Harmful algal blooms along the North American west coast region: History, trends, causes, and impacts

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A B S T R A C T

Along the Pacific coast of North America, from Alaska to Mexico, harmful algal blooms (HABs) have caused losses to natural resources and coastal economies, and have resulted in human sicknesses and deaths for decades. Recent reports indicate a possible increase in their prevalence and impacts of these events on living resources over the last 10–15 years. Two types of HABs pose the most significant threat to coastal ecosystems in this “west coast” region: dinoflagellates of the genera Alexandrium, Gymnodinium, and Pyrodinium that cause paralytic shellfish poisoning (PSP) and diatoms of the genus Pseudo-nitzschia that produce domoic acid (DA), the cause of amnesic shellfish poisoning (ASP) in humans. These species extend throughout the region, while problems from other HABs (e.g., fish kills linked to raphidophytes or Cochlodinium, macroalgal blooms related to invasive species, sea bird deaths caused by surfactant-like proteins produced by Akashiwo sanguinea, hepatotoxins from Microcystis, diarrhetic shellfish poisoning from Dinophysis, and dinoflagellate-produced yessotoxins) are less prevalent but potentially expanding. This paper presents the state-of-knowledge on HABs along the west coast as a step toward meeting the need for integration of HAB outreach, research, and management efforts.

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1. Introduction

Harmful algal blooms (HABs) are a global threat to living marine resources and human health. These events impact all coastal U.S. states and large portions of coastal Canada and Mexico (Taylor, 1993; Horner et al., 1997; Mudie et al., 2002; Hernández-Becerril et al., 2007; Anderson et al., 2008b; Band-Schmidt et al., 2010). Harmful algal blooms have had significant ecological and socioeconomic impacts on Pacific coastal communities of North America for decades, and their prevalence and impacts on living resources in this west coast region have increased markedly in frequency and geographical distribution over the last 10–15 years (Anderson et al., 2008b; Kudela et al., 2008a; Kahr et al., 2009; Band-Schmidt et al., 2010; Rensel et al., 2010b; García-Mendoza, unpubl. data). The HABs that threaten west coast water quality, the health of living resources, and the economies of its communities are diverse and often extend beyond jurisdictional boundaries. Comprehensive understanding of the causes and impacts of west coast HABs will therefore require a regionally integrated approach, and effective HAB management will depend on interstate and international collaboration and coordination.

Several policy drivers call for a regional approach to addressing marine problems (e.g., U.S. Commission on Ocean Policy, 2004; NOAA Program Planning and Integration, 2007; NSTC Joint Subcommittee on Ocean Science and Technology, 2007; Joint Ocean Commission Initiative, 2009). The 2004 Reauthorization of the Harmful Algal Bloom and Hypoxia Research Control Act also acknowledged the need for a regional approach to HAB research and response by establishing a procedure for requesting Regional Assessments of HABs. The U.S. Commission on Ocean Policy (2004) and the Pew Oceans Commission (2003) recommended regional ocean governance efforts as an effective mechanism to facilitate regional ecosystem assessment and management. Recognizing this need, the West Coast Governors’ Agreement on Ocean Health (WCGA) was established in 2006 as a proactive, regional collaboration to protect and manage ocean and coastal resources.

Table 1
Reported human illnesses and deaths due to paralytic shellfish poisonings. Additional illnesses are known from all areas, but only those associated with fatalities are reported here. Dates vary depending on state, country and when monitoring began.

<table>
<thead>
<tr>
<th>Year</th>
<th>States</th>
<th>Cases</th>
<th>Deaths</th>
<th>Counties/areas involved</th>
<th>Shellfish kind</th>
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<tr>
<td>AK</td>
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<td>100</td>
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<td>Blue mussels</td>
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<tr>
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<td>2</td>
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<tr>
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<td>1</td>
<td>Likely Sitka</td>
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<tr>
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<td>1</td>
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</tr>
<tr>
<td></td>
<td>19541</td>
<td>8</td>
<td>1</td>
<td>False Pass</td>
<td>Blue mussels</td>
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<td>1</td>
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<tr>
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<td>7</td>
<td>2</td>
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<td>6</td>
<td>Chiapas, Guerrero coasts</td>
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</table>

along the coasts of Washington (WA), Oregon (OR), and California (CA). Harmful algal blooms were highlighted as needing immediate attention by all three states.

The WCGA called for “a HAB workshop … to reach consensus on the present state-of-knowledge and prioritize the information needed by decision makers to lessen the impacts of the HAB events on humans and critical marine resources” as part of the strategy to promote interstate coordination of HAB research and monitoring efforts (Action Plan for the West Coast Governors’ Agreement on Ocean Health, 2008). The National Oceanic and Atmospheric Administration (NOAA) and the states of CA, OR, and WA convened the West Coast Regional Harmful Algal Bloom Summit on 10–12 February 2009 in Portland, Oregon, to fulfill the WCGA charge. A White Paper, Harmful Algal Blooms in the West Coast Region: History, Trends, and Impacts in California, Oregon, and Washington, was developed by the Summit Steering Committee to summarize the scope of the HAB problem in this region, in order to provide background on the state-of-knowledge for Summit attendees. Here, we expand on that White Paper, incorporating Summit findings and consensuses, and extending the geographical coverage of HAB impacts on the west coast to include Alaska (AK), British Columbia (BC), the U.S. Pacific coast states, and Mexico.

2. Paralytic shellfish poisoning

2.1. Overview of toxicity, history on the North American west coast

Paralytic shellfish poisoning is caused by a suite of biotoxins, collectively called paralytic shellfish toxins (PSTs). Taxa known to produce these toxins include species of the dinoflagellate genera Alexandrium, Gymnodinum, and Pyrodinium. The genus typically associated with toxic outbreaks along the U.S. and Canadian west coasts is Alexandrium, while Gymnodinum and Pyrodinium species are associated with outbreaks in Mexico (Ochoa et al., 1997). Symptoms of PSP are neurological, onset is rapid and can result in paralysis or death through respiratory arrest. Toxicity varies with shellfish species, and some of the west coast species most likely to be contaminated include mussel species, butter clams (Saxidomus giganteus Deshayes), geoduck clams (Panopea generosa Gould), razor clams (Siliqua patula Dixon), and Pacific oysters (Crassostrea gigas Thunberg); see Section 2.2. Several other species reportedly have also been contaminated, including northern quahogs (Mercenaria mercenaria Linnaeus), horse clams (Tresus nuttallii Conrad and Tresus capax Gould), Pacific littleneck clams (Protothaca staminea Conrad), manila clams (Venerupis philippinarum Adams & Reeves), varynish clams (Nuttallia obscurata Reese), purple-hinge rock scallops (Hinnites multirugosus Gale) and other scallop species, cockle species, whelk species, moon snails (Lunaria heros Say), gooseneck barnacles (Pollicipes polymerus Gmelin), Dungeness crabs (Metacarcinus magister Dana), and spiny lobsters (Panulirus spp.) (Shumway et al., 1990; Shumway and Cembella, 1993; Matter, 1994; Shumway, 1995; Deeds et al., 2008).

Human deaths attributed to PSP date back to 1793 (Table 1), when four members of Captain George Vancouver's Royal Navy crew became sick and one died after eating shellfish from a beach in central BC now called Poison Cove (Quayle, 1969: Fig. 2). The oldest documented apparent HAB incident in AK occurred in 1799 when the Aleut crew of the Russian fur trader, Alexander Baranof, became ill after eating blue mussels (Mytilus edulis Linnaeus) in an area near Sitka, AK, now called Peril Strait (Table 1 and Fig. 1). This incident resulted in an estimated 100 deaths (Fortuine, 1975). More recently, a June 2010 incident in southeast AK resulted in five illnesses and two deaths, the first deaths in AK since 1997. Elsewhere on the west coast, human poisonings from PSP were apparently common in CA in the last half of the 1800s (Sommer and Meyer, 1937), but the first recorded incident occurred in Sonoma County, central CA, in 1903, when 12 people became ill and five died after eating California mussels (Mytilus californianus

Fig. 1. Alaska coast line showing sites with highest concentrations of paralytic shellfish toxins (number with no units; units are μg/100 g shellfish meat) by shellfish species. The regulatory limit for paralytic shellfish toxins is 80 μg/100 g. Data sources: ADHHS-ES database, 1973–2008; http://www.epi.hss.state.ak.us/bulletins/catlist.jsp?carttype=Paralytic+Shellfish+Poisoning+(PSP), Gessner et al. (1997), Raphone (2001).
Paralytic shellfish poisoning has been recognized as a serious health risk in CA since 1927 when a major outbreak in a multi-county region north and south of San Francisco resulted in 102 illnesses and six deaths (Table 1). From 1927 through 1989, PSP related illnesses totaled 511 in CA, including 32 deaths (Price et al., 1991). The earliest reported PSP poisonings in WA and the only deaths in that state occurred in 1942 when one adult and two Native American children died after eating butter clams and blue mussels collected along the Strait of Juan de Fuca (Quayle, 1969; Table 1). Three deaths also occurred further north in Barkley Sound on the West Coast of Vancouver Island, BC (Fig. 2). Additional incidences in BC occurred in 1957 with 61 cases but no deaths, 1965 with four cases and one death, and 1980 with two illnesses and one death. The 1965 incident is especially significant because it was the first time that a human death occurred at the same time that shellfish toxicity was measured, and a bloom of the toxic species, Alexandrium acatenella (Whedon et Kofoid) Balech was recognized as the causative organism (Prakash and Taylor, 1966; Quayle, 1969). More recent events in WA include 10 illnesses in 1978, five illnesses in 1998, and nine illnesses in 2000, all in Puget Sound (Erickson and Nishitani, 1985; Trainer et al., 2003; Moore et al., 2009). PSP poisonings in OR caused 20 illnesses and one death in Coos Bay in 1933 (Sommer and Meyer, 1935; Halstead, 1965). In Pacific Mexico, the first documented report of human shellfish poisoning dates only to 1976, with seven cases and two deaths (Saldate-Castañeda et al., 1991). Between 1979 and 2008, 391 poisoning cases were recorded with 24 deaths along the Pacific coast of Mexico. Of these, 34 cases with five deaths were attributed to Gymnodinium catenatum Graham, and 357 cases with 19 deaths were attributed to Pyrodinium bahamense var. compressum (Böhm) Steidinger, Tester & Taylor (Cortés-Altamirano and Sierra-Beltrán, 2008).

2.2. Trends in prevalence and impacts

Outbreaks of Alexandrium spp., and associated shellfish toxicity and human illnesses have been a persistent problem along the west coast for decades. Outbreaks may be increasing in frequency and distribution in some locations. For example, the frequency and geographic distribution of associated shellfish closures in Puget Sound have increased in WA since monitoring first began in the 1940s and 1950s (Trainer et al., 2003) and PST-related shellfish closures have increased on the OR coast from the 1980s (Oregon Department of Fish and Wildlife, ODFW data, http://public.health.oregon.gov/HealthyEnvironments/Recreation/HarmfulAlgaeBlooms/Pages/index.aspx, Strutton and Tweddle, unpubl. data). Outbreaks have decreased in other locations (e.g., high PSP levels in Drakes Bay, CA have declined since the 1980s, California Department of Public Health, CDPH data, http://www.cdph.ca.gov/HealthInfo/Environhealth/water/Pages/Shellfish.aspx).

2.2.1. Alaska

Paralytic shellfish toxins are a pervasive problem in AK (Figs. 1 and 7A). Personal use and subsistence shellfish harvests accounted for 183 confirmed PST illnesses between 1973 and 2008 (Alaska Department of Health and Human Services-Epidemiology Section, ADHHS-ES database, 1973–2008; http://www.epi.hss.state.ak.us/bulletins/catlist.jsp?cattype=Paralytic+Shellfish+Poisoning+(PSP)), with more than half of the illnesses from consumption of butter clams that can retain the toxins for more than two years (Shumway, 1990). Blue mussels, cockles (Clinocardium sp. Keen), razor clams, Pacific littleneck clams, and other unknown clams caused the remaining illnesses. Numbers of reported illnesses may be underestimated by a factor of ~10–30, due to underreported minor illnesses, inaccurate and incomplete incident recording, and misdiagnosis (Gessner and Middaugh, 1995; Gessner and McLaughlin, 2008). Rural harvesters are particularly at risk because they underestimate the potential of illness based on trust of traditional local knowledge to determine when to consume shellfish, including use of unreliable environmental cues such as water color. Furthermore, Alaskans continue to use the myth that PSTs occur only in months that do not have an “r” in the spelling; i.e., May through August, when the reality is that PSTs and illnesses can occur year-round.

The impacts of PSTs on public health are unevenly distributed across populations; e.g., compared to other Alaskans, AK Natives
living in coastal communities are nearly 12 times more likely to encounter PSTs by consuming untested, subsistence-harvested shellfish (Gessner and Schloss, 1996). Sharing subsistence harvest is common practice among AK Natives and this practice can geographically expand the risk of illness far beyond a single community. For example, King Cove, on the Aleutian Peninsula, has a known history of PST illnesses and fatalities, and shellfish from this region are often shared with Native Americans along the U.S. west coast and interior AK (Wright et al., 2008).

