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Review

Harmful algal blooms and public health

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ABSTRACT

The five most commonly recognized Harmful Algal Bloom-related illnesses are ciguatera poisoning, paralytic shellfish poisoning, neurotoxic shellfish poisoning (NSP), amnesic shellfish poisoning, and diarrhetic shellfish poisoning. Although these exposures result from exposure to different toxins or toxin congeners, these clinical syndromes have much in common. Exposure occurs through the consumption of fish, shellfish, or through exposure to aerosolized NSP toxins. Routine clinical tests are not available for the diagnosis of harmful algal bloom related illnesses, there is no known antidote for exposure, and the risk of these illnesses can negatively impact local fishing and tourism industries. The absence of exposure risk or diagnostic certainty can also precipitate a chain of events that results in considerable psychological distress for coastal populations. Thus, illness prevention is of paramount importance to minimize human and public health risks. To accomplish this, further transdisciplinary research, close communication and collaboration are needed among HAB scientists, public health researchers, and local, state and tribal health departments at academic, community outreach, and policy levels.

Contents

1. Introduction

There is a growing appreciation of the importance of Harmful Algal Blooms (HABs) and HAB-related illnesses to public health. With the dramatic increase in the number of harmful algal blooms, as well as their frequency and intensity in coastal regions throughout the world (Glibert et al., 2005), there are more toxic algal species, more algal toxins, and more geographic areas affected than ever before. Often attributed to natural environmental factors (hurricanes, earthquakes, or sequences of ideal growth and transport conditions), anthropomorphic activity (increased eutrophication, marine transport and aquaculture) and climate change, the proliferation of these species may cause massive fish kills, destroy or poison shellfish beds, contribute to wildlife mortality, human illness and death (Lehane and Lewis, 2000; Pratchett et al., 2008; Badjeck et al., 2010). The risk of HAB-related illnesses is further amplified by shifting preferences to heart-healthy diets, increased travel to coastal destinations, increased consumption of imported fish, the growth of coastal urban communities, and growing segments of the population involved in marine recreation (Jensen, 2006; Ralston et al., 2011). Thus, in

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the absence of ongoing public health surveillance, research, and outreach into HAB-related exposures and illnesses, it is anticipated that the number of cases of HAB-related illnesses will continue to rise over the next decade. With this in mind, this Special Issue of Harmful Algae is devoted to HABs and public health.

With the exception of aerosolization of the toxins produced by "Florida Red Tide" (*Karenia brevis* blooms), the primary vector for HAB-related human health concerns is the consumption of fish and shellfish. Often due to the activity of HAB-related toxins, seafood consumption has become the leading cause of food-borne illness with known etiology. Seafood is responsible for 10–20% of outbreaks and about 5% of all individual illnesses among all food types (Huss et al., 2004; CSPI, 2007). The annual acute care costs of seafood-borne disease are estimated at approximately two-thirds of a billion dollars (Ralston et al., 2011). Persistent symptoms are seen in about 2–3% of cases, and the costs of medical care, lost productivity, and functional disability associated with chronic sequelae are thought to exceed those of acute care. These are conservative estimates as there is considerable diagnostic uncertainty and underreporting of seafood-related illnesses (Sobel and Painter, 2005; Fleming et al., 2011).

To orient the reader to the diverse scientific papers to follow, this article is organized around the five most commonly recognized HAB-related illnesses. This will include a general description of ciguatera fish poisoning (CFP), paralytic shellfish poisoning (PSP), neurotoxic shellfish poisoning (NSP), amnesic shellfish poisoning (ASP) and diarrhetic shellfish poisoning (DSP) with a quick reference table (Table 1). An overview of HABs from a public health perspective will follow, as well as highlights of important areas for future research.

1.1. Ciguatera fish poisoning (CFP)

It is generally well-accepted that ciguatera fish poisoning (CFP) is the most frequently reported seafood-related disease in the United States and most common foodborne illness related to finfish consumption in the world (Isbister and Kiernan, 2005; Lynch et al., 2006; Friedman et al., 2008; Kumar-Roiné et al., 2011). It is endemic in areas where consumption of reef fish is common. These areas include the Caribbean, southern Florida, Hawai'i, the South Pacific and Australia. Additionally, emerging data suggest expansion of the biogeographical range of ciguatoxic fish (Villareal et al., 2007; Bienfang et al., 2008; Dickey and Plakas, 2010) with recent reports of CFP from fish originating in South Carolina and the Northwestern Gulf of Mexico (CDC, 2006).

