997); and
- In phototrophs, reduced or inhibited photosynthesis.

Importantly, the adverse impacts of algal toxins are generally worse for young life stages of the affected organisms (Tables 7.1 and 7.2), attributed to factors such as a thin epithelial layer and a relatively large body surface, a high metabolic rate, and limited motility. Damage from the toxins to key developmental processes often leads to death, and the young stages of many organisms are often restricted to nearshore littoral areas where the toxic algae can accumulate (e.g., Oberemm, 1999).

Each of these effects alone could cause significant damage at the population level in a bloom area, and some blooms can cover many square kilometers. Collectively, the effects can be devastating at the population level. Food web-level impacts most commonly reported are the loss of biomass and diversity of phytoplankton assemblages, and of zooplankton or benthic invertebrate grazers. These effects, in turn, modify the structure of higher trophic levels, especially the organisms that had depended on those phytoplankton or grazers as food resources. At the ecosystem level, the most severe adverse effects are caused by toxic algae that are also high-biomass bloom formers (see Tables 7.1 and 7.2 – all planktonic cyanobacterial species listed, as well as the benthic *Lyngbya* spp.; dinoflagellates *Akashiwo sanguinea*,

Ceratium tripos, *Karenia brevis*, *K. veneficum*, *Prorocentrum micans*, and *Prorocentrum minimum*; the haptophyte *Prymnesium parvum*; the raphidophycean *Heterosigma akashiwo*; the euglenophyte *Euglena sanguinea*; and the two brown tide species). Their effects can impair ecosystem structure as well as overall trophic structure, as the toxic effects occur in combination with the loss of critical habitat such as submersed aquatic vegetation from algal overgrowth as explained above.

Food web impacts of harmful algae that *have* been studied are believed to be underestimated. For example, Shumway *et al.* (2010) noted that seabirds are among the most common members of marine food webs, and most likely to consume toxins that have bioaccumulated in other organisms. Sublethal toxin impacts may render birds more vulnerable to other environmental stressors, resulting in mortalities. This potential danger could especially affect migratory species that have spent their energy reserves, and arrive emaciated at toxin-contaminated shellfish beds or encounter schools of toxin-laden fish. In their weakened condition, even a small dose of toxin likely would impair the birds' feeding ability and lead to starvation. Research to verify these effects, however, is sparse. Few experimental studies exist because of difficulties in holding birds in captivity, the logistics of field studies, the unpredictable nature of toxic outbreaks, the short period of some outbreaks, and the lack of human awareness that seabirds might be affected. Also, cause-and-effect is difficult to establish because bird deaths often occur offshore; the carcasses drift into shore and are detected well after the toxic outbreak (see Gibble and Hoover, 2018 – Chapter 6).

While many studies have examined the effects of consumption of toxin-laden live prey, a special case of exposure via food is coprophagy or exposure to toxin-contaminated wastes (feces and pseudofeces), which can provide a mechanism for further transport of the toxins especially to benthic communities (Lehtiniemi *et al.*, 2002; Jang *et al.*, 2004; Svensen *et al.*, 2005). For example, pseudofeces of zebra mussels in Great Lake Erie are rich in cyanobacteria, and they can transfer cyanotoxins to benthic communities (Babcock-Jackson *et al.*, 2002). The same bivalve mollusc can be contaminated by its own toxic feces (e.g., MCs in Mediterranean mussels; Amorim and Vasconcelos, 1999). During and after a toxic bloom, there is no clear pattern in the detoxification, dilution, and dissipation of accumulated toxins (Doucette *et al.*, 2006b). Detoxification rates vary greatly among toxins, species, and tissues. Bacteria contribute to pathways of algal toxin transfer by enhancing or inhibiting toxin production, lysing toxic algal cells, and transforming or degrading toxins (Doucette *et al.*, 2006a). Bacteria can also affect the production of some algal toxins (e.g., DA, STXs, PfTXs), either positively or negatively, and some bacteria can modify certain algal toxins into more, or less, potent substances. In addition, bacterially mediated lysis of toxic algal cells often results in the release of dissolved toxins that can adversely affect other organisms (Doucette *et al.*, 1999, 2006a).

Many direct effects of algal toxins on aquatic biota have been documented in laboratory experiments. The effects in natural habitats are more often modulated by environmental factors or the physiological status of the biota (Ibelings and Havens, 2008). The following five examples illustrate the many indirect as well as direct effects of toxic algal blooms, and the fact that the impacts are largely controlled by environmental conditions and the characteristics of the affected species. It should be noted that, with the exception of *K. brevis*, the species involved occur in other regions as well as those considered in the examples.

7.5.1 Toxic *Microcystis aeruginosa* Blooms across North America

Blooms of *M. aeruginosa* affect eutrophic lakes, rivers, and estuaries that have high nitrogen (N) supplies, and *M. aeruginosa* cells are adept at scavenging low concentrations of inorganic phosphorus (P) above P-rich sediments (Burkholder, 2002, 2009; Davies *et al.*, 2010; O'Neill *et al.*, 2012; Gobler *et al.*, 2016). This species (as well as various other cyanobacteria) produces MCs, of which there are more than 100 congeners (Meriluoto and Spoof, 2008). Depending on the strain, *M. aeruginosa* can synthesize an array of other toxins and other bioactive substances as well (Table 7.1). Changes in MCs and other cyanotoxin concentrations often vary tenfold or more during *M. aeruginosa* blooms (Chorus and Bartram, 1999; Zurawell *et al.*, 2005) as inorganic N concentrations decrease and nontoxic strains gain predominance over toxic strains (reported as *Microcystis* or as *M. aeruginosa*; Briand *et al.*, 2009; Davis *et al.*, 2009, 2010).

The blooms typically are high biomass, although small blooms can be much more toxic (e.g., Boyer, 2007). Blooms of *M. aeruginosa* are known historically dating back to the 1800s (Chorus and Bartram, 1999 and references therein). Present-day massive

blooms in some U.S. waterbodies can often be viewed in satellite imagery (Sims, 2013; and see Burkholder *et al.*, 2018).

Havens (2008) designed a simple conceptual model summarizing ecological effects of high-biomass cyanobacteria blooms and their potential adverse impacts, applicable to blooms of *M. aeruginosa*. Bloom formation leads to reduced light availability for submersed plants, benthic algae, and other phytoplankton; elevated pH, which can adversely affect fish populations; reduced CO_2 which alters competitive interactions with other phytoplankton, as *M. aeruginosa* is adept at sequestering carbon; and production of toxins and other bioactive substances (e.g., Sychrová *et al.*, 2012) which can cause sublethal and lethal impacts for zooplankton, macroinvertebrates, fish, wading birds, and other aquatic vertebrates. The developed bloom also adversely affects zooplankton and other grazers, and food web efficiency. Reduced grazing, or avoidance of *Microcystis* by grazers (e.g., Vanderploeg *et al.*, 2001), can act as a positive feedback, promoting further bloom development until environmental conditions become unfavorable. At night, the respiring bloom can cause hypoxia/anoxia, leading to fish kills and sublethal as well as lethal effects on other biota. As the bloom senesces and dies, hypoxia/anoxia and high ammonia concentrations can stress and kill biota as well.

Blooms of toxic *M. aeruginosa* have been lethal to various zooplankton, fish, waterfowl, and mammals, including domestic animals that have orally ingested the toxins in cells and water (Table 7.1). MCs can take various routes in moving through the food web (Figure 7.4), and at relatively low concentrations they can adversely affect biota across trophic levels (Table 7.2). The effects tend to be more severe at higher temperatures (Zurawell *et al.*, 2005). Bioaccumulation commonly occurs depending on the MC congeners, which would exacerbate toxin effects (e.g., Prepas *et al.*, 1997; Lehman *et al.*, 2010; Ibelings and Havens, 2008 and references therein). As Lehman *et al.* (2010, p. 229) noted, the data suggest that "even at low abundance, *Microcystis* spp. may impact estuarine fishery production through toxic and food web impacts at multiple trophic levels." There additionally is well-known benthic-pelagic coupling in the life history of *M. aeruginosa* and other *Microcystis* spp., wherein viable populations containing MCs reside in benthic habitats until conditions are conducive for another planktonic bloom (Latour *et al.*, 2004; Schöne *et al.*, 2010; Misson *et al.*, 2012). These toxins are also found in fish feces in substantial amounts (Jang *et al.*, 2004; Xie *et al.*, 2005), and in pseudofeces of dreissenid mussels (Pires *et al.*, 2004). Thus, MCs are readily moved between the water column and benthic habitats to affect benthic as well as pelagic communities (Figure 7.4).

7.5.2 Toxic *Prymnesium parvum* Blooms and Fish Communities in Two Texas Rivers

Van Landeghem *et al.* (2013) used multiple before-after, control-impact analyses to assess whether repeated toxic *P. parvum* blooms have led to long-term declines in the relative abundance and size structure of fish populations (21-year database) in two river systems in Texas. In the upper Colorado River drainage, about 3 million fish died in 37 fish kills from toxic *P. parvum* blooms during ~2001–2007, whereas about 20 million fish died in 28 *P. parvum*-linked kills in the Brazos River drainage (Van Landeghem *et al.*, 2013 and references therein). Maximal bloom densities were fairly similar in the two rivers (40,000 to 170,000 cells mL^{-1} and 30,000 to 100,000 cells mL^{-1} in the Brazos and Colorado, respectively), but bloom duration and frequency varied substantially. In the Colorado, toxic blooms had occurred each year since 2001 and had usually lasted for six months or more. By contrast, in the Brazos, blooms had been irregular and usually had lasted for two months or less, perhaps affording more refuge availability (temporally and spatially) to allow for better recovery of fish populations. Hydrologic differences would also have influenced bloom impacts – inflows into the Colorado reservoirs were extremely low for the past few decades, in contrast with substantial flows into Brazos reservoirs. High inflows would have helped to terminate blooms by providing nutrient pulses that alleviated nutrient-imbalanced conditions which can promote *P. parvum* toxicity (Granéli and Johansson, 2003b; Granéli *et al.*, 2012). In addition, regular inflows may have reduced salinity levels in the Brazos to below thresholds for *P. parvum* growth, diluted toxin levels, and created low *P. parvum* refuge areas for fish.

The analysis indicated sustained declines in relative abundance and/or size structure, related to toxic *P. parvum* blooms, for 9 of 12 fish species in the upper Colorado River (white bass, white crappie, largemouth bass, bluegill, river carpsucker, freshwater drum, channel catfish, flathead catfish, and blue catfish), but only for 1 of 8 species (blue catfish) in the Brazos River. The authors were

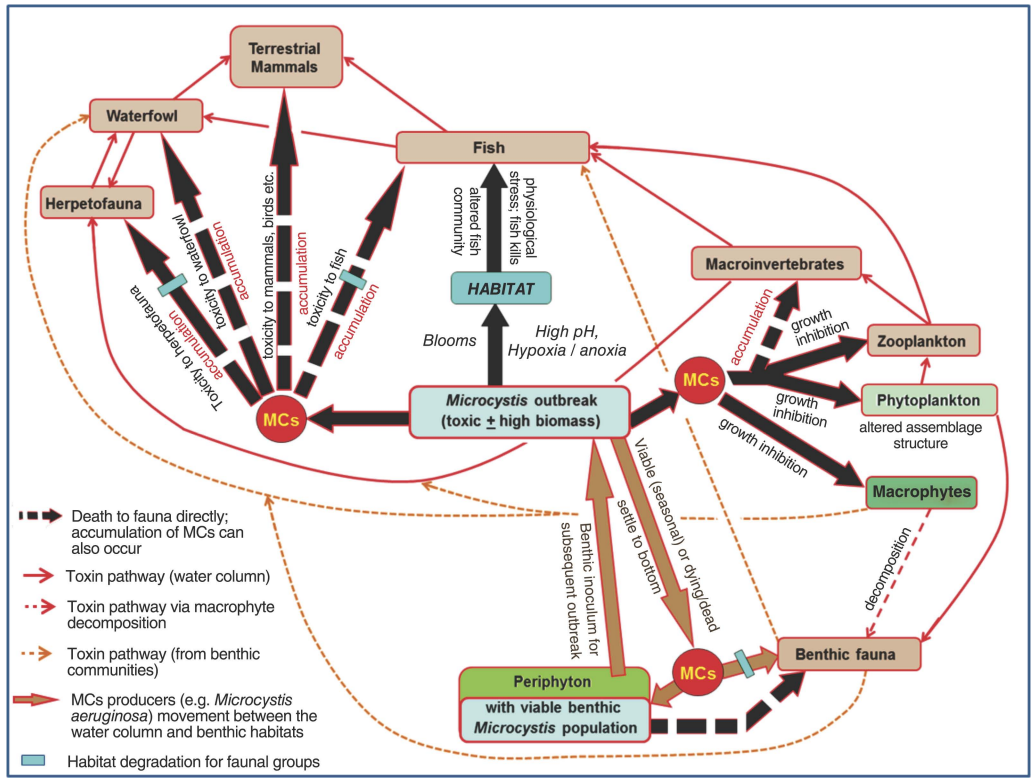

Figure 7.4 A conceptual model of microcystin (MC) pathways in aquatic ecosystems. These toxins are known to bioaccumulate in various fauna (e.g., Ibelings *et al.*, 2005; Xie *et al.*, 2005), and they are contained within cyanobacteria cells but can also be released into the surrounding medium. Not represented in this diagram are other cyanotoxins and other bioactive substances from *M. aeruginosa*, and toxins and bioactive substances from other sources that may be present. Also not shown, other than for macrophytes, are pathways for MC distribution via dead/dying flora and fauna that sink out of the water column to benthic habitats.

able to relate the varying patterns of *P. parvum* impacts on fish populations to several environmental factors, and to differences in the fish species. Previous research on aquatic disturbances had indicated that populations of species with high fecundity or high mobility recovered better and faster following a fish kill than species with low fecundity or low mobility. The much lower water levels characteristic of the Colorado likely created conditions that limited fish dispersal into smaller streams with fresh, nontoxic water, so that population recovery would have depended primarily on reproductive success or supplemental fish stocking rather than immigration from other systems. In addition, the extended duration of the toxic blooms in the Colorado overlapped with spawning periods of affected fish species, thereby severely impeding the ability of these species to recover.

For example, channel catfish in the Colorado have sustained a long-term decline despite major restocking efforts, whereas in the Brazos, a combination of stocking and natural recruitment likely was able to restore or maintain channel catfish populations. Channel catfish spawning in shallow areas (initiating in March–April, with maximal activity in June–July) considerably overlapped with toxic *P. parvum* blooms in the Colorado (from mid- to late fall through late May or June of the following year). This species resides in deeper waters before spawning, which may have provided refuge from exposure to the algal toxins. In the Brazos, by contrast, toxic blooms generally occur from January to March, overlapping only narrowly with fish spawning activity, which may allow higher natural recruitment and better recovery of catfish populations in that system after fish kills. As other examples, river carpsucker and freshwater drum usually mature at age four or later, which may have contributed to their relative inability to recover from toxic

blooms in the Colorado. Overall, a combination of ecological and physiological characteristics influenced the ability of fish species to recover from toxic *P. parvum*-related fish kills. In the upper Colorado River, fish populations have been severely affected by *P. parvum*, whereas most fish populations in the Brazos have remained stable despite toxic *P. parvum* blooms.

7.5.3 Toxic *Pseudo-nitzschia* Blooms in Coastal Upwelling Areas

Although DA is a potent water-soluble neurotoxin, it does not cause fish kills or abnormal behavior in fish during or after toxic *Pseudo-nitzschia* blooms (Lefebvre *et al.*, 2012). The toxin is usually depurated rapidly, so that the residence time in fish tissues is only 2–3 days (Lefebvre *et al.*, 2007 and references therein). Herbivorous fish species such as Pacific sardines and northern anchovies track *Pseudo-nitzschia* blooms to feed (Lefebvre *et al.*, 2002). Stomach contents of anchovies sampled during toxic *Pseudo-nitzschia* blooms, for example, contained high densities of the diatom remains (siliceous cell walls), suggesting that *Pseudo-nitzschia* was a major food resource (Lefebvre *et al.*, 1999), but the fish usually contain DA only in bloom areas or for a short time after leaving a bloom (Lefebvre *et al.*, 2002). Non-planktivorous pelagic fish such as club mackerel and jack mackerel can contain DA during toxic blooms, suggesting toxin transfer from smaller planktivorous fishes (Busse *et al.*, 2006). Filter-feeding bivalve molluscs can also accumulate DA without apparent impact.

Despite the lack of DA impacts on finfish and shellfish during natural *Pseudo-nitzschia* blooms, these animals can act as vectors for DA bioaccumulation in higher trophic levels, especially affecting birds and mammals that rely upon the *Pseudo-nitzschia* consumers as major food resources. Consequently, DA intoxication has resulted in illness and death of human consumers of blue mussels contaminated with DA (Nova Scotia, Canada; Wright *et al.*, 1989); mass mortalities of seabirds such as Brandt's cormorants and brown pelicans that had eaten fish contaminated with DA (coastal California; Work *et al.*, 1993; Sierra-Beltran *et al.*, 1997); and mortality of cetaceans and pinnipeds such as sea lions from consumption of northern anchovies contaminated with DA (Lefebvre *et al.*, 1999; Scholin *et al.*, 2000; Fire *et al.*, 2010). Planktivorous fishes such as anchovies are eaten by many aquatic animals and seabirds in marine food webs (Smith *et al.*, 2011); thus, even one species can be a major vector of DA to higher trophic levels. Limited studies available for zooplankton and phytoplankton have shown reduced feeding and fecundity of a rotifer fed *P. multiseries* (Whyte *et al.*, 1996); and growth inhibition of phytoplankters including the toxigenic dinoflagellate species *Akashiwo sanguinea*, *P. parvum*, and *Chattonella marina*, and a benign cryptophyte exposed to toxic *Pseudo-nitzschia*, as an apparent allelopathic effect (Table 7.2) (Xu *et al.*, 2015).

Diverse benthic fauna (e.g., Pacific sanddabs, longspine combfish, and bottom-feeding fish such as soles, turbots, and halibut) can also accumulate high levels of DA in association with blooms (Kvitek *et al.*, 2008; Vigilant and Silver, 2007), from dying toxic *Pseudo-nitzschia* and contaminated feces that settle out of the water column as blooms subside (Sekula-Wood *et al.*, 2009). Benthic fauna can contain detectable levels of DA even during non-bloom periods (Vigilant and Silver, 2007). Thus, there is a strong pelagic-benthic coupling of DA contamination and bioaccumulation from *Pseudo-nitzschia* blooms.

7.5.4 Toxic *Alexandrium* Blooms in the Northeast

Kills of planktivorous fish (Atlantic herring, Atlantic menhaden, sand lance) have been associated with summer blooms of STX-producing *Alexandrium* spp. in New England and eastern Canada coastal waters for decades (e.g., Smayda, 1991 and references therein). Dying fish have manifested neurological signs at the water surface, such as erratic swimming and paralysis (White, 1977). Larvae of many fish species directly depend on dinoflagellates as food items for about the first week of feeding (Smayda, 1991). In experiments, larval fish (Atlantic cod, Atlantic herring, Atlantic mackerel, capelin, red sea bream, winter flounder) fed toxic *Alexandrium tamarense* swam erratically, lost equilibrium, and/or swam on their side, upside down, or in circles prior to paralysis (Robineau *et al.*, 1991a, 1991b; Samson *et al.*, 2008). Larval fish contaminated with smaller amounts of STXs can serve as vectors for biomagnification and food web transfer of the toxins (Smayda, 1991 and references therein). Although the major route of STX exposure is the diet, STXs are water-soluble and exposure to fish can occur in several ways.

Adult fish such as Atlantic mackerel can accumulate STXs via gill absorption of dissolved toxins, without ingesting toxic cells (Montoya *et al.*, 1996). Grazers such as zooplankton can lyse toxic cells, so that a major fraction of the STXs is dissolved. Planktivorous fish mainly consume zooplankton, but incidentally take in toxic *Alexandrium* cells as well (Turner, 2006; Turner and Granéli, 2006).

Atlantic mackerel can accumulate STXs as they age, suggesting that their health could be affected, and that they could be vectors in transferring STXs to higher trophic-level consumers (Castonguay *et al.*, 1997). As STXs bioaccumulate in the food web, top predators such as piscivorous seabirds (e.g., terns; Nesbet, 1983) and marine mammals such as humpback whales (Geraci *et al.*, 1989; Anderson and White, 1992) have died in association with toxic *Alexandrium* blooms. The whales had fed mostly on Atlantic mackerel, and had acted normally until about 30 minutes before death.

Based on experimental information, organisms from lower trophic levels, while serving as STX vectors, are themselves adversely affected by toxic *Alexandrium* blooms. Bivalve molluscs (e.g., Atlantic surfclams, bay scallops, blue mussels, northern quahogs, and/or ribbed mussels) have responded to toxic *A. tamarense* with reduced clearance rates (Lesser and Shumway, 1993), shell valve closure, and increased mucus production (Shumway and Cucci, 1987), inhibited byssus production (Shumway *et al.*, 1987), and altered heart rate (Gainey and Shumway, 1988). Sea scallops have exhibited shell valve closure, increased mucus production, violent swimming activity, and reduced oxygen consumption when exposed to toxic *A. tamarense* (Shumway *et al.*, 1985; Shumway and Cucci, 1987). Softshell clams have attempted to close their siphons and have shown reduced clearance rates, impaired burrowing response, and decreased heart rate (Shumway and Cucci, 1987; Gainey and Shumway, 1988; Bricelj *et al.*, 1996). Various species of ciliates and microcrustacean zooplankton have responded to toxic *A. tamarense* with depressed feeding, feeding avoidance, feeding inhibition, reduced fecundity, aberrant swimming behavior, and/or reduced growth (Table 7.2). Some *Alexandrium* spp. are mixotrophic and have been shown to consume microalgal prey (Table 7.1). Thus, overall, *A. tamarense* has been documented to adversely affect trophic levels ranging from phytoplankton at the base of the food web to apex predators.

7.5.5 Toxic *Karenia brevis* Blooms along the Florida Coast

The toxic microalga that has been studied the longest for food web impacts in marine ecosystems of U.S. waters is *K. brevis* in the Gulf of Mexico (see Landsberg *et al.*, 2009 and references therein, from which this information is taken unless otherwise indicated). Blooms (outbreaks or red tides) affect other regions of the Gulf Coast along the U.S. and Mexico shores, but the hardest hit area is Florida. Descriptions of *K. brevis* blooms have appeared in various accounts and records since the mid-1800s; the blooms occur every year, can last for much of the year (four to nine months or longer), and annually can kill millions of marine flora and fauna, from phytoplankton to marine mammals such as dolphins and manatees, along more than 5000 km of coastline over a ~7000 km^2 area.

Water-soluble BTXs produced by delicate *K. brevis* cells are released into the surrounding medium when cells lyse, e.g., during grazing, when water carrying cells moves over fish gills, or when waves break onto shore. The toxins have killed hundreds of marine species (see examples in Table 7.1; also Broadwater *et al.*, 2018; and Gibble and Hoover, 2018 – Chapters 5 and 6, respectively). The high concentrations of dissolved BTXs can result in a time lag between a detected bloom and fish mortalities. For example, a bloom that ended late in 2005 was followed by a fish kill that lasted five months in the same area. Sampled fish had high levels of BTXs in their tissues, and the behavior of dying fish fit the symptoms expected from BTX exposure. The blooms are frequently associated with hypoxia/anoxia, likely from the oxygen demand (especially at night) created by the decomposition of dead fauna in shallow waters. Aerosolized BTXs also have affected land animals such as dogs and coyotes (Table 7.2; and see Landsberg *et al.*, 2009 and references therein for effects on humans). High levels of BTXs persist among seagrass leaves, epiphytes, and epiphytic biofilm debris for weeks, and at lower levels for months in the absence of *K. brevis* (Flewelling, 2008).

The persistent, highly toxic blooms each year and the long fish kills can have lingering effects on fish populations. During surveys conducted after these events, up to nearly 60 resident finfish species have been absent. In bloom years, declines have been reported in the annual recruitment of juvenile spotted seatrout, sand

seatrout, and red drum in some affected areas, which would alter the overall fish community structure (Flaherty and Landsberg, 2010). The prolonged blooms have led to changes in fish community structure and ecology in benthic habitats as well. Gannon *et al.* (2009) reported that during *K. brevis* outbreaks in Sarasota Bay, Florida, in 2003–2007, pelagic filter feeders became more abundant while demersal, invertebrate-feeding fish species declined by as much as 88% in comparison to their abundance in periods without blooms. The data also suggest that pelagic filter-feeding fish are more tolerant of bloom conditions or better able to avoid dense bloom areas than other fish, which would also make filter-feeding fish more effective vectors of BTXs to higher predators (Gannon *et al.*, 2009).

An outbreak and associated hypoxia/anoxia in 2005 resulted not only in massive fish kills but also in a collapse of epibenthic communities along the central west Florida shelf (DuPont and Coy, 2008). Divers provided a photographic time series (2005–2007) of the natural hard-bottom/ledge community succession after the bloom. Corals apparently bleached during the toxic bloom, but recovered fairly quickly (Table 7.2). Successional stages of fish communities generally followed a predictable progression and reverted to a pre-bloom state, but within the context that this is a disturbed area ecologically. It was hypothesized that the fluctuating environmental conditions in the shallow eastern Gulf of Mexico (major toxic blooms with extended hypoxia/anoxia, varying temperatures, turbidity, and hurricanes) limit the colonizing species and prevent communities from reaching dynamic equilibrium (DuPont and Coy, 2008).

7.6 Ecosystem-Disruptive Algal Blooms

A subcategory of HAB is sometimes referred to as *ecosystem-disruptive algal blooms* (EDAB; term from Sunda *et al.*, 2006). These EDAB are high-biomass blooms and can be either toxic or nontoxic (see Sunda *et al.*, 2006). Their impacts have been distinguished from impacts of other HAB due to direct toxicity, because EDAB effects are considered to be the result of complex feedback interactions among nutrient regulation of algal growth, population losses to grazing, and grazer-mediated recycling of nutrients (Sunda *et al.*, 2006; Gobler and Sunda, 2012). Candidate EDAB species listed by Sunda *et al.* (2006) were based on three criteria: (1) they cause massive, often-monospecific blooms that negatively affect ecosystem structure and function; (2) they adversely affect grazing rates and grazer populations through toxicity, unpalatability, or physically impeding the grazing mechanisms; and (3) positive feedbacks from reduced grazer-mediated nutrient recycling and/or shading of benthic habitats likely contribute to bloom maintenance.

Most of the candidate EDAB species suggested by Sunda *et al.* (2006) have very small cells (maximum dimension 1–5 [8] μm). They included the brown tide organisms *Aureococcus anophagefferens* and *Aureoumbra lagunensis* (Heterokontophyta, Pelagophyceae), the chlorophyte *Nannochloris atomus* (Trebouxiophyceae), the heterokontophyte *Nannochloropsis qaditana* (Eustigmatophyceae), and the cyanobacterium *Synechococcus elongatus* (Ryther, 1954, 1989; Sunda and Guillard, 1976; Phlips *et al.*, 1999; Glibert *et al.*, 2004; Gobler *et al.*, 2005; Buskey, 2008; taxonomy as in Graham *et al.*, 2016). Unlike various high-nutrient-adapted harmful algal taxa, these organisms were hypothesized to be low-nutrient-adapted, K-selected species that compete well with other algae only at low concentrations of available nutrients (Sunda *et al.*, 2006; Gobler and Sunda, 2012). Importantly, Sunda *et al.* (2006) also proposed that EDAB usually require a pre-bloom of *high*-nutrient-adapted species which reduce inorganic nutrients to low levels, stimulate population growth of grazers, and increase organic N and P availability through grazer-mediated recycling. The nutrient conditions promoting EDAB species are likely far more complex than this hypothesis, as is the diversity of nutrient strategies of EDAB species.

Like other HAB, EDAB can severely alter or degrade ecosystem functioning by disrupting nutrient and energy transfer to higher trophic levels. As their blooms develop, the resulting reduction in light penetration can shade the bottom of the typically shallow systems where they occur. The low light availability, in turn, reduces nutrient competition by benthic phototrophs, allowing sediment release of nutrients to further fuel these blooms. Thus, once the blooms become established, they can often be maintained for considerable periods of time – years as in the case of a brown tide (*Aureoumbra lagunensis*) bloom in Texas (8 years; Buskey *et al.*, 2001).

Typically, the small cells forming EDAB are not well grazed (e.g, Gobler *et al.*, 2002, 2005; Caron *et al.*, 2004), so there is little transfer of organic matter from these primary producers through the food web. As another consequence, because of the low rate of grazing, there is a reduction in water-column nutrient recycling. In Narragansett Bay, Rhode Island, U.S., during a brown tide EDAB, the

microzooplankton community of ciliates and heterotrophic flagellates was unusually sparse, suggestive of reduced grazing (Table 7.2) (Smayda and Villareal, 1989; Smayda, 2008). In Laguna Madre, Texas, the density of protozoan grazers was greatly reduced during brown tide blooms, and a thick polysaccharide layer around the cells may have made it difficult for the protozoa to feed (Buskey and Stockwell, 1993; Buskey *et al.*, 2001). The elevated pH resulting from the high algal biomass accumulation likely inhibited grazers as well (Buskey, 2008). It has also been suggested that allelopathic chemicals and toxin(s) may be important in maintaining EDAB (Sunda *et al.*, 2006; Granéli *et al.*, 2008; Robbins *et al.*, 2010).

In addition to not being well grazed by zooplankton, these picoplankton EDAB species generally are poor-quality food for bivalve molluscs and other macroinvertebrate grazers. Brown tide blooms (*Aureococcus anoffagefferens*) in Narragansett Bay led to extensive mortality and recruitment failure of scallops and mussels, die-offs of macroalgae and seagrasses, failure of the cladoceran zooplankton community to appear, failure of bay anchovies (*Anchoa mitchelli*) to spawn, and cessation of mussels to filter, which led to their starvation and death (Bricelj *et al.*, 1989; Smayda, 1991 and references therein). Brown tides (*Aureoumbra lagunensis*) in Texas lagoons (mentioned above) caused the dominant clams to virtually disappear (Montagna *et al.*, 1993). In the Coastal Bays of Maryland, U.S., growth of northern quahogs ceased during a brown tide, but recovered once the bloom subsided (Wazniak and Glibert, 2004). In all, reduced rates of grazing by both pelagic (micro- and macrozooplankton) and benthic macroinvertebrate grazers are thought to have contributed substantially to the development and persistence of brown tides in New England (Gobler *et al.*, 2002, 2004; Caron *et al.*, 2004), the mid-Atlantic U.S. coast, and the Gulf of Mexico (Buskey and Stockwell, 1993; Buskey *et al.*, 1997).

Another classic example of a small EDAB species (following Sunda *et al.*, 2006) is provided by the massive, longstanding blooms of the unicellular cyanobacteria *Synechococcus/Synechocystis*, now mostly referred to as *Synechococcus elongatus*. This organism has damaged the coastal ecosystems of south Florida (Lapointe *et al.*, 1994; Glibert *et al.*, 2004), partly by causing widespread mass mortality of various sponge species (Porifera). Sponges are the primary benthic filter feeders in these systems. When high biomass accumulations occur, the pico-cyanobacteria can release copious external polysaccharides that can obstruct the internal canal system of sponges and/or impair feeding. Thus, declines in sponge populations are thought to have been caused by *S. elongatus* via impaired grazing by sponges (Kuffner and Paul, 2004; Charpy *et al.*, 2012 and references therein). Sponges once provided the primary habitat for juvenile spiny lobsters, which also have significantly declined (Butler *et al.*, 1995). Longstanding, dense *S. elongatus* blooms have exacerbated loss of seagrasses as well (turtlegrass, shoalgrass, and manateegrass) via light reduction (Phlips *et al.*, 1995, 1999; Hall *et al.*, 1999). As described by Butler *et al.* (1995, p. 119), "This cascade of disturbances has dramatically altered the community structure of affected hard bottom areas and demonstrates the coupled dynamics of . . . shallow marine ecosystem[s]." Some strains of these unicellular cyanobacteria have been reported to be toxic (Tables 7.1 and 7.2). Toxicity would exacerbate the impacts of these high-biomass blooms (see Section 7.5).

In considerations about EDAB, Sunda *et al.* (2006) made careful interpretations based on data available at that time, and indicated that the term *EDAB* likely would be applicable to various other HAB as additional data on their ecology and impacts became available. At present, there is increasing recognition that many HAB, including both microalgae and macroalgae, can be ecosystem-disruptive.

