A Review Of The Impact Of Diseases On Crab And Lobster Fisheries

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Abstract

Diseases are a natural component of crustacean populations. Background levels of various agents are expected in fished populations, and there is good reason to establish baseline levels of pathogens in exploited fisheries before they become a problem. Such baselines are often difficult to fund or publish; nonetheless, outbreaks are an integral feature of heavily exploited populations. Mortalities or other problems can arise when an outbreak occurs, and all too often the underlying causes of an outbreak are poorly understood. A variety of stressors can lead to outbreaks of disease or contribute to their severity. Pollution, poor water quality, hypoxia, temperature extremes, overexploitation have all been implicated in various outbreaks. This review focuses on epidemic diseases of commercially important crabs and lobsters as well as a few examples of other disease issues in crustaceans that are ecologically important, but not of commercial significance.

Key words: pathogens, crustaceans, mortality, *Carcinonemertes*, PaV1, overfishing, *Rhizocephala*, *Paramoeba*

Introduction

Pathogens cause direct and indirect losses to crustacean fisheries. Direct losses are obvious, resulting in morbidity or mortality to the fished species, but they can be difficult to assess. However, mortality events can be widespread and can even damage the socioeconomics of impacted fishing communities, such as the lobster mortality event in Long Island Sound during 1999 (Pearce and Balcom 2005). Direct losses may also occur at the market where certain pathogens or syndromes, such as shell disease (Pearce and Balcom 2005), cause unappealing lesions on the carapace of lobsters rendering them less marketable in the live trade. Affected lobsters are processed in the lower-valued canned meat industry; hence they represent a direct loss to the higher grade live trade industry. Such losses can negatively influence public opinion as happened to the finfish industry during the *Pfiesteria* scare in 1997-1998, when presumably "exposed" fish were thought to contain presumptive toxins (Magnien 2001). At the time, the scare threatened the commercial fishing industry of Chesapeake Bay because consumers did not purchase fish from the region.

Direct losses are most visible to the fishery because the outcome is usually morbidity or mortality to the targeted component of the population. However, direct losses may also occur in the recruiting stock, the unfished segment of the population, which is typically include juvenile or female segments of the population. Outbreaks with resultant mortality to juveniles have been documented in at least three different fisheries that include the blue crab *Callinectes sapidus*, the snow crab *Chionoecetes opilio, C. bairdi*, and the Caribbean spiny lobster *Panulirus argus* (Messick and Shields 2000, Shields and Behringer 2004, Shields et al. 2005).

Indirect losses or indirect effects can be difficult to assess, because they are often cryptic and require long-term censuses of population trends. Basically, animals infected by one pathogen may become more susceptible to other diseases or to predation, or they may undergo loss of vigour or castration with the concomitant loss of egg or sperm production. Widespread castration and egg mortality resulting from parasites may potentially limit larval populations (Brattey et al. 1985, Wickham 1986), but these effects are difficult to establish at the population level. Nonetheless, mathematical models indicate that parasitic castrators can potentially regulate the impacted crustacean population (Blower and Roughgarden 1989 a,b). Rhizocephalan barnacles, which are known castrators, can be very prevalent in some crab populations. The castration that they inflict upon their hosts can also produce stunting and aberrant behaviors (Shields and Wood 1993). This review discusses examples of how outbreaks of parasites and diseases have impacted several crustacean fisheries. De-tails on outbreaks in cultured populations, such as shrimp, have been reviewed recently by Lightner (2005), Flegel (2006) and Walk-er and Mohan (2009). Simply defined, an outbreak is the occurrence of a pathogen at greater than background levels in a host population. In many cases, outbreaks of pathogens in crustacean fisheries were un-known or unreported from their hosts until the onset of an epizootic. This highlights the fact that baseline data has not been established on those diseases that affect many crustacean species. It also indicates that the causative agents may be newly emergent pathogens, or they are simply cryptic and remain unnoticed by fishers who might otherwise report sick or dying ani-mals.

Nemertean worms, overfishing, and the collapse of crab fisheries

The Dungeness crab (*Cancer magister*) supports several targeted fisheries off the western seaboard of the United States. The fishery off central California plummeted in the late 1950's. The once thriving fishery declined by 80-90% (Heiman and Carlisle 1970) and has never fully recovered. In 1976, three studies implicated egg mortality as a possible cause of the decline (Fisher 1976, Fisher and Wickham 1976). Egg clutches from crabs near San Francisco had high mortalities, with most clutches sustaining 10 to 50% egg mortality (Fisher and Wickham 1976). The clutches of crabs from Bodega Bay north to Washington had much less egg mortality. Fouling organisms, filamentous bacteria (e.g., *Leucothrix mucor*), and the fungus *Lagenidium* sp., were originally thought to have caused these mortalities, but the primary agent was later shown to be a nemertean worm, *Carcinonemertes errans* (Wickham 1979, Shields and Kuris 1988a). The intensities of worm infestations were highly correlated with egg mortalities; the latter often reached 100% on individual females from central California and nearly all ovigerous female crabs were infested (Wickham 1979, 1986). Moreover, the high prevalence of the worm and resultant egg mortalities correlated with the non-recovery of the central California stock of the crab (Fisher and Wickham 1976, Wickham 1979, 1980, 1986).

