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Research priorities for diseases of the blue crab Callinectes sapidus

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RESEARCH PRIORITIES FOR DISEASES OF THE BLUE CRAB *CALLINECTES SAPIDUS*

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The diseases of blue crabs have received relatively little attention compared to those of the American oyster, *Crassostrea virginica,* or the penaeid shrimps, *Penaeus* spp. This is primarily due to differences in resource management (fishery vs. aquaculture), and the magnitude of financial losses suffered by the industries from protozoal diseases in oysters and viral diseases in shrimp, respectively. Nonetheless, several agents including *Vibrio* spp., *Hematodinium perezi, Paramoeba perniciosa, Ameson michaelis* and *Loxothylacus texanus* are highly pathogenic in blue crabs, and have the capacity to severely damage certain segments of the crab population. This paper is meant to highlight priorities for critically needed research on the ecological impacts and pathological processes of these diseases in the crab host. It is not meant as a review of the literature. For more complete reviews of the parasites and diseases of blue crabs, see Johnson (1983), Couch (1983), Overstreet (1983), Messick and Sinderman (1992), Noga et al. (1998), Shields and Overstreet (in press).

Session Overview

At the 2000 Blue Crab Symposium, Wilmington, North Carolina, four papers highlighted the importance of several pathogens in the biology and economics of the blue crab. Shields (2000) gave an overview of the epizootiology of *Hematodinium* infections, and discussed aspects of the pathology, lethality, and physiology of the disease. Messick (2000) discussed the prevalence and seasonality of *Hematodinium, Paramoeba,* and *Ameson* infections along the Atlantic coast with major comparisons between Maryland and Georgia. O'Brien (2000) presented important new data on the life cycle and transmission patterns of a rhizocephalan barnacle, *Loxothylacus texanus,* and relationships with salinity and depth. Guillory and Perry (2001) provided data on the prevalence of the rhizocephalan and its association with blue crabs in Alabama and Louisiana.

Information Needs

Which Parasites and Diseases Can Potentially Damage Blue Crab Fisheries?—Several disease agents have occurred in outbreaks or epizootics in blue crabs (Table 1). In general, the outbreaks are localized to specific bays or regions (e.g., Chincoteague Bay), but widespread epizootics have occurred, especially along the Delmarva Peninsula of Maryland and Virginia. In most cases, the causative agents were not identified prior to the occurrence of the epizootics (e.g., Sprague and Beckett, 1966; Johnson, 1976; Couch, 1983; Messick, 1994), and this hampered attempts to assign causality especially after the mortalities had subsided.

Four of at least eight viruses are known to be pathogenic to the blue crab (Johnson, 1983). The pathogenic viruses typically occur in hemocytes, or epithelial cells, and are associated with significant mortalities in shedding houses (Table 1). Infected crabs are lethargic, susceptible to stress-induced mortality (e.g., capture, and handling), and often show signs of tremors, paralysis, or even blindness. Limited transmission experiments with the viral agents indicate short prepatent

Table 1. Selected pathogenic agents of the blue crab, *Callinectes sapidus*, with regional occurrence, tissues in which the disease occurs, capability of occurring in outbreaks, relation to crab mortalities, and primary reference.

Disease agent	Region	Major tissue	Outbreaks	Relation to mortality	Source
1. Viruses					
Reolike virus, RLV	Chincoteague Bay, VA, Chesapeake Bay, MD	Hemopoetic tissue, nerve cells, hemocytes, epithelial cells	Yes	Inoculation caused mortality in 3 days	Johnson and Bodammer, 1975
Rhabdolike virus A, RhVA	Atlantic, Gulf of Mexico	Nerve cells, endothelial cells, hemocytes, connective tissues	Unknown	Found with RLV, unknown, stress-related mortality	Jahromi, 1977
Picornalike virus, Chesapeake Bay Virus, CBV	Tangier Sound	Nerve cells, epithelial cells including gills	Yes	2 weeks–2 months	Johnson, 1983
Herpeslike virus, HLV (now Bifacies virus)	Chincoteague Bay, VA; Assawoman Bay, DE	Hemocytes	Yes	High prevalence, 1–2 months until mortality	Johnson, 1976
2. Bacteria					
Vibrio parahemolyticus	Chesapeake Bay	Hemolymph	Yes	High mortality in short-term shedding facilities	Krantz et al., 1969
Other bacteria	Chesapeake Bay	Hemolymph	Unknown	High prevalence	Colwell et al., 1975
3. Fungi					
Lagenidium callinectes	Atlantic	Eggs, larvae	Yes	High prevalence, 25–50% of the sponge	Couch, 1942; Rogers-Talbert, 1948
4. Protozoa					
Ameson michaelis	Atlantic, Gulf of Mexico	Muscle tissue	No	Highly pathogenic, not assoc. with outbreaks	Sprague, 1970
Paramoeba perniciosa	Mid Atlantic states	Connective tissues, hemolymph	Yes	High prevalence, late spring, and winter, 30 days after injection	Sprague et al., 1969
Hematodinium perezi	Atlantic, NE Gulf of Mexico	Hemolymph	Yes	High prevalence, juveniles up to 100%	Newman and Johnson, 1975