7.7 Future Directions

The broad overview presented in this chapter provides examples, among many, of the adverse impacts of harmful algae on food webs and ecosystems. Much is known about the effects of some major algal toxins on certain animal species but, as stated as "goals" in Section 7.2, studies are needed that assess impacts on *natural communities*, rather than on one or a few species at a time. Even for one of the best studied harmful algae to date, *Karenia brevis*, as Pierce and Henry (2008, p. 629) wrote, "an ecosystem-based approach is needed to investigate the acute and subacute impacts resulting from *K. brevis* blooms."

The fate of many algal toxins, both in the ecosystem and post-ingestion at the organism level, is not well understood. Depuration of some toxins is thought to be rapid, but depuration is seldom complete, and low concentrations can even be carried through to the next growing season (e.g., cyanotoxins; see Ibelings and Havens 2008, and references therein). Additional research should include assessment of toxin impacts in benthic

communities and benthic processes (Palmer, 2000), which have been underemphasized (e.g., Glibert *et al.*, 2012) despite the fact that benthic processes are important influences on harmful blooms (Vargo *et al.*, 1996; Vanderploeg *et al.*, 2001; Wikfors, 2005; Schöne *et al.*, 2010). From that improved knowledge base about the fate of algal toxins, models can finally be developed that reliably predict toxin concentrations of a given species/strain in natural settings, and impacts from blooms of a given HAB population on specific trophic levels and species.

Much more information is needed on the degree of bioaccumulation that occurs in aquatic communities affected by some harmful algae such as toxic cyanobacteria. There is a pressing related need to develop improved techniques to measure algal toxins in biota, and to develop biomarkers for toxin exposure. For example, all but a few published studies on toxic cyanobacteria have not accounted for covalently bound cyanotoxins in biota. Microcystins are routinely extracted using aqueous methanol, which does not extract the MCs that are covalently bound to protein phosphatases in cells of the affected organism (Ibelings and Havens, 2008). Studies that have compared the data from standard aqueous methanol extraction to extraction after Lemieux oxidation, which *does* account for covalently bound MCs, have shown that a major proportion of the total MCs in biota is covalently bound (e.g., Smith *et al.*, 2010). Thus, most published research may have significantly underestimated MC concentrations in biota (Ibelings and Havens, 2008 and references therein). Moreover, the transfer and accumulation of MCs depend on the toxin profile of the bloom (Issam *et al.*, 2010), yet most present research is still at the level of examining only one MC (usually MC-LR) or total MCs. Such research should also be extended to many other harmful algae that produce multiple toxins in varying profiles (Burkholder and Glibert, 2006 and references therein). Minor structural changes in toxin profiles produced by the dominant strain(s) in a harmful bloom may have major effects on toxin uptake, organ distribution, and excretion (Dietrich and Hoeger, 2005; Ibelings and Havens, 2008).

The chemical ecology of harmful algae should extend beyond toxins to assess much more about the roles of other potent allelochemicals that strongly influence food web functioning. As Ianora *et al.* (2011a, p. 1616) wrote, "Understanding chemical ecological responses to environmental triggers and chemically mediated species interactions [eventually] will help define crucial chemical and molecular processes that help maintain biodiversity and ecosystem functionality."

This lengthy chapter only briefly mentions certain aspects of harmful algal interactions with food webs. For example, HAB (both high-biomass and toxic) can stress and physiologically weaken aquatic fauna, which would make them more susceptible to diseases from pathogenic viruses, bacteria, fungi, and protozoans. Research is needed to unravel the complex direct and indirect effects of harmful algae on pathogenic organisms and disease in fish, macroinvertebrates, and other aquatic life.

Finally, among the most important areas for future research are the impacts to food webs from exposure to multiple algal toxins, and from simultaneous and sequential exposure to algal toxins and toxins from other sources (Codd *et al.*, 2005). Such conditions are the reality in aquatic ecosystems. Toxic cyanobacteria, dinoflagellates, haptophytes, and raphidophyceans are known to produce more than one toxin within an algal cell, and surely within a bloom (Burkholder and Glibert, 2006 and references therein). Cyanobacteria blooms commonly have been documented to contain multiple cyanotoxins (Graham *et al.*, 2010; de la Cruz *et al.*, 2013; Loftin *et al.*, 2016 and references therein). Analysis of marine mammal tissues during mortality events has shown that exposure to multiple toxins occurs, such as the presence of DA and BTXs in tissues of bottlenose dolphins, and the presence of DA and STXs in feces from North Atlantic right whales (Landsberg *et al.*, 2014 and references therein). Evidence suggests that MCs and CYLs can act additively and synergistically, both with each other and with other toxic substances (Prieto *et al.*, 2011; Rymuszka and Sierosławska, 2013; Freitas *et al.*, 2014; Pinheiro *et al.*, 2016). Algal toxins can act synergistically with heavy metals, for example (Traoré *et al.*, 1999). In addition, harmful algae excrete substances with metal-complexing properties, which can render toxic heavy metals more bioavailable (Moffett *et al.*, 1996; Krishnan *et al.*, 2007). High-biomass blooms that cause anoxic conditions also make toxic substances such as heavy metals and organohalogens much more soluble, and thereby more bioavailable to adversely affect aquatic communities (e.g., Garcia-Hernández *et al.*, 2005).

HAB caused by many high-biomass and/or toxic species are increasing in frequency and extent, due to a combination of nutrient pollution, warming trends and associated effects of climate change, and overfishing (Dale *et al.*, 2006; Casini *et al.*,

2008; Heisler *et al.*, 2008; Hallegraeff 2010; Glibert *et al.*, 2012; O'Neil *et al.*, 2012; see also Chapter 1). Evidence for range expansion of some warmwater harmful algal species is being reported (Hallegraeff, 2010 and references therein; Wells *et al.*, 2015). Research to strengthen understanding about both the obvious and the insidious major food web-level and ecosystem-level impacts of HAB will be valuable in designing reliable predictive models and more effective management strategies to mitigate their impacts and improve protection of aquatic ecosystems.

Appendix A: Scientific Names for Organisms Listed by Common Name in This Chapter, Also Indicating Species Affected by *Karenia brevis* (*Kb*)

I. Vertebrates (Phylum Chordata)

Mammals

Baleen whale (*Balaenoptera* sp.)
Bottlenose dolphin (*Tursiops truncatus*)[Kb]
California sea lion (*Zalophus californianus*)
Coyote (*Canis latrans*)[Kb]
Domestic dog (*Canis familiaris*)[Kb]
Hawaiian monk seal (*Monachus schauinslandi*)
Humpback whale (*Megaptera novaeangliae*)
Manatee (*Trichechus manatus latirostris*)[Kb]
North Atlantic right whale (*Eubalaena glacialis*)
Sea otter (southern sea otter) (*Enhydra lutris nereis*)

Birds

American coot (*Fulica americana*)
Bald eagle (*Haliaeetus leucocephalus*)
Black-crowned night heron (*Nycticorax nycticorax*)
Black oystercatcher (*Haematopus bachmani*)
Brandt's cormorant (*Phalacrocorax penicillatus*)
Brown pelican (*Pelecanus occidentalis*)
Clark's grebe (*Aechmorphorus clarkii*)
Common eider (*Somateria mollissima*)
Common murre (*Uria aalge*)
Common tern (*Sterna hirundo*)
Double-crested cormorant (*Palacrocorax auritus*)[Kb]
Glaucous-winged gull (*Larus glaucescens*)
Lesser scaup (*Aythya affinis*)[Kb]
Mallard (*Anas platyrhynchos*)
Northern fulman (*Fulmarus glacialis*)
Pacific loon (*Gavia pacifica*)
Red-breasted merganser (*Mergus merganser*)[Kb]
Red-throated loon (*Gavia stellata*)
Shag (*Phalacrocorax aristotelis*)
Surf scoter (*Melanitta perspicillata*)
Western grebe (*Aechmorphorus occidentalis*)
White wing scoter (*Melanitta deglandi*)

Reptiles

European pond turtle (*Emys orbicularis*)
Green sea turtle (*Chelonia mydas*)[Kb]
Kemp's ridley sea turtle (*Lepidochelys kempii*)[Kb]
Loggerhead sea turtle (*Caretta caretta*)[Kb]
Mediterranean turtle (*Mauremys leprosa*)[Kb]

Amphibians

Cane toad (*Bufo marinus*)

Fish

American eel (*Anguilla rostrata*)
American pollock (*Pollachius virens*)
Anchovy (*Anchoa mitchilli*)
Atlantic bumper (*Chloroscombrus chrysurus*)[Kb]
Atlantic croaker (*Micropogonais undulatus*)
Atlantic herring (*Clupea harengus harengus*)
Atlantic menhaden (*Brevoortia tyrannus*)[Kb]
Atlantic midshipman (*Porichthys porosissimus*)[Kb]
Atlantic moonfish (*Selene setapinnis*)[Kb]
Atlantic needlefish (*Strongylura marina*)[Kb]
Atlantic salmon (*Salmo salar*)
Atlantic silverside (*Menidia menidia*)
Atlantic spadefish (spadefish) (*Chaetodipterus faber*)[Kb]
Balloonfish (*Diodon holocanthus*)[Kb]
Bandtail puffer fish (*Sphoeroides spengleri*)
Bank cusk-eel (*Ophiodon holbrooki*)[Kb]
Barracuda (*Sphyraena barracuda*)[Kb]
Bay anchovy (*Anchoa mitchelli*)[Kb]
Belted sandfish (*Serranus subligarius*)[Kb]
Black bullhead (*Ictalurus melas* [*Amierus melas*])
Black crappie (*Pomoxis nigromaculatus*)
Black drum (*Pogonias chromis*)[Kb]
Black grouper (*Mycteroperca bonaci*)[Kb]
Black tip shark (*Carcharinus limbatus*)[Kb]
Blackcheek tonguefish (*Symphurus plagiusa*)[Kb]
Blindfish (*Typhlogobius californicus*)
Blue catfish (*Ictalurus furcatus*)
Blue tilapia (*Oreochromis aureus*)
Bluefish (*Pomatomos saltatrix*)
Bluegill (bluegill sunfish) (*Lepomis macrochirus*)
Bluehead wrasse (*Thalassoma bifasciatum*)
Bluerunner (*Caranx chrysos*)[Kb]

Bluestriped grunt (*Haemulon sciurus*)[Kb]
Brown trout (*Salmo trutta*)
Buffalo (*Megastomatobus cyprinella*)
Catfish (*Arius felis*)[Kb]
Channel catfish (*Ictalurus punctatus*)
Checkered puffer (checkered puffer fish) (*Sphoeroides testudineus*)[Kb]
Chinook salmon (*Oncorhynchus tshawytscha*)
Chub mackerel (*Scomber japonicus*)
Cobia (*Rachycentron canadus*)[Kb]
Cod (*Gadus morhua, Gadus* sp.)
Coho salmon (*Oncorhynchus kisutch*)
Common carp (carp) (*Cyprinus carpio*)
Common snook (*Centropomus undecimalis*)
Coney (*Epinephelus fulvus*)
Cowfish (*Lactophrys quadricornis*)[Kb]
Crested blenny (*Hypleurochilus geminatus*)
Crevalle jack (*Caranx chrysos*)[Kb]
Damselfish (genus, species not given)[Kb]
Dogfish (*Galeus californicus*)
Eel (*Ophichthus* sp.)[Kb]
European sea bass (*Dicentrarchus labrax*)
Flathead catfish (*Pylodictis olivaris*)
Freshwater drum (*Aplodinotus grunniens*)
Gafftopsail catfish (*Bagrus marinus*)[Kb]
Gag (*Mycteroperca microlepis*)[Kb]
Gar (*Lepisosteus* sp.)[Kb]
Goldfish (*Carrasius auratus*)
Goldspotted killifish (*Floridichthys carpio*)[Kb]
Grass carp (*Ctenopharyngodon idella*)[Kb]
Gray angelfish (*Pomacanthus arcuatus*)[Kb]
Gray snapper (*Lutjanus griseus*)[Kb]
Gray triggerfish (*Balistes capriscus*)[Kb]
Graysby (*Epinephelus cruentatus*)[Kb]
Great barracuda (*Sphyraena barracuda*)
Groupers (genera, species not given)[Kb]
Grunts (genera, species not given)[Kb]
Guitarfish (*Rhinobatus productus*)
Gulf flounder (*Paralichthys albiguttus*)[Kb]
Gulf kingfish (*Menticirrhus littoralis*)[Kb]
Gulf menhaden (*Brevoortia patronus*)[Kb]
Halfbeak (*Hyporhamphus unifasciatus*)[Kb]
Haller's round ray (*Urolophus halleri*)
Hardhead catfish (*Arius felis*)[Kb]
Harvestfish (*Peprilus triacanthus*)[Kb]
Hogchoker (*Trinectes maculatus*)
Hogfish (*Lachnolaimus maximus*)[Kb]
Hogsucker (*Hypentelium* sp.)
Horn shark (*Heterodontus francisci* [*Gyropleurodus francisci*])
Hybrid striped bass (cultured) (*Morone saxatilis* × *Morone chrysops*)
Inland silverside (*Menidia beryllina*)
Inshore lizardfish (*Synodus foetens*)[Kb]
Jack crevalle (*Caranx hippos*)[Kb]
Jack mackerel (California or Pacific jack mackerel) (*Trachurus symmetricus*)
Jack smelt (*Atherinopsis californiensis*)
Jacks (*Caranx* spp.)[Kb]
Jewfish (*Epinephelus itajara*)[Kb]
Ladyfish (*Elops saurus*)[Kb]
Lancet (*Branchiostoma caribbaeum*)[Kb]
Largemouth bass (*Micropterus salmoides*)
Leatherjacket (*Oligoplites saurus*)[Kb]
Leopard searobin (*Prionotus scitulus*)[Kb]
Lined sole (*Achirus lineatus*)[Kb]
Loach (*Misguruns mizolepis*)
Longnose batfish (*Ogcocephalus vespertilio*)[Kb]
Longnose gar (*Lepisosteus osseus*)
Longnose killifish (*Fundulus similis*)[Kb]
Mahogany snapper (*Lutjanus mahogoni*)
Medaka (*Oryzias latipes*)
Mississippi silversides (*Menidia audens*)
Monkfish (*Lophias americanus*)
Mosquitofish (*Gambusia affinis*)
Mummichog (*Fundulus heteroclitus*)
Needlefish (*Strongylura marina*)
Northern anchovy (*Engraulis mordax*)
Northern pike (*Esox lucius*)
Northern puffer (*Sphoeroides maculatus*)[Kb]
Orange filefish (*Aluterus schoepfi*)[Kb]
Oyster toadfish (*Opsanus tau*)[Kb]
Pacific herring (*Clupea harengus pallasi*)
Pacific sanddab (*Citharichthys sordidus*)
Pacific sardine (*Sardinops sagax*)
Palespotted eel (*Ophichthus ocellatus*)[Kb]
Planehead filefish (*Monocanthus hispidus*)[Kb]
Perch (yellow perch) (*Perca flavescens*)
Perch (*Diplectrum formosum*)[Kb]
Pikeperch (*Stizostedion lucioperca*)[Kb]
Pinfish (*Lagodon rhomboides*)[Kb]
Pompano (*Trachinotus carolinus*)[Kb]
Porgy (*Calamus* sp.)[Kb]
Porcupine fish (*Diadon hystrix*)[Kb]
Puffer (*Sphoeroides* sp.) [Kb]
Purplemouth moray (?) (*Gymnothorax vicinus*)[Kb]
Queen triggerfish (*Balistes vetula*)[Kb]
Rays (genera, species not given)[Kb]
Red drum (*Sciaenops ocellatus*)[Kb]
Red grouper (*Epenephilus morio*)[Kb]
Red perch (rose fish) (*Sebastes norvegicus*)
Red sea bream (*Pagrus major*, *Pagellus bogaraveo*)
Red snapper (*Lutjanus campechanus*)[Kb]
Redfin needlefish (*Strongylura notata*)[Kb]
Redfish (*Sciaenops ocellatus*)[Kb]
Rio Grande darter (*Etheostoma grahami*)
River carpsucker (*Carpiodes carpio*)

Roach (*Rutilus rutilus*)
Round scad (*Decapturus punctatus*)[Kb]
Sailfish (*Istiophorus platypterus*)[Kb]
Sailors choice (*Haemulon parrai*)[Kb]
Sand lances (sand eels) (*Ammodytes* spp.)
Sand perch (*Diplectrum formosum*)[Kb]
Sand seatrout (*Cynoscion arenarius*)[Kb]
Scaled sardine (*Harengula pensacolae*)[Kb]
Scamp (*Mycteroperca phenax*)[Kb]
Schoolmaster (*Lutjanus apodus*)
Sharksucker (*Echeneis naucrates*)[Kb]
Sheepshead (*Archosargus probatocephalus*)[Kb]
Sheepshead minnow (*Cyprinodon variegatus*)
Shiners (genera, species not given)[Kb]
Short bigeye (*Pristigenys alta*)[Kb]
Shortnose batfish (*Ogcocephalus radiatus*)[Kb]
Shortnose sturgeon (*Acipenser brevirostrum*)
Silver carp (*Hypophthalmichthys molitrix*)
Silver jenny (*Eucinostomus gula*)[Kb]
Silver perch (*Bairdiella chrysoura*)[Kb]
Silver trout (silver seatrout) (*Cynoscion nothus*)[Kb]
Snook (common snook) (*Centropomus undecimalis*)[Kb]
Sole (common sole) (*Solea solea*)[Kb]
Sooty eel (*Bascanichthys bascanium*)[Kb]
Southern kingfish (*Menticirrhus americanus*)[Kb]
Southern puffer fish (*Sphoeroides nephelus*)
Southern seabass (black seabass) (*Centropristis striata*)[Kb]
Southern stargazer (*Astroscopus* y-*graecum*)[Kb]
Spadefish (Atlantic spadefish) (*Chaetodipterus faber*)[Kb]
Spanish mackerel (*Scomberomorus commerson*)[Kb]
Speckled worm eel (*Myrophis punctatus*)[Kb]
Spinner shark (*Carcharhinus brevipinna*)[Kb]
Spiny boxfish (butterfish) (*Lactoria diaphana*)[Kb]
Spot (*Lieostomus xanthurus*)[Kb]
Spotted moray (?) (*Gymnothorax moringa*)[Kb]
Spotted rose snapper (*Lutjanus guttatus*)
Spotted seatrout (speckled trout) (*Cynoscion nebulosus*)[Kb]
Steelhead trout (*Oncorhynchus mykiss*)
Stingray (*Mylobatis californicus*)
Stippled clingfish (*Gobiesox punctulatus*)
Striped bass (*Morone saxatilis*)
Striped bass – reciprocal cross-hybrid (*Morone saxatilis* male × *Morone chrysops* female)
Striped burrfish (*Chilomyterus shoepfi*)[Kb]
Striped killifish (*Fundulus majalis*)
Striped mullet (*Mugil cephalis*)[Kb]
Suckers (freshwater; genera, species not given)
Tarpon (*Tarpon atlanticus*)[Kb]
Thornback guitarfish (*Platyrhinoidis triseriata* [*P. triscriatus*])
Thread herring (*Opisthonema oglinum*)[Kb]
Threadfin (*Polydactylus octonemus*)
Tidewater silverside (*Menidia beryllina*)[Kb]
Tilapia (*Oreochromis niloticus*, *O. mossambicus*)
Toadfish (*Opsanus tau*) [Kb]
Tomtate (*Haemulon aurolineatum*)[Kb]
Tripletail (*Lobotes surinamensis*)[Kb]
Trunkfish (buffalo trunkfish) (*Lactophrys trigonus*)[Kb]
Warsaw grouper (*Epinephelus nigritis*)[Kb]
Whip eel (*Basanichthyes scuticaris*)
White bass (*Morone chrysops*)
White crappie (*Pomoxis annularis*)
White grunt (*Haemulon plumieri*)[Kb]
White perch (*Morone americana*)
Whiting (genus, species unspecified)
Winter flounder (*Pseudopleuronectes americanus*)
Yellow pike (walleye) (*Sander vitreus*)
Yellowtail amberjack (*Seriola lalandi*)[Kb]
Zebrafish (*Danio rerio*)

Ascidians[Kb]

II. Invertebrates

Annelids (Phylum Annelida)

Common clam worm (*Nereis succinea*)
Polychaetes – *Americonuphis magna, Diopatra cuprea, Clymenella mucosa, Glycera americana*[Kb], *Glycera capitata*[Kb], *Laeonereis culven*[Kb], *Onuphis magna,* mudworm (*Polydora websteri*), *Scoloplos fragilis*[Kb], *S. rubra* [Kb], *S. squamata*[Kb]
Ragworm (*Branchioasychus americanus*, *Neanthes succinea*[Kb], *Nereis* sp.)

Arthropods (Phylum Arthropoda)

Chelicerates

Horseshoe crab (*Limulus polyphemus*)[Kb]

Crustaceans

American lobster (*Homarus americanus*)
Barnacle (*Balanus* sp.)
Blue crab (*Callinectes sapidus*)[Kb]
Blue shrimp (*Penaeus stylirostris*)
Brine shrimp (*Artemia salina*)
Brown rock crab (*Cancer antennarius*)
Calico crab (Dolly Varden crab) (*Hepatus epheliticus*)

Daggerblade grass shrimp (*Palaemonetes pugio*)
Depressed mud crab (*Eurypanopeus depressus*)
Dungeness crab (*Cancer magister*)
Dwarf crab (lesser blue crab) (*Callinectes similis*)
Flat porcelain crab (*Petrolisthes cinctipes*)
Florida stone crab (*Menippe mercenaria*)
Green porcelain crab (*Petrolisthes armatus*)
Ivory barnacle (*Balanus eburneus*)
Lady crab (*Hepatus epheliticus*)[Kb]
Morbid sand crab (*Emerita analoga* [*Hippa analoga*])
Pacific oyster (*Ostrea lurido*)
Pandalid shrimp (*Pandalus platyceros*)
Pea crab (*Pinnixa* sp.)[Kb]
Porcelain crab (*Petrolisthes armatus*)
Razor clam (*Siliqua patula*)
Red king crab (*Paralithodes camtschatica*)
Red swamp crayfish (*Procambarus clarkii*)
Sand flea (mole crab, sand crab) (*Emerita benedicti*)
Shrimps (*Penaeus* spp.)
Signal crayfish (*Pacifastacus leniusculus*)
Southwestern Atlantic burrowing crab (South American estuarine crab) (*Neohelice granulata* or *Chasmagnathus granulata*)
Speckled swimming crab (*Arenaeus cribarius*)
Spiny lobster (*Panulirus argus*)
Spotted porcelain crab (saltwater porcelain crab) (*Porcellana sayana*)
Stone crab (*Menippe mercenaria*)
Surf hermit crab (surf hermit) (*Isocheles wurdemanni*)
Tanner crab (*Chionoecetes bairdi*)
Thinstripe hermit crab (*Clibanarius vittatus*)

Brachiopods (Phylum Brachiopoda)

Lampshell (*Glottidia pyramidatum*)[Kb]

Cnidarians (Phylum Cnidaria)

Corals (*Oculina diffusa, Slenastrea hyades, Stephanocoenia intersepta, Siderastrea* spp.) [Kb]
Warty sea anemone (*Bunodosoma cavernata*)

Echinoderms (Phylum Echinodermata)

Brittle star (*Micropholis atra*)
Six-keyhole sand dollar (*Mellita quinquesperforata*)
Holothurians (sea cucumbers; genera, species not given)[Kb]

Molluscs (Phylum Mollusca)

Bivalves (Class Bivalvia)

Asian clam (*Corbicula fluminea*)
Atlantic deep-sea scallop (*Placopecten magellanicus*)
Atlantic surfclam (*Spisula solidissima*)
Bay scallop (*Argopectens irradians*)[Kb]
Blood ark clam (blood ark) (*Andara ovalis*)
Blue mussel (*Mytilus edulis*)
Brazil ark (incongruous ark clam) (*Anadara brasiliana*)
Butter clam (*Saxidomus giganteus*)
California mussel (*Mytilus californianus*)
California semele (*Cumingia californica*)
California tagelus (*Tagelus californianus*)
California venus (*Chione californiensis*)
Californian beanclam (*Donax californicus*)
Carpet shell clam (*Ruditapes decussatus*)
Dwarf surfclam (coot clam) (*Mulinia lateralis*)[Kb]
Eastern oyster (*Crassostrea virginica* [*Ostrea virginicus*])
European flat oyster (*Ostrea edulis*)
Farmer's scallop (*Chlamys farreri*)
Florida coquina (coquina, coquina clam) (*Donax variabilis*)
Freshwater clam (*Anodonta grandis simpsoniana*)
Freshwater mussel (*Anodonta cygnea*)
Frilled venus (*Chione undatella*)
Gaper clam (horseneck clam) (*Tresus capax*)
Gould beanclam (*Donax gouldii*)
Great scallop (*Pecten maximus*)
Green mussel (*Perna viridis*)
Hooked mussel (*Ischadium recurvum* [*Brachidontes recurvus*])
Japanese littleneck clam (*Venerupis japonica*)
Kelp scallop (*Leptopecten* [*Pecten*] *latiauratus*)
Lamellibranch bivalve (*Lasaea rubra*)
Littleneck clam (*Protothaca staminea*)
Mediterranean mussel (*Mytilus galloprovincialis*)
Minor jacknife (*Ensis minor*)[Kb]
Northern horsemussel (*Modiolus modiolus*)
Northern quahog (*Mercenaria mercenaria*)
Nuttall's cockle (basket cockle, heart cockle) (*Clinocardiurn nuttali*)
Ocean quahog (*Arctica islandica*)
Olympia oyster (*Ostrea lurida*)
Pacific eggcockle (*Laevicardium substriatum*)
Pacific oyster (*Ostrea lurido*)
Quahog (*Mercenaria* sp.)
Ribbed mussel (*Geukensia demissa*)
Rock scallop (*Hinnites multirugosus*)
Sea scallop (*Placopecten magellanicus*)
Softshell clam (*Mya arenaria*)

Spiny scallop (swimming scallop) (*Chlamys hastata*)
Straight horsemussel (*Modiolus rectus*)
Surfclam (*Spisula solidissima*)
Swam mussel (*Anodonta cygnea*)
Weathervane scallop (*Pecten caurinus*)
Zebra mussel (*Dreissenia polymorpha*)

Cephalopods (Class Cephalopoda)

Octopus (*Tevila crassatelloidis*)

Gastropods

Abalone (*Haliotis discus, Haliotis midae*)
Auger snail (*Terebra cinerea*)
Banded pheasant (*Eulithidium comptum* [as *Tricolia compta*])
Banded tulip (*Fasciolaria lilium hunteria*)
Barrel-bubble (*Acteocina californica*)
Beatic dwarf olive (*Olivella baetica*)
Blister glassy-bubble (*Haminoea viscula*)
Bruised nassa (*Nassarius vibex*)[Kb]
California cone (*Conus californicus*)
Common whelk (*Buccinum undahtum*)
Crown conch (*Melongena corona*)
Double moon shell (*Polinices duplicatus*)
Florida dogwinkle (*Thais haemastoma*)
Gray augur (*Terebra cinerea*)
Great pond snail (*Lymnaea stagnalis*)
Lettered olive (*Oliva sayana*)
Limpets (genera, species not given)
Lottia (*Acmaea conus*)
Striped false limpet (*Siphonaria benedicti*)
Tectura (*Acmaea depicta*)
Long-tailed sea hare (sea slug) (*Stylocheilus longicauda*)
Malaysian trumpet snail (*Melanoides tuberculata*)
Marsh ramshorn (*Planorbella trivolvis* [*Helisoma trivolvis*])
Mexican pyramidella (*Pyramidella mexicana*)
Moonsnail (*Neverita reclusiana* [*Polinices recluzianus imperforatus*])
Onyx slippersnail (*Crepidula onyx*)
Purple dwarf olive (*Olivella biplicata*)
Shark eye (*Polinices duplicata*)
Tadpole physa (*Physa gyrina*)
Western mud nassa (*Nassarius tiarula* [*N. tegulus*])

Phoronids (Phylum Phoronida)

Horseshoe worm (*Phoronis architecta*)[Kb]

Sponges (Phylum Porifera)

Branch candle sponge (*Vergangid longissima*)
Gray-purple sponge (*Spinosella vaginalis*)
Ircinia sp.
Loggerhead sponge (*Spheciospongia vesparium*)
Sheepswool sponge (*Hippiospongia laehna*)
Stinker sponge (*Ircinia felix*)
Vase sponge (*Ircinia campana*)

Macrophytes (Vascular Plants)

Mosses (Phylum Bryophyta)

Java moss (*Vesicularia dubyana*)

Flowering plants (Phylum Anthophyta)

Coontail (*Ceratophyllum, Ceratophyllum demersum*)
Eelgrass, marine eelgrass (*Zostera marina*)
Elodea (*Elodea canadensis*)
Eurasian watermilfoil (*Myriophyllum spicatum*)
Freshwater eelgrass (tapegrass) (*Valisneria americana*)
Hydrilla (*Hydrilla verticillata*)
Manateegrass (*Syringodium filiforme*)
Phragmites (common reed) (*Phragmites australis*)
Round-leaf seagrass (*Syringodium isoetifolium*)
Shoalgrass (*Halodule wrightii*)
Turtlegrass (*Thalassia testudinum*)

References and Further Reading

Abbott, B.C., and D. Balantine. 1957. The toxin from *Gymnodinium veneficum* Ballantine. *Journal of the Marine Biological Association UK*, **36**: 1691–1689.

Adolf, J.E., T. Bachvaroff, and A.R. Place. 2008. Can cryptophytes trigger toxic *Karlodinium veneficum* blooms in eutrophic estuaries? *Harmful Algae*, **81**: 19–128.

Adolf, J.E., D. Krupatkina, T. Bachvaroff, and A.R. Place. 2007. Karlotoxin mediates grazing by *Oxyrrhis marina* on strains of *Karlodinium veneficum*. *Harmful Algae*, **6**: 400–412.

Aguilera, A., X. Gutiérrez, J. Mayorga, F. Villanueva, and D. Varela. 2016. Effects of *Alexandrium catenella* on post smolt salmon. Available at: https://www.researchgate.net/publication/309397880_Effects_of_Alexandrium_catenella_on_post_smolt_salmon.

Ajuzie, C.C. 2007. Palatability and fatality of the dinoflagellate *Prorocentrum lima* to *Artemia salina*. *Journal of Applied Phycology*, **19**: 513–519.

Ajuzie, C.C. 2008. Toxic *Prorocentrum lima* induces abnormal behaviour in juvenile sea bass. *Journal of Applied Phycology*, **20**: 19–27.

Akin-Oriola, G.A., and L.A. Lawton. 2006. The detection and quantification of cyanobacterial toxins in water using the brine shrimp (*Artemia salina*) assay. *West African Journal of Applied Ecology*, **9**: 6 p.

Al-Sammak, M.A., K.D. Hoagland, D. Cassada, and D.D. Snow. 2014. Co-occurrence of the cyanotoxins BMAA, DABA, and anatoxin-a in Nebraska reservoirs, fish, and aquatic plants. *Toxins*, **6**: 488–508.

Amorim, A., and V. Vasconcelos. 1999. Dynamics of microcystins in the mussel *Mytilus galloprovincialis*. *Toxicon*, **37**: 1041–1052.

Anderson, D.M., and A.W. White. 1992. Marine biotoxins at the top of the food chain. *Oceanus*, **35**: 55–61.

Anonymous. 2001. Algae: Invasive Alien *Gracilaria tikvahiae* McLachlan 1987. Botany, University of Hawaíi at Manoa, Manoa. Available at: http://www.hawaii.edu/reefalgae/invasive_algae/pdf%20files/gracilaria_tikvahiae.pdf.

Anthoni, U., C. Christophersen, J.O. Madsen, S. Wium-Andersen, and N. Jacobson. 1980. Biologically active sulphur compounds from the green alga *Chara globularis*. *Phytochemistry*, **19**: 1228–1229.

Arthur, K., G. Shaw, C. Limpus, and J. Udy. 2006. A review of the potential role of tumour-promoting compounds produced by *Lyngbya majuscula* in marine turtle fibropapillomatosis. *African Journal of Marine Science*, **28**: 441–446.

Avery, D.E., K.J.K. Altland, and H.G. Dam. 2008. Sex-related differential mortality of a marine copepod exposed to a toxic dinoflagellate. *Limnology and Oceanography*, **53**: 2627–2635.