CFP is caused by the consumption of reef fish that have accumulated potent neurotoxins (ciguatoxin) in their flesh and viscera. The toxins are produced by the marine dinoflagellate *Gambierdiscus* spp., which live on various macroalgal hosts or other substrates in coral reef ecosystems. Herbivorous fish consume these dinoflagellates and, through bioaccumulation and magnification, the toxin advances through the food web through ingestion by carnivorous species. More than 400 fish species are thought to have the potential for ciguatera toxicity (Halstead, 1978; Lehane and Lewis, 2000). The risk is greatest for carnivorous, predatory fish, such as barracuda (of which >70% may be toxic). Other high-risk fish include snapper, grouper, and amberjack (Langley et al., 2009).

The diagnosis of ciguatera fish poisoning (CFP), or "ciguatera," is typically based upon clinical symptoms within the context of a carefully elicited history of recent predatory reef fish consumption. Symptoms of CFP arise within 12 h of eating the toxic fish. The initial symptoms begin with severe gastrointestinal problems (nausea, vomiting, diarrhea, abdominal pain), which usually abate within 24 h (Hokama, 1988). Cardiovascular problems (generally a combination of bradycardia with hypotension) and/or neurologic symptoms may also accompany this acute episode. In the Caribbean and Southern Florida, cardiovascular disorders often reverse within 48–72 h (Hokama, 1988; Butera et al., 2000), yet in Pacific regions, outcomes may be less favorable, as there have been reports of rapid progression to respiratory distress, coma and death (Lange, 1987; Defusco et al., 1993; Habermehl et al., 1994). From a few hours to two weeks after exposure, a diverse range of subjective neurological complaints have been reported in about 70% of cases (Lawrence et al., 1980). These may include pain and lower extremity weakness; painful tingling around the mouth, teeth, nose and throat; peripheral paresthesia, headache, metallic taste, hyporeflexia, and/or dysphagia. The hallmark of CFP neurological symptoms is an unusual paradoxical disturbance of thermal sensation, i.e., cold objects feeling hot and sometimes hot objects feeling cold (Pearn, 2001; Achaibar et al., 2007). Although detailed case reports document a wide range of neurologic symptoms, the full symptom complex of CFP remains to be fully characterized or understood.

While gastrointestinal or cardiac symptoms are generally short-lived, recovery from acute neurologic symptoms is longer and less predictable, as these symptoms can persist from approximately one week to six months (Lange et al., 1992; Butera et al., 2000; Achaibar et al., 2007). In addition, there are many patients who report symptom persistence for several years. The chronic ciguatera syndrome is typically characterized by intractable fatigue, weakness and/or paresthesias, and is accompanied by depression. Chronic symptoms may be present continuously or reappear after a period of presumed recovery. This recurrence may also be triggered by alcohol use or by repeated consumption of fish with low levels of ciguatoxin. This suggests that persons who have had one episode of ciguatera are at increased risk for repeated illness (Morris et al., 1982). In this special issue, Lopez et al. (2016) report their efforts toward developing a conceivable biomarker for chronic and recurrent ciguatera.

1.2. Paralytic shellfish poisoning (PSP)

Paralytic shellfish poisoning (PSP) is a potentially lethal clinical syndrome. It is caused by eating bivalve mollusks (mussels, scallops clams) contaminated with a group of structurally related marine toxins collectively referred to as saxitoxins (STX; Shumway, 1990; James et al., 2010). PSP toxins are concentrated in shellfish as a result of the filtration of toxic algae produced by several dinoflagellates (including *Alexandrium*, *Gymnodinium* and *Pyrodinium*) during "red tide" blooms. Predators of bivalve shellfish (scavenging shellfish, lobsters, crabs and fish) may also be vectors for saxitoxins, thus expanding the potential for human exposure (Halstead and Schantz, 1984). Geographically, the most risky regions for PSP are temperate and tropical marine coasts. In North America, this includes Alaska, the Pacific Northwest, and the St. Lawrence region of Canada; however, incidents of PSP regularly occur in the Philippines and other tropical regions. Toxic shellfish have also been found in temperate regions of southern Chile, England, Japan, and the North Sea.

The initial symptoms of PSP are numbness or tingling around the mouth and lips within 10 min to two hours after shellfish consumption. The timing of symptom onset is thought to be dose-dependent (Gessner et al., 1997a; McLaughlin et al., 2011). In mild cases, this may be the only symptom; however, in more severe cases, the numbness and tingling spread to the neck and face, and may be accompanied by headache, abdominal pain, nausea, vomiting, diarrhea, and a wide range of neurologic symptoms. These neurologic symptoms may include weakness, dizziness, dysarthria, paresthesia, double vision, loss of coordination, vertigo or dizziness, and/or a "floating" sensation. Relatively recent data reported in this special issue (Knaack et al., 2016) suggest that dysphagia and dysarthria are most likely the strongest indicators