Table 1. Continued.

Disease agent	Region	Major tissue	Outbreaks	Relation to mortality	Source
5. Helminths (worms)					
Microphallus and pepper-spot disease	Atlantic, Gulf of Mexico	Connective tissues	Yes	No mortality; market issue	Overstreet, 1978; Couch, 1974
Carcinonemertes carcinophila	Atlantic, Gulf of Mexico	Eggs	Yes	High prevalence, 5–25% of the sponge	Humes, 1942
6. Crustacea					
Loxothylacus texanus	Gulf of Mexico	Internal	Yes	High prevalence, stunting, feminization	Boschma, 1933

periods. For example, crabs inoculated with the reolike virus (RLV) died in just 3 days but crabs fed virus-infected tissues died after 30 to 40 days (Johnson and Bodammer, 1975; Johnson, 1984). Other pathogenic viruses have prepatent periods ranging from 2 weeks to 2 months. Transmission and mortality associated with viral diseases have not been well studied in blue crabs.

Bacterial infections are common in blue crabs especially in summer. *Vibrio* spp. are ubiquitous in the marine environment, and crabs are passively infected by them. *Vibrio parahaemolyticus* can occur in outbreaks in shedding facilities where it can be a significant source of mortality (Johnson, 1983). The bacterial pathogen invades the hemolymph through abrasions in the cuticle of the crab, and multiplies in the nutrient-rich environment of the crab's hemolymph. Prevalences of bacteria vary with season and are correlated with increasing water temperatures (Tubiash et al., 1975; Davis and Sizemore, 1982; Welsh and Sizemore, 1985). Stress plays a significant role in bacterial infections in crabs; hence, the association between bacterial infections and mortalities during rapid increases in water temperature and during high summer temperatures. The number and scale of crab mortalities that are caused by bacterial infections requires additional investigation.

Two egg parasites can cause considerable egg mortality in high salinity waters. The fungus *Lagenidium callinectes* assimilates large numbers of crab eggs in infected clutches. In some cases, most of the egg clutch can be destroyed by the fungus. *Lagenidinium callinectes* is widespread and tolerates moderately low salinities (Rogers-Talbert, 1948). The nemertean worm *Carcinonemertes carcinophila* ingests relatively large numbers of eggs during development. On other crab species, epizootics of nemertean worms have virtually wiped out an entire year's broodstock resulting in significant damage to the fisheries (Wickham, 1986; Kuris et al., 1991). No outbreaks have been reported from blue crabs probably because the nemertean worm is limited by low salinities (Scrocco and Fabianek, 1970), and may not accrue in large enough numbers to cause damage.

Three pathogenic protozoans are known to cause mortalities to blue crabs. Gray crab disease is caused by an amoeba, *Paramoeba perniciosa*, that invades the connective tissues, and later, the hemolymph of the crab. As the common name implies, the ventral surfaces of infected crabs turn gray in color. As in other systemic infections, the hemocytes of heavily infected crabs are virtually replaced by the trophic stage of the parasite. Crabs become lethargic and eventually die, or die from stress-related handling. The parasite occurs in the small coastal bays of the mid Atlantic states, where during late spring, it can cause mortalities in shedding facilities (Newman and Ward, 1973; Couch, 1983). Winter mortalities of crabs may also be associated with high prevalences of this amoeba (Newman and Ward, 1973; Johnson, 1977). Cannibalism is a likely avenue for the spread of the disease. For example, Johnson (1977) speculated that lethargic crabs would fall easy prey to their voracious conspecifics, and thus, effect transmission. Alternatively, the amoeba could invade crabs in the winter while their hosts are buried in the sediments. Mortality and life cycle studies along with rapid, noninvasive diagnostic techniques are research priorities for *P. perniciosa.*

The microsporidians *Ameson michaelis* and *Thelohania* sp. cause severe muscle lysis that results in a condition known as ''cotton crab.'' Crab meat infected with these microsporidians is cottony in texture, discolored with either opaque white or purple in color, and poorly flavored. *Ameson michaelis* can be transmitted via cannibalism (Overstreet, 1978). It is surprising that the parasite is only found at low to moderate prevalences (0.1 to 10%, Messick, 2000), because as much as 25% of a blue crab's diet is other blue crabs (Hsueh et al., 1992). Thus, the route of infection and potential for host resistance should be examined. In addition,

work should focus on developing detection techniques as it is difficult to diagnose microsporidians in early infections.