Babcock-Jackson, L., W.W. Carmichael, and D.A. Culver. 2002. Dreissenid mussels increase exposure of benthic and pelagic organisms to toxic microcystins. *Verhandlungen des Internationalen Verein Limnologie*, **28**: 1082–1085.

Badylak, S., and E.J. Phlips. 2008. Spatial and temporal distributions of zooplankton in Tampa Bay, Florida, including observations during a HAB event. *Journal of Plankton Research*, **30**: 449–465.

Badylak, S., E.J. Phlips, and A.L. Mathews. 2014a. *Akashiwo sanguinea* (Dinophyceae) blooms in a sub-tropical estuary: an alga for all seasons. *Plankton and Benthos Research*, **9**: 147–155.

Badylak, S., E.J. Phlips, A.L. Mathews, and K. Kelley. 2014b. *Akashiwo sanguinea* (Dinophyceae) extruding mucous from pores on the cell surface. *Algae*, **29**: 197–201.

Baganz, D., G. Staaks, and C. Steinberg. 1998. Impact of the cyanobacteria toxin, microcystin-LR on behaviour of zebra fish, *Danio rerio*. *Water Research*, **32**: 948–952.

Bagarinao, T. 1992. Sulfide as an environmental factor and toxicant: tolerance and adaptations in aquatic organisms. *Aquatic Toxicology*, **24**: 21–62.

Bagøien, E., A. Miranda, B. Reguera, and J.M. Franco. 1996. Effects of two paralytic shellfish toxin producing dinoflagellates on the pelagic harpacticoid copepod *Euterpina acutifrons*. *Marine Biology*, **126**: 361–369.

Baker, S.M., J.S. Levinton, J.P. Kurdziel, and S.E. Shumway. 1998. Selective feeding and biodeposition by zebra mussels and their relation to changes in phytoplankton composition and seston load. *Journal of Shellfish Research*, **17**: 1207–1213.

Balestra, C., L. Alonso-Sáez, J.M. Gasol, and R. Casotti. 2011. Group-specific effects on coastal bacterioplankton of polyunsaturated aldehydes produced by diatoms. *Aquatic Microbial Ecology*, **63**: 123–131.

Banack, S.A., T.G. Downing, Z. Spácil, E.L. Purdie, J.S. Metcalf, S. Downing, M. Esterhuizen, G.A. Codd, and P.A. Cox. 2010. Distinguishing the cyanobacterial neurotoxin β-*N*-methylamino-l-alanine (BMAA) from its structural isomer 2,4-diaminobutyric acid (2,4-DAB). *Toxicon*, **56**: 868–879.

Bardouil, M., M. Bohec, M. Cormerais, S. Bougrier, and P. Lassus. 1993. Experimental study of the effects of a toxic microalgal diet on feeding of the oyster *Crassostrea gigas* Thunberg. *Journal of Shellfish Research*, **12**: 417–422.

Bargu, S., E. Smith, and K. Ozhan. 2011. Toxic diatom *Pseudo-nitzschia* and its primary consumers (vectors). *The Diatom World*, **19**: 491–512.

Barica, J. 1978. Collapse of *Aphanizomenon flos-aquae* blooms resulting in massive fish kills in eutrophic lakes: effect of weather. *Verhandlungen des Internationalen Verein Limnologie*, **20**: 208–213.

Barreiro, A., C. Guisande, I. Maneiro, T.P. Lien, C. Legrand, T. Tamminen, S. Lehtinen, P. Uronen, and E. Granéli. 2005. Relative importance of the different negative effects of the toxic haptophyte *Prymnesium parvum* on *Rhodomonas salina* and *Brachionus plicatilis*. *Aquatic Microbial Ecology*, **38**: 259–267.

Basti, L., H. Hégaret, and S.E. Shumway. 2018. Harmful algal blooms and shellfish. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 135–190.

Basti, L., K. Nagai, J. Go, S. Okano, T. Oda, Y. Tanaka, and S. Nagai. 2016. Lethal effects of

ichthyotoxic raphidophytes, *Chattonella marina, C. antiqua,* and *Heterosigma akashiwo,* on post-embryonic stages of the Japanese pearl oyster, *Pinctada fucata martensii*. *Harmful Algae*, **59**: 112–122.

Bates, S.S., and V.L. Trainer. 2006. Ecology of harmful diatoms. In: *Ecology of Harmful Algae*, Vol. 189. E. Granéli and J. Turner (Eds.). Springer, Heidelberg: p. 81–93.

Bauder, A., and A.D. Cembella. 2000. Viability of the toxic dinoflagellate *Prorocentrum lima* following ingestion and gut passage in the bay scallop *Argopecten irradians*. *Journal of Shellfish Research*, **19**: 321–324.

Bauder, A., A.D. Cembella, V.M. Bricelj, and M.A. Quilliam. 2001. Uptake and fate of diarrhetic shellfish poisoning toxins from the dinoflagellate *Prorocentrum lima* in the bay scallop *Argopecten irradians*. *Marine Ecology Progress Series*, **213**: 39–52.

Beardall, J. 2008. Blooms of *Synechococcus*: An Analysis of the Problem Worldwide and Possible Causative Factors in Relation to Nuisance Blooms in the Gippsland Lakes. A report prepared for the Gippsland Task Force. School of Biological Sciences, Monash University, Victoria, South Africa.

Bergkvist, J., E. Selander, and H. Pavia. 2008. Induction of toxin production in dinoflagellates: the grazer makes a difference. *Oecologia*, **156**: 147–154.

Berry, D.L., J.A. Goleski, F. Koch, C.C. Wall, B.J. Peterson, O.R. Anderson, and C. Gobler. 2015. Shifts in cyanobacterial strain dominance during the onset of harmful algal blooms in Florida Bay, USA. *Microbial Ecology*, **70**: 361–371.

Bertin, M.J., P.V. Zimba, K.R. Beauchesne, K.M. Huncik, and P.D.R. Moeller. 2012a. Identification of toxic fatty acid amides isolated from the harmful alga *Prymnesium parvum* Carter. *Harmful Algae*, **20**: 111–116.

Bertin, M.J., P.V. Zimba, K.R. Beauchesne, K.M. Huncik, P.D.R. Moeller. 2012b. The contribution of fatty acid amides to *Prymnesium parvum* Carter toxicity. *Harmful Algae*, **20**: 117–125.

Beveridge, M.C.M., D.J. Baird, S.M. Rahmatulla, L.A. Lawton, K.A. Beattie, and G.A. Codd. 1993. Grazing rates on toxic and non-toxic strains of cyanobacteria by *Hypophthalamichtys molitrix* and *Oreochromis niloticus*. *Journal of Fish Biology*, **43**: 901–907.

Bidigare, R.R., S.J. Christensen, S.B. Wilde, and S.A. Banack. 2009. Cyanobacteria and BMAA: possible linkage with avian vacuolar myelinopathy (AVM) in the south-eastern United States. *Amyotrophic Lateral Sclerosis*, **10**: 71–73.

Black, E.A., J.N.C. Whyte, J.W. Bagshaw, and N.G. Ginther. 1991. The effects of *Heterosigma akashiwo* on juvenile *Oncorhynchus tshawytscha* and its implications for fish culture. *Journal of Applied Ichthyology*, **7**: 168–175.

Bolam, S., T.F Fernandes, P. Read, and D. Raffaelli. 2000. Effects of macroalgal mats on intertidal sandflats: an experimental study. *Journal of Experimental Marine Biology and Ecology*, **249**: 123–137.

Borowitzka, M.A. 2016. Chemically-mediated interactions in microalgae. In: *The Physiology of Microalgae. Developments in Applied Phycology*, Vol. 6. p. 321–357. Available at: http://link.springer.com/chapter/10.1007/978-3-319-24945-2_15/fulltext.html.

Bossart, G.D., D.G. Baden, R.Y. Ewing, B. Roberts, and S. Wright. 1998. Brevetoxicosis in manatees (*Trichechus manatus latirostris*) from the 1996 epizootic: gross, histologic, and immunohistochemical features. *Toxicologic Pathology*, **26**: 276–282.

Botes, L., A. Smit, and P. Cook. 2003. The potential threat of algal blooms to the abalone (*Haliotis midae*) mariculture industry situated around the South African coast. *Harmful Algae*, **2**: 247–259.

Bouvy, M., M. Pagano, and M. Troussellier. 2001. Effects of a cyanobacterial bloom (*Cylindrospermopsis raciborskii*) on bacteria and zooplankton communities in Ingazeira reservoir (northeast Brazil). *Aquatic Microbial Ecology*, **25**: 215–227.

Boyer, G. 2007. The occurrence of cyanobacterial toxins in New York lakes: lessons from the MERHAB-Lower Great Lakes program. *Lake and Reservoir Management*, **23**: 153–160.

Branch, G.M. 2008. Trophic interactions in subtidal rocky reefs on the west coast of South Africa. In: *Food Webs and the Dynamics of Marine Reefs*. T.R. McClanahan and G.M. Branch (Eds.). Oxford University Press, New York: p. 50–78.

Brand, L.E., J. Pablo, A. Compton, N. Hammerschlag, and D.C. Mash. 2010. Cyanobacterial blooms and the occurrence of the neurotoxin, beta-*N*-methylamino-L-alanine (BMAA), in South Florida aquatic food webs. *Harmful Algae*, **9**: 620–635.

Breitburg, D.L. 2002. Effects of hypoxia and the balance between hypoxia and enrichment on coastal fishes and fisheries. *Estuaries*, **25**: 767–781.

Breitburg, D.L., K.A. Rose, and J.H. Cowan Jr. 1999. Linking water quality to larval survival: predation mortality of fish larvae in an oxygen-stratified water column. *Marine Ecology Progress Series*, **178**: 39–54.

Breitburg, D.L., A. Adamack, K.A. Rose, S.E. Kolesar, M.B. Decker, J.E. Purcell, J.E. Keister, and J.H. Cowan Jr. 2003. The pattern and influence of low dissolved oxygen in the Patuxent River, a seasonally hypoxic estuary. *Estuaries*, **26**: 280–297.

Briand, E., N. Escoffier, C. Straub, M. Sabart, C. Quiblier, and J.-F. Humbert. 2009. Spatiotemporal changes in the genetic diversity of a bloom-forming *Microcystis aeruginosa* (cyanobacteria) population. *ISME Journal*, **3**: 419–429.

Bricelj, V.M., A.D. Cembella, D. Laby, S.E. Shumway, and T.L. Cucci. 1996. Comparative physiological and behavioural responses to PSP toxins in two bivalve molluscs, the softshell clam, *Mya arenaria,* and surfclam, *Spisula solidissima*. In: *Harmful and Toxic Algal Blooms*. T. Yasumoto, Y. Oshima, and Y. Fukuyo (Eds.). IOC UNESCO, Paris: p. 405–408.

Bricelj, V.M., J. Epp, and R.E. Malouf. 1987. Intraspecific variation in reproductive and somatic growth cycles of bay scallops *Argopecten irradians*. *Marine Ecology Progress Series*, **35**: 123–137.

Bricelj, V.M., and S.H. Kuenster. 1989. Effects of the "brown tide" on the feeding, physiology and growth of bay scallops and mussels. In: *Novel Phytoplankton Blooms*. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 491–509.

Bricelj, V.M., J.H. Lee, A.D. Cembella, and D.M. Anderson. 1990. Uptake kinetics of paralytic shellfish toxins from the dinoflagellate *Alexandrium fundyense* in the mussel *Mytilus edulis*. *Marine Ecology Progress Series*, **63**: 177–188.

Bricelj, V.M., and D.J. Lonsdale. 1997. *Aureococcus anophagefferens*: causes and ecological consequences of brown tides in U.S. mid-Atlantic coastal waters. *Limnology and Oceanography*, **42**: 1023–1038.

Bricelj, V.M., S.P. MacQuarrie, and R.A. Schaffner. 2001. Differential effects of *Aureococcus anophagefferens* isolates ("brown tide") in unialgal and mixed suspensions on bivalve feeding. *Marine Biology*, **139**: 605–615.

Bridgeman, T.B., and W.A. Penamon. 2010. *Lyngbya wollei* in western Lake Erie. *Journal of Great Lakes Research*, **36**: 167–171.

Britton-Simmons, K.H. 2004. Direct and indirect effects of the introduced alga *Sargassum muticum* on benthic, subtidal communities of Washington State, U.S.A. *Marine Ecology Progress Series*, **277**: 61–78.

Broadwater, M.H., F.M. Van Dolah, and S.E. Fire. 2018. Vulnerabilities of marine mammals to harmful algal blooms. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 191–222.

Brownlee, E.F., S.G. Sellner, K.G. Sellner, H. Nonogaki, J.E. Adolf, T.R. Bachvaroff, and A.R. Place. 2008. Responses of *Crassostrea virginica* (Gmelin) and *C. ariakensis* (Fujita) to bloom-forming phytoplankton including ichthyotoxic *Karlodinium veneficum* (Ballantine). *Journal of Shellfish Research*, **27**: 581–591.

Buchsbaum, R., I. Valiela, T. Swain, M. Dzierzesky, and S. Allen. 1991. Available and refractory nitrogen of coastal vascular plants and macroalgae. *Marine Ecology Progress Series*, **72**: 131–143.

Burkholder, J.M. 1998. Implications of harmful marine microalgae and heterotrophic dinoflagellates in management of sustainable marine fisheries. *Ecological Applications*, **8**: S37–S62.

Burkholder, J.M. 2002. Cyanobacteria. In: *Encyclopedia of Environmental Microbiology*. G. Bitton (Ed.). John Wiley & Sons, Inc., New York: p. 952–982.

Burkholder, J.M. 2009. Harmful algal blooms. In: *Encyclopedia of Inland Waters*, Vol. 1. G.E. Likens (Ed.). Elsevier, Oxford: p. 264–285.

Burkholder, J.M., and H.B. Glasgow. 1997. *Pfiesteria piscicida* and other toxic *Pfiesteria*-like dinoflagellates: behavior, impacts, and environmental controls. *Limnology and Oceanography*, **42**: 1052–1075.

Burkholder, J.M., H.B. Glasgow, and N.J. Deamer-Melia. 2001. Overview and present status of the toxic *Pfiesteria* complex. *Phycologia*, **40**: 186–214.

Burkholder, J.M., and P.M. Glibert. 2006. Intraspecific variability: an important consideration in forming generalizations about toxigenic algal species. *African Journal of Marine Science*, **28**: 177–180.

Burkholder, J.M., and P.M. Glibert. 2013. Eutrophication and oligotrophication. In: *Encyclopedia of Biodiversity*, 2nd ed., Vol. 3. S. Levin (Ed.). Academic Press, Waltham: p. 347–371.

Burkholder, J.M., P.M. Glibert, and H.M. Skelton. 2008. Mixotrophy, a major mode of nutrition for

harmful algal species in eutrophic waters. *Harmful Algae*, **8**: 77–93.

Burkholder, J.M., C.J. Gobler, and J.M. O'Neil. 2018. Cyanobacteria Fact Sheet. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 591–595.

Burkholder, J.M., A.S. Gordon, P.D. Moeller, J.M. Law, K.J. Coyne, A.J. Lewitus, J.S. Ramsdell, H.G. Marshall, N.J. Deamer, S.C. Cary, J.W. Kempton, S.L. Morton, and P.A. Rublee. 2005. Demonstration of toxicity to fish and to mammalian cells by *Pfiesteria* species: comparison of assay methods and multiple strains. *Proceedings of the National Academy of Sciences USA*, **102**: 3471–3476.

Burkholder, J.M., and H.G. Marshall. 2012. Toxigenic *Pfiesteria* species – updates on biology, ecology, toxins, and impacts. *Harmful Algae*, **14**: 196–230.

Burkholder, J.M., D. Tomasko, and B.W. Touchette. 2007. Seagrasses and eutrophication. *Journal of Experimental Marine Biology and Ecology*, **350**: 46–72.

Bury, N.R., G. Flik, F.B. Eddy, and G.A. Codd. 1996a. The effects of cyanobacteria and the cyanobacterial toxin microcystin-LR on Ca^{2+} transport and Na^{+}/K^{+}-ATPase in tilapia gills. *Journal of Experimental Biology*, **199**: 1319–1326.

Bury, N.R., F.B. Eddy, and G.A. Codd. 1996b. The stress responses of the brown trout, *Salmo trutta* to the cyanobacterium *Microcystis aeruginosa*. *Environmental Toxicology and Water Quality*, **11**: 187–193.

Bury, N.R., G.A. Codd, S.E. Wendelaar Bonga, and G. Flik. 1998. Fatty acids from the cyanobacterium *Microcystis aeruginosa* with potent inhibitory effects of fish gill Na^{+}/K^{+}-ATPase activity. *Journal of Experimental Biology*, **201**: 81–89.

Buskey, E.J. 1995. Growth and bioluminescence of *Noctiluca scintillans* on varying algal diets. *Journal of Plankton Research*, **17**: 29–40.

Buskey, E.J. 2008. How does eutrophication affect the role of grazers in harmful algal bloom dynamics? *Harmful Algae*, **8**: 152–157.

Buskey, E. J., and C.J. Hyatt. 1995. Effects of the Texas (USA) "brown tide" alga on planktonic grazers. *Marine Ecology Progress Series*, **126**: 285–292.

Buskey, E.J., H. Liu, C. Collumb, and J.G.F. Bersano. 2001. The decline and recovery of a persistent Texas brown tide algal bloom in the Laguna Madre (Texas, USA). *Estuaries*, **24**: 337–346.

Buskey, E.J., P.A. Montagna, A.F. Amos, and T.E. Whitledge. 1997. Disruption of grazer populations as a contributing factor to the initiation of the Texas brown tide algal bloom. *Limnology and Oceanography*, **42**: 1215–1222.

Buskey, E.J., S. Stewart, J. Peterson, and C. Collumb. 1996. Current Status and Historical Trends of Brown Tide and Red Tide Phytoplankton Blooms in the Corpus Christi Bay National Estuary Program Study Area. Publication CCBNEP-07. Texas Natural Resources Conservation Commission, Austin.

Buskey, E.J., and D.A. Stockwell. 1993. Effects of a persistent 'brown tide' on zooplankton populations in the Laguna Madre of South Texas. In: *Toxic Phytoplankton Blooms in the Sea, Proceedings of the Fifth International Conference Toxic Marine Phytoplankton*. T.J. Smayda, and Y. Shimizu (Eds.). Elsevier Science Publishers, Amsterdam: p. 659–666.

Busse, L.B., E.L. Venrick, R. Antrobus, P.E. Miller, V. Vigilant, M.W. Silver, C. Mengelt, L. Mydlarz, and B.B. Prezelin. 2006. Domoic acid in phytoplankton and fish in San Diego, CA, USA. *Harmful Algae*, **5**: 91–101.

Butler, M.J., J.H. Hunt, W.F. Herrnkind, M.J. Childress, R. Bertelsen, W. Sharp, T. Mathews, J.M. Field, and H.G. Marshall. 1995. Cascading disturbances in Florida Bay, USA: cyanobacteria blooms, sponge mortality and implications for juvenile spiny lobsters *Panulirus argus*. *Marine Ecology Progress Series*, **129**: 119–123.

Buttino, I., G. de Rosa, Y. Carotenuto, M. Mazzella, A. Ianora, F. Esposito, V. Vitiello, F. Quaglia, M.I. La Rotonda, and A. Miralto. 2008. Aldehyde-encapsulated liposomes impair marine grazer survivorship. *Journal of Experimental Biology*, **211**: 1426–1433.

Cabral, J., M. Pardal, R. Lopes, T. Murias, and J. Marques. 1999. The impact of macroalgal blooms on the use of the intertidal area and feeding behavior of waders (Charadrii) in the Mondego estuary (west Portugal). *Acta Oecologica*, **20**: 417–427.

Cabrita, M.T., C. Vale, and A.P. Rauter. 2010. Halogenated compounds from marine algae. *Marine Drugs*, **8**: 2301–2317.

Caldwell, G.S., S.B. Watson, and M.G. Bentley. 2004. How to assess toxin ingestion and post-ingestion partitioning in zooplankton? *Journal of Plankton Research*, **26**: 1369–1377.

Camacho, F.A., and R.W. Thacker. 2006. Amphipod herbivory on the freshwater cyanobacterium *Lyngbya wollei*: chemical stimulants and morphological defenses. *Limnology and Oceanography*, **51**: 1870–1875.

Caron, D.A., C.J. Gobler, D.J. Lonsdale, R.M. Cerrato, R.A. Schaffner, J.M. Rose, N. J. Buck, G. Taylor, K.R. Boissonneault, and R. Mehrane. 2004. Microbial herbivory on the brown tide alga, *Aureococcus anophagefferens*: results from natural ecosystems, mesocosms and laboratory experiments. *Harmful Algae*, **3**: 439–457.

Carpenter, S.R., D. Ludwig, and W.A. Brock. 1999. Management of eutrophication for lakes subject to potentially irreversible change. *Ecological Applications*, **9**: 751–771.

Casini, M., J. Lovgren, J. Hjelm, M. Cardinale, J. Molinero, and G. Komilovs. 2008. Multi-level trophic cascades in a heavily exploited open marine ecosystem. *Proceedings of the Royal Society B*, **275**: 1793–1801.

Castle, K.T., L.J. Flewelling, J. Bryan II, A. Kramer, J. Lindsay, C. Nevada, W. Stablein, D. Wong, and J.H. Landsberg. 2013. Coyote (*Canis latrans*) and domestic dog (*Canis familiaris*) mortality and morbidity due to a *Karenia brevis* red tide in the Gulf of Mexico. *Journal of Wildlife Diseases*, **49**: 955–964.

Castonguay, M., M. Levasseur, J.-L. Beaulieu, F. Grégoire, S. Michaud, E. Bonneau, and S.S. Bates. 1997. Accumulation of PSP toxins in Atlantic mackerel: seasonal and ontogenetic variations. *Journal of Fish Biology*, **50**: 1203–1213.

Castro, L.R., and R.K. Cowen. 1989. Growth rates of bay anchovy (*Anchoa mitchilli*) in Great South Bay under recurrent brown tide conditions, summers 1987 and 1988. In: *Novel Phytoplankton Blooms. Coastal and Estuarine Studies*, Vol. 35. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 663–674.

Cattet, M., and J.R. Geraci. 1993. Distribution and elimination of ingested brevetoxin (PBTx-3) in rats. *Toxicon*, **31**: 1483–1486.

Chang, F. H., C. Anderson, and N.C. Boustead. 1990. First record of a *Heterosigma* (Raphidophyceae) bloom with associated mortality of cage-reared salmon in Big Glory Bay, New Zealand. *New Zealand Journal of Marine and Freshwater Research*, **24**: 461–469.

Chang, Z., N. Sitachitta, J.V. Rossi, M.A. Roberts, P.M. Flatt, J. Jia, D.H. Sherman, and W.H. Gerwick. 2004. Biosynthetic pathway and gene cluster analysis of curacin A, an antitubulin natural product from the tropical marine cyanobacterium *Lyngbya majuscula*. *Journal of Natural Products*, **67**: 1356–1367.

Charpy, L., B.E. Casareto, M.J. Langlade, and Y. Suzuki. 2012. Cyanobacteria in coral reef ecosystems: a review. *Journal of Marine Biology*, 2012: art. ID 257571, 9 p. doi: 10.1155/2012/259571.

Chen, J., D. Zhang, P. Xie, Q. Wang, and Z. Ma. 2009. Simultaneous determination of microcystin contaminations in various vertebrates (fish, turtle, duck and water bird) from a large eutrophic Chinese lake, Lake Taihu, with toxic *Microcystis* blooms. *Science of the Total Environment*, **407**: 3317–3322.

Cho, H.J., and K. Matsuoka. 2000. Cell lysis of a phagotrophic dinoflagellate, *Polykrikos kofoidii* feeding on *Alexandrium tamarense*. *Plankton Biology and Ecology*, **47**: 134–136.

Chorus, I., and J. Bartram (Eds.). 1999. Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management. E&FN Spon for the World Health Organization, New York.

Christoffersen, K. 1996. Ecological implications of cyanobacterial toxins in aquatic food webs. *Phycologia*, **35**: 42–50.

Ciminiello, P., C. Dell'Aversano, E. Dello Iacovo, E. Fattorusso, and M. Forino. 2010. Complex palytoxin-like profile of *Ostreopsis ovata.* Identification of four new ovatoxins by high-resolution liquid chromatography/mass spectrometry. *Rapid Communications in Mass Spectrometry*, **24**: 2735–2744.

Ciminiello, P., C. Dell'Aversano, E. Dello Iacovo, E. Fattorusso, and M. Forino. 2012. Unique toxin profile of a Mediterranean *Ostreopsis* cf. *ovata* strain: HR LC-MS^n characterization of ovatoxin-f, a new palytoxin congener. *Chemical Research in Toxicology*, **25**: 1243–1252.

Ciminiello, P., C. Dell'Aversano, E. Fattorusso, M. Forino, and L. Grauso. 2011. A 4-decade-long (and still ongoing) hunt for palytoxins chemical architecture. *Toxicon*, **57**: 362–367.

Codd, G.A. 1995. Cyanobacterial toxins: occurrence, properties and biological significance. *Water Science and Technology*, **32**: 149–156.

Codd, G.A., L.F. Morrison, and J.S. Metcalf. 2005. Cyanobacterial toxins: risk management for health protection. *Toxicology and Applied Pharmacology*, **203**: 264–272.

Codd, G.A., C.J. Ward, and S.G. Bell. 1996. Cyanobacterial toxins: occurrence, modes of action, health effects and exposure routes. In: *Applied Toxicology: Approaches through Basic Science*. J.P. Seilerand and E. Vilanova (Eds.). Proceedings of the 1996 EUROTOX Congress Meeting held in Alicante, Spain, September 22–25, 1996. Springer, Berlin: p. 399–410.

Collie, J.S., K. Richardson, and J.H. Steele. 2004. Regime shifts: can ecological theory illuminate the mechanisms? *Progress in Oceanography*, **60**: 281–302.

Colman, J.R., M.-Y.B. Dechraoui, R.W. Dickey, and J.S. Ramsdell. 2004. Characterization of the developmental toxicity of Caribbean ciguatoxins in finfish embryos. *Toxicon*, **44**: 59–66.

Connell, C.H., and J.B. Cross. 1950. Mass mortality of fish associated with the protozoan *Gonyaulax* in the Gulf of Mexico. *Science*, **112**: 359–363.

Cox, P.A., S.A. Banack, S.J. Murch, U. Rasmussen, G. Tien, R.R. Bidigare, J.S. Metcalf, L.F. Morrison, G.A. Codd, and B. Bergman. 2005. Diverse taxa of cyanobacteria produce β-*N*-methylamino-L-alanine, a neurotoxic amino acid. *Proceedings of the National Academy of Sciences USA*, **102**: 5074–5078.

Curtiss, C.C., G.W. Langlois, L.B. Busse, F. Mazzillo, and M.W. Silver. 2008. The emergence of *Cochlodinium* along the California Coast (USA). *Harmful Algae*, **7**: 337–346.

Daan, R. 1987. Impact of egg predation by *Noctiluca miliaris* on the summer development of copepod populations in the southern North Sea. *Marine Ecology Progress Series*, **37**: 9–17.

Dale, B., M. Edwards, and P.C. Reid. 2006. Climate change and harmful algal blooms. *Ecology of Harmful Algae*, **189**: 367–378.

Dam, H.G., and S.P. Colin. 2005. *Prorocentrum minimum* (clone Exuv) is nutritionally insufficient, but not toxic to the copepod *Acartia tonsa*. *Harmful Algae*, **4**: 575–584.

Darnerud, P.O. 2003. Toxic effects of brominated flame retardants in man and in wildlife. *Environment International*, **29**: 841–853.

D'Avanzo, C., and J. Kremer. 1994. Diel oxygen dynamics and anoxic events in a eutrophic estuary. *Estuaries*, **17**: 131–139.

Davin, W.T. Jr., and C.C. Kohler. 1986. Effects of ciguatera toxins on the bluehead. *Transactions of the American Fisheries Society*, **115**: 908–912.

Davin, W.T., C.C. Kohler, and D.R. Tindall. 1988. Ciguatera toxins adversely affect piscivorous fishes. *Transactions of the American Fisheries Society*, **117**: 374–384.

Davis, T.W., D.L. Berry, G.L. Boyer, and C.J. Gobler. 2009. The effects of temperature and nutrients on the growth and dynamics of toxic and non-toxic strains of *Microcystis* during cyanobacteria blooms. *Harmful Algae*, **8**: 715–725.

Davis, T.W., M.J. Harke, M.A. Marcoval, J. Goleski, C. Orano-Dawson, D.L. Berry, and C.J. Gobler. 2010. Effects of nitrogenous compounds and phosphorus on the growth of toxic and non-toxic strains of *Microcystis* during cyanobacterial blooms. *Aquatic Microbial Ecology*, **61**: 149–162.

Dechraoui, M.-Y.B., L. Kashinsky, Z. Wang, and J.S. Ramsdell. 2011. Identification of ciguatoxins in Hawaiian monk seals *Monachus schauinslandi* from the northwestern and main Hawaiian Islands. *Environmental Science & Technology*, **45**: 5403–5409.

Deeds, J.R., and A.R. Place. 2006. Sterol-specific membrane interactions with the toxins from *Karlodinium micrum* (Dinophyceae): a strategy for self-protection? *African Journal of Marine Science*, **28**: 421–427.

Deeds, J.R., R. Reimschuessel, and A.R. Place. 2006. Histopathological effects in fish exposed to the toxins from *Karlodinium micrum*. *Journal of Aquatic Animal Health*, **18**: 136–148.

Deeds, J.R., D.E. Terlizzi, J.E. Adolf, D.K. Stoecker, and A.R. Place. 2002. Toxic activity from cultures of *Karlodinium micrum* (= *Gyrodinium galatheanum*) (Dinophyceae): a dinoflagellate associated with fish mortalities in an estuarine aquaculture facility. *Harmful Algae*, **1**: 169–189.

de Figueiredo, D.R., S.C. Antunes, M.J. Pereira, and F. Gonçalves. 2004b. Chronic effects of *Aphanizomenon flos-aquae* on the survival and reproduction of daphnids. *Fresenius Environmental Bulletin*, **13**: 665–670.

de Figueiredo, D.R., U.M. Azeiteiro, S.M. Esteves, F.J.M. Gonçalves, and M.J. Pereira. 2004a. Microcystin-producing blooms: a serious global public health issue. *Ecotoxicology and Environmental Safety*, **59**: 151–163.

de la Cruz, A.A., A. Hiskia, T. Kaloudis, N. Chernoff, D. Hill, M.G. Antoniou, X. He, K. Loftin, K. O'Shea, C. Zhao, M. Pelaez, C. Han, T.J. Lynch, and D.D. Dionysiou. 2013. A review on cylindrospermopsins: the global occurrence, detection, toxicity and degradation of a potent cyanotoxin. *Environmental Science: Processes & Impacts*, **15**: 1979–2003.

de Wit, C.A. 2002. An overview of brominated flame retardants in the environment. *Chemosphere*, **46**: 583–624.

Del Rio, R., S. Bargu, D. Baltz, S. Fire, G. Peterson, and Z. Wang. 2010. Gulf menhaden (*Brevoortia patronus*): a potential vector of domoic acid in coastal Louisiana food webs. *Harmful Algae*, **10**: 19–29.

Dela-Cruz, J., P. Ajani, R. Lee, T. Pritchard, and I. Suthers. 2002. Temporal abundance patterns of the red tide dinoflagellate *Noctiluca scintillans* along the southeast coast of Australia. *Marine Ecology Progress Series*, **236**: 75–88.

Demir, E., K.J. Coyne, M.A. Doblin, S.M. Handy, and D.A. Hutchins. 2008. Assessment of

microzooplankton grazing on *Heterosigma akashiwo* using a species-specific approach combining quantitative real-time PCR (QPCR) and dilution methods. *Microbial Ecology*, **55**: 583–594.

DeMott, W.R. 1999. Foraging strategies and growth inhibition in five daphnids feeding on mixtures of a toxic cyanobacterium and a green alga. *Freshwater Biology*, **42**: 263–274.

DeMott, W.R., and S. Dhawale. 1995. Inhibition of in vitro protein phosphatase activity in three zooplankton species by microcystin-LR, a toxin from cyanobacteria. *Archiv für Hydrobiologie*, **134**: 417–424.