The highest PST levels measured to date from various shellfish species in AK are shown in Fig. 1. The highest level, 20,600 μg/100 g shellfish meat, occurred in blue mussels from Kalsin Bay, Kodiak Island, in late May 1994, and resulted in 16 illnesses (Ostasz, 2001). Elevated PSTs in Prince William Sound are a rare event because populations of butter clams, Pacific littleneck clams, and soft shell clams (Mya arenaria Linnaeus) are depressed due to the 1964 earthquake, commercial fishery overharvesting of razor clams, sea otter (Enhydra lutris Linnaeus) predation, and impacts from the 1989 Exxon Valdez oil spill (Baxter, 1971; Rukuyama et al., 2000; Thomas et al., 2002). High PST levels, up to 7750 μg/100 g, exist along the southern shoreline of the Aleutian Peninsula, while the northern shoreline and the Bering Sea have lower levels ranging from 135 to 310 μg/100 g based on Stimpson’s surf clam (Macomeries polymyxa Stimpson) visceras (Hughes and Nelson, 1979). Commercial fisheries exist for Pacific littleneck clams in Kachemak Bay, razor clams in lower Cook Inlet, and geoduck clams in southeastern AK, where monitoring occurs during the harvest period. The following shellfish species have shown some record of PSTs above the regulatory limit of 80 μg/100 g: blue mussel, butter clam, Stimpson’s surf clam, razor clam, Pacific littleneck clam, geoduck clam, scallop species, cockle species, Pacific oyster, Dungeness crab, Tanner crab (Chionoecetes bairdi Rathbun), and snow crab (Chionoecetes opilio Fabricius), particularly in the Aleutian area, Kodiak Island, and in the Southeast Alaska region at Juneau and Ketchikan (ADHHS-ES database). Historically, the toxin appears to constitute a persistent threat to human health, in particular because while commercially harvested and farmed products are tested, recreationally harvested shellfish are not tested.

Paralytic shellfish toxins also have been measured in a number of crab species, including Dungeness, Tanner, snow, hair (Erinacrinus isnbeckii Brandt), and red king (Paralithodes camtschaticusTilesius) crabs (ADHHS-ES database, 1973–2008). Dungeness and Tanner crabs harvested in the Kodiak and Aleutian/Bering Sea fisheries must be killed, cleaned, and sectioned before being shipped to market. Testing of Dungeness crabs in the southeast AK fishery was suspended in 1996 after four years of negative PST tests.

2.2.2. British Columbia

In British Columbia, PSTs are the most prevalent biotoxins affecting shellfish growing areas. The frequency and intensity of Alexandrium blooms vary from year to year, but blooms are expected each year. Monitoring for PSTs in BC began in 1942, as did the first formal closures (Quayle, 1969). In 1982, a PST level of 30,000 μg/100 g was recorded in California mussels in Work Channel on the northern BC mainland (Chiang, 1988; Fig. 2). The highest level of PST recorded since 1994 was 10,000 μg/100 g in mussels at Ellin Point on the northeast coast of Vancouver Island in April 1994 (Fig. 2).

In the south coast of BC, PST events usually occur during the months of April to October, but may occur throughout the year (Taylor and Harrison, 2002; Canadian Food Inspection Agency data, www.inspection.gc.ca). Bloom initiation rarely occurs in winter months. The last widespread closure due to PSTs along the BC coastline occurred in 2008 when most of the main commercial growing areas were closed for a portion of the summer.

The intensity and frequency of blooms also vary among growing areas. Baynes Sound on the east coast of Vancouver Island does not often experience Alexandrium blooms, and the highest level of PST recorded there between 1994 and 2008 was 430 μg/100 g in California mussels (Canadian Food Inspection Agency data). Areas such as Barkley Sound on the west coast of Vancouver Island have PST events nearly every year. The north and central coasts can have extensive Alexandrium spp. blooms, and at least one PST event has been recorded along the mainland coast each year (Taylor and Harrison, 2002). Paralytic shellfish toxin activity is less common in the areas monitored on the Haida Gwaii (formerly named Queen Charlotte Islands).

Illnesses due to PST are relatively rare in BC, with none reported since 2005 (Canadian Food Inspection Agency data). The low number of PST illnesses may be attributed to the biotoxin monitoring program, prompt and effective closures of harvest areas, and education of the public to the hazards of shellfish poisoning. The majority of recorded illnesses have been attributed to butter clams. As noted previously, these clams can retain toxins for longer than two years (Quayle, 1969; Kvitek et al., 2008). Due to the likelihood of high levels of PST in butter clams, this species remains closed for harvest in most areas in BC. The impacts of PST closures on the commercial shellfish industry are difficult to quantify. Anecdotally, the impacts are considered extensive. Harvest areas can be shut down for months at a time, resulting in significant layoffs for staff and harvesters, and markets can be lost. Butter clams are the preferred shellfish species for Food Social Ceremonial harvesting for First Nations people on the north coast, but it can be difficult to find areas that can be opened for their harvest because these clams retain toxins for long periods of time.

2.2.3. Washington

In Washington (Fig. 3), PST-related closures of recreational shellfish harvesting have been imposed since an incident in 1942 that led to three Native American fatalities on the Strait of Juan de Fuca (Trainer et al., 2003; Table 1). The Washington Department of Health (WDOH; http://www.doh.wa.gov/CommunityandEnvironment/Shellfish/BiotoxinsIllnessPrevention/Biotoxins.aspx) imposed a harvesting closure at that time for all bivalve species except razor clams from Dungeness Spit to the mouth of the Columbia River from 1 April to 31 October. The coastal closure is reissued every year, but the Strait of Juan de Fuca closures are now regulated by toxin monitoring (F. Cox, pers. comm.). Routine monitoring for toxins in commercial shellfish in waters north and west of Admiralty Inlet and in Willapa Bay and Grays Harbor began in 1957 following a severe outbreak of PST in BC (Nishitani and Chew, 1988). Washington Department of Health records indicate that PST closures occur in these coastal bays on an irregular (sporadic) basis.

In the 1950s and 1960s, PSTs occurred in the northern regions of Puget Sound (e.g., Sequim and Discovery bays), extending southward during the 1970s and 1980s to the inner Sound (Quayle, 1969; Nishitani and Chew, 1988; Rensel, 1993; Trainer et al., 2003; Trainer and Hickey, 2003; Cox et al., 2008). Prior to 1978, illnesses due to PST were not reported in Puget Sound including Hood Canal, and Whidbey, Central and South basins, but widespread toxicity occurred in September 1978, beginning in Whidbey Basin and spreading as far south as Des Moines (south of Seattle) in the Central Basin (Nishitani and Chew, 1988; Fig. 7b). Toxin levels in bay mussels (Mytilus trossulus Gould) were as high as 30.360 μg/100 g shellfish meat (Fig. 3). Ten people reported PST symptoms after eating recreationally harvested mussels and pink scallops (Chlamys rubida Hinds), but no deaths occurred. The first shellfish harvest closures in the South Basin occurred in October 1988 when toxin levels in Pacific oysters reached 2000 μg (Trainer et al., 2003). Since then, repeated closures have occurred in most
years throughout the Puget Sound basins, except south Hood Canal, but not always in the same time or place each year (Cox et al., 2008).

Ceremonial, subsistence, and commercial harvests by WA tribal communities have been greatly impacted by PST-associated shellfish closures. The Puyallup, Suquamish, and Jamestown S’Klallam tribes have experienced severe economic losses from their commercial geoduck fisheries, based on frequent and lengthy seasonal harvest closures (Wekell and Trainer, 2002). Recalls of geoduck related to PST events have cost the tribes about $30,000. Commercial harvesting of Pacific oysters and Dungeness crabs by the Jamestown S’Klallam Tribe, and of manila clams, Pacific oysters, and basket cockles (Climaciardium nuttallii Conrad) by the Lummi Nation have also been significantly affected. Subsistence and ceremonial harvesting by the Jamestown S’Klallam Tribe have been impacted by PST toxicity of butter, Pacific littleneck, horse, and manila clams. Beach closures have also impacted Puyallup tribal cultural practices by restricting the use of clams for ceremonial dinners at weddings and funerals.

2.2.4. Oregon

Irregular monitoring of shellfish for saxitoxins began in OR in 1958 after high levels of PSTs were reported along the WA coast (Nishitani and Chew, 1988). Changes in monitoring sites, shellfish species monitored, and the possibility that blooms initiated offshore make it difficult to compare the early data with later values (Nishitani and Chew, 1988). More consistent monitoring (conducted since 1979 by the Oregon Department of Agriculture, ODA; http://oregon.gov/ODA/FSD/shellfish_status.shtml) at more sites has improved the coverage and has led to frequent closures, primarily of razor clams and mussel species shellfisheries. Paralytic shellfish toxins have severely impacted shellfish harvests at Clatsop Beach in northern OR (Fig. 4). The severity of a PST-associated HAB outbreak varies annually between northern and southern OR coastal areas. In 1992, a PST event affected the central and northern coast, but not the southern beaches, while in 2001 PST affected only the southern beaches.

The frequency and duration of PST-related shellfish closures on the OR coast increased from 1979 to 1996 (ODFW data, http://public.health.oregon.gov/HealthyEnvironments/Recreation/HarmfulAlgaeBlooms/Pages/index.aspx, Strutton and Tweddle, unpubl. data, Fig. 7C). Twice as many closures occurred from 1990 to 1996 as in all previous years, and most of the recent closures lasted more than 50 days. In the 2000s, the total number of closures, considering the northern and southern coasts separately, was 23, compared with 15 in the 1990s and 6 in the 1980s.

2.2.5. California

Paralytic shellfish poisoning events have occurred along the CA coast (Fig. 5) since before written records were maintained, with “mussel poisoning” being recognized by coastal tribes (Meyer et al., 1928). Paralytic shellfish toxin levels have been highly variable and unpredictable during the decades that monitoring has been conducted, as has the breadth of geographic range involved (Price et al., 1991; Langlois, 2001). Despite the temporal and geographic variability, in every year since 1999, PSP toxins have been observed in Drakes Bay along the Marin County coast, north of San Francisco (CDPH data).

In general, Alexandrium is absent or constitutes a minor component of the marine phytoplankton community along the CA coast. This dinoflagellate has been observed in approximately 3500 of the 24,000 phytoplankton samples collected by the CDPH monitoring program since 1993. It has comprised less than 10% of the phytoplankton assemblage in 93% of these samples and 55% of the observations have been at <1% relative abundance (CDPH data). Visible blooms of *Alexandrium* are rarely seen along the CA coast, with only one documented visible event in the past 19 years. A massive ‘red tide’ due to *Alexandrium* covered Drakes Bay for a brief period in July 1991 (Langlois, 2001; G. Langlois, pers. comm.).

The greatest frequency of *Alexandrium* observations has been recorded along the Marin County coast, consistent with the general pattern of PST frequency, followed by sites along the San Luis Obispo County coast (Langlois, 2001; CDPH data). Each of these
regions experiences more than twice the frequency of *Alexandrium* observations of any other coastal county. There was an apparent increase in *Alexandrium* along the Santa Barbara coast beginning in 1999, with the greatest number of observations occurring in 2006; 15 of 52 phytoplankton samples (29%) collected at Goleta Pier contained *Alexandrium* cells, coinciding with an increase in PSP activity in the region (see below). Low abundances of *A. catenella* were detected in all seasons from a weekly monitoring program conducted in a small harbor of Santa Monica Bay from 2006 to 2009 using a quantitative PCR method (Garneau et al., 2011).

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**Fig. 4.** Oregon coast line with sites of highest concentrations of paralytic shellfish toxins (number with no units; units are μg/100 g shellfish meat) and domoic acid (ppm). Data sources: ODA data, http://oregon.gov/ODA/FSD/shellfish_status.shtml, ODFW data, http://public.health.oregon.gov/HealthyEnvironments/Recreation/HarmfulAlgaeBlooms/Pages/index.aspx, M. Hunter (unpubl. data).

**Fig. 5.** California coast line with sites of highest concentrations of paralytic shellfish toxins (number with no units; units are μg/100 g shellfish meat) and domoic acid (ppm). Data sources: CDPH data, http://www.cdph.ca.gov/HealthInfo/environhealth/water/Pages/Shellfish.aspx, Price et al. (1991).
Highest abundances of the dinoflagellate coincided with low but measurable concentrations of PSTs in the plankton.

Data from the CDPH biotoxin monitoring program show that a major period of PSP activity extended through the 1980s into 1991 (Fig. 7D). The highest toxin levels detected in shellfish during this time were 26,000 μg/100 g in rock scallops from Sonoma County, associated with the last documented fatality in August 1980, 16,000 μg/100 g in California mussels from Marin County (July 1980), 14,000 μg/100 g in California mussels from Humboldt County (September 1989) and 10,000 μg/100 g in California mussels from Marin County (July 1991) (Price et al., 1991; CDPH data, Fig. 5 and Table 1). Each of the subsequent 18 years has experienced significant levels of PST in shellfish, but these have been well below the 1980s maxima and have been restricted in geographic range and duration (CDPH data, Fig. 7D). During this latter period, the concentration of PSTs has exceeded 3000 μg/100 g only once in California mussels from Marin County (in August 1998).

The majority of PSP activity has historically occurred in the central and northern portions of CA (Price et al., 1991; CDPH data). Despite this general pattern, over the years alert levels of PST have been detected in shellfish from each of the coastal counties (CDPH data). In recent years, an increase in PSP activity has been suggested in some southern CA sites, most notably in commercial shellfish growing areas in Santa Barbara and San Diego counties. Sampling sites in Santa Barbara experienced alert levels of PST every year between 2005 and 2008, peaking in 2006 (744 μg/100 g). Prior to this recent activity and a moderate event in 1998, there had not been alert levels for these toxins in this region since the 1980s. The San Diego aquaculture site experienced PST concentrations in excess of the federal alert level for the first time in 2008. The last time the alert level was exceeded anywhere in San Diego County was during 1985 in a mussel sample from Scripps Pier (La Jolla) and 2006 inside San Diego Bay (G. Langlois, pers. comm.).

2.2.6. Mexico

Paralytic shellfish poisoning is the most important toxic syndrome related to HABs in Mexico, and PSTs are the only toxins associated with human fatalities (Fig. 6 and Table 1). Paralytic shellfish toxin accumulations are associated with blooms of G. catenatum and P. bahamense var. compressum. The former species has been observed from the upper Gulf of California in the north to Guerrero in the south, while P. bahamense var. compressum is responsible for PSP outbreaks in the southern states of Pacific Mexico (Ochoa et al., 2002).