Hematodinium perezi is an unusual parasitic dinoflagellate that occurs in major outbreaks. It lives in the hemolymph of crabs and rapidly proliferates to overwhelm the defenses of the host. Outbreaks of *H. perezi* have been reported from the high salinity waters of the lower Chesapeake Bay, coastal bays in Maryland and Virginia, Georgia, and Florida (Newman and Johnson, 1975; Couch, 1983; Messick, 1994; Messick and Shields, 2000). The disease is most prevalent in fall (Messick, 1994; Messick and Shields, 2000). During epizootics, crab mortality can reach 50% in set pots, and 75% in shedding facilities on the Eastern Shore of Virginia. During one outbreak, prevalence reached of 100% in juvenile crabs and up to 70% in mature crabs (Messick, 1994). Infections are generally fatal with crabs dying from energy depletion or disruption of the tissues (Shields and Squyars, 2000; Shields et al., 2003). Correlative evidence indicates that temperature and salinity play key roles in limiting epizootics of the dinoflagellate (Messick et al., 1999).

Hematodinium perezi may have several alternate or reservoir hosts. At present, rock crabs (*Cancer* spp.), lady crabs (*Ovalipes ocellatus*) (MacLean and Ruddell, 1978), mud crabs (*Rhithropanopeus* spp., *Neopanope* spp.), and green crabs (*Carcinus maenas*) are known to be infected with parasites resembling *H. perezi* (Messick and Shields, 2000). In addition, several species of amphipods have been reported with *Hematodinium*-like infections (Johnson, 1986). Hudson and Shields (1994) and Shields (1994) speculated that amphipods may act as intermediate or alternate hosts for the disease. PCR primers and molecular probes would help solve life cycle questions by identifying life history stages. Molecular studies would also facilitate the taxonomy of this troublesome group of pathogens as there are few morphological or life cycle features that distinguish species.

While not pathogenic, pepper spot or buck-shot crab is a parasitic condition that affects the quality of crab meat. Pepper spot occurs when innocuous microscopic trematode cysts of *Microphallus bassodactylus,* and other microphallid trematodes, are attacked by the hyperparasitic haplosporidan protozoan, *Urosporidium crescens* (Couch, 1974; Overstreet, 1978). The infected trematode cysts enlarge and turn black due to the multitude of black spores of the protozoan. Crabs are not affected by the disease but it can affect the aesthetics of the meat. Pepper spot is common along the mid Atlantic and Gulf coasts. The environmental factors that affect the distribution of the protozoan are not understood.

WHICH DISEASES CAN BE ADDED TO POPULATION MONITORING PROGRAMS?—The rhizocephalan, *L. texanus,* is abundant in blue crabs in the Gulf of Mexico. In certain localities it reaches prevalences of 30% to 70% (Wardle and Tirpak, 1991; Alvarez and Calderon, 1996; Guillory and Perry, 2001). Infected crabs are stunted and rarely reach marketable size; and are, therefore, lost to the fishery. Castration is a side effect of infection. However, stunting and castration are only physical manifestations of infection. Sand crabs, *Portunus pelagicus,* infected with a related rhizocephalan, *Sacculina granifera,* exhibit morbid behavioral changes such as docility (Bishop and Cannon, 1979), sterile matings, and male-male precopulatory embraces which may further affect these crab populations (Shields and Wood, 1993). Similar behaviors may occur in *C. sapidus* but they have yet to be reported. Given that blue crabs have multiple broods with planktonic larvae, the population-level effects of rhizocephalans are generally thought to be low (but see Blower and Roughgarden, 1989a, 1989b). Connectivity of populations and recruitment of juveniles may more than make up for the losses (castration) in the adult breeding populations. Recent models have, however, suggested that diseases

can regulate closed populations of crabs (Blower and Roughgarden, 1989a, 1989b; Kuris and Lafferty, 1993), and that marine populations with larval dispersal can, in some cases, be hydrodynamically or demographically closed for purposes of immigration (Cowen et al., 2000). The potential impacts of rhizocephalans on the connectivity between fecundity, larval dispersal and juvenile recruitment represents an exciting opportunity for productive research.