DeMott, W.R., Q.-X. Zhang, and W.W. Carmichael. 1991. Effects of toxic cyanobacteria and purified toxins on the survival and feeding of a copepod and three species of *Daphnia*. *Limnology and Oceanography*, **36**: 1346–1357.

Dennison, W.C., G.J. Marshall, and W. Wigand. 1989. Effect of "brown tide" shading on eelgrass (*Zostera marina*) distributions. In: *Novel Phytoplankton Blooms*. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Coastal and Estuarine Studies, Vol. 35. Springer, Berlin: p. 675–692.

Desbiens, M., F. Coulombe, J. Gaudreault, and R. Larocque. 1989. PSP toxicity of wild and cultured blue mussels induced by *Alexandrium excavatum* in Gaspé Bay (Canada): implications for aquaculture. In: *Toxic Marine Phytoplankton*. E. Granéli, B. Sundstrom, L. Edler, and D.M. Anderson (Eds.). Elsevier, Amsterdam: p. 459–468.

Desforges, J.P.W., P.S. Ross, and L.L. Loseto. 2012. Transplacental transfer of polychlorinated biphenyls and polybrominated diphenyl ethers in Arctic beluga whales (*Delphinapterus leucas*). *Environmental Toxicology and Chemistry*, **31**: 296–300.

deYoung, B., M. Barange, G. Beaugrand, R. Harris, R.I. Perry, M. Scheffer, and F. Werner. 2008. Regime shifts in marine ecosystems: detection, prediction and management. *Trends in Ecology and Evolution*, **23**: 402–409.

Diaz, R.J. 2001. Overview of hypoxia around the world. *Journal of Environmental Quality*, **30**: 275–281.

Diaz, R., and R. Rosenberg. 1995. Marine benthic hypoxia: a review of its ecological effects and the behavioral responses of benthic macrofauna. *Oceanography and Marine Biology: An Annual Review*, **33**: 245–303.

Diersing, N. 2009. Phytoplankton blooms in Florida Bay. Florida Keys National Marine Sanctuary, Key Largo.

d'Ippolito, G., N. Lamari, M. Montresor, G. Romano, A. Cutignano, A. Gerecht, G. Cimino, and A. Fontana. 2009. 15*S*-lipoxygenase metabolism in the marine diatom *Pseudo-nitzschia delicatissima*. *New Phytologist*, **183**: 1064–1071.

Dixit, R.B., and M.R. Suseela. 2013. Cyanobacteria: potential candidates for drug discovery. *Antonie van Leeuwenhoek*, **103**: 947–961.

Dobberfuhl, D.R. 2003. *Cylindrospermopsis raciborskii* in three central Florida lakes: population dynamics, controls, and management implications. *Lake and Reservoir Management*, **19**: 341–348.

Dodds, W.K. 2006. Eutrophication and trophic state in rivers and streams. *Limnology and Oceanography*, **51**: 671–680.

Dorantes-Aranda, J.J., L.M. Garcia-de la Parra, R. Alonso-Rodriguez, L. Morquecho, and D. Voltolina. 2010. Toxic effect of the harmful dinoflagellate *Cochlodinium polykrikoides* on the spotted rose snapper *Lutjanus guttatus*. *Environmental Toxicology*, **25**: 319–326.

Dorantes-Aranda, J.J., A. Seger, J.I. Mardones, P.D. Nichols, and G.M. Hallegraeff. 2015. Progress in understanding algal bloom-mediated fish kills: the role of superoxide radicals, phycotoxins and fatty acids. *PLoS ONE*, **10**(7): e0133549. doi: 10.1371/journal.pone.0133549.

Doucette, G.J., A. Cembella, J. Martin, J. Michaud, T. Cole, and R. Rolland. 2006a. Paralytic shellfish poisoning (PSP) toxins in North Atlantic right whales *Eublanaena glacialis* and their zooplankton prey in the Bay of Fundy, Canada. *Marine Ecology Progress Series*, **306**: 303–313.

Doucette, G.J., E.R. McGivern, and J.A. Babinchak. 1999. Algicidal bacteria active against *Gymnodinium breve* (Dinophyceae). I. Bacterial isolation and characterization of killing activity. *Journal of Phycology*, **35**: 1447–1454.

Doucette, G.J., I. Maneiro, I. Riveiro, and C. Svensen. 2006b. Phycotoxin pathways in aquatic food webs: transfer, accumulation, and degradation. In: *Ecology of Harmful Algae*. E. Granéli and J. Turner (Eds.). Springer, New York: p. 283–295.

Doyle, R.D., and R.M. Smart. 1998. Competitive reduction of noxious *Lyngbya wollei* mats by rooted aquatic plants. *Aquatic Botany*, **61**: 17–32.

Duarte, C.M. 1995. Submerged aquatic vegetation in relation to different nutrient regimes. *Ophelia*, **41**: 87–112.

Duffy, J.E., and M.E. Hay. 1990. Seaweed adaptations to herbivory. *BioScience*, **40**: 368–375.

Dunker, S., T. Jakob, and C. Wilhelm. 2013. Contrasting effects of the cyanobacterium

Microcystis aeruginosa on the growth and physiology of two green algae, *Oocystis marsonii* and *Scenedesmus obliquus,* revealed by flow cytometry. *Freshwater Biology*, **58**: 1573–1587.

Dupont, J.M., and C. Coy. 2008. Only the strong will survive: red tides as community-structuring forces in the eastern Gulf of Mexico. In: *Proceedings of the American Academy of Underwater Sciences 27th Symposium*. P. Brueggeman and N.W. Pollock (Eds.). AAUS, Dauphin Island.

Dupont, J.M., P. Hallock, and W.C. Jaap. 2010. Ecological impacts of the 2005 red tide on artificial reef epibenthic macroinvertebrate and fish communities in the eastern Gulf of Mexico. *Marine Ecology Progress Series*, **415**: 189–200.

Dupuy, J.L., and A.K. Sparks. 1967. *Gonyaulax washingtonensis,* its relationship to *Mytilus californianus and Crassostrea gigas* as a source of paralytic shellfish toxin in Sequim Bay, Washington. *Proceedings of the National Shellfisheries Association*, **58**: 2.

Durbin, A.G., and E.G., Durbin. 1989. Effect of the 'brown tide' on feeding, size and egg laying rate of adult female *Acartia tonsa*. In: *Novel Phytoplankton Blooms*. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 625–646.

Echevarria, M., J.P. Naar, C. Tomas, and J.R. Pawlik. 2012. Effects of *Karenia brevis* on clearance rates and bioaccumulation of brevetoxins in benthic suspension feeding invertebrates. *Aquatic Toxicology*, **106–107**: 85–94.

Edmunds, J.S.G., R.A. McCarthy, J.S. Ramsdell. 1999. Ciguatoxin reduces larval survivability in finfish. *Toxicon*, **37**: 1827–1832.

Edvardsen, B., and I. Imai. 2006. The ecology of harmful flagellates within Prymnesiophyceae and Raphidophyceae. In: *Ecology of Harmful Algae*. E. Granéli and J.T. Turner (Eds.). Ecological Studies, Vol. 189. Springer, Berlin: p. 67–79.

Edwards, D.J., B.L. Marquez, L.M. Nogle, K. McPhail, D.E. Goeger, M.A. Roberts, and W.H. Gerwick. 2004. Structure and biosynthesis of the jamaicamides, new mixed polyketide-peptide neurotoxins from the marine cyanobacterium *Lyngbya majuscula*. *Chemical Biology*, **11**: 817–833.

Egloff, D.A. 1986. Effects of *Olisthodiscus luteus* on the feeding and reproduction of the marine rotifer *Synchaeta cecilia*. *Journal of Plankton Research*, **8**: 263–274.

Engelke, C.J., L.A. Lawton, and M. Jaspars. 2003. Elevated microcystin and nodularin levels in cyanobacteria growing in spent medium of *Planktothrix agardhii*. *Archiv für Hydrobiologie*, **158**: 541–550.

Engene, N., E.C. Rottacker, J. Kaštovský, T. Byrum, H. Choi, M.H. Ellisman, J. Komárek, and W.H. Gerwick. 2012. *Moorea producens* gen. nov., sp. nov. and *Moorea bouillonii* comb. nov., tropical marine cyanobacteria rich in bioactive secondary metabolites. *International Journal of Systematic and Evolutionary Microbiology*, **62**: 1171–1178.

Eppley, R.W., and C.R. Bovell. 1958. Sulfuric acid in *Desmarestia*. *Biology Bulletin*, **115**: 101–106.

Eriksson, J.E., J.A.O. Meriluoto, and T. Lindholm. 1989. Accumulation of a peptide toxin from the cyanobacterium *Oscillatoria agardhii* in the freshwater mussel *Anodonta cygnea*. *Hydrobiology*, **183**: 211–216.

Escalera, L., Y. Pazos, Á. Moroño, and B. Reguera. 2007. *Noctiluca scintillans* may act as a vector of toxigenic microalgae. *Harmful Algae*, **6**: 317–320.

Estrella, S.M., A.W. Storey, G. Pearson, and T. Piersma. 2011. Potential effects of *Lyngbya majuscula* blooms on benthic invertebrate diversity and shorebird foraging ecology at Roebuck Bay, Western Australia: preliminary results. *Journal of the Royal Society of Western Australia*, **94**: 171–179.

Ettoumi, A., F. El Khalloufi, I. El Ghazali, B. Oudra, A. Amrani, H. Nasri, and N. Bouaïcha. 2011. Bioaccumulation of cyanobacterial toxins in aquatic organisms and its consequences for public health. In: *Zooplankton and Phytoplankton*. G. Kattel (Ed.). Nova Science Publishers, Hauppauge: p. 1–33.

Evans, J., A. Wilkie, J. Burkhardt, and R. Haynes. 2007. Rethinking exotic plants: using citizen observation in a restoration proposal for Kings Bay, Florida. *Ecological Research*, **25**: 199–210.

Fabrowska, J., B. Łeska, and G. Schroeder. 2015. Freshwater *Cladophora glomerata* as a new potential cosmetic raw material. *Chemik*, **69**: 645–647.

Faimali, M., V. Guissani, V. Piazza, and M. Chiantore. 2011. Toxic effects of harmful benthic dinoflagellate *Ostreopsis ovata* on invertebrate and vertebrate marine organisms. *Marine Environmental Research*, **76**: 97–107.

Fauquier, D.A., L.J. Flewelling, J.M. Maucher, M. Keller, M.J. Kinsel, C.K. Johnson, M. Henry, J.G. Gannon, J.S. Ramsdell, and J.H. Landsberg. 2013a. Brevetoxicosis in seabirds naturally exposed to

Karenia brevis blooms along the central west coast of Florida. *Journal of Wildlife Diseases*, **49**: 246–260.

Fauquier, D.A., L.J. Flewelling, J. Maucher, C.A. Manire, V. Socha, M.J. Kinsel, B.A. Stacy, H.M. Gannon, J.S. Ramsdell, and J.H. Landsberg. 2013b. Brevetoxin in blood, biological fluids, and tissues of sea turtles naturally exposed to *Karenia brevis* blooms in central west Florida. *Journal of Zoo and Wildlife Medicine*, **44**: 364–375.

Ferrão-Filho, A.S., and B. Kozlowsky-Suzuki. 2011. Cyanotoxins: bioaccumulation and effects on aquatic animals. *Marine Drugs*, **9**: 2729–2772.

Ferrão-Filho, A.S., R. Cunha, V.F. Magalhães, M.C.S. Soares, and D.F. Baptista. 2007. Evaluation of sub-lethal toxicity of cyanobacteria on the swimming activity of aquatic organisms by image analysis. *Journal of the Brazilian Society of Ecotoxicology*, **2**: 93–100.

Ferrão-Filho, A. da S., S.M. da Costa, M.G.L. Ribeiro, and S.M.F.O. Azevedo. 2008. Effects of a saxitoxin-producer strain of *Cylindrospermopsis raciborskii* (Cyanobacteria) on the swimming movements of cladocerans. *Environmental Toxicology*, **23**: 161–168.

Ferrão-Filho, A.S., B. Kozlowsky-Suzuki, and S.M.F.O. Azevedo. 2002. Accumulation of microcystins by a tropical zooplankton community. *Aquatic Toxicology*, **59**: 201–208.

Fiamali, M., V. Giussani, V. Piazza, F. Garaventa, C. Corrà, V. Asnaghi, D. Privitera, L. Gallus, R. Cattaneo-Vietti, L. Mangialajo, and M. Chiantore. 2012. Toxic effects of the harmful benthic dinoflagellate *Ostreopsis ovata* on invertebrate and vertebrate marine organisms. *Marine Environmental Research*, **76**: 97–107.

Fíedler, P.C. 1982. Zooplankton avoidance and reduced grazing responses to *Gymnodinium splendens* (Dinophyceae). *Limnology and Oceanography*, **27**: 961–965.

Figueredo, C.C., A. Giani, and D.F. Bird. 2007. Does allelopathy contribute to *Cylindrospermopsis raciborskii* (cyanobacteria) bloom occurrence and geographic expansion? *Journal of Phycology*, **43**: 256–265.

Fire, S.E., L.J. Flewelling, J. Naar, M.J. Twiner, M.S. Henry, R.H. Pierce, D.P. Gannon, Z. Wangm, L. Davidson, and R.S. Wells. 2008. Prevalence of brevetoxins in prey fish of bottlenose dolphins in Sarasota Bay. Florida. *Marine Ecology Progress Series*, **368**: 283–294.

Fire, S.E., J. Pruden, D. Couture, Z. Wang, M.-Y. Dechraoui Botten, B.L. Haynes, T. Knott, D. Bouchard, A. Lichtenwalner, and G. Wippelhauser. 2012. Saxitoxin exposure in an endangered fish: association of a shortnose sturgeon mortality event with a harmful algal bloom. *Marine Ecology Progress Series*, **460**: 145–153.

Fire, S.E., Z.H. Wang, M. Berman, G.W. Langlois, S.L. Morton, E. Sekula-Wood, and C.R. Benitez-Nelson. 2010. Trophic transfer of the harmful algal toxin domoic acid as a cause of death in a Minke whale (*Balaenoptera acutorostrata*) stranding in Southern California. *Aquatic Mammals*, **36**: 342–350.

Fistarol, G.O., C. Legrand, and E. Granéli. 2003. Allelopathic effect of *Prymnesium parvum* on a natural planktonic community. *Marine Ecology Progress Series*, **255**: 115–125.

Fistarol, G.O., C. Legrand, E. Selander, C. Hummert, W. Stolte, and E. Granéli. 2004. Allelopathy in *Alexandrium* spp.: effect on a natural plankton community and on algal monocultures. *Aquatic Microbial Ecology*, **35**: 45–56.

Fitch, C.P. L.M. Bishop, and W.L. Boyd. 1934. "Waterbloom" as a cause of poisoning in domestic animals. *The Cornell Veterinarian*, **24**: 30–39.

Flaherty, K.E., and J.H. Landsberg. 2010. Effects of a persistent red tide (*Karenia brevis*) bloom on community structure and species-specific relative abundance of nekton in a Gulf of Mexico estuary. *Estuaries Coasts*, **34**: 417–439.

Flewelling, L.J. 2008. Vectors of brevetoxins to marine mammals. PhD dissertation, University of South Florida.

Flewelling, L.J., J.P. Naar, J.P. Abbott, D.G. Baden, N.B. Barros, G.D. Bossart, M.Y.D. Bottein, D.G. Hammond, E.M. Haubold, C.A. Heil, M.S. Henry, H.M. Jacocks, T.A. Leighfield, R.H. Pierce, T.D. Pitchford, S.A. Rommel, P.S. Scott, K.A. Steidinger, E.W. Truby, F.M. Van Dolah, and J.H. Landsberg. 2005. Red tides and marine mammal mortalities. *Nature*, **435**: 755–756.

Foote, J.J., N.L. Park, and J.M. Sprinke. 1998. An increase in marine turtle deaths along the west central coast of Florida (1995–1996): is red tide the culprit? In: *Proceedings of the Seventeenth Annual Sea Turtle Symposium*. S.P. Epperly and J. Braun (Comps.). NOAA Technical Memorandum. NMFS-SEFSC-415. National Marine Fisheries Service, Miami: p. 180–182.

Foss, A.J., E.J. Philips, M. Yilmaz, and A. Chapman. 2012. Characterization of paralytic shellfish toxins from *Lyngbya wollei* dominated mats collected from two Florida springs. *Harmful Algae*, **16**: 98–107.

Frangópulos, M., C. Guisande, I. Maneiro, I. Riveiro, and J. Franco. 2000. Short-term and long-term

effects of the toxic dinoflagellate *Alexandrium minutum* on the copepod *Acartia clausi*. *Marine Ecology Progress Series*, **203**: 161–169.

Frangópulos, M., E. Spyrakos, and C. Guisande. 2011. Ingestion and clearance rates of the red *Noctiluca scintillans* fed on the toxic dinoflagellate *Alexandrium minutum* (Halim). *Harmful Algae*, **10**: 304–309.

Franz, D.R., and R.D. LeClaire. 1989. Respiratory effects of brevetoxin and saxitoxin in awake guinea pigs. *Toxicon*, **27**: 647–654.

Freitas, E.C., C. Pinheiro, O. Rocha, and S. Loureiro. 2014. Can mixtures of cyanotoxins represent a risk to the zooplankton? The case study of *Daphnia magna* Straus exposed to hepatotoxic and neurotoxic cyanobacterial extracts. *Harmful Algae*, **31**: 143–152.

Fristachi, A., and J.L. Sinclair. 2008. Occurrence of cyanobacterial harmful algal blooms workgroup report. In: *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs.* K.H. Hudnell (Ed.). Advances in Experimental Medicine and Biology, Vol. 619. Springer, New York: p. 45–103.

Fritz, L., M.A. Quilham, J.L.C. Wright, and T.M. Work. 1992. An outbreak of domoic acid poisoning attributed to the pennate diatom *Pseudo-nitzschia australis*. *Journal of Phycology*, **28**: 439–442.

Fulco, K. 2007. Harmful effects of the toxic dinoflagellate *Alexandrium tamarense* on the tintinnids *Favella taraikaensis* and *Eutintinnus* sp. *Journal of the Marine Biological Association UK*, **87**: 1085–1088.

Fulton, R.S. III. 1988a. Grazing on filamentous algae by herbivorous zooplankton. *Freshwater Biology*, **20**: 263–271.

Fulton, R.S. III. 1988b. Resistance to blue-green algal toxins by *Bosmina longirostris*. *Journal of Plankton Research*, **10**: 771–778.

Fulton, R.S. III and H.W. Paerl. 1987. Toxic and inhibitory effects of the blue-green alga *Microcystis aeruginosa* on herbivorous zooplankton. *Journal of Plankton Research*, **9**: 837–855.

Fulton, R.S. III and H.W. Paerl. 1988. Effects of the blue-green alga *Microcystis aeruginosa* on zooplankton competitive relations. *Oecologia*, **76**: 383–389.

Gaete, V., E. Canelo, N. Lagos, and F. Zambrano. 1994. Inhibitory effects of *Microcystis aeruginosa* toxin on ion pumps of the gill of freshwater fish. *Toxicon*, **32**: 121–127.

Gainey, L.F., and S.E. Shumway. 1988. Physiological effects of *Protogonyaulax tamarensis* on cardiac activity in bivalve molluscs. *Comparative Biochemistry and Physiology*, **91C**: 159–164.

Gainey, L.F., and S.E. Shumway. 1991. The physiological effect of *Aureococcus anophagefferens* ("brown tide") on the lateral cilia of bivalve mollusks. *Biology Bulletin*, **181**: 298–306.

Galimany, E., A.R. Place, M. Ramón, M. Jutson, and R.K. Pipe. 2008a. The effects of feeding *Karlodinium veneficum* (PLY #103; *Gymnodinium veneficum* Ballantine) to the blue mussel *Mytilus edulis*. *Harmful Algae*, **7**: 91–98.

Galimany, E., I. Sunila, H. Hégaret, M. Ramón, and G.H. Wikfors. 2008b. Pathology and immune response of the blue mussel (*Mytilus edulis* L.) after an exposure to the harmful dinoflagellate *Prorocentrum minimum*. *Harmful Algae*, **7**: 630–638.

Gallager, S.M., D.K. Stoecker, and V.M. Bricelj. 1989. Effects of the brown tide alga on growth, feeding physiology and locomotory behavior of scallop larvae (*Argopecten irradians*). In: *Novel Phytoplankton Blooms*. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 511–541.

Gannon, D.P., E.J.B. McCabe, S.A. Camilleri, J.G. Gannon, M.K. Brueggen, A.A. Barleycorn, V.I. Paluboki, G.J. Kirkpatrick, and R.S. Wells. 2009. Effects of *Karenia brevis* harmful algal blooms on nearshore fish communities in southwest Florida. *Marine Ecology Progress Series*, **378**: 171–186.

Gao, L., L. Zhang, J. Hou, Q. Wei, F. Fu, and H. Shao. 2013. Decomposition of macroalgal blooms influences phosphorus release from the sediments and implications for coastal restoration in Swan Lake, Shandong, China. *Ecological Engineering*, **60**: 19–28.

Garcia-Hernández, J., L. Garcia-Rico, M.E. Jara-Marini, R. Barraza-Guardado, and A. Hudson Weaver. 2005. Concentration of heavy metals in sediment and organisms during a harmful algal bloom (HAB) at Kuk Kaak Bay, Sonora, Mexico. *Marine Pollution Bulletin*, **50**: 733–739.

Gates, J.A., and W.B. Wilson. 1960. The toxicity of *Gonyaulax monilata* Howell to *Mugil cephalus*. *Limnology and Oceanography*, **5**: 171–174.

Gauthier, M.J., P. Bernard, and M. Aubert. 1978. Modification de la fonction antibiotique de deux Diatomées marines, *Asterionella japonica* (Cleve) *et Chaetoceros lauderi* (Raifs), par le dinoflagellé *Prorocentrum micans* (Ehrenberg). *Journal of Experimental Marine Biology and Ecology* **33**: 37–50.

Gaydos, J.K. 2012. Deadly diatoms: the latest on harmful algal blooms. Proceedings of the 2012 North American Veterinary Conference, Orlando.

Gélinas, M., A. Lajeunesse, C. Gagnon, and F. Gagné. 2013. Temporal and seasonal variation in acetylcholinesterase activity and glutathione-S-transferase in amphipods collected in mats of *Lyngbya wollei* in the St. Lawrence River (Canada). *Ecotoxicology and Environmental Safety*, **94**: 54–59.

Genkai-Kato, M. 2011. Regime shifts in response to human impacts and management in ecosystems. *Kuroshio Sciences*, **5**: 33–37.

Genkai-Kato, M., Y. Vadeboncoeur, L. Liboriussen, and E. Jeppesen. 2012. Benthic-planktonic coupling, regime shifts, and whole-lake primary production in shallow lakes. *Ecology*, **93**: 619–631.

Geraci, J.A., D.M. Anderson, R.J. Timperi, D.J. St Aubin, G.A. Early, J.A. Prescott, and C.A. Mayo. 1989. Humpback whales (*Megaptera novaeangliae*) fatally poisoned by dinoflagellate toxin. *Canadian Journal of Fisheries and Aquatic Sciences*, **46**: 1895–1898.

Gibble, C.M., and B.A. Hoover. 2018. Interactions between seabirds and harmful algal blooms. In: *Harmful Algal Blooms: A Compendium Desk Reference.* S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 223–242.

Gilbert, J.J. 1994. Susceptibility of planktonic rotifers to a toxic strain of *Anabaena flos-aquae*. *Limnology and Oceanography*, **39**: 1286–1297.

Gilby, B.L., D.D. Burfeind, and I.R. Tibbetts. 2011. *Lyngbya majuscula* blooms and the diet of small subtropical benthivorous fishes. *Marine Biology*, **158**: 245–255.

Gill, C.W., and R.P. Harris. 1987. Behavioural responses of the copepods *Calanus helgolandicus* and *Temora longicornis* to dinoflagellate diets. *Journal of the Marine Biological Association UK*, **67**: 785–801.

Glibert, P.M., J. Alexander, D.W. Meritt, E.W. North, and D.K. Stoecker. 2007. Harmful algae pose additional challenges for oyster restoration: impacts of the harmful algae *Karlodinium veneficum* and *Prorocentrum minimum* on early life stages of the oysters *Crassostrea virginica* and *Crassostrea ariakensis*. *Journal of Shellfish Research*, **26**: 919–925.

Glibert, P.M., and J.M. Burkholder. 2018a. Causes of harmful algal blooms. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 1–38.

Glibert, P.M., and J.M. Burkholder. 2018b. *Prorocentrum* Fact Sheet. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 625–628.

Glibert, P.M., J.M. Burkholder, and T.M. Kana. 2012. Recent insights about relationships between nutrient availability, forms, and stoichiometry, and the distribution, ecophysiology, and food web effects of pelagic and benthic *Prorocentrum* species. *Harmful Algae*, **14**: 231–259.

Glibert, P.M., J.M. Burkholder, T.M. Kana, J. Alexander, H. Skelton, and C. Shillings. 2009. Grazing by *Karenia brevis* on *Synechococcus* enhances its growth rate and may help to sustain blooms. *Aquatic Microbial Ecology*, **55**: 17–30.

Glibert, P.M., D. Fullerton, J.M. Burkholder, J.C. Cornwell, and T.M. Kana. 2011. Ecological stoichiometry, biogeochemical cycling, invasive species and aquatic food webs: San Francisco Estuary and comparative systems. *Reviews in Fisheries Science*, **19**: 358–417.

Glibert, P.M., C.A. Heil, D. Hollander, M. Revilla, A. Hoare, J. Alexander, and S. Murasko. 2004. Evidence for dissolved organic nitrogen and phosphorus uptake during a cyanobacterial bloom in Florida Bay. *Marine Ecology Progress Series*, **280**: 73–83.

Gobler, C.J., D.L. Berry, O.R. Anderson, A. Burson, F. Koch, B.S. Rodgers, L.K. Moore, J.A. Goleski, B. Allam, P. Bowser, Y. Tang, and R. Nuzzi. 2008. Characterization, dynamics, and ecological impacts of harmful *Cochlodinium polykrikoides* blooms on eastern Long Island, NY, USA. *Harmful Algae*, **7**: 293–307.

Gobler, C.J., J.M. Burkholder, T.W. Davis, M.J. Harke, T. Johengen, C. Stow, and D.B. Van de Waal. 2016. The dual role of nitrogen supply in controlling the growth *and* toxicity of cyanobacterial blooms. *Harmful Algae*, **54**: 87–97.

Gobler, C.J., S. Deonarine, J. Leigh-Bell, M.D. Gastrich, O.R. Anderson, & S.W. Wilhelm. 2004. Ecology of phytoplankton communities dominated by *Aureococcus anophagefferens*: the role of viruses, nutrients, and microzooplankton grazing. *Harmful Algae*, **3**: 471–483.

Gobler, C.J., F. Koch, Y. Kang, D.L. Berry, Y.Z. Tang, M. Lasi, L. Walters, L. Hall, and J.D. Miller. 2013. Expansion of harmful brown tides caused by the pelagophyte, *Aureoumbra lagunensis* DeYoe et Stockwell, to the U.S. east coast. *Harmful Algae*, **27**: 29–41.

Gobler, C.J., D.J. Lonsdale, and G.L. Boyer. 2005. A review of the causes, effects, and potential management of harmful brown tide blooms caused by *Aureococcus anophagefferens* (Hargraves et Sieburth). *Estuaries*, **28**: 726–749.

Gobler, C.J., M.J. Renaghan, N.J. Buck. 2002. Impacts of nutrients and grazing mortality on the abundance of *Aureococcus anophagefferens* during a New York brown tide bloom. *Limnology and Oceanography*, **47**: 129–141.

Gobler, C.J., and W.G. Sunda. 2012. Ecosystem disruptive algal blooms of the brown tide species, *Aureococcus anophagefferens* and *Aureoumbra lagunensis*. *Harmful Algae*, **14**: 36–45.

Goldberg, J.D. 2003. Domoic acid in the benthic food web of Monterey Bay, California. MSc thesis, California State University Monterey Bay.

Goldstein, T., J.A.K. Mazet, T.S. Zabka, G. Langlois, K.M. Colegrove, M. Silver, S. Bargu, F. Van Dolah, T. Leighfield, P.A. Conrad, J. Barakos, D.C. Williams, S. Dennison, M. Haulena, and F.M.D. Gulland. 2008. Novel symptomatology and changing epidemiology of domoic acid toxicosis in California sea lions (*Zalophus californianus*): an increasing risk to marine mammal health. *Proceedings of the Royal Society B*, **275**: 267–276.

Goldstein, T., T.S. Zabka, R.L. DeLong, E.A. Wheeler, G. Ylitalo, S. Bargu, M. Silver, T. Leighfield, F. Van Dolah, G. Langlois, I. Sidor, J.L. Dunn, and F.M.D. Gulland. 2009. The role of domoic acid in abortion and premature parturition of California sea lions (*Zalophus californianus*) on San Miguel Island, California. *Journal of Wildlife Diseases*, **45**: 91–108.

Gómez, F., M.L. Richlen, and D.M. Anderson. 2017. Molecular characterization and morphology of *Cochlodinium strangulatum*, the type species of *Cochlodinium*, and *Margalefidinium* gen. nov. for *C. polykrikoides* and allied species (Gymnodiniales, Dinophyceae). *Harmful Algae*, **63**: 32–44.

Goodlet, R.O., F.M.V. Dolah, and J.A. Babinchak. 1994. Re-occurrence of a ciguatera outbreak in a coral reef microcosm at the Pittsburg Zoo. In: *Proceedings of the International Symposium on Ciguatoxin and Marine Natural Products*, by Y. Hokama, P.J. Scheuer, and T. Yasumoto (Eds.). Asian Pacific Research Foundation, S. Kohala.

Gordon, A.S., B.J. Dyer, D. Seaborn, and H.G. Marshall. 2002. Comparative toxicity of *Pfiesteria* spp., prolonging toxicity of *P. piscicida* in culture and evaluation of toxin(s) stability. *Harmful Algae*, **1**: 85–94.

Graham, J.L., K.A. Loftin, M.T. Meyer, and A.C. Ziegler. 2010. Cyanotoxin mixtures and taste-and-odor compounds in cyanobacterial blooms from the midwestern United States. *Environmental Science & Technology*, **44**: 7361–7368.

Graham, L.E., J.M. Graham, L.W. Wilcox, and M.E. Cook. 2016. *Algae*, 3rd ed. LJLM Press, LLC.

Graham, S.L., and S.L. Strom. 2010. Growth and grazing of microzooplankton in response to the harmful alga *Heterosigma akashiwo* in prey mixtures. *Aquatic Microbial Ecology*, **59**: 111–124.

Granéli, E., B. Edvardsen, D.L. Roelke, and J.A. Hagström. 2012. The ecophysiology and bloom dynamics of *Prymnesium* spp. *Harmful Algae*, **14**: 260–270.

Granéli, E., and N. Johansson. 2003a. Effect of the toxic haptophyte *Prymnesium parvum* on the survival and feeding of a ciliate: the influence of different nutrient conditions. *Marine Ecology Progress Series*, **254**: 49–56.

Granéli, E., M. Weberg, and P.S. Salomon. 2008. Harmful algal blooms of allelopathic microalgal species: the role of eutrophication. *Harmful Algae*, **8**: 94–102.

Gray, J.S. 2002. Biomagnification in marine systems: the perspective of an ecologist. *Marine Pollution Bulletin*, **45**: 46–52.

Gray, J.S., R.S. Wu, and Y.Y. Or. 2002. Effects of hypoxia and organic enrichment on the coastal marine environment. *Marine Ecology Progress Series*, **238**: 249–279.

Green, L., D.T. Blumstein, and P. Fong. 2013. Macroalgal mats in a eutrophic estuary obscure visual foraging cues and increase variability in prey availability for some shorebirds. *Estuaries and Coasts*, **38**: 917–926.

Green, R.L. 2011. Macroalgal mats control trophic structure and shorebird foraging behavior in a southern California estuary. Dissertation, University of California, Los Angeles.

Greenfield, D.I., and D.J. Lonsdale. 2002. Mortality and growth of juvenile hard clams *Mercenaria mercenaria* during brown tide. *Marine Biology*, **141**: 1045–1050.

Greenfield, D.I., D.J. Lonsdale, R.M. Cerrato, and G.R. Lopez. 2004. Effects of background concentrations of *Aureococcus anophagefferens* (brown tide) on growth and feeding in the bivalve *Mercenaria mercenaria*. *Marine Ecology Progress Series*, **274**: 171–181.