The dinoflagellate, G. catenatum, was first described from the central Gulf of California coast at cell abundances up to 10⁶/l (Graham, 1943), but the first documented PSP event occurred near the mouth of the Gulf in 1979 when three human deaths and an extensive fish kill occurred (Mee et al., 1986, Table 1). Toxin levels during that event ranged from <20 to 7640 μg/100 g in the tropical rocky oyster (Ostrea iridescent Hanley), with cell densities up to 6.6 × 10⁶ cells/l (Mee et al., 1986, Fig. 6). Additional blooms have occurred in Bahia Mazatlán, Colima, Guerrero, and Oaxaca. Most blooms occur between February and May when the water temperature ranges between 17 and 25 °C (Manrique and Molina, 1997; Gárate-Lizárraga et al., 2004, 2006).

Bloom of Gymnodinium species have impacted public health. The number of humans affected varies in different reports. Hernández-Becerril et al. (2007) mentioned 561 intoxications and 38 fatalities from 1970 to 2004 related to PSTs of Gymnodinium. In contrast, Cortés-Altimirano and Sierra-Beltrán (2008) recognized only 34 intoxications and five deaths. No intoxications associated with Gymnodinium have been reported recently, which may be attributed to increased attention by Mexican health authorities. Periodic bans on harvesting cultured or wild shellfish have been imposed by health authorities in Mexico since 2004. The majority of those closures were associated with the presence of Gymnodinium species (Federal Commission for the Protection against Sanitary Risks, COFEPRIS: http://www.cofepris.gob.mx/AZ/Paginas/Marea%20Roja/MareaRoja.aspx).

Three major toxic outbreaks of P. bahamense var. compressum involving human poisoning have been documented on the Pacific coast of Mexico (Hernández-Becerril et al., 2007). In November 1989 in the Gulf of Tehuantepec region, three persons died and 99 persons were poisoned as a consequence of a bloom that reached a maximum abundance of 1.7 × 10⁶ cells/l (Fig. 6 and

![Fig. 6](http://www.cofepris.gob.mx/AZ/Paginas/Marea%20Roja/MareaRoja.aspx)
Table 1). During this bloom, PST concentrations as high as 811 μg/100 g were recorded in tropical rocky oyster (Saldate-Castañeda et al., 1991). On the Michoacán and Guerrero coasts in central Mexico, 600 people were affected and six died during a bloom from November 2001 to February 2002. From November 2001 to August 2002 on the Chiapas (south) and Guerrero (central) coasts, 101 persons were poisoned and six died when patches of *P. bahamense* var. *compressum* were present in this region. Thirteen other toxic outbreaks associated with *P. bahamense* var. *compressum* were documented from 1979 to 2006 but did not result in fatalities (Corte´s-Altamirano and Sierra-Beltrán, 2008).

Several species of *Alexandrium* have been reported from Pacific waters off Mexico (Okolodkov and Garate-Lizárraga, 2006; Hernández-Becerril et al., 2007), but none have been associated with toxic events. One species, *A. catenella*, has been reported in the phytoplankton assemblage of the Todos Santos Bay region (northern part of the Baja California peninsula), and resting cysts of this species exist in the sediments of the bay (Peña-Manjarrez et al., 2005); however, high densities of *A. catenella* have not been reported in this region or in other areas of Pacific Mexico.

Monitoring of phycotoxins related to HABs started in the 1980s in high risk areas of the Pacific coasts of southern states of Mexico. Unfortunately, data on the variation of microalgal phycotoxin concentrations were not available until 2001 when a nationwide program was implemented by the COFEPRIS (Fig. 7E). A consistent monitoring of PSTs started in Baja California, Sonora, and southern
states of the Pacific coast of Mexico. Concentration of toxins in shellfish from Baja California and Sonora were never above the alert level (“Proyecto Marea Roja” of the COFEPRIS). In contrast, the presence of PSTs is a recurrent problem in Chiapas, Oaxaca, and Guerrero. Concentrations above 80 µg/100 g are frequent in coastal areas of these states and a maximum concentration of 7309 µg/100 g was detected in the Acapulco area in the winter of 2001. A trend for the presence of PSTs is difficult to assess in the Acapulco area. However, toxin outbreaks seemed to diminish in frequency (number of events above the alert level) but increase in magnitude (maximum concentration measured in shellfish samples) from 2003 to 2011.

2.3. Factors promoting blooms

2.3.1. Overview: anthropogenic vs. natural factors

There is little evidence to support anthropogenic factors as primary promoters of *Alexandrium* blooms and PSTs in most areas along the Pacific west coast. In CA, blooms are stronger in the drier seasons and it appears that blooms usually start offshore and move onshore when upwelling winds relax (Langlois and Smith, 2001; Anderson et al., 2008b). Although some blooms start nearshore in BC and WA, in general blooms occur during periods of warmer surface temperatures, which typically characterize periods of stratification in upwelling regions. Thus, these PSP events appear related more to large-scale oceanographic forcing, although there may be potential influence from local nutrient inputs when cells reach the shore (Anderson et al., 2008a). Presently, the extent and possible role of local nutrient pulses in stimulating blooms of *Alexandrium* are not known. In WA, little is known about the origin of coastal blooms of *Alexandrium*, but it is possible that they may also be brought inshore when upwelling winds relax or downwelling winds occur and enter coastal bays; e.g., Grays Harbor and Willapa Bay (Roegner et al., 2002), although this is not always the case (Cox, 2001). The relaxation or reversal of upwelling-favorable winds is also likely an important mechanism for bringing blooms into contact with the Oregon coast, and Tweddle et al. (2010) showed that elevated toxin levels were associated with late summer upwelling. Anthropogenic nutrient sources are more likely to be relevant in inland waters along the Strait of Juan de Fuca and in Puget Sound (Rensel, 2007), although with the exception of some shallow bays, the nutrient source is still more likely upwelled waters.

2.3.2. Alaska

Alaska has a long history of encounters with PSTs that occur along much of the Gulf of Alaska coast from the BC border in the southeast to the Aleutian chain and into the Bering Sea on the west and more northerly coasts. Human health problems persist despite better understanding of these events, as many coastal residents continue to consume potentially toxic shellfish. There is no evidence to support anthropogenic factors as promoters of *Alexandrium* blooms or toxic events in this region. The shoreline is long and complex, human populations are remote and widely disbursed, and there are many streams, rivers, islands, and extreme weather events that produce a complex marine ecosystem. The nearshore Alaska Coastal Current, with a seaward boundary near the edge of the continental shelf of the Gulf of Alaska, is greatly affected by the local shoreline topography and by freshwater input from fjords and estuaries (Roey, 1979, 1981; Schumacher and Reed, 1980). The North Pacific High in summer allows intrusion of deep, nutrient-rich water into coastal waters resulting in the development of seasonal algal blooms (Horner et al., 1997); however, there has been little historical phytoplankton monitoring with only a few sparse reports of the presence of *Alexandrium* (as *Gonyaulax*) (Horner et al., 1997). A new phytoplankton monitoring program (Alaska HAB monitoring program or AHAB, sponsored by the University of Alaska Southeast and Fairbanks, and NOAA) has trained shellfish growers, tribal members and volunteers to sample and identify HAB species in coastal areas near Ketchikan, Juneau, and Kachemak Bay. This program, which began in 2008, will add to the knowledge of HAB species in AK waters.

2.3.3. British Columbia

There does not appear to be a link between pollution (or other anthropogenic effects) and promotion of *Alexandrium* blooms in BC. Many fairly unpopulated, remote areas along the BC coast experience significant *Alexandrium* blooms, while some areas that are relatively heavily populated are not as affected by *Alexandrium* blooms (Taylor, 1993). In BC, blooms often originate nearshore in shallow areas and then spread to larger bodies of water (Taylor et al., 1994). There is some evidence for increased *Alexandrium* blooms during El Niño years; however, there have been significant blooms also in non El Niño years (Erickson and Nishitani, 1985; Taylor and Harrison, 2002). There may also be a link between warmer water temperatures and increased bloom activity (Yan et al., 2003).

2.3.4. Washington

Paralytic shellfish toxin events in Puget Sound are thought to originate primarily from local shallow areas of the Sound and not from offshore cys or motile cell populations advected into the Sound (Cox et al., 2008). Paralytic shellfish toxin occurrences throughout Puget Sound have been documented since 1978, when a bloom of *A. catenella* spread from the Whidbey Basin through central Puget Sound and into the southern extremities of the Sound (Nishitani and Chew, 1988). A survey in 1981 found motile cells, cysts, or low levels of toxin in all areas of the southern Sound (Nishitani and Chew, 1988). More recently, in a 2005 survey, cysts were found throughout Puget Sound, with highest abundances in the northern and central regions (Horner et al., 2008, 2011). Abundance was highest in Quartermaster Harbor, considered a possible “breeding bay” for *Alexandrium* (Nishitani and Chew, 1984), but little correlation of cyst abundance with physical or chemical properties of the sediment was found.

Bloom of *A. catenella* in Puget Sound generally occur from late spring through summer (Trainer et al., 2003; Dyrman et al., 2010). Like many other dinoflagellates, its growth is favored by a stable water column and warm temperatures (Nishitani and Chew, 1984), consistent with the hypothesis that blooms are stimulated by large precipitation events followed by warm and calm weather (Erickson and Nishitani, 1985; Determan, 1998). Moore et al. (2009), however, did not find a correlation between precipitation-induced freshwater runoff (i.e., elevated Skagit River streamflow) and PST events. They hypothesized that long residence time in surface waters (i.e., low streamflow) would favor PST events because toxin accumulation by shellfish would be enhanced. These authors determined that warm air and water temperatures as well as low streamflow conditions preceded exceptional PST events in blue mussels from 1993 to 2007 at four Puget Sound “hot spots” (sites of high PST incidence), Mystery, Discovery, and Sequim bays, and Kingston Marina, all in the northern and central parts of Puget Sound.

Moore et al. (2008, 2009) also assessed the relationship of large-scale and local climatic factors and PST occurrence in Puget Sound shellfish. In contrast to previous hypotheses linking large-scale climatic variations (e.g., ENSO) with PST events (e.g., Erickson and Nishitani, 1985), they found no such correlation. Their statistical analyses of a 15-year continuous dataset of mussel toxicity indicated that local climatic variability was more important than large-scale variation in explaining shellfish toxicity in Puget Sound.

The source(s) of *Alexandrium* blooms on the open WA coast has not been identified, and little is currently known about its occurrence and distribution offshore (F. Cox and R. Horner, pers.)
comm.). If such blooms develop in the Juan de Fuca eddy or any other offshore region, they are likely to impact the coast only during periods of downwelling winds (storms, which does not appear to be the case). Blooms occur sporadically in coastal embayments such as Grays Harbor and Willapa Bay, and impact the local shellfish growing areas, but it is not known if these events originate within the bays or are advected from offshore. Razor clams and mussels on the coastal beaches are sometimes affected when shellfish in the embayments are not.

2.3.5. Oregon

Observations of PSTs indicate a steady increase in frequency and in both California mussels (M. californianus) and razor clams (S. patula) since the inception of the Oregon shellfish toxin sampling program in 1979. This increase was markedly so from 1992 to 1997 and from 2008 to 2010 (ODF data, http://oregon.gov/ODA/FSD/shellfish_status.shtml, M. Hunter, unpubl. data). Since 2007, Oregon has been monitoring surf zone phytoplankton for the presence of HABs and Alexandrium is commonly observed in samples, especially from the middle of June through September when the water temperature is >12.5 °C (ODF data at http://bioweb.coas.oregonstate.edu/~mocha/odfdata.html). High levels of saxitoxin in shellfish tissues are also often associated with late summer upwelling and higher chlorophyll concentrations (Tweddle et al., 2010). Comparing the tissue PST data with the surf zone Alexandrium data indicates that a very low abundance of cells can result in elevated levels of tissue toxins (ODA and ODFW data at http://bioweb.coas.oregonstate.edu/~mocha/odfabdata.html).

2.3.6. California

There is no clear evidence to link the occurrence of PST events off CA with El Niño or La Niña periods – in fact, the last two major PST events in CA occurred during opposite conditions: 1989 during a strong La Niña period and 1991 during a strong El Niño period (Langlois, 2001).

The source of Alexandrium responsible for PST events along the CA coast is in question, but two likely scenarios are possible (Kudela et al., 2005; GEOHAB, 2011). First, this dinoflagellate may be transported in offshore warm water masses that can move onshore under calm conditions. This advection process could potentially result in either a quick increase in PSP toxicity if the number of transported cells is high, or it may simply provide the cells necessary for a bloom to initiate. Second, resting cysts of Alexandrium in local sediments can, under favorable conditions, produce vegetative cells that have the ability to reproduce both sexually and asexually, resulting in localized “hot spots” of PSP toxicity in shellfish. Regardless of the origins of the toxin-producing dinoflagellates, the general pattern has been for these blooms to be detected first along the open coast and in bays (e.g., Drakes Bay), followed by transport into enclosed estuaries (e.g., Drakes Estero) (Price et al., 1991; Langlois and Smith, 2001; Anderson et al., 2008b). The degree to which coastal phytoplankton blooms are found in bays and estuaries depends on the source waters for the bay/estuary (e.g., Banas et al., 2007; Kimbro et al., 2009), consistent with the occurrence of HABs in Drakes Estero following high levels observed in the open waters of Drakes Bay. The depth of penetration into the bay/estuary is controlled by tidal mixing and stratification inside the bay (Largier et al., 1997; Kimbro et al., 2009).

The majority of historical PSP events along the CA coast have occurred in central and northern regions (Price et al., 1991). The most common occurrences were in Drakes Bay off the Marin coast north of San Francisco (CDPH data), which is sheltered from upwelling (Largier, 2004). High-PSP events are most likely to occur either early or late in the upwelling season (i.e., early spring or during the fall), and they typically occur during periods of weaker winds following an upwelling event (unpubl. analysis of CDPH data, S. Piedracoba and J. Largier, pers. comm.).