Monitoring for *L. texanus* should be incorporated into the crab monitoring programs of the Gulf states. Trawl, dredge and pot surveys could easily include data such as the presence/absence of the external stage of the parasite (sacculina externa), the size and relative sex of the crab (feminization occurs in most juvenile males), and a standardized color scheme for the sacculina externae (for maturity information). Trawl and dredge surveys may be unbiased sampling techniques for the parasite as they have a smaller size selectivity than pot surveys, and may more accurately represent subpopulations of crabs stunted by the infection. Long-term data sets of prevalence, host factors, and parasite maturity represent a veritable gold mine of new biological information on the life history and impact of the parasite on the fishery. For example, increases in prevalence may show an increase in the salinity regime of an area, or may indicate a significant increase in transmission that would otherwise be underreported by the fishery. Color schemes for the externa of the parasite may help pinpoint seasonality in transmission.

Shell disease may also warrant inclusion in monitoring programs. Shell disease is caused by chitinoclasitic bacteria in the genus *Vibrio* that colonize the exocuticle of the crustacean host. Shell disease is common in crustaceans that have been subjected to stress. Injuries sustained from high stocking densities, long-term confinement, molting, and environmental pollutants have been implicated as stressors inducing shell disease in many decapods (Rosen, 1967; Iversen and Beardsley, 1976; Overstreet, 1978; Johnson, 1983; Getchell, 1989; Sinderman, 1989; Smolowitz et al., 1992). While bacteria are clearly involved in the etiology of the disease, pollutants (i.e., sewage sludge, dredge spoils, heavy metals, organic debris) and other external symbionts can play a significant role in the syndrome (Young and Pearce, 1975; Couch, 1983; Morado et al., 1988; Gemperline et al., 1992; Weinstein et al., 1992; Ziskowski et al., 1996). In general, shell disease is not a significant mortality factor to wild stocks of crustaceans, but severe cases do result in host death. However, high prevalences of shell disease may indicate significant water quality or stress issues in resident crustacean populations (McKenna et al., 1990). Shell disease does have a small economic impact in that afflicted animals are not aesthetically pleasing to eat; thus there may be a lower grading of meat value (Rosen, 1970; Getchell, 1989).

Shell disease is relatively innocuous in blue crabs. There are, however, signficant research questions to be addressed through study of the disease. Stress is a significant issue in the onset of numerous diseases and conditions in invertebrates. Shell disease typically indicates a significant problem with water quality. Study of the association between different stressors and the onset of shell disease may provide a useful model to study the effects of stress on crustaceans. Indeed, the decrease in immune function and decline in hemocyanin in diseased crabs from polluted waters (e.g., Engel et al., 1993; Noga et al., 1994, 1996) highlights the need for just such studies. In addition, the prevalence of shell disease may be an excellent indicator of water quality (pollution, nutrient enrichment, etc.). Thus, monitoring for shell disease may provide an inexpensive early warning tool for pollutants or other stressors.

What Are The Impacts of Parasites on the Population Dynamics of the

BLUE CRAB?—The importance of diseases in regulating the population dynamics of the host is the subject of considerable debate, even though host regulation has been clearly demonstrated in numerous examples of biological control of insect pests. Parasitic diseases can negatively impact, and possibly regulate crustacean populations (Blower and Roughgarden, 1989a, 1989b; Hobbs and Botsford, 1989). Hence, pathogenic diseases may play a key role in the population dynamics of the blue crab. In addition to direct mortality from disease, parasitized crabs are weakened by infections, and often succumb to sublethal stressors such as temperature (high or low), hypoxia, or increased predation or cannibalism. Measuring the indirect effects of the diseased state on predation rates may be possible using tethering experiments as has been done for juvenile blue crabs in the field (Hines and Ruiz, 1995; Pile et al., 1996).

Outbreaks of egg predatory nemerteans, rhizocephalan castrators, and dinoflagellates on crabs and lobsters have seriously affected or even devastated host populations in California, British Columbia, Alaska and Scotland (e.g., Sloan, 1984, 1985; Wickham, 1986; Meyers et al., 1987; Shields et al., 1989, 1990; Kuris et al., 1991; Field et al., 1992). During most outbreaks, environmental factors such as hydrographic conditions and seasonal increases in water temperature contributed to the development of the epizootics. Hydrographic features may enhance transmission rates through entrainment of water masses and amplification or retention of infectious stages. The entrainment of water and the reduction in water mixing/flushing in the isolated fjords maintained relatively high levels of infectious larval stages and were considered primary factors in the spread of rhizocephalans and nemerteans on king crabs (Sloan, 1984, 1985; Kuris et al., 1991).