Grzebyk, D., A. Denardou, B. Berland, and Y.F. Pouchus. 1997. Evidence of a new toxin in the red-tide dinoflagellate *Prorocentrum*

minimum. *Journal of Plankton Research*, **19**: 1111–1124.

Günter, G., R.H. Williams, C.C. Davis, and F.G. Walton Smith. 1948. Catastrophic mass mortality of marine animals and coincident phytoplankton bloom on the west coast of Florida, November 1946–May 1947. *Ecological Monographs*, **18**: 309–324.

Guo, C., and P.A. Tester. 1994. Toxic effect of the bloom-forming *Trichodesmium* sp. (Cyanophyta) to the copepod *Acartia tonsa*. *Natural Toxins*, **2**: 222–227.

Gutiérrez-Praena, D., A. Jos, S. Pichardo, R. Moyano, A. Blanco, J.G. Monterde, and A.M. Cameán. 2012. Time-dependent histopathological changes induced in tilapia (*Oreochromis niloticus*) after acute exposure to pure cylindrospermopsin by oral and intraperitoneal route. *Ecotoxicology and Environmental Safety*, **76**: 102–113.

Haglund, P., A. Malmvärn, S. Bergek, A. Bignert, L. Kautsky, T. Nakano, K. Wiberg, and L. Asplund. 2007. Brominated dibenzo-*p*-dioxins: a new class of marine toxins? *Environmental Science & Technology*, **41**: 3069–3074.

Hall, M.O., M.J. Durako, J.W. Fourqurean, and J.C. Zieman. 1999. Decadal changes in seagrass distribution and abundance in Florida Bay. *Estuaries*, **22**: 445–459.

Hallegraeff, G.M. 2010. Ocean climate change, phytoplankton community responses, and harmful algal blooms: a formidable predictive challenge. *Journal of Phycology*, **46**: 220–235.

Hammer, U.T. 1968. Toxic blue-green algae in Saskatchewan. *Canadian Veterinary Journal*, **9**: 221–229.

Handy, S.M., E. Demir, D.A. Hutchins, K.J. Portune, E.B. Whereat, C.E. Hare, J.M. Rose, M. Warner, M. Farestad, S.C. Cary, and K.J. Coyne. 2008. Using quantitative real-time PCR to study competition and community dynamics among Delaware Inland Bays harmful algae in field and laboratory studies. *Harmful Algae*, **7**: 599–613.

Haney, J.E., J.J. Sasner, and M. Ikawa. 1995. Effects of products released by *Aphanizomenon flos-aquae* and purified saxitoxin on the movements of *Daphnia carinata* feeding appendages. *Limnology and Oceanography*, **40**: 263–272.

Hansen, P.J. 1989. The red tide dinoflagellate *Alexandrium tamarense*: effects on behaviour and growth of a tintinnid ciliate. *Marine Ecology Progress Series*, **53**: 105–116.

Harding, J.M., R. Mann, P. Moeller, and M.S. Hsia. 2009. Mortality of the veined rapa whelk, *Rapana venosa,* in relation to a bloom of *Alexandrium monilatum* in the York River, United States. *Journal of Shellfish Research*, **28**: 363–367.

Hastings, A. 2013. Multiple stable states and regime shifts in ecological systems. *Mathematics Today*, February: 37–39.

Hattori, S. 1962. Predatory activity of *Noctiluca* on anchovy eggs. *Bulletin of the Tokai Regional Fisheries Research Laboratory*, **9**: 211–220.

Hauxwell, J., J. Cebrián, C. Furlong, and I. Valiela. 2001. Macroalgal canopies contribute to eelgrass (*Zostera marina*) decline in temperate estuarine ecosystems. *Ecology*, **82**: 1007–1022.

Havens, K.E. 2008. Cyanobacteria blooms: effects on aquatic ecosystems. In: *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. H.K. Hudnell (Ed.). Advances in Experimental Medicine and Biology, Vol. 619. Springer, New York: p. 733–747.

Havens, K.E., J. Hauxwell, A.C. Tyler, S. Thomas, K.J. McGlathery, J. Cebrian, I. Valiela, A.D. Steinman, and S.-J. Hwang. 2001. Complex interactions between autotrophs in shallow marine and freshwater ecosystems: implications for community responses to nutrient stress. *Environmental Pollution*, **113**: 95–107.

Hawkins, P.R., N.R. Chandrasena, G.J. Jones, A.R. Humpage, and I.A. Falconer. 1997. Isolation and toxicity of *Cylindrospermopsis raciborskii* from an ornamental lake. *Toxicon*, **35**: 314–346.

Hawkins, P.R., and W. Lampert. 1989. The effect of *Daphnia* body size on filtering rate inhibition in the presence of a filamentous cyanobacterium. *Limnology and Oceanography*, **34**: 1084–1089.

Hawser, S.P., J.M. O'Neill, M.R. Roman, and G.A. Codd. 1992. Toxicity of blooms of the cyanobacterium *Trichodesmium* to zooplankton. *Journal of Applied Phycology*, **4**: 79–86.

Hay, M.E. 2009. Marine chemical ecology: chemical signals and cues structure marine populations, communities, and ecosystems. *Annual Review of Marine Science*, **1**: 193–212.

Hay, M.E., and W. Fenical. 1988. Marine plant-herbivore interactions: the ecology of chemical defense. *Annual Review of Ecology and Systematics*, **19**: 111–145.

Hayden, H.S., J. Blomster, C.A. Maggs, P.C. Silva, M.J. Stanhope, and J.R. Waaland. 2003. Linnaeus was right all along: *Ulva* and *Enteromorpha* are not distinct genera. *European Journal of Phycology*, **38**: 277–294.

Hégaret, H., and G.H. Wikfors. 2005a. Time-dependent changes in haemocytes of eastern

oysters, *Crassostrea virginica,* and northern bay scallops, *Argopecten irradians irradians,* exposed to a cultured strain of *Prorocentrum minimum*. *Harmful Algae*, **4**: 187–199.

Hégaret, H., and G.H. Wikfors. 2005b. Effects of natural and field-simulated blooms of the dinoflagellate *Prorocentrum minimum* upon hemocytes of eastern oysters, *Crassostrea virginica,* from two different populations. *Harmful Algae*, **4**: 201–209.

Hégaret, H., G.H. Wikfors, and S.E. Shumway. 2007b. Diverse feeding responses of five species of bivalve mollusc when exposed to three species of harmful algae. *Journal of Shellfish Research*, **26**: 549–559.

Hégaret, H., G.H. Wikfors, P. Soudant, C. Lambert, S.E. Shumway, J.B. Bérard, and P. Lassus. 2007a. Toxic dinoflagellates (*Alexandrium fundyense* and *A. catenella*) have minimal apparent effect on oyster hemocytes. *Marine Biology*, **152**: 441–447.

Heisler, J., P. Glibert, J. Burkholder, D. Anderson, W. Cochlan, W. Dennison, C. Gobler, Q. Dortch, C. Heil, E. Humphries, A. Lewitus, R. Magnien, H. Marshall, D. Stockwell, and M. Suddleson. 2008. Eutrophication and harmful algal blooms: a scientific consensus. *Harmful Algae*, **8**: 3–13.

Henrikson, J.C., M.S. Gharfeh, A.C. Easton, J.D. Easton, K.L. Glenn, M. Shadfan, S.L. Mooberry, K.D. Hambright, and R.H. Cichewicz. 2010. Reassessing the ichthyotoxin profile of cultured *Prymnesium parvum* (golden algae) and comparing it to samples collected from recent freshwater bloom and fish kill events in North America. *Toxicon*, **55**: 1396–1404.

Hershner, C. 2011. The certainty of confronting uncertainty in the Chesapeake Bay restoration effort. *Choices*, 3rd Quarter. Available at: http://www.choicesmagazine.org/choices-magazine/theme-articles/innovating-policy-for-chesapeake-bay-restoration/the-certainty-of-confronting-uncertainty-in-the-chesapeake-bay-restoration-effort.

Heil, C.A., P.M. Glibert, and C. Fan. 2005. *Prorocentrum minimum* (Pavillard) Schiller: a review of a harmful algal bloom species of growing worldwide importance. *Harmful Algae*, **4**: 449–470.

Hietala, J., C. Laurón-Maata, and M. Walls. 1997. Life history responses of *Daphnia* clones to toxic *Microcystis* at different food levels. *Journal of Plankton Research*, **19**: 917–926.

Hietala, J., M. Reinkainen, and M. Walls. 1995. Variation in life history responses of *Daphnia* to toxic *Microcystis aeruginosa*. *Journal of Plankton Research*, **17**: 2307–2318.

Higgins, S.N., S.Y. Malkin, E.T. Howell, S.J. Guildford, L. Campbell, V. Hiriart-Baer, and R.E. Hecky. 2008. An ecological review of *Cladophora glomerata* (Chlorophyta) in the Laurentian Great Lakes. *Journal of Phycology*, **44**: 1–16.

Hinton, M., and J.S. Ramsdell. 2008. Brevetoxin in two planktivorous fishes after exposure to *Karenia brevis*: implications for food-web transfer to bottlenose dolphins. *Marine Ecology Progress Series*, **356**: 251–258.

Ho, M.-S., and P.L. Zubkoff. 1979. The effects of a *Cochlodinium heterolobatum* bloom on the survival and calcium uptake by larvae of the American oyster, *Crassostrea virginica*. In: *Toxic Dinoflagellate Blooms*. D.L. Taylor and H.H. Seliger (Eds.). Elsevier, New York: p. 409–412.

Holtcamp, W. 2012. The emerging science of BMAA. *Environmental Health Perspectives*, **120**: A110–A116.

Holmer, M., and R.M. Nielsen. 2007. Effects of filamentous algal mats on sulfide invasion in eelgrass (*Zostera marina*). *Journal of Experimental Marine Biology and Ecology*, **353**: 245–252.

Horner, R.A., D.L. Garrison, and F.G. Plumley. 1997. Harmful algal blooms and red tide problems on the U.S. west coast. *Limnology and Oceanography*, **42**: 1076–1088.

Howell, J.P. 1953. *Gonyaulax monilata,* sp. nov., the causative dinoflagellate of a red tide on the east coast of Florida in August–September, 1951. *Transactions of the American Microscopical Society*, **72**: 153–156.

Hsia, M.H., S.L. Morton, L.L. Smith, K.R. Beauchesne, K.M. Huncik, and P.D.R. Moeller. 2005. Production of goniodomin A by the planktonic, chain-forming dinoflagellate *Alexandrium monilatum* (Howell) Balech isolated from the Gulf Coast of the United States. *Harmful Algae*, **5**: 290–299.

Hudon, C., A. Cattaneo, A.-M. Tourville Poirier, P. Brodeur, P. Dumont, Y. Mailhot, J.-P. Amyot, S.-P. Despatie, and Y. de Lafontaine. 2012. Oligotrophication from wetland epuration alters the riverine trophic network and carrying capacity for fish. *Aquatic Sciences*, **74**: 495–511.

Hudon, C., M. De Sève, and A. Cattaneo. 2014. Increasing occurrence of the benthic filamentous cyanobacterium *Lyngbya wollei*: a symptom of freshwater ecosystem degradation. *Freshwater Science*, **33**: 606–618.

Huntley, M.E., P. Ciminello, and M.D.G. Lopez. 1987. Importance of food quality in determining development and survival of *Calanus pacificus* (Copepods: Calanoida). *Marine Biology*, **95**: 103–113.

Huntley, M.E., P. Sykes, P., S. Rohan, and V. Marin. 1986. Chemically-mediated rejection of dinoflagellate prey by copepods *Calanus pacificus* and *Paracalanus parvus*: mechanism, occurrence and significance. *Marine Ecology Progress Series*, **28**: 105–120.

Ianora, A., M.G. Bentley, G.S. Caldwell, R. Casotti, A.D. Cembella, J. Engström-Öst, C. Halsband, E. Sonnenschein, C. Legrand, C.A. Llewellyn, A. Paldavičienë, R. Pilkaityte, G. Pohnert, A. Razinkovas, G. Romano, U. Tillmann, and D. Vaiciute. 2011a. The relevance of marine chemical ecology to plankton and ecosystem function: an emerging field. *Marine Drugs*, **9**: 1625–1648.

Ianora, A., A. Miralto, S.A. Poulet, Y. Carotenuto, I. Buttino, G. Romano, R. Casotti, G. Pohnert, T. Wichard, L. Colucci-D'Amato, G. Terrazzano, and V. Smetacek. 2004. Aldehyde suppression of copepod recruitment in blooms of a ubiquitous planktonic diatom. *Nature*, **429**: 403–407.

Ianora, A., G. Romano, Y. Carotenuto, F. Esposito, V. Roncalli, I. Buttino, and A. Miralto. 2011b. Impact of the diatom oxylipin 15S-HEPE on the reproductive success of the copepod *Temora stylifera*. *Hydrobiologia*, **666**: 265–275.

Ibelings, B.W., K. Bruning, J. De Jonge, K. Wolfstein, L.M.D. Pires, J. Postma, and T. Burger. 2005. Distribution of microcystins in a lake food web: no evidence for biomagnification. *Microbial Ecology*, **49**: 487–500.

Ibelings, B.W., and K.E. Havens. 2008. Cyanobacterial toxins: a qualitative meta-analysis of concentrations, dosage and effects in freshwater, estuarine and marine biota. In: *Cyanobacterial Harmful Algal Blooms: State of the Science and Research Needs*. K. Hudnell (Ed.). Springer, New York: p. 675–732.

Ieno, E.N., M. Solan, P. Batty, and G.J. Pierce. 2006. How biodiversity affects ecosystem functioning: roles of infaunal species richness, identity and density in the marine benthos. *Marine Ecology Progress Series*, **311**: 263–271.

International Council for the Exploration of the Sea (ICES). 1999. Report of the ICES/IOC Working Group on Harmful Algal Bloom Dynamics (WGHABD), Jena, Germany, March 16–20, 1999. C.M. 1999/C: 4: 1–89. ICES, Copenhagen.

Imai, I., and S. Kimura. 2008. Resistance of the fish-killing dinoflagellate *Cochlodinium polykrikoides* against algicidal bacteria isolated from the coastal Sea of Japan. *Harmful Algae*, **7**: 360–367.

Indeck, K.L., P. Simard, S. Gowans, S. Lowerre-Barbieri, and D.A. Mann. 2015. A severe red tide (Tampa Bay, 2005) causes an anomalous decrease in biological sound. *Royal Society Open Science*, **2**: 150337. doi: 10.1098/rsos.150337.

Inderjit and S.O. Duke. 2003. Ecophysiological aspects of allelopathy. *Planta*, **217**: 529–539.

Ishida, K., and M. Murakami. 2000. Kasumigamide, an antialgal peptide from the cyanobacterium *Microcystis aeruginosa*. *Journal of Organic Chemistry*, **65**: 5898–5900.

Issam, E.G., I., S. Saqrane, A.P. Carvalho, Y. Ouahid, F.F. Del Campo, B. Oudra, and V. Vasconcelos. 2010. Effect of different microcystin profiles on toxin bioaccumulation in common carp (*Cyprinus carpio*) larvae via *Artemia* nauplii. *Ecotoxicology and Environmental Safety*, **73**: 762–770.

Iverson, F., J. Truelove, L. Tryphonas, and E. Nera. 1990. The toxicology of domoic acid administered systemically to rodents and primates. *Canada Diseases Weekly Report*, **16S1E**: 15–19.

Ives, J.D. 1985. The relationship between *Gonyaulax tamarensis* cell toxin levels and copepod ingestion rates. In: *Toxic Dinoflagellates*. D.M. Anderson, A.W. White, and D.G. Baden (Eds.). Elsevier, New York: p. 413–418.

Ives, J.D. 1987. Possible mechanisms underlying copepod grazing responses to levels of toxicity in red tide dinoflagellates. *Journal of Experimental Marine Biology and Ecology*, **112**: 131–145.

Izagirre, O., M. Bermejo, J. Pozo, and A. Elosegi. 2007. RIVERMET (c): an Excel-based tool to calculate river metabolism from diel oxygen-concentration curves. *Environmental Modelling & Software*, **22**: 24–32.

Jacquet, C., V. Thermes, A. de Luze, S. Puiseux-Dao, C. Bernard, J.S. Joly, F. Bourrat, and M. Edery. 2004. Effects of microcystin-LR on development of medaka fish embryos (*Oryzias latipes*). *Toxicon*, **43**: 141–147.

Jang, M.H., K. Ha, M.C. Lucas, G.J. Joo, and N. Takamura. 2004. Changes in microcystin production by *Microcystis aeruginosa* exposed to phytoplanktivorous and omnivorous fish. *Aquatic Toxicology*, **68**: 51–59.

Jang, M.H., K. Ha, and N. Takamura. 2007. Reciprocal allelopathic responses between toxic cyanobacteria (*Microcystis aeruginosa*) and duckweed (*Lemna japonica*). *Toxicon*, **49**: 727–733.

Jeong, H.J., J.Y. Park, J.H. Nho, M.O. Park, J.H. Ha, K.A. Seong, C. Jeng, C.N. Seong, K.Y. Lee, and W.H. Yih. 2005b. Feeding by red-tide dinoflagellates on the cyanobacterium *Synechococcus*. *Aquatic Microbial Ecology*, **41**: 131–143.

Jeong, H.J., J.Y. Song, C.H. Lee, and S.T. Kim. 2004b. Feeding by larvae of the mussel *Mytilus galloprovincialis* on red-tide dinoflagellates. *Journal of Shellfish Research*, **23**: 185–195.

Jeong, H.J., Y.D. Yoo, J.S. Kim, and T.H.T.H. Kim. 2010. Growth, feeding and ecological roles of the mixotrophic and heterotrophic dinoflagellates in marine planktonic food webs. *Ocean Science Journal*, **45**: 65–91.

Jeong, H.J., Y.D. Yoo, J.S. Kim, T.H. Kim, J.H. Kim, N.S. Kang, and W. Yih. 2004a. Mixotrophy in the phototrophic harmful alga *Cochlodinium polykrikoides* (Dinophycea[e]): prey species, the effects of prey concentration, and grazing impact. *Journal of Eukaryotic Microbiology*, **51**: 563–569.

Jeong, H.J., Y.D. Yoo, J.Y. Park, J.Y. Song, S.T. Kim, S.H. Lee, K.Y. Kim, and W.H. Yih. 2005a. Feeding by phototrophic red-tide dinoflagellates: five species newly revealed and six species previously known to be mixotrophic. *Aquatic Microbial Ecology*, **40**: 133–150.

Jessup, D.A., M.A. Miller, J.P. Ryan, H.M. Nevins, H.A. Kerkering, A. Mekebris, D.B. Crane, T.A. Johnson, and R.M. Kudela. 2009. Mass stranding of marine birds caused by a surfactant-producing red tide. *PLoS ONE*, **4**: e4550. doi: 10.1371/journal.pone.0004550.

Ji, X., X. Han, L. Zheng, and J. Zou. 2011. Allelopathic interactions between *Prorocentrum micans* and *Skeletonema costatum* or *Karenia mikimotoi* in laboratory cultures. *Chinese Journal of Oceanology and Limnology*, **29**: 840–848.

Jiang, X., Y. Tang, D. Lonsdale, and C. Gobler. 2009. Deleterious consequences of a red tide dinoflagellate *Cochlodinium polykrikoides* for the calanoid copepod *Acartia tonsa*. *Marine Ecology Progress Series*, **390**: 105–116.

Jiang, X., Q. Li, H. Liang, S. Zhao, L. Zhang, Y. Zhao, L. Chen, W. Yang, and X. Xiang. 2013. Clonal variation in growth plasticity within a *Bosmina longirostris* population: the potential for resistance to toxic cyanobacteria. *PLoS ONE*, **8**(9): e73540. doi: 10.1371/journal.pone.0073540.

Jiang, X., W. Yang, X. Xiang, Y. Niu, L. Chen, and J. Zhang. 2014. Cyanobacteria alter competitive outcomes between *Daphnia* and *Bosmina* in dependence on environmental conditions. *Fundamental and Applied Limnology/Archiv für Hydrobiologie*, **184**: 11–22.

Johansson, N. 2000. Ecological implications of the production of toxic substances by fish-killing phytoplankton species grown under variable N:P-ratios. Dissertation, Lund University.

Johnson, M.D., M. Rome, and D.K. Stoecker. 2003. Microzooplankton grazing on *Prorocentrum minimum* and *Karlodinium micrum* in Chesapeake Bay. *Limnology and Oceanography*, **48**: 238–248.

Jones, A.C., E.A. Monroe, S. Podell, W.R. Hess, S. Klages, E. Esquenaz, S. Niessen, H. Hoover, M. Rothmann, R.S. Lasken, J.R. Yates III, R. Reinhardt, M. Kube, M.D. Burkart, E.E. Allen, P.C. Dorrestein, W.H. Gerwick, and L. Gerwick. 2011. Genomic insights into the physiology and ecology of the marine filamentous cyanobacterium *Lyngbya majuscula*. *Proceedings of the National Academy of Sciences USA*, **108**: 8815–8820.

Jones, T.O., J.N.C. Whyte, N.G. Ginther, L.D. Townsend, and G.K. Iwama. 1995a. Haemocyte changes in the Pacific oyster, *Crassostrea gigas,* caused by exposure to domoic acid in the diatom *Pseudo-nitzschia pungens* f. *multiseries*. *Toxicon*, **33**: 347–353.

Jones, T.O., J.N.C. Whyte, L.D. Townsend, N. Ginther, and G.K. Iwama. 1995b. Effects of domoic acid on haemolymph pH, PCO_2 and PO_2 in the Pacific oyster, *Crassostrea gigas* and the California mussel, *Mytilus californianus*. *Aquatic Toxicology*, **31**: 43–55.

Juhel, G., J. Davenport, J. O'Halloran, S.C. Culloty, R.M. O'Riordan, K.F. James, A. Furey, and O. Allis. 2006. Impacts of microcystins on the feeding behaviour and energy balance of zebra mussels, *Dreissena polymorpha*: a bioenergetics approach. *Aquatic Toxicology*, **79**: 391–400.

Junk, W.J. 1973. Investigations on the ecology and production-biology of the "floating meadows" (Paspalo-Echinochloetum) in the Middle Amazon. *Amazoniana*, **4**: 9–102.

Kamjunke, N., K. Schmidt, S. Pflugmacher, and T. Mehner. 2002. Consumption of cyanobacteria by roach (*Rutilus rutilus*): useful or harmful to the fish? *Freshwater Biology*, **47**: 243–250.

Karjalainen, M., J. Engström-Öst, S. Korpinen, H. Peltonen, J.P. Pääkkönen, S. Rönkkönen, S. Suikkanen, and M. Vitasalo. 2007. Ecosystem consequences of cyanobacteria in the northern Baltic Sea. *Ambio*, **36**: 195–202.

Keppler, C.J., J. Hoguet, K. Smith, A.H. Ringwood, and A.J. Lewitus. 2005. Sublethal effects of the toxic alga *Heterosigma akashiwo* on the southeastern oyster (*Crassostrea virginica*). *Harmful Algae*, **4**: 275–285.

Kerbrat, A.-S., H.T. Darius, S. Pauillac, M. Chinain, and D. Laurent. 2010. Detection of ciguatoxins-like and paralysing toxins in *Trichodesmium* spp. from New Caledonia lagoon. *Marine Pollution Bulletin*, **61**: 360–366.

Keshavanath, P., M.C.M. Beveridge, D.J. Baird, L.A. Lawton, A. Nimmo, and G.A. Codd. 1994. The functional grazing response of a phytoplankton fish *Oreochromis niloticus* to mixtures of toxic and nontoxic strains of the cyanobacterium *Microcystis aeruginosa*. *Journal of Fish Biology*, **45**: 123–129.

Kearns, K.D., and M.D. Hunter. 2000. Green algal extracellular products regulate antialgal toxin production in a cyanobacterium. *Environmental Microbiology*, **2**: 291–297.

Kearns, K.D., and M.D. Hunter. 2001. Toxin-producing *Anabaena flos-aquae* induces settling of *Chlamydomonas reinhardtii,* a competing motile alga. *Microbial Ecology*, **42**: 80–86.

Keating, K.I. 1977. Allelopathic influence of blue-green bloom sequence in a eutrophic lake. *Science*, **196**: 885–887.

Keating, K.I. 1978. Blue-green algal inhibition of diatom growth: transition from mesotrophic to eutrophic community structure. *Science*, **199**: 971–973.

Kempton, J.W., A.J. Lewitus, J.R. Deeds, J.M. Law, and A.R. Place. 2008. Toxicity of *Karlodinium micrum* (Dinophyceae) associated with a fish kill in a South Carolina brackish retention pond. *Harmful Algae*, **1**: 233–241.

Kim, C.S., S.G. Lee, C.K. Lee, H.G. Kim, and J. Jung. 1999. Reactive oxygen species as causative agents in the ichthyotoxicity of the red tide dinoflagellate *Cochlodinium polykrikoides*. *Journal of Plankton Research*, **21**: 2105–2115.

Kim, D., T. Oda, T. Muramatsu, D. Kim, Y. Matsuyama, and T. Honjo. 2002. Possible factors responsible for the toxicity of *Cochlodinium polykrikoides,* a red tide phytoplankton. *Comparative Biochemistry and Physiology (Part C)*, **132**: 415–423.

Kimor, B. 1979. Predation by *Noctiluca miliaris* Souriray on *Acartia tonsa* Dana eggs in the inshore waters of southern California. *Limnology and Oceanography*, **24**: 568–572.

Kiørboe, T., and J. Titelman. 1998. Feeding, prey selection and prey encounter mechanisms in the heterotrophic dinoflagellate *Noctiluca scintillans*. *Journal of Plankton Research*, **20**: 1615–1636.

Kinnear, S. 2010. Cylindrospermopsin: a decade of progress on bioaccumulation. *Marine Drugs*, **8**: 542–564.

Kinnear, S.H.W., L.J. Duivenvoorden, and L.D. Fabbro. 2007. Sublethal responses in *Melanoides tuberculata* following exposure to *Cylindrospermopsis raciborskii* containing cylindrospermopsin. *Harmful Algae*, **6**: 642–650.

Kirchner, M., G. Sahling, G. Uhling, W. Gunkel, and K.W. Klings. 1996. Does the red tide forming dinoflagellate *Noctiluca scintillans* feed on bacteria? *Sarsia*, **81**: 45–55.

Kraberg, A.C., N. Wasmund, J. Vanaverbeke, D. Schiedek, K.H. Wiltshire, and N. Mieszkowska. 2011. Regime shifts in the marine environment: the scientific basis and political context. *Marine Pollution Bulletin*, **62**: 7–20.

Kramer, D.L. 1987. Dissolved oxygen and fish behavior. *Environmental Biology of Fishes*, **18**: 81–92.

Kreuder, C., M.A. Miller, L.J. Lowenstine, P.A. Conrad, T.E. Carpenter, D.A. Jessup, and J.A.K. Mazet. 2005. Evaluation of cardiac lesions and risk factors associated with myocarditis and dilated cardiomyopathy in southern sea otters (*Enhydra lutris nereis*). *American Journal of Veterinary Research*, **66**: 289–299.

Krishnan, A.A., P.K. Krishnakumar, and M. Rajagopalan. 2007. *Trichodesmium erythraeum* (Ehrenberg) bloom along the southwest coast of India (Arabian Sea) and its impact on trace metal concentrations in seawater. Estuarine, *Coastal and Shelf Science*, **71**: 641–646.

Kubanek, J., M.K. Hicks, J. Naar, and T.A. Villareal. 2005. Does the red tide dinoflagellate *Karenia brevis* use allelopathy to outcompete other phytoplankton? *Limnology and Oceanography*, **50**: 883–895.

Kuffner, I.B., and V.J. Paul. 2004. Effects of the benthic cyanobacterium *Lyngbya majuscula* on larval recruitment of the reef corals *Acropora surculosa* and *Pocillopora damicornis*. *Coral Reefs*, **23**: 455–458.

Kuffner, I.B., L.J. Walters, M.A. Becerro, V.J. Paul, R. Ritson-Williams, and K.S. Beach. 2006. Inhibition of coral recruitment by macroalgae and cyanobacteria. *Marine Ecology Progress Series*, **323**: 107–117.

Kvetik, R., and C. Bretz. 2004. Harmful algal bloom toxins protect bivalve populations from sea otter predation. *Marine Ecology Progress Series*, **271**: 233–243.

Kvetik, R., and C. Bretz. 2005. Shorebird foraging behavior, diet, and abundance vary with harmful algal bloom toxin concentrations in invertebrate prey. *Marine Ecology Progress Series*, **293**: 303–309.

Kvitek, R.G., A.R. DeGunge, and M.K. Beitler. 1991. Paralytic shellfish poisoning toxins mediate feeding behavior of sea otters. *Limnology and Oceanography*, **36**: 393–404.

Kvitek, R.G., J.D. Goldberg, G.J. Smith, G.J. Doucette, and M.W. Silver. 2008. Domoic acid

contamination within eight representative species from the benthic food web of Monterey Bay, California, USA. *Marine Ecology Progress Series*, **67**: 35–47.

Landsberg, J.H. 2002. The effects of harmful algal blooms on aquatic organisms. *Reviews in Fisheries Science*, **10**: 113–390.

Landsberg, J.H., S. Hall, J.N. Johannessen, K.D. White, S.M. Conrad, J.P. Abbott, L.J. Flewelling, R.W. Richardson, R.W. Dickey, E.L.E. Jester, S.M. Etheridge, J.R. deeds, F.M. Van Dolah, T.A. Leighfield, Y. Zou, C.G. Beaudry, R.A. Benner, P.L. Rogers, P.S. Scott, K. Kawabata, J.L. Wolny, and K.A. Steidinger. 2006. Saxitoxin puffer fish poisoning in the United States, with the first report of *Pyrodinium bahamense* as the putative toxin source. *Environmental Health Perspectives*, **114**: 1502–1507.

Landsberg, J.H., L.J. Flewelling, and J. Naar. 2009. *Karenia brevis* red tides, brevetoxins in the food web, and impacts on natural resources: decadal advancements. *Harmful Algae*, **8**: 598–607.

Landsberg, J.H., K.A. Lefebvre, and L.J. Flewelling. 2014. Effects of toxic microalgae on marine organisms. In: *Toxins and Biologically Active Compounds from Microalgae*, Vol. 2. G.P. Rossini (Ed.). CRC Press, Boca Raton: p. 379–449.

Lapointe, B.E., J.M. Burkholder, and K.L. Van Alstyne. 2018. Harmful macroalgal blooms in a changing world: causes, impacts, and management. In: *Harmful Algal Blooms: A Compendium Desk Reference*. S.E. Shumway, J.M. Burkholder, and S.L. Morton (Eds.). Wiley Blackwell: p. 515–560.

Lapointe, B.E., and M.W. Clark. 1992. Nutrient inputs from the watershed and coastal eutrophication in the Florida Keys. *Estuaries*, **15**: 465–476.

Lapointe, B.E., W.R. Matzie, and M.W. Clark. 1994. Phosphorus inputs and eutrophication on the Florida reef tract. In: *Proceedings of the Colloquium on Global Aspects of Coral Reefs: Health, Hazards and History, 1993*. Rosenstiel School of Marine and Atmospheric Science, University of Miami, Miami: p. 106–112.

Lapointe, B.E., W.R. Matzie, and P.J. Barile. 2002. Biotic phase-shifts in Florida Bay and fore reef communities of the Florida Keys: linkages with historical freshwater flows and nitrogen. In: *The Everglades, Florida Bay, and Coral Reefs of the Florida Keys: An Ecosystem Sourcebook*. J.W. Porter and K.G. Porter (Eds.). CRC Press, Boca Raton: p. 629–648.

Lassus, P., M. Bardouil, B. Beliaeff, P. Masselin, M. Naviner, and P. Truquet. 1999. Effect of a continuous supply of the toxic dinoflagellate *Alexandrium minutum* Balim on the feeding behaviour of the Pacific oyster (*Crassostrea gig*as Thunberg). *Journal of Shellfish Research*, **18**: 211–216.

Lassus, P., and J.P. Berthome. 1988. Status of 1987 algal blooms in IFREMER [l'Insitut Francais de Recherché Pour l'Exploitation de La Mer, or the French Research Institute for Exploitation of the Sea]. *International Council for the Exploration of the Sea (ICES)/annes III*, C.M. 1988/F: 33A. 5–13.

