D. Abstract ECOHAB 2006-Targeted project EPA-G2006-STAR-B1

Title: Spread of a sodium channel mutation in softshell clam, *Mya arenaria*, populations: Implications for risk assessment and management of PSP toxins.

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Paralytic shellfish toxins (PSTs) are potent neurotoxins produced by dinoflagellates, *Alexandrium* spp. on both coasts of North America, and accumulated by suspension-feeding shellfish. Consumption of toxic shellfish can cause paralytic shellfish poisoning (PSP) in humans. Shellfish may also be affected by PSTs, exhibiting paralysis, burrowing and feeding inhibition, leading to indirect mortalities, and even direct mortalities during severe blooms. The softshell clam, *Mya arenaria*, is a commercially important bivalve with wide latitudinal distribution in N. America. Clam populations with a history of exposure to PSP have developed natural resistance to PSTs. Our prior work identified a sodium (Na^+) channel mutation in some *M. arenaria* conferring resistance to PSTs and allowing clams to accumulate toxin at 8x the rate of wild-type clams. PSTs may thus act as potent natural selection agents, leading to a spread of toxin resistance in *M. arenaria* populations associated with higher toxin levels. Furthermore, global expansion of PSP to previously unaffected coastal areas might result in long-term changes to shellfish communities and increased trophic transfer of PSTs in the ecosystem.

Objectives: This project will establish the range and extent of the mutation found in wild *Mya arenaria* populations in the NW Atlantic, including populations in southern New England which experienced a massive PSP outbreak in 2005, the first of this intensity since 1972 when PSP spread to this region. We will determine from laboratory and field studies the selective pressure that variable bloom scenarios of *Alexandrium* spp. impose on these populations, thereby altering their fitness and capacity to accumulate toxins. In addition to these population studies, we will undertake selected breeding trials to determine the inheritance of toxin resistance, and explore the physiological mechanism (hypoxia of the mantle cavity) responsible for toxin-induced mortality.

Approach: The methods used for this project have largely been well developed. They include a nerve trunk assay for the determination of resistance to STX in individual clams, established cDNA and DNA sequencing protocols to conduct a phylogeographic survey of the prevalence of Na^+ channel mutations, and toxification protocols. Selectively bred *M. arenaria* will be exposed in the laboratory to *Alexandrium* spp. of varying cell toxicity and toxin composition as well as to a range of cell concentrations representative of natural HABs to determine their effects on population genetic structure at the targeted Na^+ channel locus. Field trials at PSP-affected and unaffected sites will also be conducted to further validate laboratory trials. Oxygen microprobes will be used to determine oxygen levels in both resistant and sensitive clams that have been exposed to PST in order to determine if hypoxia is the primary mechanism of mortality.

Expected results: The increased prevalence of clams carrying a toxin resistant mutation can significantly affect their ability to accumulate PSTs and food-web toxin transfer. Genotype information can be used in regional risk-assessment to predict potential toxin load of an individual clam, or population, during red tides as well as the impact of red tides on clam populations. Results of this study will be useful for monitoring and management of shellfish toxins as well as management of the softshell clam resource.

Supplemental keywords: food web, biogeography, PSP toxicity, genotype, fisheries