In general, dinoflagellate blooms in the central and northern CA upwelling area are strongest in the fall (A. Paquin, K. Nielsen, and J. Largier, pers. comm.), when winds are weaker and near-surface thermal stratification can develop (Largier et al., 1993; Garcia-Reyes and Largier, 2012). The highest chlorophyll a levels in waters off Bodega Bay are observed in the fall (Garcia-Reyes and Largier, 2012), during periods of onshore flow (unpubl. mooring data, E. Dever and J. Largier, pers. comm.). During calm periods or southerly winds, warm, low-salinity water that flows out of San Francisco Bay can enhance stratification and fronts along the Marin and Sonoma coasts (Send et al., 1987; Wing et al., 1998; R. Fontana and J. Largier, pers. comm.). In addition, southerly/westerly breezes in fall lead to weak downwelling conditions, which can concentrate dinoflagellate and other upward-motile plankton near to the coast (A. Paquin, K. Nielsen, and J. Largier, pers. comm.), particularly along south-facing coasts like those in Drakes Bay (unpubl. drifter data and High Frequency–Radar surface current data, J. Largier and C. Halle, pers. comm.). The dinoflagellate, A. catenella, is a strong swimmer, in part due to the formation of long chains of cells (Fraga et al., 1988), and it can be expected to be concentrated in buoyancy fronts and downwelling circulation near the coast. From preliminary analysis of PST records, it appears that these events indeed correlate with large-scale oceanographic events, such as the upwelling-relaxation cycle (B. Keafer, D. Anderson, and J. Largier, pers. comm.), and during onshore flow. These results suggest that blooms are accumulated by interactions with coastal flows during calm or downwelling periods, whether initiated offshore or inshore.

In an embayment such as Drakes Bay, which is sheltered from upwelling and receives low-salinity outflow from San Francisco Bay, stratification is more persistent (Largier, 2004), presumably an important factor in the frequent occurrence of high PST levels there. High PST concentrations observed in contiguous estuarine environments (Drakes Estero) occur some days after high concentrations are detected on the open coast, consistent with the scenario that PSP outbreaks initiate on the open coast and are subsequently transported into estuaries (Langlois, 2001; Banas et al., 2007; Kimbro et al., 2009). Central San Francisco Bay is not well suited to the development of dinoflagellate blooms owing to strong vertical mixing driven by tides (Cloern et al., 2005), but coastal blooms could be imported to and spread in the Bay during calm periods in the dry season.

The tendency for dinoflagellate bloom concentration and nearshore distributions to occur during weak, southerly, or westerly winds suggests a tendency for blooms to spread northward – currents nearshore off northern and central CA are typically northward and onshore at such times (Largier et al., 2006). An apparent northward spread has been observed during large-scale outbreaks of PST toxicity, with PST events often initiating near San Francisco and extending north to Point Arena (Rogers-Bennett et al., 2012) and beyond, as far north as the CA–OR border (Langlois, 2001), consistent with northward transport of warm waters and planktonic larvae from Drakes Bay (Send et al., 1987; Wing et al., 1998). While bloom populations may find refuge in bays during brief upwelling events, it appears that bloom concentrations dissipate coast-wide during periods of persistent upwelling, presumably due to the twin negative influences of offshore Ekman transport and strong vertical mixing due to surface wind stress (Botsford et al., 2003; Largier et al., 2006).

Although there is no evidence that nutrients due to land runoff are a principal factor in triggering or promoting these blooms (Price et al., 1991; Langlois and Smith, 2001; Anderson et al., 2008b), new work on phytoplankton productivity in ammonium-rich outflow
from San Francisco Bay re-opens this question; e.g., the idea that elevated ammonium levels preclude nitrate uptake by diatoms, allowing dinoflagellates to bloom (Wilkerson et al., 2006; Dugdale et al., 2007; Gilbert, 2010; Gilbert et al., 2011). A recent summary of nutrient use by harmful algae in upwelling systems, however, suggests that while *Alexandrium* may prefer ammonium as a nitrogen source, chain-forming HABs (including *Alexandrium*) also are well adapted to use upwelling-derived nitrate (Kudela et al., 2010). The relative influences of upwelled vs. anthropogenic nutrients on *Alexandrium* bloom properties (e.g., initiation, magnitude, duration) in coastal areas receiving high nutrient loads (e.g., San Francisco Bay) is unresolved.

2.3.7. Mexico

The best studied toxic species present in Mexican coastal waters is *G. catenatum*, but its ecology is still not well understood (Band-Schmidt et al., 2010). There is general agreement that HABs have increased in recent years (Hernández-Becerril et al., 2007; Cortés-Altamirano and Sierra-Beltrán, 2008) and *G. catenatum*-related blooms are no exception (Band-Schmidt et al., 2010); however, the increase in *G. catenatum* blooms along the Mexican Pacific coast does not appear to be related to anthropogenic activities. For example, extensive aquaculture and agriculture activities are present on the east coast of the Gulf of California, but there is no clear evidence that PST-producing blooms have increased due to pollution or nutrient runoff from these activities (Flores-Trujillo et al., 2009). Blooms of *G. catenatum* in the Gulf of California appear to be related to other environmental forcing factors. It is recognized that blooms occur more frequently in late winter and early spring when upwelling events are present in the south part of the Gulf (reviewed by Band-Schmidt et al., 2010). Moreover, the paleographic record of absolute and relative abundances of the resting cyst of this species (as an indicator of its abundance in the water column) is correlated to sea surface temperatures (Flores-Trujillo et al., 2009). Therefore, in the southern part of the Gulf of California this species seems to respond to interdecadal forcing phenomena, being more abundant in La Niña than in El Niño warm conditions (Flores-Trujillo et al., 2009).

3. *Pseudo-nitzschia* ssp. and domoic acid poisoning

3.1. Overview of toxicity, history on North American west coast

The genus *Pseudo-nitzschia* (mostly reported before ~1990 as *Nitzschia seriata* P.T. Cleve) has been present on the west coast since at least the 1920s (Fryxell et al., 1997). Several species of the diatom genus, *Pseudo-nitzschia*, produce domoic acid (DA), a toxin causing amnesic shellfish poisoning (ASP). Of the 12 species of *Pseudo-nitzschia* known to produce DA, 10 have been reported from west coast waters (Horner et al., 1997; Anderson et al., 2008a). The species, *Pseudo-nitzschia australis* Frenguelli and *Pseudo-nitzschia multiseries* (Hasle) Hasle, are most commonly associated with toxic events throughout this region, with *Pseudo-nitzschia pseudodelicatissima* (Hasle) Hasle, and *Pseudo-nitzschia cuspidata* (Hasle) Hasle also implicated in toxic events in WA waters (Adams et al., 2000; Trainer et al., 2009a). Amnesic shellfish poisoning results in gastrointestinal and neurological disorders within 24–48 h of consumption of toxic shellfish by humans, and can be life-threatening (Perl et al., 1990; Teitelbaum et al., 1990; Jeffery et al., 2004; Goldstein et al., 2008; Lefebvre and Robertson, 2010). The disease can lead to short-term memory loss that may become permanent. Some symptoms are similar to other diseases and thus lead to misdiagnoses.

Shellfish toxicity due to DA was discovered in 1987 in Canada, when three people died and 105 became ill from eating contaminated cultivated blue mussels from Prince Edward Island in the Gulf of St. Lawrence (Bates et al., 1989). Since then, however, most of the reported DA events have occurred on the U.S. west coast. The first documented outbreak on the west coast occurred in 1991, causing the deaths of dozens of brown pelicans (*Pelecanus occidentalis Linnaeus*) and Brandt’s cormorants (*Phalacrocorax penicillatus Brandt*) in Monterey Bay, CA (Fritz et al., 1992; Work et al., 1993) and contaminating razor clams and Dungeness crabs in WA, OR, and northern CA (Weckel et al., 1994). In southwest WA alone, crab fishing losses were estimated at $7 million. It was originally thought that 25 human illnesses in WA were attributable to ASP in the 1991 event (Washington Department of Fish and Wildlife, 2004). None of these illnesses were ever officially confirmed and no mortalities occurred (Quick, 1992).

After these early ASP events, monitoring efforts and regulations to prevent harvest of toxin-contaminated shellfish have succeeded in preventing human incidents of ASP, but numerous cases of DA toxicity of finfish, marine mammals, and birds have been documented (Landsberg, 2002; Shumway et al., 2003; Goldstein et al., 2008; Fire et al., 2010; Bargu et al., 2012). A number of shellfish and finfish have been reported as potential vectors of the disease, including razor clams, blue mussels, Pacific littleneck, geoduck, and manila clams, Pacific oysters, Dungeness, rock, and pelagic red king crab, sprint lobster viscera, Pacific sardines (Sardinops sagax Jenyns), northern anchovies (Engraulis mordax Girard), krill (*Euphausia pacifica* Hansen, Thyasoea spinfera Holmes), market squid (*Loligo opalescens* Berry), and benthic invertebrates (Bargu et al., 2002, 2008, 2010, 2012; Landsberg, 2002; Lefebvre et al., 2002; Shumway et al., 2003; Schnetzer et al., 2007; Kvitek et al., 2008; Mazzillo et al., 2010). In addition to commercially harvested species, many other animals can accumulate DA, leading to widespread transfer through marine food webs (e.g., Lefebvre et al., 2002; Bargu and Silver, 2003; Bargu et al., 2008; Kvitek et al., 2008; Mazzillo et al., 2010). Death or strandings have been reported in California sea lions (*Zalophus californianus* Lesson), northern fur seals (*Callorhinus ursinus* Linnaeus), harbor porpoises (*Phocoena phocoena* Linnaeus), common dolphins (*Delphinus delphis* Linnaeus), sea otters, gray whales (*Eschrichtius robustus* Lilljeborg), minke whales (*Balaenoptera acutorostrata* Lacepede), brown pelicans, Brandt’s cormorants, ruddy ducks (*Oxyura jamaicensis* Gmelin), and western grebes (*Aechmophorus occidentalis* Lawrence) (Work et al., 1993; Scholin et al., 2000; Landsberg, 2002; Shumway et al., 2003; Goldstein et al., 2008; Fire et al., 2010; Bargu et al., 2012; D. Caron, pers. obs.).

3.2. Trends in prevalence and impacts

During the last 15 years, numerous blooms of *Pseudo-nitzschia* ssp. have been reported, and associations between DA and animal deaths and illnesses frequently documented (Scholin et al., 2000; Gulland et al., 2002; Trainer and Hickey, 2003; Goldstein et al., 2008). The next exceptional and widespread event after 1991 occurred in 1998, when marine mammal deaths attributed to DA were first reported (e.g., 81 California sea lions from San Luis Obispo to Santa Cruz), and high levels of DA were measured in WA and OR razor clams (*Adams et al., 2000; Scholin et al., 2000*). In CA, DA outbreaks have occurred in almost every year over the last decade (CDPH data, Fig. 8C) and increasingly south of Point Conception. DA levels associated with *Pseudo-nitzschia* blooms were exceptionally high in coastal OR and CA in 2010 (CDPH data, ODA and ODFW data at http://bioweb.coas.oregonstate.edu/~mocha/odadata.html). Off OR, *Pseudo-nitzschia* cell counts were as high as 10^6 cells/l in June 2010, associated with elevated DA levels in razor clams that led to harvesting closures. In CA, DA levels in Monterey Bay during fall 2010 were exceptionally high in water (dissolved and particulate) and California mussels.
3.2.1. Alaska

The AHAB (Alaska HAB) program monitors the occurrence of several HABs including *Pseudo-nitzschia* blooms in southeast AK and the Homer region, and is beginning to work with the State of Alaska to implement screening methods for the measurement of DA in recreationally harvested shellfish. The Alaska Department of Environmental Conservation (ADEC) is the only agency responsible for marine biotoxin testing in shellfish, with sampling restricted to commercially harvested and aquaculture products. The ADEC tests between 600 and 700 samples annually; however, testing is primarily conducted on Pacific oysters and geoduck clams, both poor candidates for monitoring DA. An intensive ADEC study between 1992 and 1996 tested for DA in 5123 individual molluscs and found measurable levels (ADEC Database, unpubl. data, R. RaLonde, pers. comm.). The study, however, was limited in scope to locations and times where commercial mollusc harvest and farming occurred, and the highest DA concentration measured was 18.8 ppm. Testing for DA of 4262 commercially harvested Dungeness crabs showed highest DA concentration of 1.37 ppm in a snow crab harvested in the Aleutian/Bering Sea commercial fishery. A monitoring program, funded by the North Pacific Research Board to determine the occurrence and geographical distribution of DA in shellfish, conducted intensive sampling at Annette Island and Sea Otter Sound, both near Ketchikan, Sitka Sound, eastern Prince William Sound, Kachemak Bay, and Unalaska, with additional opportunistic sampling along the entire coast (RaLonde and Wright, 2011). DA concentrations were near the undetectable level.

Phytoplankton monitoring in AK has historically concentrated on the spring bloom period, primarily to understand the influence...
of ocean productivity on marine fish survival. Because *Pseudo-nitzschia* is not a major component of the spring bloom, data on the locations, timing, and intensity of *Pseudo-nitzschia* blooms are rare, but see Schandelmeier and Alexander (1981) for early data from the Bering Sea. Phytoplankton sampling as part of research programs in AK waters (GLOBEC, Global Ocean Ecosystem Dynamics; BEST, Bering Sea Ecosystem Study) has shown that *Pseudo-nitzschia* is widespread on the Gulf of Alaska and Bering Sea shelves and can reach high abundances. For example, in June 2010 near St. Lawrence Island, large-sized *Pseudo-nitzschia* spp. were present at >10^4 cell/l (E. Lessard, unpubl. data). The ADHHS-ES database has no record of illness from DA. Since AK has little data from DA monitoring other than screening commercially harvested product, and because toxin illnss is generally underreported, the actual impact of DA on human and marine ecosystem health is unknown (Gessner and Middaugh, 1995).

3.2.2. British Columbia

British Columbia began monitoring for DA after the 1987 outbreak on the east coast of Canada and currently analyzes approximately 3000 blue mussel and razor clam samples for DA each year (Canadian Food Inspection Agency data). Domoic acid is rarely detected in samples in BC and no confirmed illnesses due to DA have been reported. The highest level of DA detected since 1994 was 106 ppm on the west coast of Vancouver Island in Port Eliza, off Nootka Sound, during March 2002 (Fig. 2). Blooms of *Pseudo-nitzschia* have been documented along the west coast of Vancouver Island and on the north end of the Haida Gwaii (Forbes and Denman, 1991).