Lassus, P., D.J. Wildish, M. Bardouil, J.L. Martin, M. Bohec, and S. Bougrier. 1996. Ecophysiological study of toxic *Alexandrium* spp. effects on the oyster *Crassostrea gigas*. In: T. Yasumoto, Y. Oshima, and Y. Fukuyo (Eds.), *Harmful and Toxic Algal Blooms*. UNESCO, Paris: p. 409–412.

Latour, D., O. Sabido, M.-J. Saleçon, and H. Giraudet. 2004. Dynamics and metabolic activity of the benthic cyanobacterium *Microcystis aeruginosa* in the Grangent reservoir (France). *Journal of Plankton Research*, **26**: 719–726.

Laurén-Määttä, C., J. Hietala, and M. Walls. 1997. Responses of *Daphnia pulex* populations to toxic cyanobacteria. *Freshwater Biology*, **37**: 635–647.

Leão, P.N., M.T.S.D. Vasconcelos, and V.M. Vasconcelos. 2009. Allelopathy in freshwater cyanobacteria. *Critical Reviews in Microbiology*, **35**: 271–282.

Lefebvre, K.A., N. Elder, P.K. Hershberger, V.L. Trainer, C.M. Stehr, and N.L. Scholz. 2005. Dissolved saxitoxin causes transient inhibition of sensorimotor function in larval Pacific herring (*Clupea harengus pallasi*). *Marine Biology*, **147**: 1393–1402.

Lefebvre, K.A., E.R. Frame, and P.S. Kendrick. 2012. Domoic acid and fish behavior: a review. *Harmful Algae*, **13**: 126–130.

Lefebvre, K.A., D.P. Noren, I.R. Schultz, S.M. Bogard, J. Wilson, and B.T. Eberhart. 2007. Uptake, tissue distribution and excretion of domoic acid after oral exposure in coho salmon (*Oncorhynchus kisutch*). *Aquatic Toxicology*, **81**: 266–274.

Lefebvre, K.A., C.L. Powell, M. Busman, G.J. Doucette, P.D.R. Moeller, J.B. Silver, P.E. Miller, M.P. Hughes, S. Singaram, M.W. Silver, and R.S. Tjeerdema. 1999. Detection of domoic acid in northern anchovies and California sea lions associated with an unusual mortality event. *Natural Toxins*, **7**: 85–92.

Lefebvre, K.A., and A. Robertson. 2010. Domoic acid and human exposure risks: a review. *Toxicon*, **56**: 218–230.

Lefebvre, K.A., M.W. Silver, S.L. Coale, and R.S. Tjeerdema. 2002. Domoic acid in planktivorous fish in relation to toxic *Pseudo-nitzschia* cell densities. *Marine Biology*, **140**: 625–631.

Leflaive, J., L. Ten-Hage. 2007. Algal and cyanobacterial secondary metabolites: a comparison of allelopathic compounds and toxins. *Freshwater Biology*, **52**: 199–214.

Legler, J. 2008. New insights into the endocrine disrupting effects of brominated flame retardants. *Chemosphere*, **73**: 216–222.

Legrand, C., K. Rengefors, G.O. Fistarol, and E. Granéli. 2003. Allelopathy in phytoplankton: biochemical, ecological and evolutionary aspects. *Phycologia*, **42**: 406–419.

Lehman, P.W., S.J. Teh, G. Boyer, M.L. Nobriga, E. Bass, and C. Hogle. 2010. Initial impacts of *Microcystis aeruginosa* blooms on the aquatic food web in the San Francisco estuary. *Hydrobiologia*, **637**: 229–248.

Lehtiniemi, M., J. Engstrom-Ost, M. Karjalainen, B. Kozlowsky-Suzuki, and M. Vitasalo. 2002. Fate of cyanobacterial toxins in the pelagic food web: transfer to copepods or to faecal pellets? *Marine Ecology Progress Series*, **241**: 13–21.

Lelong, A., H. Haberkorn, N. Goïc, H. Hégaret, and P. Soudant. 2011. A new insight into allelopathic effects of *Alexandrium minutum* on photosynthesis and respiration of the diatom *Chaetoceros neogracile* revealed by photosynthetic-performance analysis and flow cytometry. *Microbial Ecology*, **62**: 919–930.

Lembi, C.A. 2003. Control of nuisance algae. In: *Freshwater Algae of North America – Ecology and Classification*. J.D. Weir and R.G. Sheath (Eds.). Academic Press, New York: p. 805–834.

Leonard, J.A., and H.W. Paerl. 2005. Zooplankton community structure, micro-zooplankton grazing impact, and seston energy content in the St. Johns River system, Florida as influenced by the toxic cyanobacterium *Cylindrospermopsis raciborskii*. *Hydrobiologia*, **537**: 89–97.

Lesser, M.P., and S.E. Shumway. 1993. Effects of toxic dinoflagellates on clearance rates and survival in juvenile bivalve molluscs. *Journal of Shellfish Research*, **12**: 377–381.

Leverone, J.R., N.J. Blake, R.H. Pierce, and S.E. Shumway. 2006. Effects of the dinoflagellate *Karenia brevis* on larval development in three species of bivalve mollusc from Florida. *Toxicon*, **48**: 75–84.

Lévesque, D., A. Cattaneo, C. Hudon, and P. Gagnon. 2012. Predicting the risk of proliferation of the benthic cyanobacterium *Lyngbya wollei* in the St. Lawrence River. *Canadian Journal of Fisheries and Aquatic Sciences*, **69**: 1585–1595.

Lewin, R.A. 1962. *Physiology and Biochemistry of Algae*. Academic Press, New York.

Lewis, R.J. 1992. Ciguatoxins are potent ichthyotoxins. *Toxicon*, **30**: 207–211.

Lewitus, A.J., M.S. Wetz, B.M. Willis, J.M. Burkholder, and H.B. Glasgow. 2006. Grazing activity of *Pfiesteria piscicida* (Dinophyceae) and susceptibility to ciliate predation vary with toxicity status. *Harmful Algae*, **5**: 427–434.

Lewitus, A.J., R.A. Horner, D.A. Caron, E. Garcia-Mendoza, B.M. Hickey, M. Hunter, D.D. Huppert, R.M. Kudela, G.W. Langlois, J.L. Largier, E.J. Lessard, R. RaLonde, J.E.J. Rensel, P.G. Strutton, V.L. Trainer, and J.F. Tweddle. 2012. Harmful algal blooms along the North American west coast region: history, trends, causes, and impacts. *Harmful Algae*, **19**: 133–159.

Li, J., P.M. Glibert, and Y. Gao. 2015. Temporal and spatial changes in Chesapeake Bay water quality and relationships to *Prorocentrum minimum, Karlodinium veneficum,* and CyanoHAB events, 1991–2008. *Harmful Algae*, **42**: 1–14.

Li, J., P.M. Glibert, J.A. Alexander, and M.E. Molina. 2012b. Growth and competition of several harmful dinoflagellates under different nutrient and light conditions. *Harmful Algae*, **13**: 112–125.

Li, Y., I. Sunila, and G.H. Wikfors. 2012a. Bioactive effects of *Prorocentrum minimum* on juvenile bay scallops (*Argopecten irradians irradians*) are dependent upon algal physiological status. *Botanica Marina*, **55**: 19–29.

Lin, J., T. Yan, Q. Zhang, and M. Zhou. 2016. Impact of several harmful algal bloom (HAB) causing species, on life history characteristics of rotifer *Brachionus plicatilis* Müller. *Chinese Journal of Oceanology and Limnology*, **34**: 642–653.

Liu, H., and E.J. Buskey. 2000. The extracellular polymeric substance (EPS) layer surrounding *Aureoumbra lagunensis* cells reduces grazing by protozoa. *Limnology and Oceanography*, **45**: 1187–1191.

Liu, J., A.J. Lewitus, P. Brown, and S.B. Wilde. 2008. Growth-promoting effects of a bacterium on raphidophytes and other phytoplankton. *Harmful Algae*, **7**: 1–10.

Liu, Y., L. Song, X. Li, and T. Liu. 2002. The toxic effects of MC-LR on embryo-larval and juvenile development of loach, *Misguruns mizolepis* Gunthe. *Toxicon*, **40**: 395–399.

Liu, Y., P. Xie, and X.-P. Wu. 2006. Effects of toxic and nontoxic *Microcystis aeruginosa* on survival, population-increase, and feeding of two small

cladocerans. *Bulletin of Environmental Contamination and Toxicology*, **77**: 566–573.

Lightner, D.V. 1978. Possible toxic effects of the marine blue-green alga, *Spirulina subsalsa,* on the blue shrimp, *Panaesus stylirostis*. *Journal of Invertebrate Pathology*, **32**: 139–150.

Liras, V., M. Lindberg, P. Nystrom, H. Annadotter, L.A. Lawton, and B. Graf. 1998. Can ingested cyanobacteria be harmful to the signal crayfish (*Pacifastacus leniusculus*)? *Freshwater Biology*, **39**: 233–242.

Llewellyn, L.E., A.P. Negri, J. Doyle, P.D. Baker, E.C. Beltran, and B.A. Neilan. 2001. Radioreceptor assays for sensitive detection and quantitation of saxitoxin and its analogues from strains of the freshwater cyanobacterium, *Anabaena circinalis*. *Environmental Science & Technology*, **35**: 1445–1451.

Loftin, K.A., J.L. Graham, E.D. Hilborn, S.C. Lehmann, M.T. Meyer, J.E. Dietze, and C.B. Griffith. 2016. Cyanotoxins in inland lakes of the United States: occurrence and potential recreational health risks in the EPA National Lakes Assessment 2007. *Harmful Algae*, **56**: 77–90.

Lonsdale, D. J., E. M. Cosper, W.S. Kim, and S.H. Jónasdóttir. 1996. Food web interactions in the plankton of Long Island bays, with preliminary observations on brown tide effects. *Marine Ecology Progress Series*, **134**: 247–263.

Łotocka, M. 2001. Toxic effects of cyanobacterial blooms on the grazing activity of *Daphnia magna* Straus. *Oceanologia*, **43**: 441–453.

Lu, Z., and S.M. Tomchik. 2002. Effects of a red-tide toxin on fish hearing. *Journal of Comparative Physiology*, **188**: 807–813.

Luckenbach, M.W., K.G. Sellner, S.E. Shumway, and K. Greene. 1993. Effects of two bloom forming dinoflagellates, *Prorocentrum minimum* and *Gyrodinium uncatenatum,* on the growth and survival of the eastern oyster, *Crassostrea virginica*. *Journal of Shellfish Research*, **12**: 411–415.

Lürling, M., E.J. Faassen, and J.S. Van Eenennaam. 2011. Effects of the cyanobacterial neurotoxin β-N-methylamino-L-alanine (BMAA) on the survival, mobility and reproduction of *Daphnia magn*a. *Journal of Plankton Research*, **33**: 333–342.

Lürling, M., and G.E. van der Grinten. 2003. Life–history characteristics of *Daphnia* exposed to dissolved microcystin–LR and to the cyanobacterium *Microcystis aeruginosa* with and without microcystins. *Environmental Toxicology and Chemistry*, **6**: 1281–1287.

Lyczkowski, E.R., and L. Karp-Boss. 2014. Allelopathic effects of *Alexandrium fundyense* (Dinophyceae) on *Thalassiosira* cf. *gravida* (Bacillariophyceae): a matter of size. *Journal of Phycology*, **50**: 376–387.

Lyons, D.A., C. Arvanitidis, A.J. Blight, E. Chatzinikolaou, T. Guy-Haim, J. Kotta, H. Orav-Kotta, A. Queiros, G. Rilov, P.J. Somerfield, and T.P. Crowe. 2014. Macroalgal blooms alter community structure and primary productivity in marine ecosystems. *Global Change Biology*, **20**: 2712–2724.

Ma, H., B. Krock, U. Tillmann, and A. Cembella. 2009. Preliminary characterization of extracellular allelochemicals of the toxic marine dinoflagellate *Alexandrium tamarense* using a *Rhodomonas salina* assay. *Marine Drugs*, **7**: 497–522.

Ma, H., B. Krock, U. Tillmann, A. Muck, N. Wielsch, A. Svatoš, and A. Cembella. 2011. Isolation of activity and partial characterization of large non-proteinaceous lytic allelochemicals produced by the marine dinoflagellate *Alexandrium tamarense*. *Harmful Algae*, **11**: 65–72.

Mackenthum, K.M., E.F. Herman, and A.F. Bartsch. 1948. A heavy mortality of fishes resulting from the decomposition of algae in the Yahara River, Wisconsin. *Transactions of the American Fisheries Society*, **75**: 175–180.

Mahoney, J.B. 1978. The Seasonal Maxima of *Ceratium tripos* with Particular Reference to a Major New York Bight Bloom. Technical Report No. 16. Sandy Hook Laboratory, Northeast Fisheries Center, National Marine Fisheries Service, NOAA – U.S. Department of Commerce, Highlands.

Mahoney, J.B., and F.W. Steimle Jr. 1979. A mass mortality of marine animals associated with a bloom of *Ceratium tripos* in the New York Bight. In: *Toxic Dinoflagellate Blooms*. D.L. Taylor and H.H. Seliger (Eds.). Elsevier, New York: p. 225–230.

Malbrouck, C., and P. Kestemont. 2006. Effects of microcystins on fish. *Environmental Toxicology and Chemistry*, **25**: 72–86.

Mallin, M.A., J.M. Burkholder, L.M. Larsen, and H.B. Glasgow. 1995. Response of two zooplankton grazers to an ichthyotoxic estuarine dinoflagellate. *Journal of Plankton Research*, **17**: 351–363.

Malmvärn, A., G. Marsh, L. Kautsky, M. Athanasidou, A. Bergman, and L. Asplund. 2005. Hydroxylated and methoxylated brominated diphenyl ethers in the red alga[e] *Ceramium tenuicorne* and blue mussels from the Baltic Sea. *Environmental Science & Technology*, **39**: 2990–2997.

Manire, C.A., E.T. Anderson, L. Byrd, and D.A. Fauquier. 2013. Dehydration as an effective treatment for brevetoxicosis in loggerhead sea turtles. *Journal of Zoo and Wildlife Medicine*, **44**: 447–452.

Manning, S.R., and J.W. La Claire. 2010. Prymnesins: toxic metabolites of the golden alga, *Prymnesium parvum* Carter (Haptophyta). *Marine Drugs*, **8**: 678–704.

Marcoval, M.A., J. Pan, Y. Tang, and C.J. Gobler. 2013. The ability of the branchiopod, *Artemia salina,* to graze upon harmful algal blooms caused by *Alexandrium fundyense, Aureococcus anophagefferens,* and *Cochlodinium polykrikoides*. *Estuarine, Coastal and Shelf Science*, **131**: 235–244.

Marshall, H.G., P.E. Hargraves, M. Elbrächter, J.M. Burkholder, M.W. Parrow, E.H. Allen, V.M. Knowlton, P.A. Rublee, W.L. Hynes, T.A. Egerton, D.L. Remington, K.B. Wyatt, K.J. Coyne, A.J. Lewitus, and V.C. Henrich. 2006. Taxonomy of *Pfiesteria* (Dinophyceae). *Harmful Algae*, **5**: 481–496.

Martin-Cereceda, M., G. Novarino, and J.R. Young. 2003. Grazing by *Prymnesium parvum* on small planktonic diatoms. *Aquatic Microbial Ecology*, **33**: 191–199.

Martins, J.C., P.N. Leão, and V. Vasconcelos. 2009. Differential protein expression in *Corbicula fluminea* upon exposure to a *Microcystis aeruginosa* toxic strain. *Toxicon*, **53**: 409–416.

Martins, R., P. Pereira, M. Welker, J. Fastner, and M.V. Vasconcelos. 2005. Toxicity of culturable cyanobacteria strains isolated from the Portuguese coast. *Toxicon*, **46**: 454–464.

Mason, C.P., K.R. Edwards, R.E. Carlson, J. Pignatello, F.K. Gleason and J.M. Wood. 1982. Isolation of chlorine-containing antibiotic from the freshwater cyanobacterium *Scytonema hofmanni*. *Science*, **215**: 400–402.

Matsuyama, Y., H. Usuki, T. Uchida, and Y. Kotani. 2001. Effects of harmful algae on the early planktonic larvae of the oyster *Crassostrea gigas*. In: *Proceedings of the Ninth International Conference on Harmful Algal Blooms*. G.M. Hallegraeff, S.I. Blackburn, C.J. Bolch, and R.J. Lewis (Eds.). IOC UNESCO, Paris: p. 411–414.

May, S.E., J.M. Burkholder, S.E. Shumway, H. Hégaret, G.H. Wikfors, and D. Frank. 2010. Effects of the toxic dinoflagellate *Alexandrium monilatum* on survival, grazing and behavioral response of three ecologically important bivalve molluscs. *Harmful Algae*, **9**: 281–293.

Meeuse, B.J.D. 1956. Free sulfuric acid in the brown alga, *Desmarestia*. *Biochimica et Biophysica Acta*, **19**: 373–374.

Mendoza, W.G., R.N. Mead, L.E. Brand, and D. Shea. 2008. Determination of brevetoxin in recent marine sediments. *Chemosphere*, **73**: 1373–1377.

Meriluoto, J. A., and L.E. Spoof. 2008. Cyanotoxins: sampling, sample processing and toxin uptake. *Advances in Experimental Medicine and Biology*, **619**: 483–499.

Metcalf, J.S., K.A. Beattie, S. Birmingham, M.L. Saker, A.K. Torokne, and G.A. Codd. 2002. Toxicity of cylindrospermopsin to the brine shrimp *Artemia salina*: comparisons with protein synthesis inhibitors and microcystins. *Toxicon*, **40**: 1115–1120.

Metcalf, J.S., G.A. Codd, and V.M. Vasconcelos. 2004. Accumulation and depuration of the cyanobacterial toxin cylindrospermopsin in the freshwater mussel *Anodonta cygnea*. *Toxicon*, **43**: 185–194.

Miller, M.A., R.M. Kudela, A. Mekebri, D. Crane, S.C. Oates, M.T. Tinker, M. Staedler, W.A. Miller, S. Toy-Choutka, C. Dominik, D. Hardin, G. Langlois, M. Murray, K. Wards, and D.A. Jessup. 2010. Evidence for a novel marine harmful algal bloom: cyanotoxin (microcystin) transfer from land to sea otters. *PLoS ONE*, **59**(9): e12576. doi: 10.1371/journal.pone.0012576

Miralto, A., G. Barone, G. Romano, S.A. Poulet, A. Ianora, G.L. Russo, I. Buttino, G. Mazzarella, M. Laabir, M. Cabrini, and M.G. Giacobbe. 1999. The insidious effect of diatoms on copepod reproduction. *Nature*, **402**: 173–176.

Misson, B., M. Sabart, C. Amblard, and D. Latour. 2012. Benthic survival of *Microcystis*: long-term viability and ability to transcribe microcystin genes. *Harmful Algae*, **13**: 20–25.

Mitch, W.J., and J.G. Gosselink. 2007. *Wetlands*, 4th ed. John Wiley & Sons, Inc., New York.

Mitrovic, S.M., O. Allis, A. Furey, and K.J. James. 2005. Bioaccumulation and harmful effects of microcystin-LR in the aquatic plants *Lemna minor* and *Wollfia arrhiza* and the filamentous alga *Cladophora fracta*. *Ecotoxicology and Environmental Safety*, **61**: 345–352.

Moeller, P.D.R., K.R. Beauchesne, K.M. Huncik, W.C. Davis, S.J. Christopher, P. Riggs-Gelasco, and A. Gelasco. 2007. Metal complexes and free radical toxins produced by *Pfiesteria piscicida*. *Environmental Science & Technology*, **41**: 1166–1172.

Moestrup, Ø., G. Hansen, N. Daugbjerg, N. Lundholm, J. Overton, M. Verstergård, S.J. Steenfeldt, A.J. Calado, and P.J. Hansen. 2014. The dinoflagellates *Pfiesteria shumwayae* and *Luciella masanensis* cause fish kills in

recirculation fish farms in Denmark. *Harmful Algae*, **32**: 33–39.

Moffett, J.W., and L.E. Brand. 1996. The production of strong, extracellular Cu chelators by marine cyanobacteria in response to Cu stress. *Limnology and Oceanography*, **41**: 288–293.

Mohamed, A., and M. Al-Shehri. 2012. The link between shrimp farm runoff and blooms of toxic *Heterosigma akashiwo* in Red Sea coastal waters. *Oceanologia*, **54**: 287–309.

Montagna, P.A., D.A. Stockwell, and R.D. Kalke. 1993. Dwarf surfclam *Mulinia lateralis* (Say, 1822) populations and feeding during the Texas brown tide event. *Journal of Shellfish Research*, **12**: 433–442.

Montoya, N.G., R. Akselman, J. Franco, and J.I. Carreto. 1996. Paralytic shellfish toxins and mackerel (*Scomber japonicus*) mortality in the Argentine sea. In: *Harmful and Toxic Algal Blooms*. T. Yasumoto, Y. Oshima, and Y. Fukuyo (Eds.). IOC UNESCO, Paris: p. 417–420.

Moore, R.E. 1977. Toxins from blue-green algae. *BioScience*, **27**: 797–802.

Morgan, A.M., T.V. Royer, M.B. David, and L.E. Gentry. 2006. Relationships among nutrients, chlorophyll-*a,* and dissolved oxygen in agricultural streams in Illinois. *Journal of Environmental Quality*, **35**: 1110–1117.

Morton, B.E., C.F. Fraser, M.T. Suhartono, and M.D. Rayner. 1982. Potent inhibition of sperm motility by palytoxin. *Experimental Cell Research*, **140**: 261–265.

Murray, M.M., and N.H. Marcus. 2002. Survival and diapause egg production of the copepod *Centropages hamatus* raised on dinoflagellate diets. *Journal of Experimental Marine Biology and Ecology*, **270**: 39–56.

Nakamura, Y. 1998. Growth and grazing of a large heterotrophic dinoflagellate, *Noctiluca scintillans,* in laboratory cultures. *Journal of Plankton Research*, **20**: 1711–1720.

Naar, J.P. L.J. Flewelling, A. Lenzi, J.P. Abbott, A. Granholm, H.M. Jacocks, D. Gannon, M. Henry, R. Pierce, D.G. Baden, J. Wolny, and J.H. Landsberg. 2007. Brevetoxins, like ciguatoxins, are potent ichthyotoxic neurotoxins that accumulate in fish. *Toxicon*, **50**: 707–723.

Nasri, H., S. El Herry, and N. Bouaicha. 2008. First reported case of turtle deaths during a toxic *Microcystis* spp. bloom in Lake Oubeira, Algeria. *Ecotoxicology and Environmental Safety*, **71**: 535–544.

National Oceanographic and Atmospheric Administration (NOAA). 2006. Notice – availability of new criteria for designation of marine mammal unusual mortality events (UMEs). *Federal Register*, **71**: 75234–75236.

Nejstgaard, J.C., and P.T. Solberg. 1996. Repression of copepod feeding and fecundity by the toxic haptophyte *Prymnesium patelliferum*. *Sarsia*, **81**: 339–344.

Nelson, T.A., and A. Lee. 2001. A manipulative experiment demonstrates that ulvoid algal blooms reduce eelgrass shoot density. *Aquatic Botany*, **71**: 149–154.

Nelson, T.A., A.V. Nelson, and M. Tjoelker. 2003. Seasonal and spatial patterns of "green tides" (ulvoid algal blooms) and related water quality parameters in the coastal waters of Washington State, USA. *Botanica Marina*, **46**: 263–275.

Nesbet, I.C.T. 1983. Paralytic shellfish poisoning: effects on breeding terns. *The Condor*, **85**: 338–345.

Ng, C.S.L., L.J. Ong, and L.M. Chou. 2012. *Lyngbya majuscula* blooms in an enclosed marine environment. *Environment Asia*, **5**: 93–98.

Nielsen, M.V. 1993. Toxic effect of the marine dinoflagellate *Gymnodinium galatheanum* on juvenile cod *Gadus morhua*. *Marine Ecology Progress Series*, **95**: 273–277.

Nielsen, M.V., and T. Strømgren. 1991. Shell growth response of mussels (*Mytilus edulis*) exposed to toxic microalgae. *Marine Biology*, **108**: 263–267.

Nikishina, A.B., A.V. Drits, Y.V. Vasilyeva, A.G. Timonin, K.A. Solovyev, T.N. Ratkova, and V.M. Sergeeva. 2011. Role of the *Noctiluca scintillans* population in the trophic dynamics of the Black Sea plankton over the spring period. *Oceanology*, **51**: 1029–1039.

Nizan, S., C. Dimentman, and M. Shilo. 1986. Acute toxic effects of the cyanobacterium *Microcystis aeruginosa* on *Daphnia magna*. *Limnology and Oceanography*, **31**: 497–502.

Nogueira, I.C.G., M.L. Saker, S. Pflugmacher, C. Wiegand, and V.M. Vasconcelos. 2004. Toxicity of the cyanobacterium *Cylindrospermopsis raciborskii* to *Daphnia magna*. *Environmental Toxicology*, **19**: 453–459.

Nygaard, K. and A. Tobiesen. 1993. Bacterivory in algae: a survival strategy during nutrient limitation. *Limnology and Oceanography*, **38**: 273–279.

Oberemm, A., J. Becker, G.A. Codd, and C. Steinberg. 1999. Effects of cyanobacterial toxins and aqueous crude extracts of cyanobacteria on the development of fish and amphibians. *Environmental Toxicology*, **14**: 77–88.

O'Clair, R.M., and S.C. Lindstrom. 2000. *North Pacific Seaweeds*. Plant Press, Juneau.

Okaichi, T., and S. Nishio. 1976. Identification of ammonia as the toxic principle of red tide of *Noctiluca miliaris*. *Bulletin of the Plankton Society of Japan*, **23**: 75–80.

O'Neill, J.M., T.W. Davis, M.A. Burford, and C.J. Gobler. 2012. The rise of harmful cyanobacteria blooms: the potential roles of eutrophication and climate change. *Harmful Algae*, **14**: 313–334.

O'Neil, J.M., and M.R. Roman. 1994. Ingestion of the cyanobacterium[a] *Trichodesmium* spp. by pelagic harpacticoid copepods *Macrosetella, Miracia* and *Oculostella*. *Hydrobiologia*, **293**: 235–240.

Onoue, Y., and K. Nozawa. 1989. Zinc-bound PSP toxins separated from *Cochlodinium* red tide. In: *Mycotoxins and Phycotoxins 88*. S. Natori, K. Hashimoto, and Y. Ueno (Eds.). Elsevier, Amsterdam: p. 359–366.

Onuf, C.P. 1996. Seagrass responses to long-term light reduction by brown tide in upper Laguna Madre, Texas: distribution and biomass patterns. *Marine Ecology Progress Series*, **138**: 219–231.

Osborne, N.J.T., P.M. Webb, and G.R. Shaw. 2001. The toxins of *Lyngbya majuscula* and their human and ecological health effects. *Environment International*, **27**: 381–392.

Osman, R.W., P. Munguia, and R.N. Zajac. 2010. Ecological thresholds in marine communities: theory, experiments and management. *Marine Ecology Progress Series*, **413**: 185–187.

O'Shea, T.J., G.B. Rathbun, R.K. Bonde, C.D. Buergelt, and D.K. Odell. 1991. An epizootic of Florida manatees associated with a dinoflagellate bloom. *Marine Mammal Science*, **7**: 165–179.

Osswald, J., S. Rellán, A.P. Carvalho, A. Gago, and V. Vasconcelos. 2007. Acute effects of an anatoxin-a producing cyanobacterium on juvenile fish – *Cyprinus carpio* L. *Toxicon*, **49**: 693–698.

Osterling, M., and L. Pihl. 2001. Effects of filamentous green algal mats on benthic macrofaunal functional feeding groups. *Journal of Experimental Marine Biology and Ecology*, **263**: 159–183.

Ou, D., L. Song, N. Gan, and W. Chen. 2005. Effects of microcystins on and toxin degradation by *Poterioochromonas* sp. *Environmental Toxicology*, **20**: 373–380.

Padilla, D.K., M.H. Doall, C.J. Gobler, A. Hartson, and K. O'Boyle. 2006. Brown tide alga, *Aureococcus anophagefferens,* can affect growth but not survivorship of *Mercenaria mercenaria* larvae. *Harmful Algae*, **5**: 736–748.

Palikova, M., F. Kovářů, S. Navrátil, L. Kubala, S. Pešák, and V. Piackova. 1998. The effects of pure microcystin-LR and biomass of blue-green algae on selected immunological indices of carp (*Cyprinus carpio* L.) and silver carp (*Hypophthalmichthys molitrix* Val.). *Acta Veterinaria Brunensis*, **67**: 265–272.

Palmer, M.A. 2000. Linkages between aquatic sediment biota and life above sediments as potential drivers of biodiversity and ecological processes. *BioScience*, **50**: 1062–1075.

Parrow, M.W., J.M. Burkholder, N.J. Deamer, and J.S. Ramsdell. 2005. Contaminant-free cultivation of *Pfiesteria shumwayae* (Dinophyceae) on a fish cell line. *Aquatic Microbial Ecology*, **39**: 97–105.

Parsons, M.L., and Q. Dortch. 2002. Sedimentological evidence of an increase in *Pseudo-nitzschia* (Bacillariophyceae) abundance in response to coastal eutrophication. *Limnology and Oceanography*, **47**: 551–558.

Paul, N.A., R. De Nys, and P.D. Steinberg. 2006. Chemical defense against bacteria in the red alga *Asparagopsis armata*: linking structure with function. *Marine Ecology Progress Series*, **306**: 87–101.

Paul, V.J., R.W. Thacker, K. Banks, and S. Golubic. 2005. Benthic cyanobacterial bloom impacts the reefs of South Florida (Broward County, USA). *Coral Reefs*, **24**: 693–697.

Paz, B., P. Riobó, M.L. Fernández, S. Fraga, and J.M. Franco. 2004. Production and release of yessotoxins by the dinoflagellates *Protoceratium reticulatum* and *Lingulodinium polyedrum* in culture. *Toxicon*, **44**: 251–258.

Paz, B., A.H. Daranas, M. Norte, P. Riobó, J.M. Franco, and J.J. Fernández. 2008. Yessotoxins, a group of marine polyether toxins: an overview. *Marine Drugs*, **6**: 73–102.

Pelletreau, K., and G. Muller-Parker. 2002. Sulfuric acid in the phaeophyte alga *Desmarestia munda* deters feeding by the sea urchin *Strongylocentrotus droebachiensis*. *Marine Biology*, **141**: 1–9.

Peuthert, A., and C. Wiegand. 2004. Glutathione involvement in the biotransformation of microcystins in *Dreissena polymorpha*. In: *6th International Conference on Toxic Cyanobacteria, Bergen, Norway (abstract)*.

Pflugmacher, S., C. Wiegand, A. Oberemm, K.A. Beattie, E. Krause, G.A. Codd, and C.E.W. Steinberg. 1998. Identification of an enzymatically formed glutathione conjugate of the cyanobacterial hepatotoxin microcystin-LR: the first step of detoxication. *Biochimica et Biophysica Acta*, **1425**: 527–533.

Pierce, R.H., and M.S. Henry 2008. Harmful algal toxins of the Florida red tide (*Karenia brevis*): natural chemical stressors in South

Florida coastal ecosystems. *Ecotoxicology*, **17**: 623–631.

Phlips, E.J., S. Badylak, and T.C. Lynch. 1999. Blooms of the picoplanktonic cyanobacterium *Synechococcus* in Florida Bay, a subtropical inner-shelf lagoon. *Limnology and Oceanography*, **44**: 1166–1175.

Phlips, E.J., T.C. Lynch, and S. Badyluk. 1995. Chlorophyll *a,* tripton, color, and light availability in a shallow tropical inner-shelf lagoon, Florida Bay, USA. *Marine Ecology Progress Series*, **127**: 223–234.

Pihl, L., S.P. Baden, R.J. Diaz, and L.C. Schaffner. 1991. Hypoxia-induced structural changes in the diet of bottom-feeding fish and Crustacea. *Marine Biology*, **112**: 349–361.

Pihl, L., I. Isaksson, H. Wennhage, and P. Moksnes. 1995. Recent increase of filamentous algae in shallow Swedish bays: effects on the community structure of epibenthic fauna and fish. *Netherlands Journal of Aquatic Ecology*, **29**: 349–358.