The small bays, shallow backwaters and lagoons of coastal mid Atlantic and Gulf states of the U.S.A. are ideal for the growth and spread of parasitic diseases in the blue crab (Shields, 1994). For example, outbreaks of *Hematodinium* and *Paramoeba* occur in regions that feature relatively closed crab populations (i.e., those with little immigration and emigration of juveniles and adults), high salinities with, in some cases, entrainment of water through lagoonal areas (e.g., narrow channels with shallow sills and barrier islands/bars), and stressful physicochemical conditions (temperature and salinity stress, seasonal hypoxia) (Messick and Shields, 2001).

Salinity and temperature limit the spread of *Hematodinium* in blue crabs (Messick et al., 1999; Messick and Shields, 2000). The ranges of several marine parasites and symbionts of portunid crabs are limited by low salinities (e.g., *Octolasmis mulleri*—Walker, 1974; *Loxothylacus texanus*—Ragan and Matherne, 1974; *Choniosphaera indica* and *Carcinonemertes mitsukurii*—Shields and Wood, 1993) and low temperatures (e.g., *Carcinonemertes mitsukurii* on *P. pelagicus*—Shields and Wood, 1993). Gauging impacts of salinity, temperature and hypoxia on infections should be further examined in laboratory and mesocosm studies; such studies would help to define the extent and range of disease impacts on the blue crab.

The role of stressors in crab mortalities cannot be overstated. Seasonal hypoxia and temperature extremes are often associated with crab mortalities (Van Engel, 1982, 1987), but neither stressor has received much attention. The sudden mortalities in *Hematodinium*-infected crabs could be related to hypoxic events, especially given the oxygen demands of the parasite and the moribund host (e.g., Taylor et al., 1996; Shields et al., 2003). Low temperatures are often cited by watermen as the cause of winter mortalities of blue crabs (Van Engel, 1982, 1987). However, parasitic diseases may also be involved. *Paramoeba perniciosa* is

known to overwinter in blue crabs (Newman and Ward, 1973; Johnson, 1977), and *H. perezi* may overwinter or kill overwintering crabs (Messick et al., 1999). These parasites may represent proximal causes of winter mortalities, especially in mid to high salinity areas. Thus, the role of diseases in winter mortalities should be examined further.

How Are Immune Functions Compromised by Diseases?—While much is known about the functional defensive responses in decapods, the interactions between pathogens and immune defenses are not well understood. The effects of bacterial and fungal pathogens on components of the immune system have been examined in crayfishes, crabs, lobsters, and shrimps. For general reviews on crustacean immunity, see Sindermann (1971, 1990) for pertinent observations on the older literature, and Smith and Chisholm (1992), Söderhäll and Cerenius (1992), Bachère et al. (1995), Holmblad and Söderhäll (1999) for newer syntheses.

Absolute and relative decreases in hemocyte densities are diagnostic features of pathogenic infections in blue crabs (Shields and Squyars, 2000). Hemocyte densities may decline via several mechanisms including phagocytosis by *P. perniciosa* (Johnson, 1977), lysis from viral infections (Johnson, 1984), and physical disruption or extracellular enzymatic degradation by *H. perezi* (Shields and Squyars, 2000). Absolute declines in cell density have been correlated with time-to-death in blue crabs infected with *H. perezi* (Shields and Squyars, 2000). Given that many of the defensive reactions are cell-bound or cell-mediated (Smith and Chisholm, 1992; Söderhäll and Cerenius, 1992), it is no surprise that morbidity and mortality are closely associated with the decline and loss of the hemocytes, the primary defense of the host. The underlying causes of hemocyte loss or sequestration, and the differential changes in hemocyte densities, coupled with the further characterization of cell-bound defensive molecules, are critical avenues for future research.

Agglutinins are lectins that function in molecular recognition of self versus non-self. The role of agglutinins or other humoral factors in response to disease or the development of resistance in crustaceans is not well understood. Hemagglutination was not affected by infection with *Hematodinium* (Shields et al., 2003), but agglutination was not examined in crabs that were found to be refractory to the infection. Foreign bodies, such as human red blood cells, induced increased agglutination titers in the blue crab, but the utility of this response was not documented (Pauley, 1973). The presence of agglutinins was not correlated with resistance in lobsters challenged with gaffkemia (Cornick and Stewart, 1973). Understanding the role of agglutinins in the defensive responses of marine invertebrates is a major area of research.