On the south coast of BC, only 10 blue mussel samples, all collected on the west coast of Vancouver Island, have had measurable levels of DA above the action level of 20 ppm since 1994 (Canadian Food Inspection Agency data). Domoic acid is rarely seen in samples collected in the Strait of Georgia and the levels have not exceeded 6 ppm. There does not appear to be any regularity to the seasonality of DA detection in monitoring samples on the south coast (D. Kelly, pers. obs.).

The highest level of DA detected on the north coast of BC was 37 ppm found in razor clams in August 1995 (Canadian Food Inspection Agency data). Detectable levels of DA are found most years on the Haida Gwaii; however, only during the September 1995 to May 1996 period were the levels greater than the action level of 20 ppm (Fig. 2). Blooms tend to start developing in the spring/summer months on the north coast.

Populations of *Pseudo-nitzschia* spp. are usually a minor component of the phytoplankton community off the southwest coast of Vancouver Island in late summer, but there is substantial variability and species may occur throughout the year (Forbes and Denman, 1991). Species are also found in the Strait of Georgia and north of Vancouver Island. They are generally found in water temperatures between 8 and 14 ºC and salinity between 30.0 and 32.5 with reduced concentrations of dissolved inorganic nutrients, particularly silicate.

3.2.3. Washington

Toxic *Pseudo-nitzschia* blooms have caused severe economic losses for coastal WA (including tribal) communities from beach and shellfish harvest closures (Fig. 3). Impacted areas span the coast, with a demonstrated “hot spot” in the Juan de Fuca eddy area, where *Pseudo-nitzschia* blooms occur relatively frequently, associated with high DA concentrations (Trainer et al., 2002; MacFadyen et al., 2005). This area is a chronic upstream source of toxic *Pseudo-nitzschia* for the WA coast (Trainer et al., 2002, 2009a; Trainer and Hickey, 2003; Marchetti et al., 2004; MacFadyen et al., 2005). Results from six cruises conducted over four years suggest that toxin occurs in the eddy in any 21-d period, although razor clam beaches have clamping closures due to toxin roughly every two years (MacFadyen et al., 2005). For toxin to reach coastal beaches, toxic patches must first develop, the patches must escape the eddy and travel downcoast toward the clamping beaches, and the patches must move onto coastal beaches, remaining long enough to be ingested by the clams. Recent research shows that concentration, as well as onshore transport, occurs during periods of downwelling winds (storms), whereas escape from the eddy and travel downcoast occurs during periods of upwelling winds (MacFadyen and Hickey, 2010).

The relatively few toxic events on Washington’s coastal beaches in comparison to the regular appearance of DA in the source regions suggests that significant impediments to transport occur between source regions and the beaches. One important impediment may be the buoyant plume from the Columbia River estuary (Hickey et al., 2010). A plume from the Columbia is generated along the WA coast north of the estuary mouth each time winds on the WA coast switch to a northward direction (a storm period) (Hickey et al., 2005). The plume has density fronts along its edges and such fronts can be a barrier to onshore transport of toxic blooms (Hickey and Banas, 2003). A new study led by the University of Washington (PNWTOX, “Pacific Northwest Toxins”) is using numerical models of coastal circulation and also ecosystem variability in association with previously collected data to determine the effects of the Columbia on HABs for both OR and WA coasts.

Exceptional years of DA-associated beach, razor clam, and Dungeness crab closures in WA include 1991, 1998–1999, 2002–2003, and 2005 (see Fig. 1 in MacFadyen et al., 2005, Fig. 8A). In 1991, closure of beaches to recreational and commercial razor clam and Dungeness crab harvesting resulted in a $15–20 million revenue loss to fisheries (Anderson, 1995). During 1998–1999 (over a year and a half), fishery closures caused Washington’s Quinault tribe to lose all of their razor clam income and a large portion of their Dungeness crab income, and the Quileute tribe to lose 50% of their Dungeness crab income (Wekell and Trainer, 2000). From 2002 to 2003, another prolonged closure period (>1.5 years) resulted in a $10.4 million loss in revenue (Wekell and Trainer, 2002). In 2005, toxic blooms of *P. pseudodelicatissima* and *P. australis* caused significant commercial, recreational, and tribal shellfish harvest losses in Sequim Bay and Penn Cove areas of Puget Sound, respectively (Trainer et al., 2007). Although DA producing blooms of *Pseudo-nitzschia* species have been known previously in Puget Sound (e.g., Hood Canal, Horner et al., 1996), blooms with toxin levels above the regulatory limit of 20 ppm have been reported only since 2003 (Bill et al., 2006), causing some concern that continued escalation/ expansion will impact the many valuable fisheries there (Trainer et al., 2007). The total estimated impacts of a hypothetical coast-wide seasonal closure of the recreational razor clam fishery for 2008 was estimated to be $21.9 million, and the income impact of the recreational razor clam fishery in WA for 2008 was estimated at $13.5 million (Huppert and Dyson, 2008, see Section 6).

3.2.4. Oregon

In addition to Washington’s Juan de Fuca area, other chronic sources of toxigenic *Pseudo-nitzschia* (i.e., “hot spots”) include Heceta Bank (Trainer et al., 2001; B. Hickey, pers. comm.; P. Strutton, unpubl. data) and south of the Columbia River estuary (Clatsop) areas of OR (Tweddle et al., 2010). Like WA, the 1998 and 2003 events caused beach closures of razor clam harvesting that lasted >1.5 years and led to a $4.8 million loss in estimated income to coastal communities around Clatsop Beach alone in 2003 (Tweddle et al., 2010; ODFW data, http://public.health.oregon.gov/HealthyEnvironments/Recreation/HarmfulAlgaeBlooms/Pages/index.aspx, Fig. 8B). The impact of DA toxicity on razor clam, mussel species, and Dungeness crab industries appears to be
increasing in recent years, with exceptionally extensive and prolonged razor clam and mussel closures occurring from 2002 to 2006 (Tweddle et al., 2010, Fig. 8B). The Dungeness crab industry in OR has never had a closure due to toxins, but orders for evisceration occurred in 1999 when levels of toxins in S. patula warranted the precaution (ODA data, http://oregon.gov/ODA/FSD/shellfish_status.shtml).

3.2.5. California

Since the 1998 event when DA was first linked to sea lion deaths, toxic blooms and associated mammal and bird illnesses on the CA coast have occurred in nearly every year (CDPH data, Fig. 8C). For example, toxic *Pseudo-nitzschia* blooms have been recorded in the Santa Barbara Channel in every year since 2002, and off Los Angeles in every year since 2003 (Schnetzer et al., 2007; Anderson et al., 2008a; CDPH data). Blooms of *P. australis* off southern CA in 2006 and 2007 were characterized by some of the highest planktonic DA concentrations measured; e.g., 14.39 µg pDA/l on March 17, 2006 just inside and 26.97 µg/l on April 26, 2007 just outside the breakwater at Long Beach (A. Schnetzer et al., unpubl. data, Fig. 5). Since 2003, hundreds of marine mammal and bird strandings or deaths from central to southern CA have been attributed to DA, and there is evidence that these poisonings are increasing. For example, Caron (2008) reported DA toxicity in several bird species for the first time in 2006–2007. Spring 2007 was cited as the worst season for marine mammal and bird mortality on the southern CA coast (International Bird Rescue Research Center, http://www.ibrrc.org/pr_04_25_2007.html). The link between upper water column blooms and potential exposure of benthic organisms to DA through rapid downward flux has also been demonstrated through toxin measurements in sediment traps from as deep as 500 and 800 m in the Santa Barbara and San Pedro Channels, respectively (Schnetzer et al., 2007; Sekula-Wood et al., 2009).

Domoic acid has been detected in seafood species along the CA coast (bivalve shellfish, sardines, anchovies) almost every year since the 1991 epidemic (CDPH data). Concentrations of DA exceeding the federal public health alert level (20 ppm) have been detected in seafood species every year between 2000 and 2007, primarily between San Luis Obispo and Los Angeles counties. In 2007, a DA concentration of 610 ppm was detected in California mussels from Santa Barbara, the highest level ever recorded in CA (Fig. 8). The most persistent problems with elevated DA concentrations have been in the Santa Barbara- Ventura region, extending offshore to the Channel Islands.

Based on examination of 715 sea lions with neurological symptoms collected between 1998 and 2006, Goldstein et al. (2008) identified two DA syndromes, an acute DA toxicity and a chronic epileptic syndrome. Clusters of strandings of acute syndrome cases occurred in 1998 (centered in Monterey Bay), 2000, 2001, 2002, and 2005 (centered off San Luis Obispo and Santa Barbara counties). While an increasing trend in acute cases from 1998 to 2006 was not found, chronic cases were found to increase in every year, from four cases in 1999 to 45 cases in 2006. These data indicate that chronic effects of DA on sea lions have continually increased in recent years. Bargu et al. (2012) also found evidence for increasing chronic cases over time in their examination of 82 sea lions stranded in Monterey Bay from 2004 to 2007.

3.2.6. Mexico

Domoic acid detection and the presence of potentially toxic *Pseudo-nitzschia* species have been documented in Pacific waters off Mexico. Of the 12 species identified as potential producers of DA (Moestrup and Lundholm, 2007), *P. australis, Pseudo-nitzschia delicatissima* (Cleve) Heiden, *Pseudo-nitzschia fraudulenta* (Cleve) Hasle, *P. multiseries* and *Pseudo-nitzschia pungens* (Grunow ex Cleve) Hasle have been reported in Pacific Mexico (Hernández-Becerril et al., 2007). Only two DA-associated blooms have been documented (Fig. 6). Domoic acid was detected in net phytoplankton samples and in chocolate clams (*Megapidaria squalida* Sowerby) during a *P. fraudulenta* bloom in June-July 2006 at La Paz, in the Gulf of California, but the concentration of DA in the clams (0.55 ppm) was well below the action limit for shellfish (Gárate-Lizárraga et al., 2007). Another toxic bloom occurred in the Todos Santos Bay in 2007, in the northern part of the Baja California Peninsula (García-Mendoza et al., 2009). Here, the maximum DA concentration in particulate matter (0.86 ppm) was associated with the presence of *P. australis* that reached a maximum abundance of 3.02 × 10^6 cells/l (García-Mendoza et al., 2009). Adverse biological effects related to DA in the environment were not detected during these events.

There are only two incidents where DA toxicity of animals was confirmed in Mexico, both occurring in the Gulf of California (Fig. 6). The deaths of approximately 150 brown pelicans during the winter of 1996 at the tip of the Baja California Peninsula were associated with DA toxicity after the animals consumed contaminated mackerel (Scomber japonicus Houttuyn; Sierra-Beltrán et al., 1997). Another massive mortality of sea birds and marine mammals associated with DA poisoning occurred in the winter of 1997 when 766 sea birds (common loon, *Gavia immer* Brunnich), 168 dolphins, nine sea lions, and four fin whales (*Balaenoptera physalus* Linnaeus) died. This is the only such event described in an official technical report issued by SEMARNAP (Former Mexican Secretary of the Environment and Natural Resources) (SEMARNAP-PROFEPA, 1997). The dead animals were dispersed in several locations on the east coast of the Gulf of California. The responsible species was not identified.

The limited number of DA field measurements or confirmed DA poisonings does not necessarily indicate that blooms associated with the toxin are unusual in Mexico. Other events consistent with DA toxicity have been identified, for example, mass mortalities of sea mammals and birds in 1995 and 2004 in the Gulf of California; however, no hard data were provided to corroborate the cause of mortality (Ochoa et al., 2002). Anecdotal evidence from the northern part of the Baja California Peninsula also suggests that some sea lion strandings might have been related to DA toxicity. In 2002, 87 sea lions were found stranded on beaches from Tijuana to Ensenada, and it was assumed that DA toxicity was the cause of this event (Hernández-Becerril et al., 2007). Also in 2006, nine corpses and three sea lions with symptoms of DA toxicity were found near Ensenada, Northern Baja California (media report CP052-06 issued by SEMARNAT-PROFEPA; http://www.profepa.gob.mx/). These findings indicate that DA toxicity might be a recurrent phenomenon for the northern part of the Baja California Peninsula. Further evidence suggests that DA occurrence in this region may be on the rise. Domoic acid was not reported in the northern region of the Baja California peninsula before 2007 (García-Mendoza et al., 2009). Domoic acid content in Pacific sardines (*S. sagax caerulea* Jenyns) collected every two weeks from December 2007 to February 2009 was relatively high (>100 ppm viscera) during winter of 2007–2008 and from July to August of 2008 (G. Cabrales-Talavera and E. García-Mendoza, unpubl. data). Also, some positive samples were detected in April 2008, but the concentration was <20 ppm in viscera (G. Cabrales-Talavera and E. García-Mendoza, unpubl. data). Furthermore, during the last week of August and through September of 2010, sardines collected near the Todos Santos Bay region had DA concentrations as high as 800 ppm in viscera (E. García-Mendoza, unpubl. data) and in September of 2011, the first ban in the region associated with the presence of DA in shellfish was implemented by COFEPRIS.
3.3. Factors promoting blooms

3.3.1. Overview: anthropogenic vs. natural factors

At present, there appears to be little evidence to support anthropogenic nutrient loading as the primary promoter of *Pseudo-nitzschia* blooms along the west coast of North America. Blooms often occur in offshore areas, e.g., the Juan de Fuca eddy off WA and Heceta Bank off OR (Hickey and Banas, 2003; MacFadyen et al., 2005), where anthropogenic influence is neither expected nor found. Even in the Southern California Bight, the most highly populated area along the U.S. west coast, *Pseudo-nitzschia* abundances and DA concentrations were higher at offshore stations and were not associated with higher nutrient concentrations at coastal stations where Toxic bloom levels were inversely correlated with nutrient levels (Schnetzer et al., 2007). Also, high concentrations of the micronutrients Cu and Fe, generally linked to anthropogenic activities (e.g., Johnson et al., 2001) were inversely related to DA production in laboratory and field studies (Rue and Bruland, 2001; Trainer et al., 2009b). Urea might also be a N source for *Pseudo-nitzschia* and would have an anthropogenic source (Cochlan et al., 2008). There is no direct evidence, however, that *Pseudo-nitzschia* blooms are related to run-off or eutrophication. Instead, they are more likely related to large scale physical forcing such as upwelling that brings in high quantities of macronutrients (Kudela et al., 2004; Anderson et al., 2008b; MacFadyen et al., 2008).