Pinheiro, C., J. Azevedo, A. Campos, V. Vasconcelos, and S. Loureiro. 2016. The interactive effects of microcystin-LR and cylindrospermopsin on the growth rate of the freshwater alga[e] *Chlorella vulgaris*. *Ecotoxicology*, **25**: 745–758.

Pires, L.M.D., K.M. Karlsson, J.A.O. Meriluoto, E. Kardinaal, P.M. Visser, K. Siewertsen, E. Van Donk, and B.W. Ibelings. 2004. Assimilation and depuration of microcystin-LR by the zebra mussel, *Dreissena polymorpha*. *Aquatic Toxicology*, **69**: 385–396.

Pires, L.M.D., and E. Van Donk. 2002. Comparing grazing by *Dreissena polymorpha* on phytoplankton in the presence of toxic and nontoxic cyanobacteria. *Freshwater Biology*, **47**: 1855–1865.

Poulson, K.L., R.D. Sieg, E.K. Prince, and J. Kubanek. 2010. Allelopathic compounds of a red tide dinoflagellate have species-specific and context-dependent impacts on phytoplankton. *Marine Ecology Progress Series*, **416**: 69–78.

Poulson-Ellestad, K.L., C.M. Jones, J. Roy, M.R. Viant, F.M. Fernández, J. Kubanek, and B.L. Nunn. 2014. Metabolomics and proteomics reveal impacts of chemically mediated competition on marine plankton. *Proceedings of the National Academy of Sciences USA*, **111**: 9009–9014.

Prado-Alvarez, M., F. Flórez-Barrós, J. Méndez, and J. Fernandez-Tajes. 2013. Effect of okadaic acid on carpet shell clam (*Ruditapes decussatus*) haemocytes by in vitro exposure and harmful algal bloom simulation assays. *Cell Biology and Toxicology*, **29**: 189–197.

Pratt, D.M. 1966. Competition between *Skeletonema costatum* and *Olisthodiscus luteus* in Narragansett Bay and in culture. *Limnology and Oceanography*, **11**: 447–455.

Prepas, E.E., B.G. Kotak, L.M. Campbell, J.C. Evans, S.E. Hrudey, and C.F.B. Holmes. 1997. Accumulation and elimination of cyanobacterial hepatotoxins by the freshwater clam *Anodonta grandis simpsoniana*. *Canadian Journal of Fisheries and Aquatic Sciences*, **54**: 41–46.

Preston, N.P., M.A. Burford, and D.J. Stenzel. 1998. Effects of *Trichodesmium* blooms on penaeid prawn larvae. *Marine Biology*, **131**: 671–679.

Prieto, A., A. Campos, A. Cameán, and V. Vasconcelos. 2011. Effects on growth and oxidative stress status of rice plants (*Oryza sativa*) exposed to two extracts of toxin-producing cyanobacteria (*Aphanizomenon ovalisporum* and *Microcystis aeruginosa*). *Ecotoxicology and Environmental Safety*, **74**: 1973–1980.

Priyadarshani, I., and B. Rath. 2012. Bioactive compounds from microalgae and cyanobacteria: utility and applications. *International Journal of Pharmaceutical Sciences and Research*. Available at: http://ijpsr.com/bft-article/bioactive-compounds-from-microalgae-and-cyanobacteria-utility-and-applications/?view=fulltext.

Prince, E.K., F. Irmer, and G. Pohnert. 2013. Domoic acid improves the competitive ability of *Pseudo-nitzschia delicatissima* against the diatom *Skeletonema marinoi*. *Marine Drugs*, **11**: 2398–2412.

Prince, E.K., T.L. Myers, and J. Kubanek. 2008. Effects of harmful algal blooms on competitors: allelopathic mechanisms of the red tide dinoflagellate *Karenia brevis*. *Limnology and Oceanography*, **53**: 531–541.

Prince, E.K., K.L. Poulson, T.L. Myers, R.D. Sieg, and J. Kubanek. 2010. Characterization of allelopathic compounds from the red tide dinoflagellate *Karenia brevis*. *Harmful Algae*, **10**: 39–48.

Provan, J.D., N.P. Todd, G.E. Beatty, and C.A. Maggs. 2007. Tracking biological invasions in space and time: elucidating the invasive history of the green alga *Codium fragile* using old DNA. *Diversity and Distributions*, **14**: 343–354.

Pryor, D., E. Saarman, D. Murray, and W. Prell. 2007. Nitrogen Loading from Wastewater Treatment Plants to Upper Narragansett Bay. Narragansett Bay Estuary Program, Brown University, Providence.

Puerto, M., A. Jos, S. Pichardo, R. Moyano, A. Blanco, and A.M. Cameán. 2012. Acute exposure to pure cylindrospermopsin results in oxidative stress and pathological alterations in tilapia (*Oreochromis niloticus*). *Environmental Toxicology*, **29**: 371–385.

Puschner, B., F.D. Galey, B. Johnson, C.W. Dickie, M. Vondy, T. Francis, and D.M. Hostege. 1998. Blue-green algae toxicosis in cattle. *Journal of the American Veterinary Medical Association*, **213**: 1605–1607.

Quevedo, M., R. González-Quirós, and R. Anadón. 1999. Evidence of heavy predation by *Noctiluca scintillans* on *Acartia clausi* (Copepoda) eggs off the central Cantabrian coast (NW Spain). *Oceanologica Acta*, **22**: 127–131.

Raffaelli, D., S. Hull, and H. Milne. 1989. Long-term changes in nutrients, weed mats and shorebirds in an estuarine system. *Cahiers de Biologie Marine*, **30**: 259–270.

Raffaelli, D., J. Limia, S. Hull, and S. Pont. 1991. Interactions between the amphipod *Corophium volutator* and macroalgal mats on estuarine mudflats. *Journal of the Marine Biological Association UK*, **71**: 899–908.

Ramamurthy, V., S. Raveendran, and H.V.A. Kumars. 2014. Effect of *Lyngbya majuscula* on the immunity and survival of *Aeromonas hydrophylia* infected *Mugil cephalus*. *International Journal of Innovative Research in Science, Engineering, and Technology (IJIRSET)*, **3**: 9782–9789.

Rangel, L., K.A. Ger, L.H. Sampaio da Silva, and M. Lurling. 2016. Toxicity overrides morphology on *Cylindrospermopsis raciborskii* grazing resistance to the calanoid copepod *Eudiaptomus gracile*. *Microbial Ecology* **71**: 835–844.

Rasher, D.B., and M.E. Hay. 2014. Competition induces allelopathy but suppresses growth and anti-herbivore defence in a chemically rich seaweed. *Proceedings of the Royal Society B: Biological Sciences*, **281**: 20132615.

Rastogi, R.P., R.P Sinha, and A. Incharoensakdi. 2014. The cyanotoxin microcystins: current overview. *Reviews in Environmental Science and Bio/Technology*, **13**: 215–249.

Red-Tide. 2011. *Prorocentrum micans* Ehrenberg 1833. Available at: http://www.red-tide.org/new_site/pmin.htm.

Reiffenstein, R.I., W.C. Hulbert, and S.H. Roth. 1992. Toxicology of hydrogen sulfide. *Annual Review of Pharmacology and Toxicology*, **32**: 109–134.

Reinikainen, M., J. Hietala, and M. Walls. 1999. Reproductive allocation in *Daphnia* exposed to toxic cyanobacteria. *Journal of Plankton Research*, **21**: 1553–1564.

Rensel, J.E.J., N. Haigh, and T.J. Tynan. 2010. Fraser river sockeye salmon marine survival decline and harmful blooms of *Heterosigma akashiwo*. *Harmful Algae*, **10**: 98–115.

Rhodes, L. 2011. Worldwide occurrence of the toxic dinoflagellate genus *Ostreopsis* Schmidt. *Toxicon*, **57**: 400–407.

Ribalet, F., J.A. Berges, A. Ianora, and R. Casotti. 2007. Growth inhibition of cultured marine phytoplankton by algal-derived polyunsaturated aldehydes. *Aquatic Toxicology*, **85**: 219–227.

Ribalet, F., L. Intertaglia, P. Lebaron, and R. Casotti. 2008. Differential effect of three polyunsaturated aldehydes on marine bacterial isolates. *Aquatic Toxicology*, **86**: 249–255.

Richardson, R.W. 2004. Florida Bay microalgae blooms: physiological characteristics and competitive strategies of bloom forming cyanobacteria and diatoms of Florida Bay. PhD thesis, University of South Florida.

Richlen, M.L., M.L. Parsons, and D.M. Anderson. 2012. Ecology and impacts of ciguatera on coral reef ecosystems. In: *Advances in Environmental Research*, Vol. 26. J.A. Daniels (Ed.). Nova Science Publishers, Hauppauge: p. 41–76.

Roberts, B.S., G.E. Henderson, and R.A. Medlyn. 1979. The effect of *Gymnodinium breve* toxin(s) on selected mollusks and crustaceans. In: *Toxic Dinoflagellate Blooms*. D.L. Taylor and H.H. Seliger (Eds.). Elsevier, New York: p. 419–424.

Robineau, B., J.A. Gagné, L. Fortier, and A.D. Cembella. 1991a. Potential impact of a toxic dinoflagellate (*Alexandrium excavatum*) bloom on survival of fish and crustacean larvae. *Marine Biology*, **108**: 293–301.

Robineau, B., L. Fortier, J.A. Gagné, and A.D. Cembella. 1991b. Comparison of the response of five larval fish species to the toxic dinoflagellate *Alexandrium excavatum* (Braarud) Balech. *Journal of Experimental Marine Biology and Ecology*, **152**: 225–242.

Robbins, H.M., V.M. Bricelj, and J.E. Ward. 2010. In vivo effects of brown tide on the feeding function of the gill of the northern quahog *Mercenaria mercenaria* (Bivalvia: Veneridae). *Biology Bulletin*, **219**: 61–71.

Rogers, E.D. 2010. Sublethal toxicity of Microcystis and microcystin-LR in fish. PhD dissertation, University of Tennessee. Available at: http://trace.tennessee.edu/utk_graddiss/909.

Rohrlack, T., K. Christoffersen, E. Dittmann, I. Nogueira, V. Vasconcelos, and T. Börner. 2005. Ingestion of microcystins by *Daphnia*: intestinal uptake and toxic effects. *Limnology and Oceanography*, **50**: 440–448.

Rohrlack, T., E. Dittmann, T. Börner, and K. Christoffersen. 2001. Effects of cell-bound microcystins on survival and feeding of *Daphnia*

spp. *Applied and Environmental Microbiology*, **67**: 3523–3529.

Rohrlack, T., E. Dittmann, M. Henning, T. Börner, and J.-G. Kohl. 1999. Role of microcystins in poisoning and food ingestion inhibition of *Daphnia galeata* caused by the cyanobacterium *Microcystis aeruginosa*. *Applied and Environmental Microbiology*, **65**: 737–739.

Romanowska-Duda, Z., and M. Tarczynska. 2002. The influence of microcystin-LR and hepatotoxic cyanobacterial extract on the water plant *Spirodela oligorrhiza*. *Environmental Toxicology*, **17**: 434–440.

Romero-Geraldo, R. de J., and N.Y. Hernandez-Saavedra. 2014. Stress gene expression in *Crassostrea gigas* (Thunberg, 1793) in response to experimental exposure to the toxic dinoflagellate *Prorocentrum lima* (Ehrenberg) Dodge, 1975. *Aquaculture Research*, **45**: 1512–1522.

Rose, E.T. 1953. Toxic algae in Iowa lakes. *Proceedings of the Iowa Academy of Science*, **60**: 738–745.

Rosetta, C.H., and G.B. McManus. 2003. Feeding by ciliates on two harmful algal bloom species, *Prymnesium parvum* and *Prorocentrum minimum*. *Harmful Algae*, **2**: 109–126.

Rothhaupt, K.O. 1991. The influence of toxic and filamentous blue-green algae on feeding and population growth of the rotifer *Brachionus rubens*. *Internationale Revue der gesamten Hydrobiologie und Hydrographie*, **76**: 67–72.

Rymuszka, A., and A. Sierosławska. 2013. Cytotoxic and immunotoxic effects of the mixture containing cyanotoxins on carp cells following in vitro exposure. *Central European Journal of Immunology*, **38**: 159–163.

Ryther, J.H. 1954. The ecology of phytoplankton blooms in Moriches Bay and Great South Bay, Long Island, New York. *Biology Bulletin*, **106**: 198–209.

Ryther, J.H. 1989. Historical perspective of phytoplankton blooms on long island and the green tides of the 1950s. In: *Novel Phytoplankton Blooms*. E. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 375–381.

Rzymski, P., B. Poniedziałek, M. Kokociński, T. Jurczak, D. Lipski, and K. Wiktorowicz. 2014. Inter-specific allelopathy in cyanobacteria: Cylindrospermopsin and *Cylindrospermopsis raciborskii* effect on the growth and metabolism of *Microcystis aeruginosa*. *Harmful Algae*, **35**: 1–8.

Saha, M., M. Rempt, S.B. Stratil, M. Wahl, and F. Weinberger. 2014. Defence chemistry modulation by light and temperature shifts and the resulting effects on associated epibacteria of *Fucus vesiculosus*. *PLoS ONE*, **9**(10): e105333. doi: 10.1371/journal.pone.0105333.

Saker, M.L., and G.K. Eaglesham. 1999. The accumulation of cylindrospermopsin from the cyanobacterium *Cylindrospermopsis raciborskii* in tissues of the redclaw crayfish *Cherax quadricarinatus*. *Toxicon*, **37**: 1065–1077.

Saker, M.L., J.S. Metcalf, G.A. Codd, and V.M. Vasconcelos. 2004. Accumulation and depuration of the cyanobacterial toxin cylindrospermopsin in the freshwater mussel *Anodonta cygnea*. *Toxicon*, **43**: 185–194.

Saker, M.L., A.D. Thomas, and J.H. Norton. 1999. Cattle mortality attributed to the toxic cyanobacterium *Cylindrospermopsis raciborskii* in an Outback region of North Queensland. *Environmental Toxicology*, **14**: 179–182.

Salierno, J.D. 2005. Harmful algal bloom stressors alter behavior and brain activity in the killifish, *Fundulus heteroclitus*. University of Maryland, College Park.

Samson, J.C., S.E. Shumway, and J.S. Weis. 2008. Effects of the toxic dinoflagellate, *Alexandrium fundyense* on three species of larval fish: a food-chain approach. *Journal of Fish Biology*, **72**: 168–188.

Saqrane, S., I. El Ghazali, Y. Ouahid, M. El Hassni, I. El Hadrami, L. Bouarab, F.F. del Campo, B. Oudra, and V. Vasconcelos. 2007. Phytotoxic effects of cyanobacteria extract on the aquatic plant *Lemna gibba*: microcystin accumulation, detoxication and oxidative stress induction. *Aquatic Toxicology*, **83**: 284–294.

Schaefer, A.L., E.P. Greenberg, C.M. Oliver, Y. Oda, J.J. Huang, G. Bittan-Banin, C.M. Peres, S. Schmidt, K. Juhaszova, J.R. Sufrin, and C.S. Harwood. 2008. A new class of homoserine lactone quorum-sensing signals. *Nature*, **454**: 595–599.

Scheffer, M.S., S.H. Hosper, M.L. Meijer, B. Moss, and E. Jeppesen. 1993. Alternative equilibria in shallow lakes. *Trends in Ecology and Evolution*, **8**: 275–279.

Scholin, C.A., F. Gulland, G.J. Doucette, S. Benson, M. Busman, F.P. Chavez, J. Cordaro, R. DeLong, A. De Vogelaere, J. Harvey, M. Haulena, K. Lefebvre, T. Lipscomb, S. Loscutoff, L.J. Lowenstine, R. Marin III, P.E. Miller, W.A. McLellan, P.D.R. Moeller, C.L. Powell, T. Rowleskk, P. Silvagni, M. Silver, T. Spraker, V. Trainer, and F.M. Van Dolah. 2000. Mortality of sea lions along the central California coast linked to a toxic diatom bloom. *Nature*, **403**: 80–84.

Schmidt, R.J., and A.R. Loeblich III. 1979. Distribution of paralytic shellfish poison among

Pyrrhophyta. *Journal of the Marine Biological Association UK*, **59**: 479–487.

Schmidt, K., and S.H. Jónasdóttir. 1997. Nutritional quality of two cyanobacteria: how rich is 'poor' food? *Marine Ecology Progress Series*, **151**: 1–10.

Schöne, K., S. Jähnichen, T. Ihle, F. Ludwig, and J. Benndorf. 2010. Arriving in better shape: Benthic *Microcystis* as inoculum for pelagic growth. *Harmful Algae*, **9**: 494–503.

Schwimmer, D., and M. Schwimmer. 1968. Medical aspects of phycology. In: *Algae, Man and the Environment*. D. Jackson (Ed.). Syracuse University Press, Syracuse: p. 279–358.

Seifert, M. 2007. The ecological effects of the cyanobacterial toxin cylindrospermopsin. PhD thesis, School of Medicine, University of Queensland. Available at: http://espace.library.uq.edu.au/view/UQ:158799.

Sekula-Wood, E., A. Schnetzer, C.R. Benitez-Nelson, C. Anderson, W.M. Berelson, M.A. Brzezinski, J.M. Burns, D.A. Caron, I. Cetinic, J.L. Ferry, E. Fitzpatrick, B.H. Jones, P.E. Miller, S.L. Morton, R.A. Schaffner, D.A. Siegel, and R. Thunell. 2009. Rapid downward transport of the neurotoxin domoic acid in coastal waters. *Nature Geoscience*, **2**: 272–275.

Selander, E., P. Thor, G. Toth, and H. Pavia. 2006. Copepods induce paralytic shellfish toxin production in marine dinoflagellates. *Proceedings of the Royal Society B: Biological Sciences*, **273**: 1673–1680.

Sellner, K.G., M.M. Olson, and K. Olli. 1996. Copepod interactions with toxic and nontoxic cyanobacteria from the Gulf of Finland. *Phycologia*, **35**: 177–182.

Seong, K.A., H.J. Jeong, S. Kim, G.H. Kim, and J.H. Kang. 2006. Bacterivory by co-occurring red-tide algae, heterotrophic nanoflagellates, and ciliates on marine bacteria. *Marine Ecology Progress Series*, **322**: 85–97.

Seyedsayamdost, M.R., R.J. Case, R. Kolter, and J. Clardy. 2011a. The Jekyll-and-Hyde chemistry of *Phaeobacter gallaeciensis*. *Nature Chemistry*, **3**: 331–335.

Seyedsayamdost, M.R. G. Carr, R. Kolter, and J. Clardy. 2011b. Roseobacticides: small molecule modulators of an algal-bacterial symbiosis. *Journal of the American Chemical Society*, **133**: 18343–18349.

Sheng, J., E. Malkiel, J. Katz, J.E. Adolf, and A.R. Place. 2010. A dinoflagellate exploits toxins to immobilize prey prior to ingestion. *Proceedings of the National Academy of Sciences USA*, **107**: 282–287.

Shimada, H., I.V. Motylkova, T. Alexandrovna, K. Mikami, and M. Kimura. 2011. Toxin profile of *Alexandrium tamarense* (Dinophyceae) from Hokkaido, northern Japan and southern Sakhalin, eastern Russia. *Plankton and Benthos Research*, **6**: 35–41.

Sharp, K., K.E. Arthur, L. Gu, C. Ross, G. Harrison, S.P. Gunasekera, T. Meickle, S. Matthew, H. Luesch, R.W. Thacker, D.H. Sherman, and V.J. Paul. 2009. Phylogenetic and chemical diversity of three chemotypes of bloom-forming *Lyngbya* species (Cyanobacteria: Oscillatoriales) from reefs of southeastern Florida. *Applied Environmental Microbiology*, **75**: 2879–2888.

Shears, N.T., and P.M. Ross. 2010. Toxic cascades: multiple anthropogenic stressors have complex and unanticipated interactive effects on temperate reefs. *Ecology Letters*, **13**: 1149–1159.

Shimps, E.L., J.A. Rice, and J.A. Osborne. 2005. Hypoxia tolerance in two juvenile estuary-dependent fishes. *Journal of Experimental Marine Biology and Ecology*, **325**: 146–162.

Shin, K., M.C. Jang, P.K. Jang, S.J. Ju, T.K. Lee, and M. Chang. 2003. Influence of food quality on egg production and viability of the marine planktonic copepod *Acartia omorii*. *Progress in Oceanography*, **57**: 265–277.

Shumway, S.E. 1990. A review of the effects of algal blooms on shellfish and aquaculture. *Journal of the World Aquaculture Society*, **21**: 65–104.

Shumway, S.E., S.M. Allen, and P.D. Boersma. 2003. Marine birds and harmful algal blooms: sporadic victims or under-reported events? *Harmful Algae*, **2**: 1–17.

Shumway, S.E., J.M. Burkholder, and J. Springer. 2006. Effects of the estuarine dinoflagellate *Pfiesteria shumwayae* (Dinophyceae) on survival and grazing activity of several shellfish species. *Harmful Algae*, **5**: 442–458.

Shumway, S.E., and T.L. Cucci. 1987. The effects of the toxic dinoflagellate *Protogonyaulax tamarensis* on the feeding and behavior of bivalve molluscs. *Aquatic Toxicology*, **10**: 9–27.

Shumway, S.E., T.L. Cucci, L. Gainey, and C.M. Yentsch. 1985. A preliminary study of the behavioral and physiological effects of *Gonyaulax tamarensis* on bivalve molluscs. In: *Toxic Dinoflagellates*. D.M. Anderson, A.W. White, and D.G. Baden (Eds.). Elsevier, New York: p. 389–394.

Shumway, S.E., F.C. Pierce, and K. Knowlton. 1987. The effects of *Protogonyaulax tamarensis* on byssus production in *Mytilus edulis* L., *Modiolus modiolus* Linnaeus, 1758 and *Geukensia demissa*

Dillwyn. *Comparative Biochemistry and Physiology A*, **87**: 1021–1023.

Shumway, S.E., T.M. Scott, and J.M. Shick. 1983. The effects of anoxia and hydrogen sulphide on survival, activity and metabolic rate in the coot clam, *Mulinia lateralis* (Say). *Journal of Experimental Marine Biology and Ecology*, **71**: 135–146.

Sieburth, J.M. 1961. Antibiotic properties of acrylic acid, a factor in the gastrointestinal antibiosis of polar marine animals. *Journal of Bacteriology*, **82**: 72–79.

Sieburth, J.M., and J.T. Conover. 1965. *Sargassum* tannin, an antibiotic which retards fouling. *Nature*, **208**: 52–53.

Sierra-Beltran, A., M. Palafox-Uribe, J. Grajales-Montiel, A. Cruz-Villacorta, and J.L. Ochoa. 1997. Sea bird mortality at Cabo San Lucas, Mexico: evidence that toxic diatom blooms are spreading. *Toxicon*, **35**: 447–453.

Sievers, A.M. 1969. Comparative toxicity of *Gonyaulax monilata* and *Gymnodinium breve* to annelids, crustaceans, molluscs, and fish. *Journal of Protozoology*, **16**: 401–404.

Simon, J.L., and D.M. Dauer. 1972. A quantitative evaluation of red tide induced mortalities of benthic invertebrates in Tampa Bay, Florida. *Environmental Letters*, **3**: 229–234.

Sims, D. 2013. Troubled Waters. Institute for the Study of Earth, Oceans, and Space. University of New Hampshire, Hanover. Available at: http://www.eos.unh.edu/Spheres_1113/moore.shtml.

Singh, D.P., M.B. Tyagi, A. Kumar, J.K. Thakur, and A. Kumar. 2001. Antialgal activity of a hepatoxin-producing cyanobacterim *Microcystis aeruginosa*. *World Journal of Microbiology & Biotechnology*, **17**: 15–22.

Skovgaard, A. 2000. A phagotrophically derivable growth factor in the plastidic dinoflagellate *Gyrodinium resplendens* (Dinophyceae). *Journal of Phycology*, **36**: 1068–1078.

Skovgaard, A., and P.J. Hansen. 2003. Food uptake in the harmful alga *Prymnesium parvum* mediated by excreted toxins. *Limnology and Oceanography*, **48**: 1161–1166.

Smayda, T.J. 1991. Global epidemic of noxious phytoplankton blooms and food chain consequences. In: *Food Chains, Yields, Models, and Management of Large Marine Ecosystems*. K. Sherman, L.M. Alexander, and B.D. Gold (Eds.). Westview Press, Boulder: p. 275–307.

Smayda, T.J. 1997. Harmful algal blooms: their ecophysiology and general relevance to phytoplankton blooms in the sea. *Limnology and Oceanography*, **42**: 1137–1153.

Smayda, T.J. 2008. Complexity in the eutrophication-harmful algal bloom relationship, with comment on the importance of grazing. *Harmful Algae*, **8**: 140–151.

Smayda, T.J., and T.A. Villareal. 1989. The 1985 "brown-tide" and the open phytoplankton niche in Narragansett Bay during the summer. In: *Novel Phytoplankton Blooms*. E.M. Cosper, V.M. Bricelj, and E.J. Carpenter (Eds.). Springer, Berlin: p. 159–187.

Smith, A.D.M., C.J. Brown, C.M. Bulman, E.A. Fulton, P. Johnson, I.C. Kaplan, H. Lozano-Montes, S. Mackinson, M. Marzloff, L.J. Shannon, Y.-J. Shin, and J. Tam. 2011. Impacts of fishing low-trophic level species on marine ecosystems. *Science*, **333**: 1147–1150.

Smith, G.B. 1975. The 1971 red tide and its impact on certain reef communities in the mid-eastern Gulf of Mexico. *Environmental Letters*, **9**: 141–152.

Smith, J.K., D.J. Lonsdale, C.J. Gobler, and D.A. Caron. 2008. Feeding behavior and development of *Acartia tonsa* nauplii on the brown tide alga *Aureococcus anophagefferens*. *Journal of Plankton Research*, **30**: 937–950.

Smith, J.L., K.L. Schulz, P.V. Zimba, and G.L. Boyer. 2010. Possible mechanism for the foodweb transfer of covalently bound microcystins. *Ecotoxicology and Environmental Safety*, **73**: 757–761.

Smithsonian Institution. 2011. *Prorocentrum micans* Ehrenberg 1833. Available at: http://botany.si.edu/references/dinoflag/Taxa/Pmicans.htm.

Solan, M., B.J. Cardinale, A.L. Downing, K.A.M. Engelhardt, J.L. Ruesink, and D.S. Srivastava. 2004. Extinction and ecosystem function in the marine benthos. *Science*, **306**: 1177–1180.

Sopanen, S., M. Koski, P. Uronen, P. Kuuppo, S. Lehtinen, C. Legrand, and T. Tamminen. 2008. *Prymnesium parvum* exotoxins affect the grazing and viability of the calanoid copepod *Eurytemora affinis*. *Marine Ecology Progress Series*, **361**: 191–202.

Sopanen, S., M. Koski, P. Kuuppo, P. Uronen, C. Legrand, and T. Tamminen. 2006. Toxic haptophyte *Prymnesium parvum* affects grazing, survival, egestion and egg production of the calanoid copepods *Eurytemora affinis* and *Acartia bifilosa*. *Marine Ecology Progress Series*, **327**: 223–232.

Sotton, B., I. Domaizon, O. Anneville, F. Cattane, and J. Guillard. 2015. Nodularin and cylindrospermopsin: a review of their effects on

fish. *Reviews in Fish Biology and Fisheries*, **25**: 1–19.

Springer, J., S.E. Shumway, J.M. Burkholder, H.B. Glasgow. 2002. Interactions between the toxic estuarine dinoflagellate, *Pfiesteria piscicida* and two species of bivalve molluscs. *Marine Ecology Progress Series*, **245**: 1–10.

Stachowicz, J.J., M. Graham, M.E.S. Bracken, and A.I. Szoboszlai. 2007. Diversity enhances cover and stability of seaweed assemblages: the role of heterogeneity and time. *Ecology*, **89**: 3008–3019.

Stangenberg, M. 1968. Toxic effects of *Microcystis aeruginosa* KG extract on *Daphnia longispina* O.F. Muller and *Eucypris virens* Jurine. *Hydrobiologia*, **32**: 81–87.

Starkweather, P.L., and P.E. Kellar. 1987. Combined influences of particulate and dissolved factors in the toxicity of *Microcystis aeruginosa* (NRC-SS-17) to the rotifer *Brachionus calyciflorus*. *Hydrobiologia*, **147**: 375–378.

Stein, J.R., and C.A. Borden. 1984. Causative and beneficial algae in human disease conditions: a review. *Phycologia*, **23**: 485–501.

Steinke, M., J. Stefels, and E. Stamhuis. 2006. Dimethyl sulfide triggers search behavior in copepods. *Limnology and Oceanography*, **51**: 1925–1930.

Stoecker, D., R.R.L. Guillard, and R.M. Kavee. 1981. Selective predation by *Favella ehrenbergii* (Tintinnia) on and among dinoflagellates. *Biology Bulletin*, **160**: 136–145.

Stoecker, D.K., M.W. Parrow, J.M. Burkholder, and H.B. Glasgow. 2002. Grazing by microzooplankton on *Pfiesteria piscicida* cultures with different histories of toxicity. *Aquatic Microbial Ecology*, 28: 79–85.

Stoecker, D.K., J.E. Adolf, A.R. Place, P.M. Glibert, and D.W. Meritt. 2008. Effects of the dinoflagellates *Karlodinium veneficum* and *Prorocentrum minimum* on early life history stages of the eastern oyster (*Crassostrea virginica*). *Marine Biology*, **1154**: 81–90.

Stouder, D.J., and M.L. McMullin. 2006. Disturbance. In: *The Ecology of Marine Fishes: California and Adjacent Waters*. L.G. Allen and M.H. Horn (Eds.). University of California Press, Berkeley.

Stumm, W., and J.J. Morgan. 1996. *Aquatic Chemistry: Chemical Equilibria and Rates in Natural Waters*, 3rd ed. Wiley Inter-Science, New York.

Sudek, S., M.G. Haygood, D.T. Youssef, and E.W. Schmidt. 2006. Structure of trichamide, a cyclic peptide from the bloom-forming cyanobacterium *Trichodesmium erythraeum,* predicted from the genome sequence. *Applied and Environmental Microbiology*, **72**: 4382–4387.

Suikkanen, S., G.O. Fistarol, and E. Granéli. 2004. Allelopathic effects of the Baltic cyanobacteria *Nodularia spumigena, Aphanizomenon flos-aquae,* and *Anabaena lemmermannii* on algal monocultures. *Journal of Experimental Marine Biology and Ecology*, **308**: 85–101.

Sukenik, A., R. Eshkol, A. Livne, O. Hadas, M. Rom, D. Tchernov, A. Vardi, and A. Kaplan. 2002. Inhibition of growth and photosynthesis of the dinoflagellate *Peridinium gatunense* by *Microcystis* sp. (cyanobacteria): a novel allelopathic mechanism. *Limnology and Oceanography*, **47**: 1656–1663.

Summerson, H.C., and C.H. Peterson. 1990. Recruitment failure of the bay scallop, *Argopecten irradians concentricus,* during the first red tide, *Ptychodiscus brevis,* outbreak recorded in North Carolina. *Estuaries*, **13**: 322–331.

Sunda, W.G., E. Granéli, and C.J. Gobler. 2006. Positive feedback and the development and persistence of ecosystem disruptive algal blooms. *Journal of Phycology*, **42**: 963–974.

Svensen, C., E. Strogyloudi, C. Wexels Riser, J. Dahlmann, C. Legrand, P. Wassmann, E. Granéli, and K. Pagou. 2005. Reduction of cyanobacterial toxins through coprophagy in *Mytilus edulis*. *Harmful Algae*, **4**: 329–333.

Sychrová, E., T. Štěpánková, K. Nováková, L. Bláha, J.P. Giesy, and K. Hilscherová. 2012. Estrogenic activity in extracts and exudates of cyanobacteria and green algae. *Environment International*, **39**: 134–140.

Sykes, P.F., and M.E Huntley. 1987. Acute physiological reactions of *Calanus pacificus* to selected dinoflagellates: direct observations. *Marine Biology*, **94**: 19–24.

Tan, L.T. 2007. Bioactive natural products from marine cyanobacteria for drug discovery. *Phytochemistry*, **68**: 954–979.

Tang, Y.Z., and C.J. Gobler. 2009a. Characterization of the toxicity of *Cochlodinium polykrikoides* isolates from northeast U.S. estuaries to finfish and shellfish. *Harmful Algae*, **8**: 454–462.