Insect defensins have broad specificity, do not react with eukaryotic cells, are easily synthesized (no specialization required), and are inducible (Smith and Chisholm, 1992). Crab defensins are likely to be similar to insect defensins, but work is only beginning to elucidate the role of such defensins in crustaceans. Callinectin is a low molecular weight peptide that occurs on the cell membranes of hemocytes of crabs (Khoo et al., 1996; Noga et al., 1996). The peptide shows specific activity against several marine bacteria, including *Vibrio* spp. (Noga et al., 1996). Declines in callinectin have been correlated with the presence of shell disease in crabs from sites polluted with high levels of phosphates and metals (Noga et al., 1994). Continued work on these low molecular weight peptides and their relation to insect defense systems will clearly be an avenue worth exploring.

Future Research Priorities

Diseases are important to the blue crab fishery. Yet, studying a pathogen because it affects a fishery is never enough to attract funds. As in all fisheries, the

conundrum lies in convincing resource managers that diseases do indeed impact the fishery. Pathogenic diseases have obvious negative impacts on individuals, but extrapolation to host populations and their dynamics can be difficult. Increasingly, research must show how to manage diseases in fished populations. Simple strategies may work such as taking precautions to limit the spread of disease (e.g., destroying infected animals, culling on station), or by incorporating changes in stock assessment models that require lowering catch limits in epizootic years, or by incorporating disease-induced mortality in overall mortality rates. That is, there must be application to the management of the resource. Other resource strategies, such as overfishing enclosed regions, and harvesting females to reduce the prevalence of pathogens, have received attention in the literature (Kuris and Lafferty, 1992).

Understanding the biology of the pathogen is critical to the success of any control efforts to mitigate the impact of the disease. Information is urgently needed on the transmission of several disease agents and the resulting mortality of the blue crab. Transmission and mortality studies are clearly the required first step in identifying the potential threat of a pathogen to blue crabs. Understandably, most studies will have to rely on artificial methods of infection like inoculation, or feeding infected tissues. However, water-borne transmission via contact and direct invasion of the host are probably the major avenues for many of the disease agents. Cannibalism may serve as a route of transmission of several pathogens as crabs are known cannibals, and infected crabs are often more susceptible to predation. The effect of diseases on predation rates is clearly important, but few, if any studies have examined possible relationships.

The development of molecular probes would enhance our understanding the life history, pathology and host-pathogen relationships for several agents. For example, the life cycle, life history and taxonomy of *Hematodinium perezi* may be clarified through specific PCR primers and DNA probes. Amphipods and other crabs may serve as hosts in the life cycle of *H. perezi* and molecular probes will help to identify whether these hosts are involved in transmission. Further, molecular probes could resolve potential affinities between *Paramoeba perniciosa* from the blue crab and the *Paramoeba*-like organism from lobsters from Long Island Sound. The rapid diagnosis of new pathogens such as *Paramoeba*-like organisms is critical to evaluating the potential threat of the disease to that fishery.

Identification of the environmental factors that contribute to epizootics should also be considered a high research priority. Laboratory studies should continue to identify the effects of specific factors such as temperature, salinity, dissolved oxygen on the growth and survival of the pathogens of blue crabs. When the roles of these factors are understood, they should be coupled with GIS to predict transmission, scale of impact, and spread of the disease agent.

In conclusion, epizootics of several pathogens occur in blue crabs with some regularity. They are frequently associated with high salinity waters, and, in some cases, with high water temperatures. The hydrography of the coastal region of the Eastern United States (mid Atlantic and Gulf of Mexico) may facilitate the spread of epizootics by retaining infected crabs in lagoonal waters, and by entraining or focusing the infectious agents in dense populations of the blue crab. The impact of disease agents on populations of the blue crab are not well understood, but as shown for *H. perezi,* infections in juveniles can reach very high prevalences (Messick, 1994; Messick and Shields, 2000) with potentially high mortality rates (Shields and Squyars, 2000). Given that agencies in several states annually survey blue crab populations, it would be prudent to incorporate monitoring of certain agents such as *Loxothylacus texanus,* shell disease, and, if possible, *Hematodinium*

perezi, in these surveys to enhance our understanding of the impacts of these agents in populations of the blue crab.

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