Although anthropogenic sources of nutrients have not been clearly linked to *Pseudo-nitzschia* blooms on the WA open coast, in smaller, more encased areas and embayments in Puget Sound and along the Strait of Juan de Fuca, anthropogenic factors might play a role in promoting blooms. For example, a bloom of *P. pseudodecactisima* in Sequim Bay, WA, in September 2005 was attributed in part to a pulse of ammonium from an outdated sewage system at a state park (Trainer et al., 2007). A bloom of *P. australis* occurring at roughly the same time at nearby Penn Cove, WA, was attributed to water column stratification caused by high precipitation, high stream flow, and strong local winds (Trainer et al., 2007), similar to the causes of a bloom in the same area in 1997 (Trainer et al., 1998).

Laboratory studies have demonstrated that several *Pseudo-nitzschia* species are able to use both organic and inorganic nitrogen sources for growth, and there is often an uptake preference for ammonium that can be derived from both natural and anthropogenic sources (Howard et al., 2007; Cochlan et al., 2008; Kudela et al., 2008a, 2010). Cochlan et al. (2008) suggest that anthropogenic sources such as ammonium and urea may sustain non-bloom concentrations of *Pseudo-nitzschia* during periods of relaxed upwelling when low nitrate concentrations can be expected within 10–20 km of the coastline (MacFadyen et al., 2008). The potential remains for anthropogenic sources to promote or sustain blooms for inland waters such as Puget Sound and along the Strait of Juan de Fuca, or may be linked to the possible spread of toxic HAB species into new areas although proof is still lacking (Anderson et al., 2008b).

3.3.2. Alaska

Species of *Pseudo-nitzschia* occur along the whole AK coast from at least Point Barrow in the north (Bursa, 1963; R. Horner, pers. obs. as *N. seriata*), throughout the Bering Sea (Schandelmeier and Alexander, 1981 as *Nitzchia* spp., section *Pseudo-nitzschia*), and into the Gulf of Alaska, including along the Aleutian chain (Cupp, 1943) and into south central AK at Port Valdez (Horner et al., 1973), all as *Nitzchia* spp. Note that species identifications may not always be correct because they were made well before the need to use electron microscopy for this purpose was recognized. The same oceanographic factors discussed above for PSTs (Section 2.3.2) probably also affect the distribution of *Pseudo-nitzschia* species.

The sparse and widespread population areas preclude the possibility of much anthropogenic influence.

3.3.3. British Columbia

Species of *Pseudo-nitzschia* are present and often abundant in all BC marine waters in summer and fall, with the largest blooms occurring on the outer continental shelf (Forbes and Denman, 1991; Taylor and Harrison, 2002). These may be advected into coastal inlets such as Barkley Sound (Taylor and Haigh, 1996). Further, blooms of *Pseudo-nitzschia* spp. in BC usually occur first in more southerly regions before more northerly ones (Taylor and Harrison, 2002).

3.3.4. Washington

Bloom of toxic *Pseudo-nitzschia* on the WA coast in summer and fall are generally associated with a recent history in the Juan de Fuca eddy and a retentive region just offshore of the Strait of Juan de Fuca (Trainer et al., 2009a), and are more related to physical forcings than to anthropogenic factors. This region is known for its high concentration of chlorophyll in summer (Hickey and Banas, 2008); i.e., the region is favorable to phytoplankton blooms in general, but *Pseudo-nitzschia* is typically less than 20% of the phytoplankton community by biomass (Trainer et al., 2009a). The factors contributing to phytoplankton blooms in this region include a steady source of high nutrient waters, a circulation pattern that favors retention rather than loss, and low grazing relative to growth rates (MacFadyen et al., 2005, 2008; Foreman et al., 2008; Olson et al., 2008; MacFadyen and Hickey, 2010). As in other portions of the California Current System, the high nutrient waters are derived from large-scale seasonal upwelling of deep coastal waters. In the Juan de Fuca eddy region however, the upwelled waters travel up the coastal canyon system into the Strait of Juan de Fuca, where they mix and then exit back out as surface waters into the eddy region, providing as much nitrate as that due to traditional upwelling along the entire WA coast (see Table 1 in Hickey and Banas, 2008). In addition, water is upwelled directly into the eddy as the season progresses (MacFadyen et al., 2008). Thus, in contrast to the open WA coast, the nutrient source does not disappear during periods of wind relaxation or wind direction reversal to downwelling-favorable. Moreover, nutrients passing through the Strait are transported much farther offshore than occurs via traditional coastal upwelling (~50 km vs. 10 km) (Hickey and Banas, 2008). Although the high nutrient concentrations favor phytoplankton growth in general, no significant relationship was found between toxin production and macronutrient supply (Trainer et al., 2009a). Significant DA concentrations were observed on six 21-d surveys over four years, but the factors that determine the transition from toxigenic to toxic have not been determined to date (Trainer et al., 2009a; B. Hickey, unpubl. data).

The timing, frequency, and magnitude of toxic *Pseudo-nitzschia* blooms impacting the WA coast in summer/fall is therefore largely a function of the dynamics of the Juan de Fuca eddy. The eddy forms in the spring transition period, increases in spatial extent throughout the summer, and decreases in the fall. Under the typical northerly wind conditions, the eddy is “leaky” to the south, but under weak wind conditions or when southerly wind reversals occur, the eddy is more retentive. These conditions promote maintenance of high abundances of *Pseudo-nitzschia* and also their shoreward advection.

The influence of wind conditions (timing, speed, direction, magnitude) on the Juan de Fuca eddy circulatory patterns is a critical determinant of toxic *Pseudo-nitzschia* bloom impact, and thus is a focus of bloom forecasting models, including the longterm effects of climate change (e.g., storminess). In spring, high concentrations of pDA in razor clams are more likely to originate from southern sources, such as Heceta Bank, OR (Hickey et al.,
2010; see below). This is because regional coastal currents are toward the south in summer/fall, but toward the north in early spring.

3.3.5. Oregon

Analogous to the Juan de Fuca eddy off WA, Oregon’s Heceta Bank is a “hot spot” for DA. It is the dominant bathymetric feature off the mid-OR coast that enhances retention of highly productive waters, and may provide a source of toxic *Pseudo-nitzschia* blooms to the OR nearshore coastal zone and to the southern WA coast in spring (B. Hickey, pers. comm.). Like the Juan de Fuca eddy, southerly winds result in more retention and also shoreward advection of nutrients and phytoplankton (Barth, 2003; Barth et al., 2005). The northern OR coast, in particular the Clatsop Beach region, experiences higher cell counts of *Pseudo-nitzschia* and greater instances of increased DA levels in shellfish, compared to southern regions of OR (Tweddle et al., 2010). Upwelling effects, however, are not greatly different between Clatsop and the Tillamook region immediately to the south. This suggests that the increased toxins at Clatsop may be related to the presence of the Columbia River plume, possibly a result of enhanced retention in this region. High concentrations in summer and fall could also be a result of southward advection from the Juan de Fuca eddy. Domoic acid contamination of shellfish along the OR coast most closely corresponds to periods of transition from upwelling to downwelling (Tweddle et al., 2010) and not to any anthropogenic input.

3.3.6. California

*Pseudo-nitzschia* spp. are common in CA waters, but major toxin events occur only at specific times, although they often occur over large spatial scales (e.g., Southern California Bight to Monterey Bay, Trainer et al., 2000) and may persist for weeks. Of interest here are not only the factors that control the population bloom, but also the factors that control the large-scale production of DA. Following Section 3.2.5, the present-day widespread and annually occurring DA problem in CA appears to have only emerged in the last decade. Specifically in southern CA, Lange et al. (1994) considered toxigenic blooms both rare and unusual, but in recent years ASP has become increasingly important (e.g., Trainer et al., 2000; Anderson et al., 2006; Busse et al., 2006; Schnetzer et al., 2007). While DA outbreaks are most common in the sheltered waters in the Southern California Bight (including the Santa Barbara Channel) and Monterey Bay, they also occur along the open coast of central CA (south of Monterey Bay) but are not regularly found in the sheltered waters of Drakes Bay (or on the open coast north of Monterey Bay). Given the retentive and stratified nature of Monterey Bay and the Santa Barbara Channel, these regions may act as source regions comparable with the Juan de Fuca Eddy and Heceta Bank regions described for WA and OR.

The marked shift to DA events in recent years in southern CA may be related to changes in the oceanographic climate. For example, there was a significant change in ocean climate in the eastern Pacific in 1999 as both the PDO and North Pacific Gyre Oscillation (NPGO) reversed sign in a manner that would enhance upwelling effects off central and southern CA. Just as +PDO may correspond with higher DA off cooler OR (see Section 3.3.5), so PDO and +NPGO may correspond with higher DA off warmer southern CA (Sekula-Wood et al., 2011). Although this –PDO/+NPGO period was interrupted by anomalous years in 2005 and 2006, the tendency for cooler conditions has continued to the present. The possibility of large-scale ecosystem change since 1999 is supported by recent studies that document a dramatic and persistent response in demersal fish, crab, and shrimp populations in San Francisco Bay (Cloern et al., 2010).

As noted in Section 3.3.1, *Pseudo-nitzschia* blooms are most likely related to large-scale physical forcing that brings in excess quantities of macronutrients (Kudela et al., 2004; Anderson et al., 2008b; MacFadyen et al., 2008), with outbreaks typically occurring following upwelling events as nutrients become less available to a well-developed multi-species phytoplankton bloom (Kudela et al., 2004). The argument for DA events being controlled by large-scale oceanic forcing is based on the apparent synchrony of ASP events observed in CA. Multiple factors have been shown to trigger the production of DA by *Pseudo-nitzschia* (cf. reviews by Bates et al., 1989; Bates, 1998, 2000; Bates and Trainer, 2006; Trainer et al., 2008), but the most thoroughly characterized is macronutrient limitation by either phosphate or silicate in cultures (Pan et al., 1996a,b,c). More recently, Anderson et al. (2006) reported a correspondence between limiting Si concentrations indexed by the ratios of Si(OH)₄: NO₃ and Si(OH)₄:PO₄ in *Pseudo-nitzschia* and particulate DA; however, they concluded that the relationship is complex, with added variability caused by mesoscale circulation (see also review by Kudela, 2008).

A link between ASP events and land runoff has been postulated (e.g., that the massive DA event in Monterey Bay in 1998 was triggered by post-El Niño runoff, Scholin et al., 2000), but the evidence remains circumstantial and the relationship between ASP and coastal runoff and/or eutrophication remains unclear. In addition to the possible importance of land-derived macronutrients, the micro-nutrients in land runoff may be critical. Recent laboratory and field data suggest that *Pseudo-nitzschia* may increase toxicity when growing on urea as a nitrogen source (Howard et al., 2007; Kudela et al., 2008a), a source of N without a concomitant source of Si. Urea is primarily from anthropogenic sources and thus cultural eutrophication may have the unanticipated consequence of both selecting for *Pseudo-nitzschia* spp. and promoting toxin production in this organism. In addition, DA production by *Pseudo-nitzschia* spp. has also been linked to Fe and Cu stress. Iron limitation directly modulates Si:N ratios in diatoms, and DA may serve as an Fe-acquisition mechanism either directly (Rue and Bruland, 2001; Maldonado et al., 2002) or through the stimulation of a Cu-mediated high affinity transport system (Wells et al., 2005). Anthropogenic changes in runoff amounts and timing, and Fe or Cu loading (e.g., Johnson et al., 2001; Ladizinsky, 2003) thus may have amplified effects on coastal waters by triggering or suppressing DA outbreaks.

3.3.7. Mexico

Water properties associated with wind-driven upwelling were related to the accumulation of *P. australis* cells in the Todos Santos Bay region (García-Mendoza et al., 2009). As in more northern locations (Trainer et al., 2000; Taylor and Trainer, 2002; Anderson et al., 2006), the injection of nutrients to upper layers associated with upwelling events appears to promote the formation of toxic *Pseudo-nitzschia* blooms in the Baja California northern region (García-Mendoza et al., 2009). Specifically, it was documented that a high Si(OH)₄:N ratio instead of the absolute concentration of each nutrient was an important factor for the accumulation of *P. australis* (García-Mendoza et al., 2009). Toxic blooms of *Pseudo-nitzschia* have been related to prominent oceanographic features such as eddies, fronts, and upwelling events (GEOHAB, 2005). The west coast of Baja California has a number of these mesoscale oceanographic features that could offer the conditions that encourage the growth of toxigenic diatom species; e.g., upwelling conditions that occur in late spring and in summer in the Mexican part of the California Current System (Hickey, 1998; Pérez-Brunius et al., 2006).

In summary, the generality emerging from observations of *Pseudo-nitzschia* blooms and outbreaks of DA along the North American west coast indicates that these diatom species bloom in response to classical upwelling conditions, potentially along with numerous other species of diatoms and non-diatom taxa. The
specific conditions leading to dominance of *Pseudo-nitzschia* during these blooms, the factors leading to production of DA, and the oceanographic/meteorological conditions leading to exposure of coastal communities to toxic blooms once they have developed are less clear, possibly complex, and clearly in need of further study.