Tang, Y.Z., and C.J. Gobler. 2009b. *Cochlodinium polykrikoides* blooms and clonal isolates from the northwest Atlantic coast cause rapid mortality in larvae of multiple bivalve species. *Marine Biology*, **156**: 2601–2611.

Tang, Y.Z., and C.J. Gobler. 2010. Allelopathic effects of *Cochlodinium polykrikoides* isolates and blooms from the estuaries of Long Island, New York, on co-occurring phytoplankton. *Marine Ecology Progress Series*, **406**: 19–31.

Tang, Y.Z., and C.J. Gobler. 2012. Lethal effects of northwest Atlantic Ocean isolates of the dinoflagellate, *Scrippsiella trochoidea,* on eastern oyster (*Crassostrea virginica*) and northern quahog (*Mercenaria mercenaria*) larvae. *Marine Biology*, **159**: 199–210.

Taylor, M.S., W. Stahl-Timmins, C.H. Redshaw, and N.J. Osborne. 2014. Toxic alkaloids in *Lyngbya majuscula* and related tropical marine cyanobacteria. *Harmful Algae*, **31**: 1–8.

Teegarden, G.J., and A.D. Cembella. 1996. Grazing of toxic dinoflagellates, *Alexandrium* spp., by adult copepods of coastal Maine: implications for the fate of paralytic shellfish toxins in marine food webs. *Journal of Experimental Marine Biology and Ecology*, **196**: 145–176.

Tencalla, F.G., D.R. Dietrich, and C. Schlatter. 1994. Toxicity of *Microcystis aeruginosa* peptide toxin to yearling rainbow trout (*Oncorhynchus mykiss*). *Aquatic Toxicology*, **30**: 215–224.

Thacker, R.W., D.G. Nagle, and V.J. Paul. 2007. Effects of repeated exposures to marine cyanobacterial secondary metabolites on feeding by juvenile rabbitfish and parrotfish. *Marine Ecology Progress Series*, **147**: 21–29.

Thomsen, M.S., and K. McGlathery. 2006. Effects of accumulations of sediments and drift algae on recruitment of sessile organisms associated with oyster reefs. *Journal of Experimental Marine Biology and Ecology*, **328**: 22–34.

Tidgewell, K., B.R. Clark, and W.H. Gerwick. 2010. The natural products chemistry of cyanobacteria. In: *Comprehensive Natural Products II Chemistry and Biology*, Vol. 2. L. Mander and H.W. Lui (Eds.). Elsevier, Oxford: p. 141–188.

Tilling, K.A. 2007. The effects of the cyanobacterium *Lyngbya majuscula* and artificial shading on the seagrass *Halodule wrightii*. Florida Atlantic University, Jupiter. Available at: http://fau.digital.flvc.org/islandora/object/fau%3A1329.

Tillmann, U. 2004. Interactions between planktonic microalgae and protozoan grazers. *Journal of Eukaryotic Microbiology*, **51**: 156–168.

Tillmann, U., and P.J. Hansen. 2009. Allelopathic effects of *Alexandrium tamarense* on other algae: evidence from mixed growth experiments. *Aquatic Microbial Ecology*, **57**: 101–112.

Tomascik, T., and F. Sander. 1985. Effects of eutrophication on reef-building corals. I. Growth rate of the reef-building coral *Montastrea annularis*. *Marine Biology*, **87**: 143–155.

Tomascik, T., and F. Sander. 1987. Effects of eutrophication on reef building corals: reproduction of the reef-building coral *Porites porites*. *Marine Biology*, **94**: 77–94.

Tracey, G.A. 1988. Feeding reduction, reproductive failure, and mortality in the mussel, *Mytilus edulis,* during the 1985 "brown tide" in Narragansett Bay, Rhode Island. *Marine Ecology Progress Series*, **50**: 73–81.

Traoré, A., M. Bonini, S.D. Dano, and E.E. Creppy. 1999. Synergistic effects of some metals contaminating mussels on the cytotoxicity of the marine toxin okadaic acid. *Archives of Toxicology*, **73**: 289–295.

Tobiesen, A. 1991. Growth rates of *Heterophrys marina* (Heliozoa) on *Chrysochromulina polylepis* (Prymnesiophyceae). *Ophelia*, **33**: 205–212.

Tomas, C.R., and E.E. Deacon. 1981. The influence of grazing by two *Acartia* species on *Olisthodiscus luteus* Carter. *P.S.Z.N.I. Marine Ecology*, **2**: 215–223.

Trainer, V.L., S.S. Bates, W.P. Cochlan, and C.G. Trick. 2012. *Pseudo-nitzschia* physiological ecology, phylogeny, toxicity, monitoring and impacts on ecosystem health. *Harmful Algae*, **14**: 271–300.

Tryphonas, L., J. Truelove, E. Nera, and F. Iverson. 1990. Acute neurotoxicity of domoic acid in the rat. *Toxicologic Pathology*, **18**: 1–9.

Turner, J.T. 2006. Harmful algae interactions with marine planktonic grazers. In: *Ecology of Harmful Algae*. E. Granéli and J. Turner (Eds.). Springer, New York: p. 259–270.

Turner, J.T., and E. Granéli. 2006. "Top-down" predation control on marine harmful algae. In: *Ecology of Harmful Algae*. E. Granéli and J. Turner (Eds.). Springer, New York: p. 355–366.

Turner, J.T., J.A. Lincoln, and A.D. Cembella. 1998. Effects of toxic and nontoxic dinoflagellates on copepod grazing, egg production and egg hatching success. In: *Harmful Algae*. B. Reguera, J. Blanco, M.L. Fernández, and T. Wyatt (Eds.). Xunta de Galicia, IOC, Paris: p. 379–381.

Turriff, N., J.A. Runge, and A.D. Cembella. 1995. Toxin accumulation and feeding behaviour of the planktonic copepod *Calanus finmarchicus* exposed to the red-tide dinoflagellate *Alexandrium excavatum*. *Marine Biology*, **123**: 55–64.

Twiner, M.J., L.J. Flewelling, S.E. Fire, S.R. Bowen-Stevens, J.K. Gay, C.K. Johnson, J.H. Landsberg, T.A. Leighfield, B. Mase-Guthrie, L. Schwake, F.M. Van Dolah, Z. Wang, and T.K. Rowles. 2012. Comparative analysis of three brevetoxin-associated bottlenose dolphin (*Tursiops truncatus*) mortality events in the Florida Panhandle region (USA). *PLoS ONE*, **7**(8): e42974. doi: 10l1371/journal.pone.0042974.

Uchida, T. 1977. Excretion of a diatom-inhibitory substance by *Prorocentrum micans* Ehrenberg. *Japanese Journal of Ecology*, **27**: 1–4.

Ungerer, J.R., and P. Thomas. 1996. Role of very low density lipoproteins in the accumulation of o,p'-DDT in fish ovaries during gonadal recrudescence. *Aquatic Toxicology*, **35**: 183–195.

Ureña-Boeck, F. 2008. *Prymnesium parvum* effects on green algae, cladocerans and fish in field and laboratory studies. Thesis, Department of Environmental Science, Baylor University.

Uronen, P., P. Kuuppo, C. Legrand, and T. Tamminen. 2007. Allelopathic effects of toxic haptophyte *Prymnesium parvum* lead to release of dissolved organic carbon and increase in bacterial biomass. *Microbial Ecology*, **54**: 183–193.

Usup, G., A. Ahmad, K. Matsuoka, P.T. Lim, and C. P. Leaw. 2012. Biology, ecology and bloom dynamics of the toxic marine dinoflagellate *Pyrodinium bahamense*. *Harmful Algae*, **14**: 301–312.

Ulitzer, S., and M. Shilo. 1966. Mode of action of *Prymnesium parvum* ichthyotoxin. *Journal of Protozoology*, **13**: 332–336.

Uye, S., and K. Takamatsu. 1990. Feeding interactions between planktonic copepods and red-tide flagellates from Japanese coastal waters. *Marine Ecology Progress Series*, **59**: 97–107.

Valiela, I., K. Foreman, M. LaMontagne, D. Hersh, J. Costa, B. Peckol, B. DeMeo-Anderson, C. D'Avanzo, M. Babione, C. Sham, J. Rawley, and K. Lajtha. 1992. Couplings of watersheds and coastal waters: sources and consequences of nutrient enrichment in Waquoit Bay, Massachusetts. *Estuaries*, **15**: 443–457.

Valiela, I. J. McClelland, J. Hauxwell, P.J. Behr, D. Hersh, and K. Foreman. 1997. Macroalgal blooms in shallow coastal estuaries: controls and ecophysiological and ecosystem consequences. *Limnology and Oceanography*, **42**: 1105–1118.

Viaroli, P., M. Bartoli, G. Giordani, M. Naldi, S. Orfanidis, and J.M. Zaldívar. 2008. Community shifts, alternative stable states, biogeochemical controls and feedbacks in eutrophic coastal lagoons: a brief overview. *Aquatic Conservation: Marine and Freshwater Ecosystems*, **18**: S105–S117.

Vanderploeg, H.A., J.R. Liebig, W.W. Carmichael, M.A. Agy, T.H. Johengen, G.L. Fahnenstiel, and T.F. Nalepa. 2001. Zebra mussel (*Dreissena polymorpha*) selective filtration promoted toxic *Microcystis* blooms in Saginaw Bay (Lake Huron) and Lake Erie. *Canadian Journal of Fisheries and Aquatic Sciences*, **58**: 1208–1221.

Van Alstyne, K.L. 1988. Herbivore grazing increases polyphenolic defenses in the intertidal brown alga *Fucus distichus*. *Ecology*, **69**: 655–663.

Van Alstyne, K.L. 2008. Ecological and physiological roles of dimethylsulfoniopropionate (DMSP) and its products in marine macroalgae. In: *Algal Chemical Ecology*. C.D. Amsler (Ed.). Springer, Berlin: p. 173–194.

Van Alstyne, K., K.J. Anderson, A.K. Winans, and S.A. Gifford. 2011. Dopamine release by the green alga *Ulvaria obscura* after simulated release by incoming tides. *Marine Biology*, **158**: 2087–2094.

Van Alstyne, K.L., E.L. Harvey, and M. Cataldo. 2014. Effects of dopamine, a compound released by the green tide macroalga *Ulvaria obscura,* on marine algae and invertebrate larvae and juveniles. *Phycologia*, **53**: 195–202.

Van Alstyne, K.L., and L.T. Houser. 2003. Dimethylsulfide release during macroinvertebrate grazing and its role as an activated chemical defense. *Marine Ecology Progress Series*, **250**: 175–181.

Van Alstyne, K.L., L. Koellermeier, and T. Nelson. 2007. Spatial variation in dimethylsulfoniopropionate (DMSP) production in *Ulva lactuca* (Chlorophyta) from the Northeast Pacific. *Marine Biology*, **150**: 1127–1135.

Van Alstyne, K.L., A.V. Nelson, J. Vyvyan, and D. Cancilla. 2006. Dopamine functions as an antiherbivore defense in the temperate green alga *Ulvaria obscura*. *Oecologia*, **148**: 304–311.

Van Alstyne, K.L., T. Nelson, and R.L. Ridgway. 2015. Environmental chemistry and chemical ecology of "green tide" seaweed blooms. *Integrative and Comparative Biology*, 1–15. doi: 10.1093/ich/icv035.

Van Alstyne, K.L., and V.J. Paul. 1989. The role of secondary metabolites in marine ecological interactions. *Proceedings of the 6th International Coral Reef Symposium*, **1**: 175–186.

van Deventer, M., K. Atwood, G.A. Vargo, L.J. Flewelling, J.H. Landsberg, J.P. Naar, and D. Stanek. 2012. *Karenia brevis* red tides and brevetoxin-contaminated fish: a high risk factor for Florida's scavenging shorebirds? *Botanica Marina*, **55**: 31–37.

Vanderploeg, H.A., J.R. Liebig, W.W. Carmichael, M.A. Agy, T.H. Johengen, G.L. Fahnenstiel, and T.F. Nalepa. 2001. Zebra mussel (*Dreissena polymorpha*) selective filtration promoted toxic

Microcystis blooms in Saginaw Bay (Lake Huron) and Lake Erie. *Canadian Journal of Fisheries and Aquatic Sciences*, **58**: 1208–1221.

van Hees, D., and K. Van Alstyne. 2013. Effects of emersion, temperature, dopamine, and hypoxia on extracellular oxidant accumulations surrounding the bloom-forming seaweeds *Ulva lactuca* and *Ulvaria obscura*. *Journal of Experimental Marine Biology and Ecology*, **448**: 207–213.

Van Landeghem, M.M., M. Farooqi, B. Farquhar, and R. Patiño. 2013. Impacts of golden alga *Prymnesium parvum* on fish populations in reservoirs of the upper Colorado River and Brazos River basins, Texas. *Transactions of the American Fisheries Society*, **142**: 581–595.

Van Wagoner, R.M., J.R. Deeds, M. Satake, A.A. Ribeiro, A.R. Place, and J.L.C. Wright. 2008. Isolation and characterization of karlotoxin 1, a new amphipathic toxin from *Karlodinium veneficum*. *Tetrahedron Letters*, **49**: 6457–6461.

Vardi, A., F. Formiggini, R. Casotti, A. de Martino, F. Ribalet, A. Miralro, and C. Bowler. 2006. A stress surveillance system based on calcium and nitric oxide in marine diatoms. *PLoS Biology*, **4**: 411–419.

Vargo, G.A., R. Erdman, G. Kleppel, and C. Burkhart. 1996. Controls on phytoplankton populations in Florida Bay: clams and eggs. In: *Florida Bay Science Conference, December 10–12*. University of Florida and Sea Grant of Florida, Key Largo: p. 60–62. Available at: http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.110.7743&rep=rep1&type=pdf.

Vasconcelos, V.M. 1995. Uptake and depuration of the heptapeptide toxin microcystin-LR in *Mytilus galloprovincialis*. *Aquatic Toxicology*, **32**: 227–237.

Vasconcelos, V., S. Oliveira, and F.O. Teles. 2001. Impact of a toxic and a nontoxic strain of *Microcystis aeruginosa* on the crayfish *Procabarus clarkii*. *Toxicon*, **39**: 1461–1470.

Verity, P.G., and D. Stoecker. 1982. Effects *of Olisthodiscus luteus* on the growth and abundance of tintinnids. *Marine Biology*, **72**: 79–87.

Vernoux, J.P., N. Lahlou, S.A. Elandaloussi, N. Riyeche, and L.P. Magras. 1985. A study of the distribution of ciguatoxin in individual Caribbean fish. *Acta Tropica*, **42**: 225–233.

Viaroli, P., M. Bartoli, G. Giordani, M. Naldi, S. Orfanidis, and J.M. Zaldívar. 2008. Community shifts, alternative stable states, biogeochemical controls and feedbacks in eutrophic coastal lagoons: a brief overview. *Aquatic Conservation*, **18**: S105–S117.

Vidoudez, C., R. Casotti, M. Bastianini, and G. Pohnert. 2011. Quantification of dissolved and particulate polyunsaturated aldehydes in the Adriatic Sea. *Marine Drugs*, **9**: 500–513.

Vidoudez, C., and G. Pohnert. 2008. Growth phase-specific release of polyunsaturated aldehydes by the diatom *Skeletonema marinoi*. *Journal of Plankton Research*, **30**: 1305–1313.

Vigilant, V.L., and M.W. Silver. 2007. Domoic acid in benthic flatfish on the continental shelf of Monterey Bay, California, USA. *Marine Biology*, **151**: 2053–2062.

Vinagre, T.M., J.C. Alciati, J.S. Yunes, J. Richards, A. Bianchi, and J.M. Monserrat. 2002. Effects of extracts from the cyanobacterium *Microcystis aeruginosa* on ion regulation and gill Na^+, K^+-ATPase and K^+-dependent phosphatase activities of the estuarine crab *Chasmagnathus granulata* (Decapoda, Grapsidae). *Physiological and Biochemical Zoology*, **75**: 600–608.

Waggett, R.J., P.A. Tester, and A.R. Place. 2008. Anti-grazing properties of the toxic dinoflagellate *Karlodinium veneficum* during predator-prey interactions with the copepod *Acartia tonsa*. *Marine Ecology Progress Series*, **366**: 31–42.

Walsh, C.J., M. Butawan, J. Yordy, R. Ball, L. Flewelling, M. deWit, and R.K. Bonde. 2015. Sublethal red tide toxin exposure in free-ranging manatees (*Trichechus manatus*) affects the immune system through reduced lymphocyte proliferation responses, inflammation, and oxidative stress. *Aquatic Toxicology*, **161**: 73–84.

Walters, S., S. Lowerre-Barbieri, J. Bickford, J. Tustison, and J.H. Landsberg. 2013. Effects of *Karenia brevis* red tide on the spatial distribution of spawning aggregations of sand seatrout *Cynoscion arenarius* in Tampa Bay, Florida. *Marine Ecology Progress Series*, **479**: 191–202.

Ward, L.A., P. Montagna, R.D. Kalke, and E. Buskey. 2000. Sublethal effects of Texas brown tide on *Streblospio benedicti* (Polychaeta) larvae. *Journal of Experimental Marine Biology and Ecology*, **248**: 121–129.

Wardle, W.J., S.M. Ray, and A.S. Aldrich (1975) Mortality of marine organisms associated with offshore summer blooms of the toxic dinoflagellate *Gonyaulax monilata* Howell at Galveston, Texas. In: *Proceedings of the First International Conference on Toxic Dinoflagellate Blooms*. V.R. LoCicero (Ed.).

Science and Technology Foundation, Wakefield: p. 257–263.

Wasniak, C.E., and P.M. Glibert. 2004. Potential impacts of brown tide, *Aureococcus anophagefferens,* on juvenile hard clams, *Mercenaria mercenaria,* in the Coastal Bays of Maryland, USA. *Harmful Algae*, **3**: 321–329.

Watanabe, M.F., H.D. Park, F. Kondo, K.I. Harada, H. Hayashi, and T. Okino. 1997. Identification and estimation of microcystins in freshwater mussels. *Natural Toxins*, **5**: 31–35.

Watkinson, A.J., J.M. O'Neil, and W.C. Dennison. 2005. Ecophysiology of the marine cyanobacterium, *Lyngbya majuscula* (Oscillatoriaceae) in Moreton Bay, Australia. *Harmful Algae*, **4**: 697–715.

Weckesser, J., C. Martin, and C. Jakobi. 1996. Dipsipeptides from cyanobacteria: structures and biological activities. *Journal of Systematic and Applied Microbiology*, **19**: 133–138.

Weijs, L., A.C. Dirtu, K. Das, A. Gheorghe, P.J.H. Reijnders, H. Neels, R. Blust, and A. Covaci. 2009. Inter-species differences for polychlorinated biphenyls and polybrominated diphenyl ethers in marine top predators from the southern North Sea: Part 1. Accumulation patterns in harbour seals and harbour porpoises. *Environmental Pollution*, **157**: 437–444.

Wells, M.L., V.L. Trainer, T.J. Smayda, B.S.O. Karlson, C.G. Trick, R.M. Kudela, A. Ishikawa, S. Bernard, A. Wulff, D.M. Anderson, and W.P. Cochlan. 2015. Harmful algal blooms and climate change: learning from the past and present to forecast the future. *Harmful Algae*, **49**: 68–83.

Wennhage, H., and L. Pihl. 1994. Substratum selection by juvenile plaice (*Pleuronectes platessa* L.): impact of benthic microalgae and filamentous macroalgae. *Netherlands Journal of Sea Research*, **32**: 343–351.

Wetzel, R.G. 2001. *Limnology*: *Lake and River Ecosystems*, 3rd ed. Academic Press, New York.

White, A.W. 1977. Dinoflagellate toxins as probable cause of an Atlantic herring (*Clupea horengus harengus*) kill, and pteropods as apparent vector. *Journal of the Fisheries Research Board of Canada*, **34**: 2421–2424.

White, A.W. 1980. Recurrence of kills of Atlantic herring (*Clupea harengus harengus*) caused by dinoflagellate toxins transferred through herbivorous zooplankton. *Canadian Journal of Fisheries and Aquatic Sciences*, **37**: 2262–2265.

White, A.W. 1981a. Marine zooplankton can accumulate and retain dinoflagellate toxins and cause fish kills. *Limnology and Oceanography* **26**: 103–109.

White, A.W. 1981b. Sensitivity of marine fishes to toxins from red tide dinoflagellate *Gonyaulax excavata* and implications for fish kills. *Marine Biology*, **65**: 255–260.

White, S.H., L.J. Duivenvoorden, L.D. Fabbro, and G.K. Eaglesham. 2006. Influence of intracellular toxin concentrations on cylindrospermopsin bioaccumulation in a freshwater gastropod (*Melanoides tuberculata*). *Toxicon*, **47**: 497–509.

White, S.H., L.J. Duivenvoorden, L.D. Fabbro, and G.K. Eaglesham. 2007. Mortality and toxin bioaccumulation in *Bufo marinus* following exposure to *Cylindrospermopsis raciborskii* cell extracts and live cultures. *Environmental Pollution*, **147**: 158–167.

Whyte, J.N.C., L.D. Townsend, and N.G. Ginther. 1996. Fecundity, toxin and trophic levels of the rotifer *Brachionus plicatilis* fed *Pseudo-nitzschia pungens* f. *multiseries*. In: *Harmful and Toxic Algal Blooms*. T. Yasumoto, Y. Oshima, and Y. Fukuyo (Eds.). Intergovernmental Oceanographic Commission of UNESCO, Paris: p. 401–404.

Wikfors, G.H. 2005. A review and new analysis of trophic interactions between *Prorocentrum minimum* and clams, scallops, and oysters. *Harmful Algae*, **4**: 585–592.

Wikfors, G.H., and E. Fernandez. 2013. Induced bacterivory in a declining culture of the mixotrophic dinoflagellate *Prorocentrum minimum* (Pavillard) Schiller. *International Journal of Ecology*, **2013**: art. ID 234372, 4 p. Available at: http://dx.doi.org/10.1155/2013/234372.

Wikfors, G.H., and R.M. Smolowitz. 1995. Experimental and histological studies of four life-history stages of the eastern oyster, *Crassostrea virginica,* exposed to a cultured strain of the dinoflagellate *Prorocentrum minimum*. *Biology Bulletin*, **188**: 313–328.

Wilcock, R.J., J.W. Nagels, G.B. McBride, K.J. Collie, B.T. Wilson, and B.A. Huser. 2010. Characterisation of lowland streams using a single-station diurnal curve analysis model with continuous monitoring data for dissolved oxygen and temperature. *New Zealand Journal of Marine and Freshwater Research*, **32**: 67–79.

Williams, D.E., S.C. Dawe, M.L. Kent, R.J. Andersen, M. Craig, and C.F.B. Holmes. 1997. Bioaccumulation and clearance of microcystins from salt water mussels, *Mytilus edulis,* and in vivo evidence for covalently bound

microcystins in mussel tissues. *Toxicon*, **35**: 1617–1625.

Williams, J., and R.M. Ingle. 1972. Ecological notes on *Gonyaulax monilata* (Dinophyceae) blooms along the west coast of Florida. *Florida Department of Natural Resources Marine Research Laboratory Leaflet Series*, **1**: 1–12.

Wiegand, C., and S. Pflugmacher. 2005. Ecotoxicological effects of selected cyanobacterial secondary metabolites a short review. *Toxicology and Applied Pharmacology*, **203**: 201–218.

Wilde, S.B., T.M. Murphy, C.P. Hope, S.K. Habrun, J. Kempton, A. Birrenkott, F. Wiley, W.W. Bowerman, and A.J. Lewitus. 2005. Avian vacuolar myelinopathy linked to exotic aquatic plants and a novel cyanobacterial species. *Environmental Toxicology*, **20**: 348–353.

Wium-Andersen, S., U. Anthoni, C. Christophersen, and G. Houen. 1982. Allelopathic effects on phytoplankton by substances isolated from aquatic macrophytes (Charales). *Oikos*, **39**: 187–190.

Wohirab, S., M.H. Iversen, and U. John. 2010. A molecular and co-evolutionary context for grazer induced toxin production in *Alexandrium tamarense*. *PLoS ONE*, **5**(11): e15039. doi: 10.1371/journal.pone.0015039.

Wolfe, G.V. 2000. The chemical defense ecology of marine unicellular plankton: constraints, mechanisms, and impacts. *Biology Bulletin*, **198**: 225–244.

Wood, P.C., and J. Mason. 1968. Paralytic shellfish poisoning: a short account of an outbreak occurring on the north-east coast of Britain in May 1968. ICES CM 1968/K:16; 6 p.

Work, T.M., B. Barr, A.M. Beale, L. Fritz, M.A. Quilliam, and J.L. Wright. 1993. Epidemiology of domoic acid poisoning in brown pelicans (*Pelecanus occidentalis*) and Brandt's cormorants (*Phalacrocorax penicillatus*) in California. *Journal of Zoo and Wildlife Medicine*, **24**: 54–62.

Wright, J.L.C., R.K. Boyd, A.S.W. de Freitas, M. Falk, R.A. Foxall, W.D. Jamieson, M.V. Laycock, A.W. McCulloch, A.G. McInnes, P. Odense, V.P. Pathak, M.A. Quilliam, M.A. Ragan, P.G. Sim, P. Thibault, J.A. Walter, M. Gilgan, D.J.A. Richard, and D. Dewar. 1989. Identification of domoic acid, a neuroexcitatory amino acid, in toxic mussels from eastern Prince Edward Island. *Canadian Journal of Chemistry*, **67**: 481–490.

Xiaoqing, J.I., H. Xiaotian, Z. Li, Y. Baijuan, Y. Zhiming, and Z. Jingzhong. 2011. Allelopathic interactions between *Prorocentrum micans* and *Skeletonema costatum* or *Karenia mikimotoi* in laboratory cultures. *Chinese Journal of Oceanology and Limnology*, **29**: 840–848.

Xie, L., P. Xie, K. Ozawa, T. Honma, A. Yokoyama, and H.-D. Park. 2004. Dynamics of microcystins-LR and -RR in the phytoplanktivorous silver carp in a sub-chronic toxicity experiment. *Environmental Pollution*, **127**: 431–439.

Xie, L.Q., P. Xie, L.G. Guo, L. Li, Y. Miyabara, and H.-D. Park. 2005. Organ distribution and bioaccumulation of microcystins in freshwater fish at different trophic levels from the eutrophic Lake Chaohu, China. *Environmental Toxicology*, **20**: 293–300.

Xie, Z., H. Xiao, X. Tang, and H. Cai. 2008. Interactions between red tide microalgae and herbivorous zooplankton: effects of two bloom-forming species on the rotifer *Brachionus plicatilis* (O.F. Muller). *Hydrobiologia*, **600**: 237–245.

Xu, N., Y.Z. Tang, J. Qin, S. Duan, and C.J. Gobler. 2015. Ability of the marine diatoms *Pseudo-nitzschia* multiseries and *P. pungens* to inhibit the growth of co-occurring phytoplankton via allelopathy. *Aquatic Microbial Ecology*, **74**: 29–41.

Yamasaki, Y., D.-I. Kim, Y. Matsuyama, T. Oda, and T. Honjo. 2004. Production of superoxide anion and hydrogen peroxide by the red tide dinoflagellate *Karenia mikimotoi*. *Journal of Bioscience and Bioengineering*, **97**: 212–215.

Yamasaki, Y., S. Nagasoe, T. Matsubara, T. Shikata, Y. Shimasaki, Y. Oshima, and T. Honjo. 2007. Allelopathic interactions between the bacillariophyte *Skeletonema costatum* and the raphidophyte *Heterosigma akashiwo*. *Marine Ecology Progress Series*, **339**: 83–92.

Yamasaki, Y., Y. Ohmichi, M. Hirose, T. Shikata, Y. Shimasaki, Y. Oshima, and T. Honjo. 2012. Low molecular weight allelochemicals produced by the diatom, *Skeletonema costatum*. *Thalassas*, **28**: 9–17.

Yamasaki, Y., T. Shikata, A. Nukata, S. Ichiki, S. Nagasoe, T. Matsubara, Y. Shimasaki, M. Nakao, K. Yamaguchi, Y. Oshima, T. Oda, M. Ito, I.R. Kenkinson, M. Asakawa, and T. Honjo. 2009. Extracellular polysaccharide-protein complexes of a harmful alga mediate the allelopathic control it exerts within the phytoplankton community. *ISME Journal*, **3**: 808–817.

Yan, T., M. Zhou, M. Fu, Y. Wang, R. Yu, and J. Li. 2001. Inhibition of egg hatching success and larvae of the scallop, *Chlamys farreri*, associated with exposure to cells and cell fragments of the dinoflagellate *Alexandrium tamarense*. *Toxicon*, **39**: 1239–1244.

Yang, J., X. Deng, Q. Xian, X. Qian, and A. Li. 2014. Allelopathic effect of *Microcystis aeruginosa* on

Microcystis wesenbergii: Microcystin-LR as a potential allelochemical. *Hydrobiologia*, **727**: 65–73.

Yasuno, M., and T. Sugaya. 1991. Toxicities of *Microcystis viridis* and the isolated hepatotoxic polypeptides on cladocerans. *Verhandlungen der Internationalen Vereinigung für Theoretische und Angewandte Limnologie*, **24**: 2622–2626.

YSI Environmental, Inc. 2009. *The Dissolved Oxygen Handbook*. YSI Environmental, Inc., Yellow Springs.

Zabka, T.S., T. Goldstein, C. Cross, R.W. Mueller, C. Kreuder-Johnson, S. Gill, and F.M.D. Gulland. 2009. Characterization of a degenerative cardiomyopathy associated with domoic acid toxicity in California sea lions (*Zalophus californianus*). *Veterinary Pathology*, **46**: 105–119.

Zagatto, P.A., S.V. Buratini, M.A. Aragão, and A.S. Farrão-Filho. 2012. Neurotoxicity of two *Cylindrospermopsis raciborskii* (cyanobacteria) strains to mice, *Daphnia,* and fish. *Environmental Toxicology and Chemistry*, **31**: 857–862.

Zambrano, F., and E. Canelo. 1996. Effects of microcystin-LR on the partial reactions of the Na^+-K^+ pump on the gill of carp (*Cyprinus carpio* Linneo). *Toxicon*, **34**: 451–458.

Zanchett, G., and E.C. Oliveira-Filho. 2013. Cyanobacteria and cyanotoxins: from impacts on aquatic ecosystems and human health to anticarcinogenic effects. *Toxins*, **5**: 1896–1917.

Żak, A., and A. Kosakowska. 2014. Allelopathic influence of cyanobacteria *Microcystis aeruginosa* on green algae *Chlorella vulgaris*. In: *Insights on Environmental Changes*, T. Zielinski, K. Pazdro, A. Dragan-Górska, and A. Weydmann (Eds.). Springer International, Cham: p. 141–150.

Zhang, X., T.P. Warming, and K.S. Christoffersen. 2009. Life history responses of *Daphnia magna* feeding on toxic *Microcystis aeruginosa* alone and mixed with a mixotrophic *Poterioochromonas* species. *Water Research*, **43**: 5053–5062.

Zhao, S., Y. Wang, and D. Li. 2014. Effects of toxic and non-toxic *Microcystis aeruginosa* in different mixtures with *Scenedesmus obliquus* on growth of *Brachionus calyciflorus*. *Journal of Freshwater Ecology*, **29**: 377–386.

Zimba, P.V., P.D. Moeller, K. Beauchesne, H.E. Lane, and R.E. Triemer. 2010. Identification of euglenophycin – a toxin found in certain euglenoids. *Toxicon*, **55**: 100–104.

Zimba, P.V., I.-S. Huang, D. Gutierrez, W.-G. Shin, M. Bennett, and R. Triemer. 2017. Euglenophycin is produced in at least six species of euglenoid algae and six of seven strains of *Euglena sanguinea*. *Harmful Algae*, **63**: 79–84.

Zingone, A., and H.O. Enevoldsen. 2000. The diversity of harmful algal blooms: a challenge for science and management. *Ocean and Coastal Management*, **43**: 725–748.

Zurawell, R.W., B.G. Kotak, and E.E. Prepas. 1999. Influence of lake trophic status on the occurrence of microcystin-LR in the tissue of pulmonate snails. *Freshwater Biology*, **42**: 707–718.

Zurawell, R.W., H. Chen, J.M. Burke, and E. Prepas. 2005. Hepatotoxic cyanobacteria: a review of the biological importance of microcystins in freshwater environments. *Journal of Toxicology and Environmental Health, Part B*, **8**: 1–37.