4. *Heterosigma*, *Chattonella*, and fish kills

Blooms of the raphidophyte, *Heterosigma akashiwo* (Hada) Sournia, have been associated with massive fish kills in temperate waters worldwide and are known for their antagonistic effects on organisms with sizes ranging from bacteria to fish (Smayda, 2006). In WA and BC, losses to commercial fisheries, particularly aquaculture, have been substantial since the late 1980s, and concurrently wild fish have been affected (Horner et al., 1997; Taylor and Haigh, 1993; Taylor et al., 1994; Rensel and Whyte, 2003; Rensel et al., 2010b). The mechanism for *Heterosigma* toxicity is not well established. Several modes of toxicity have been proposed and investigated in laboratory settings, including production of brevetoxin-like compounds (Khan et al., 1997; Keppler et al., 2006), mucus or lectin-like polysaccharides (Pratt, 1966; Chang et al., 1990), reactive oxygen species such as superoxide and hydrogen peroxide (Yang et al., 1995; Twiner and Trick, 2000) and hemaglutinating and hemolysing compounds (Onoue and Nozouwa, 1989).

In coastal marine waters of WA and BC, finfish aquaculture kills due to *Heterosigma* began in 1976 near Lummi Island but did not become substantial until the late 1980s (Taylor et al., 1994; Horner et al., 1997; Rensel, 2007; Rensel et al., 2010a,b). A major kill of coho salmon (*Oncorhyncus kisutch* Walbaum) and chinook salmon (*Oncorhyncus tshawytscha* Walbaum) occurred in Sechelt Inlet, BC, in 1986 resulting in a loss of approximately 1/3 of the salmon population and $2.5 million in revenue. In 1989, another massive bloom led to a $4 million loss of caged chinook salmon in BC and another $4 million of the same species at Cypress Island, WA. Aquaculture fish losses were also severe in central Puget Sound in 1990, when 1.3 million fish and $5 million revenue were lost (85–100% population losses per pen of Atlantic salmon, *Salmo salar* Linnaeaus), including an endangered species, wild White River spring chinook (*O. tshawytscha* Walbaum) brood stock. Widespread blooms in 2006 ($2 million loss) and 2007 also caused substantial losses of farmed Atlantic salmon (*S. salar* Linnaeaus) (Rensel, 2007; Rensel et al., 2010b).

Mortalities of wild salmon and marine fish species associated with *Heterosigma* blooms have been documented since 1994 in Puget Sound (Hershberger et al., 1997; Horner et al., 1997; Rensel, 2007; Rensel et al., 2010b), particularly in shallow, warm bays where the dead fish are more visible and likely to float and accumulate on beaches than in the colder main basins (Rensel et al., 2010a). More recently, evidence has been found linking *Heterosigma* blooms in the Strait of Georgia and North Puget Sound to a 2-decade decline of a key stock of Fraser River sockeye salmon (*Oncorhynchus nerka* Walbaum), historically the most valuable west coast Canadian and U.S. salmon fishery (Rensel et al., 2010a,b). Since 1989, marine survival of Chilko sockeye salmon stock averaged 2.7% in years when juvenile sockeye salmon seawater migration in the Strait coincided with major *Heterosigma* blooms vs. 10.9% in years with no or minor blooms. Strong correlations were also seen between major *Heterosigma* bloom years and young-of-the-year Pacific herring (*Clupea pallasi* Valenciennes) abundance a few months later. A panel of U.S. and BC fisheries experts assembled by the Pacific Salmon Commission reviewed the evidence (i.e., Rensel et al., 2010a,b) and concluded that at least some years of very poor Fraser River sockeye salmon returns, such as 2009, were likely due to *Heterosigma* blooms and that future risk necessitates the need to further study the issue (Peterman et al., 2010).

Although *Heterosigma* blooms have been reported on the CA coast, impacts to fisheries have only been documented in WA and northward. This may be related to the relative lack of year-round marine finfish aquaculture facilities in affected CA regions (e.g., San Francisco Bay, Monterey Bay, and Southern California).

The presence of *Heterosigma* has been reported from the Gulf of California (Band-Schmidt et al., 2004), but noxious effects have not been associated with this genus off Mexico; however, blooms of other raphidophyceans have caused massive fish mortalities in the Gulf of California. Specifically, in April 2003, a bloom of *Chattonella marina* (Subrahmanyan) Har & Chihara and *Chattonella cf. ovata* Y. Har & M. Chihara was associated with massive mortalities of fish, although it was not possible to evaluate the magnitude of the impact (Nuñez-Vázquez et al., 2011 and references therein). Benthic fauna were killed in Kun Kan Bay, Sonora, located on the east coast of the Gulf of California. Another bloom of *C. marina* during April and May of 2006 in a southern location (Sinaloa coast) on the east coast of the Gulf of California was also associated with mortality of approximately 48–60 tons of fish (Nuñez-Vázquez et al., 2011 and references therein).

Blooms of *Heterosigma* in inland coastal marine waters of WA are typically associated with summertime warm weather and high river discharge, resulting in brackish salinities and a stable surface layer (Taylor and Haigh, 1993; Rensel, 1995, 2007; Rensel et al., 2010b). Blooms in WA and BC may be expanding in range and magnitude, and contribution of anthropogenic factors is possible (Anderson et al., 2008b). The role of aquaculture effluent in promoting *Heterosigma* increases has been proposed in other countries (e.g., Scotland; Smayda, 2006), but all commercial finfish aquaculture sites in WA are sited in non-nutrient sensitive areas where naturally occurring background concentrations and flux of dissolved inorganic nitrogen from the Pacific Ocean is very high compared to the half saturation constants for growth of this species, implicating other factors, such as light and advection, in controlling algal bloom dynamics (Rensel, 1991, 2007; Anderson et al., 2008b). Rensel et al. (2010a,b) showed that the most frequent and intense blooms of *Heterosigma* in the region occur in the southern Strait of Georgia, where there are no commercial fish farms but pronounced influence of the spring/early summer Fraser River peak discharge that creates ideal growth conditions for the alga. Other anthropogenic activities are potential stimulatory factors; for example, sewage effluent spills were correlated with several *Heterosigma* blooms in poorly flushed and nutrient-sensitive central Puget Sound backwaters (Rensel, 2007; Anderson et al., 2008b). The raphidophyte’s high capacity for $\text{NH}_4^+$ uptake, that may originate from many sources, has been highlighted as an important factor (Anderson et al., 2008b) but the alga is equally adept at using other forms of dissolved inorganic nitrogen such as nitrate (Herndon and Cochlan, 2007).

The ecophysiology of *Heterosigma* is complex, and numerous behavioral and nutritional adaptive characteristics can contribute to bloom initiation. Niche-defining criteria proposed for *Heterosigma* include: temperature-regulated excystment, trace-metal (e.g., Fe) stimulation of growth, stimulation by organic compounds, allelopathic deterrence of competitor growth or predator activity, nutrient retrieval via vertical migration, halotolerance, shade adaptation, and occurrence of different ecotypes within a given region (Smayda, 1997, 1998; Hallegraeff, 1998; Anderson et al., 2002; Zhang et al., 2006; Fredrickson et al., 2011). Understanding *Heterosigma* toxic bloom ecology is further complicated by the uncertain knowledge of the species’ mechanism for toxicity (see above), hindering management capabilities to predict, prepare for, and respond to these toxic events.
5. Other HABs

Other HAB species are widespread along the North American west coast, but historically, bloom formation, toxin production, and ecosystem or human health impacts by these species have been rarely reported. Recent observations suggest possible future impacts (discussed below), and west coast regional monitoring programs should include emphasis on their detection. Such HAB species include the dinoflagellates, Dinophysis sp. (diarrhetic shellfish poisoning), Cochlodinium sp. (fish kills, toxic mechanism unclear), Protoceratium reticulatum (Claparède et Lachmann) Bütschli, Lingulodinium polyedrum (Stein) Dodge, and Gonylonax spinifera (Claparède et Lachmann) Diesing (yessotoxin producers), and Akashiwo sanguinea (K. Hirasaka) G. Hansen & Ø. Moestrup (sea bird kills due to surfactant-like proteins that coat feathers and neutralize water repellency and insulation). Also, in the upper reaches of some CA estuaries, the cyanobacterium, Microcystis aeruginosa (Kützing) Kützing, has emerged as a major bloom-former formerly and Trichodesmium spp. are listed as potential harmful species in Mexico (Hernández-Becerril et al., 2007). Also on the Mexican west coast, problems associated with potentially toxic benthic dinoflagellates should be considered. There are two descriptions of ‘ciguatera-like’ intoxications caused by the consumption of fish captured at Alijos Rocks, located 300 miles from the East coast of Southern Baja California (Lechuga-Deveze and Sierra-Beltrán, 1995) and from El Pardito Island in the Gulf of California (Heredia-Tapia et al., 2002).

Several species of Dinophysis (e.g., D. acuminata Claparède et Lachmann, D. acuta Ehrenberg, D. fortii Pavillard, D. norvegica Claparède et Lachmann, and D. rotundata Claparède et Lachmann) that have been shown to produce okadaic acid and cause diarrhetic shellfish poisoning (DSP) in other parts of the world are commonly found in west coast waters (Hernández-Becerril, 1988; Horner et al., 1997; Jester et al., 2009; Trainer et al., 2010). Symptoms of DSP include mild to severe gastrointestinal illnesses and, while deaths have not been documented, okadaic acid is known to be a strong tumor promoter (Suganuma et al., 1988; Domínguez et al., 2010). Okadaic acid was first detected in BC shellfish in 2003 in manila clams at low levels (Canadian Food Inspection Agency data), and in Monterey Bay, CA water samples and phytoplankton extracts in 1999, where Dinophysis – primarily D. acuminata – abundance correlated with inhibition of protein phosphatase activity in an assay for okadaic acid (Weber, 2000). Later, Southerland (2008) showed that DSP toxins occurred in sentinel California mussels collected from Monterey Bay in 2004–2005. One DSP toxin, okadaic acid, was positively associated with D. fortii and a second DSP toxin, dinophysistoxin-1 (DTX-1), also appeared to be produced by that species. Recently, okadaic acid was linked to three cases of human DSP illness from Sequim Bay, WA blue mussels (June 2011) and 62 illnesses from Gorge Harbour, BC blue mussels (August 2011; V. Trainer, pers. comm.). In Mexico, several species of Dinophysis are also present along the Pacific littoral but no cases of DSP have been officially recognized (Hernández-Becerril et al., 2007; Cortés-Altamirano and Sierra-Beltrán, 2008). Diarrhetic shellfish poisoning toxins have been measured since 2009 in Mexico, but they were officially regulated in 2011 with an action level of 160 μg eq okadaic acid per kg of shellfish (NORMA Oficial Mexicana, 2011). In 2010, two sanitary bans associated with the presence of DSP toxins in cultured oysters (C. gigas Thunberg) were implemented in Baja California (COFEPRES data). The toxins were detected by the mouse bioassay method but the presence of DSP toxins was not confirmed by an analytical approach. Toxic effects from these species had not been demonstrated on the west coast until very recently, and toxic effects can be mild and misdiagnosed. British Columbia and WA are developing more comprehensive DSP monitoring programs (D. Kelly and J. Borchert, pers. comm.).

Two species of Cochlodinium have widespread distribution on the west coast but only recently have they been linked to fish kills. Blooms of Cochlodinium polykrikoides Margeleff have been a major cause of fish kills off Japan, China, and Korea, while C. sp., recently identified as Cochlodinium fulvescens Iwatsuki, Kawai and Matsuoka using LSU rDNA sequences (Iwatsuki et al., 2008), was linked to a kill of aquacultured Atlantic salmon (S. salar Linnaeus) in BC in 1999–2000 (Whyte et al., 2001). More recently, a bloom of Cochlodinium (species not determined, but possibly C. fulvescens, see Iwatsuki et al., 2008) that extended over 800 km of CA coastline was linked to a 2004–2005 California mussel mortality event in Monterey Bay (Curtiss et al., 2008). Genetic characterization confirmed the presence of C. fulvescens in southern and central CA during this period (Howard et al., 2012). Since the 2004–2005 bloom event, Cochlodinium has emerged as a common bloom-forming organism along the CA coastline (Curtiss et al., 2008; Jester et al., 2009; Kudela et al., 2010; Howard et al., 2012; Kudela and Göbler, 2012; CDPH data). Based on nutrient uptake kinetic analyses conducted on samples from a 2006 Monterey Bay Cochlodinium bloom, Kudela et al. (2008b) estimated that from 55% (August) to 62% (September) of N uptake was derived from urea, suggesting a role of urea in the bloom increase in bloom prevalence. In September 2007, a Cochlodinium bloom cost the Monterey Abalone Company over $60,000 worth of abalone (D. Caron, pers. comm.). Along the Pacific coast of Mexico, Cochlodinium spp. are important ichthyotoxic species. Two recent reviews mention that since the beginning of the present decade, fish mortalities associated with C. polykrikoides and Cochlodinium cf. catenatum Okamura are common phenomena in the central Mexico coastal areas of Colima, Jalisco, and Nayarit, and in the southern Gulf of California, specifically in Sinaloa and Baja California Sur coasts (Hernández-Becerril et al., 2007; Cortés-Altamirano and Sierra-Beltrán, 2008).

Yessotoxins (YTX) are a new class of lipophilic biotoxins shown to be tumor promoters in mice but with unknown effects on humans. They are produced by three cosmopolitan, bloom-forming dinoflagellates, P. reticulatum, L. polyedrum, and G. spinifera, all common and sometimes abundant at various times and locations along the Pacific coast. Yessotoxins were detected in west coast waters, near Grays Harbor, WA, in summer 2004 when P. reticulatum was abundant (Howard et al., 2008), in isolates of L. polyedrum and G. spinifera from coastal CA (Armstrong and Kudela, 2006; Howard et al., 2008), and in California mussels from Scripps Pier during a bloom of L. polyedrum and from Monterey Bay in 2005 (Howard et al., 2008). One species, P. reticulatum, which is known to have significant impacts on the shellfish industry in BC because it reduces the ability of oyster seed to feed (Cassis, 2007), was documented in bloom abundance in north Puget Sound in 2006 and 2008 (Horner et al., 2010; R. Horner, unpubl. obs.). In the Todos Santos Bay region (northern Baja California), L. polyedrum blooms are recurrent phenomena that can cover the whole bay area (Peña-Manjarrez et al., 2005). Production of YTX was not measured during these events; however, due to the magnitude and recurrence of potentially toxic blooms, a phycotoxins monitoring program started in the region (project FICOTOX funded by the CONACyt-FORDECYT program; P.I. García-Mendoza) will monitor for YTX and other microalgal toxins. An extensive bloom of G. spinifera occurred off the WA/BC coasts extending into inland waters of WA and BC in August to September 1990 with shellfish deaths reported in Barkley Sound, BC due to low oxygen (Taylor and Horner, 1994). Another bloom occurred in northern Puget Sound in August 2011, but with no reports of impacts (R. Horner, pers. comm.). At the same time, August and September 2011, a bloom occurred on the Sonoma County, CA coast causing massive mortalities of abalone and sea urchins. The cause of the mortalities is not known, but low dissolved oxygen was not thought to be a factor (Rogers-Bennett et al., 2012).
The death of thousands of seabirds (e.g., surf scoters – *Melanitta perspicillata* Linnaeus, white wing scoters – *Melanitta deglandi* Bonaparte, common murre – *Uria aalge* Pontoppidan, Pacific loon – *Gavia pacifica* Lawrence, northern fulmar – *Fulmarus glacialis* Linnaeus, and western grebes) during September and October 2009 off WA, OR, and CA was attributed to surfactant-like substances produced by a bloom of *A. sanguinea* (Jessup et al., 2009; Du et al., 2011; Phillips et al., 2011). The dinoflagellate-produced foam destroys the waterproof layer of feathers that keeps seabirds dry, restricting flight and leading to hypothermia. This bloom extended into Oregon in October to November 2009 and was possibly related to a combination of a prior diatom bloom, a stratified water column with low nutrient concentrations, and an active upwelling event in October. The source of the bloom was thought to be the WA coast (Du et al., 2011).

Starting in the late 1990s, massive blooms of the cyanobacterium *M. aeruginosa* have recurred in the upper San Francisco (Lehman and Waller, 2003; Lehman et al., 2005, 2008; Moisander et al., 2009) and Klamath River estuaries, CA (Fetch, 2007). These are not restricted to very low salinities; e.g., *M. aeruginosa* colonies were found in salinities as high as 18 during an October 2003 bloom in San Francisco Estuary (Lehman et al., 2005) and *M. aeruginosa* was detected in salinities up to 9.1 in San Francisco Bay Delta waters during August and September 2007 (Moisander et al., 2009). A 2004 San Francisco Estuary bloom was associated with microcystin detection in the water, zooplankton (mesozooplankton including *Eurytemora affinis* Poppe and *Pseudodiaptomus forbesii* Poppe & Richard, amphipods, jellyfish, and worms), and clam tissue (Lehman et al., 2008). A massive bloom occurred along the entire length of the Klamath River in 2005 (Fetch, 2006, 2007). The cyanobacterium is a major concern for the Yurok Tribe because the timing of the bloom coincides with the adult Chinook salmon run, which has subsistence and commercial value for this tribal fishery. Microcystins were not detected in Chinook salmon livers or fillets, but trace amounts were detected in one adult steelhead (*Onchorhyncus mykiss* Walbaum) liver in 2005, the only year salmon and steelhead were tested (K. Fetch, pers. comm.). Recently, Miller et al. (2010) presented the results of investigations into the 2007 death of 21 southern sea otters recovered along the shore of Monterey Bay. The authors confirmed the cause of death as microcystin intoxication, and provided strong evidence that the toxin was derived from freshwater cyanobacteria transported from eutrophic rivers and accumulated by marine shellfish (i.e., hepatotoxic shellfish poisoning).

An increase in *Microcystis* abundance and bloom frequency in the San Francisco Estuary starting in the late 1990s and escalating over the last decade (Lehman et al., 2005, 2008, 2010) has been linked to the influence of anthropogenic activities on alterations in food web structure and trophodynamics (Gilbert et al., 2011). The authors used a 30-year data set to examine the relationship between nutrient dynamics and ecosystem properties. They found that changes in nutrient loading were linked to a cascade of changes in biogeochemical processes and other ecosystem properties. Both N and P loading increased from the mid-1980s to mid-1990s, but after that, P load reductions (i.e., through removal of P from laundry detergents and loss of canneries using P in their processing; Van Nieuwenuyse, 2007; Gilbert, 2010) without concomitant N reductions led to changes in nutrient stoichiometry (increased N:P). The authors concluded that increased nutrient loads (eutrophication) together with changes in nutrient ratios (stoichiometry) has led to changes in biogeochemical conditions (e.g., high N, high N:P) and trophic cascades (e.g., abundance of the invasive macrophyte, *Egeria densa* (Planch.) that increases pH, and the invasive clam, *Corbula amurensis* Schrenck that removes macrozooplankton and regenerates nutrients) favoring proliferation of *Microcystis*.

In addition to nutrient supply, *Microcystis* has several physiological characteristics that make it a superior competitor in the Bay Delta. It is a superior algal competitor under elevated pH; like *Egeria*, it has highly effective carbon concentrating mechanisms, allowing it to sustain photosynthesis when other algae may become C-limited (Jähnichen et al., 2007, and references therein, Gilbert et al., 2011). Like many cyanobacteria HAB formers, it also preferentially uses chemically reduced over oxidized nitrogen forms (e.g., Gilbert et al., 2004). With loads in the Sacramento River of effluent NH4+ exceeding 14 tons day⁻¹ (Jassby, 2008; Gilbert, 2010; Gilbert et al., 2011), ambient concentrations of NH4+ in the upper Bay Delta (where *Microcystis* occurs) exceed several μM-N, the threshold for inhibition of NO3 uptake, throughout much of the year (e.g., Dugdale et al., 2007). It has also been suggested that *Microcystis* may also have the capability to reduce its P requirement by lipid substitution, as shown for other cyanobacteria (Van Moyo et al., 2009; Gilbert et al., 2011). Thus, it can tolerate elevated N:P ratios, and its dominance under high N:P ratios may also reflect the decline in other species that lack such tolerance. As noted by Gilbert (2010), cyanobacteria do not have to grow faster at elevated N:P than at lower N:P values to become abundant; they merely have to grow faster than competing species groups.

Macroalgal blooms have been documented in a number of Pacific coast estuaries, but data are lacking for many areas (Bricker et al., 2007). These blooms develop high biomass in shallow water and small sea grass habitats, shading other vegetation and negatively impacting animals through hypoxia formation or possibly by production of toxic secondary metabolites. Examples of macroalgal blooms from invasive species have been documented recently in the San Juan Archipelago of WA (*Sargassum muticum* (Yendo) Fensholt; Britton-Simmons, 2004; Klinger et al., 2006), lagoons in southern CA (*Caulerpa taxifolia* (Vahl) C. Ag.; Jousson et al., 2000; Walters et al., 2006), and Baja California (*Ulva* spp.; Jorgensen et al., 2010; Zertuche-González et al., 2009) that have displaced native algal species and modified habitat, leading to losses in living resources and economic costs for eradication.

## 6. Economics and harmful algal blooms

Economists have focused on at least three aspects of HABs: (1) the negative regional economic impacts due to HABs; (2) the economic costs of human illness caused by HABs; and (3) human perceptions of risks associated with seafood due to HABs. Each research area applies specific methodologies and concepts, and careful utilization of these economic studies requires a basic understanding of the economic concepts. This section reviews the concepts and findings of some published studies.

The economic impacts are usually driven by reduced commercial fishery harvest, reduced aquaculture harvest, and/or reduced participation in and expenditures on marine recreation. As explained by Radtke et al. (1987), a simple estimate of economic impact can be calculated using a regional economic model (typically an Input–Output model) to calculate the “direct impact” of reduced fishing or recreational activity, the “indirect impacts” caused by reduced purchases of supplies and inputs by the directly impacted sectors, and the “induced impact” resulting from decreased purchases of consumer goods and services due to the combined direct and indirect reductions in regional incomes. The Input–Output (I–O) model is a simple linear model of regional economy that documents the aggregate outputs of each economic sector, the inter-sectoral transactions, and the resulting regional incomes (Miernyk, 1986). The I–O model is used to estimate changes in regional income, regional employment, and overall gross expenditures in the region. The income and employment impacts may be relevant to policy, but the gross expenditure does not coincide with the usual economic indicators. Further, the
income and employment impacts are not valid measures of economic benefits or costs. So, the impact analysis is an informative but largely a descriptive measure.

A recent study by Dyson and Huppert (2010) estimated that the regional impact of a year-long closure of all four WA coast razor clam beaches due to *Pseudo-nitzschia* blooms would cause an $11.36 million/year reduction in coastal county incomes due to reduced recreational activity, and nearly $2 million/year reduction in incomes due to lack of tribal and non-tribal commercial harvest of razor clams. Much lower impacts are estimated for single beach or shorter lasting closures. For example, an annual closure of the popular Long Beach peninsula razor clam fishery would have a regional income impact of negative $4.4 million. If the closure were for but one clam fishery opening (typically 2–5 days) the negative income impact would be just $1.4 million (Dyson and Huppert, 2010). These are hypothetical impacts that could be used in assessing the effects of avoiding beach closures through better monitoring of HABs.

Other economic studies use a simpler approach to assess effects of HABs on commercial landings and value (direct effects). Jin et al. (2008) estimated a negative relationship between red tides and landings of soft shell clams in Maine. They compared annual landings of northern quahogs, soft shell clams, blue mussels, and oysters in New England over the period 1990–2005 (with some years missing) to determine whether the extensive red tide bloom in 2005 was associated with a decline in the fishery. Overall, they estimated total direct impacts of up to ~$18 million. They also showed that imports of shellfish to New England increased during 2005, partly compensating for the loss of local harvests and reducing the indirect and direct impacts of lost harvest.

In their broad survey of economic effects of HABs, Hoagland et al. (2002) include estimates of the cost of human sickness and death caused by shellfish poisoning and ciguatera fish poisoning in the U.S. during 1987–1992. These are rough approximations based on a range of values estimated by others, including $1400 per reported illness, $1100 per unreported illness (estimated to be 90% of total illnesses), and $1 million per death. The estimates represent direct medical expenses and lost work time. Overall, they estimate that the human health costs of shellfish poisoning varied between $11 thousand and $1.1 million, averaging $400 thousand per year, over the 1987–1992 period. The human health costs of ciguatera fish poisoning were estimated for Florida, Hawaii, Puerto Rico, the Virgin Islands, Guam, American Samoa, and the Northern Mariana Islands using rough estimates of $1000 per reported case and $700 per unreported case to come up with an overall estimate of $15–$22 million per year, averaging $19 million annually over 1987–1992. These are rough, first-cut estimates which give some indication of the negative economic effects due to illness.

Whitehead et al. (2003) used a telephone and mail survey to investigate the effects of public information about *Pfiesteria* blooms in the mid-Atlantic states on seafood consumer’s perceptions and decisions. They found that survey respondents who received a brochure about the dangers of *Pfiesteria* expressed significantly increased perception of the health risks; however, more than 50% of respondents said that they would not alter their seafood consumption even when fish kills due to *Pfiesteria* were reported. If there were a seafood inspection program to identify health risks, the percentage of respondents who would maintain their usual seafood consumption level jumped to about 80%. This shows that HABs cause reduced demand for seafood. The authors also estimated that the average seafood consumer would be willing to pay $7/year for a seafood inspection program, and that the expressed value of seafood consumption increased by $3 per meal on average if a seafood inspection program is implemented.

These examples illustrate the wide range of possible economic aspects of HABs and ciguatera fish poisoning. The number of economic studies on HABs is very few, but this summary suggests the types of studies that might contribute to decisions regarding monitoring, information dissemination, and control of HABs.

7. Conclusions

By virtue of jurisdictional delineation, our summary knowledge of HAB distribution, causes, and impacts along the Pacific coasts of AK through Mexico is based on a collection of disparate sources of information, with diverse histories of HAB problem recognition and response. Despite this disparity, coastal HAB monitoring and mitigation has become a management priority throughout the region, reflecting a common recognition in each jurisdiction that HABs and their impacts are increasing and can have a profound effect on the health and economies of their coastal communities. It is tempting to lump HAB trends in this region with worldwide trends of increasing HABs related to eutrophication, but the two primary types of HABs (dinoflagellates causing PSP and *Pseudo-nitzschia* spp. producing DA) apparently do not follow this rule along the North American west coast. They are primarily derived from offshore waters and carried inshore, where it is possible that anthropogenic nutrient sources affect their dynamics, including increasing magnitude and prolonging duration. The primary nutrient drivers for bloom initiation are more consistent with an upwelling source.

Systematic economic assessments of HAB impacts and cost-benefit analyses for management strategic planning purposes remain important needs for the west coast region. Impacts to the shellfish industry in the Pacific Northwest region are significant, annually persistent, and well-documented. The effect of *Heterosigma* on the salmon aquaculture industry is another well-recognized economic threat (Rensel et al., 2010b); however, much of the impact of west coast HABs on coastal communities may not translate as well to economic losses. The threat to human health is always present, and the June 2010 death of two Alaskans attributed to PSP is a tragic reminder. The August 2011 discovery of DSP with human illnesses for the first time in BC and WA indicates that continued vigilance is necessary. Also, DA poisoning has led to thousands of sick or dead seals, sea lions, sea otters, dolphins, birds, and whales along the west coast in the last decade.

Pacific west coast districts of AK, BC, WA, OR, CA, and Mexico each has increased HAB monitoring infrastructure and improved detection and response capabilities in recent years; however, coastal HABs and their impacts frequently traverse state and federal boundaries, emphasizing the need for effective exchange of information on HAB ecology and impacts across districts. This paper presents the state of the knowledge of HAB research along the west coast of North America, as a step toward meeting the need for integration of HAB outreach, research, and management efforts.

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