Correlations between worm prevalence, intensity and egg mortality over time led Wickham (1979, 1986) to speculate that three factors contributed to the outbreak of *C. errans*. The exploitation of the central California crab stock was extraordinarily

high in the late 1950's, with record landings in 1957. The intense fishing pressure and large biomass of landed crabs essentially led to mass settlement of worms on the few remaining crabs. The worm population was so large that every female crab became infected, but more importantly, high intensity infestations caused wide-scale egg mortality (c.f., population-wide castration), which was further exacerbated by fouling agents. With fewer crabs and increased exploitation, crab recruitment decreased, with the positive feedback of higher intensity worm infestations and additional egg predation fueling the decline in recruitment. Interestingly, in terms of fishing, central California is the southern-most part of the range for Dungeness crab, and this stock may be subject to large variations in recruitment. If this is the case, then the outbreak of *C. errans* would represent a new lower ecologically stable state, non-recovery of the stocks continuing into the future, as has happened, albeit the fishery has increased to about one quarter of its landings compared to its peak periods.

Given the possibility that nemerteans were contributing to the non-recovery of the fishery, worm infestations were included in a model to examine abundance of Dungeness crabs in a simulated population from northern California. Worm density did not cause destabilization of the crab population in the model, but worm density contributed to depressed population levels (Botsford and Wickham 1979). Later, an age- and sex-structured model of the crab population indicated that worms as a single factor could not destabilize the population model, but in combination with density-dependent recruitment, the parasites could have a destabilizing effect (Hobbs and Botsford 1989).

Infestations of *C. errans* on the Dungeness crab have not been studied since the mid-1980s. Because egg mortalities were generally lower in more northerly waters, and the fishery has only marginally recovered, continued surveillance of the worms and their related fouling agents would be integral to sound management of that fishery. Unfortunately, review of the published literature does not indicate that any such databases are under development.

Outbreaks of nemertean egg predators have occurred in other fisheries. In the early 1980's, the red king crab fishery (*Paralithodes camtschaticus*) off southern Alaska collapsed. The collapse occurred directly after a peak in crab abundance, which co-occurred with a peak in fishing pressure (Blau 1986, see also http://www.nmfs.noaa.gov/fishwatch/species/red_king_crab.htm). Interestingly, a herpes-like viral infection was reported from blue king crabs, *Paralithodes platypus*, and it was conjectured to have contributed to the decline of the red king crabs (Sparks and Morado 1986), but infections were not reported from the red king crab host. As with the outbreak in Dungeness crabs, egg mortalities were quite high in ovigerous red king crabs, reaching an average 90% egg mortality in all of the females within certain fjord or coastal areas (Kuris et al. 1991). Egg mortalities were caused by high intensity infestations of *Carcinonemertes regicides* and several other undescribed species of *Carcinonemertes* (Shields et al. 1989, 1990, Kuris et al. 1991). The infestations were seasonal and peaked in intensity during the early summer (Kuris et al. 1991). Interestingly, egg mortality was only correlated with worm intensity over the summer months because the worms did not remain on their hosts at high levels in other months (Shields et al. 1990). Fjord systems may trap or entrain the parasitic larvae into the more enclosed "closed system" of the fjord. Thus, the loss of eggs negatively affects larval recruitment into "closed" fjords

that have high worm abundances. The landings of red king crabs have not since returned to their former levels.

It is interesting to speculate that fishing pressure has somehow contributed to the outbreaks of nemertean worms on Dungeness and red king crabs. That is, removal of one segment of the fished population somehow served to amplify infestations on the remaining hosts. Fishing pressure was certainly high on these crab hosts, and the reduction of females, or the loss of males, might have led to amplified parasite intensities on remaining brood stock. However, fishing activity is not necessarily required for this amplification. An outbreak of *C. epialti* was reported from an unfished or lightly fished population of *Hemigrapsus oregonensis* (Shields and Kuris 1988b). The fishing pressure on *H. oregonensis* was light prior to the outbreak, as they are used as bait by the recreational fishers and little else.

Pseudocarcinonemertes homari is a nemertean that infects the American lobster, *Homarus americanus*. This worm is usually found at low intensities on its host, but high intensity infestations have been reported (Aiken et al. 1985). Grooming activity by the female reduces the burden of the worms, but some lobsters are not fastidious groomers and have heavy infestations with resultant egg mortality. Prevalences of *P. homari* are usually quite low (Aiken et al. 1983), but one apparent outbreak was reported from lobsters off Grand Manaan Island, Nova Scotia, Canada (Brattey et al. 1985). In that instance, the prevalence among ovigerous females was 78.7% with a mean intensity of 102.4 worms per infected host. Egg mortality due to *P. homari* correlated with the intensity of infection (Campbell and Brattey, 1986). More than 16% of the clutches of lobsters from off Grand Manaan Island had >50% egg mortality. Mean egg mortality over time ranged from 38% to 43% of the clutch with complete brood loss reported among heavily infested lobsters (Aiken et al. 1985, Brattey et al. 1985).

The ecological effects of *P. homari* on the fisheries for the American lobster do not appear to be as severe as those for *C. errans* and *C. regicides*; and it is interesting to speculate on why this may be the case. Certain life history characteristics play a role in the outbreaks of both *C. errans* and *C. regicides*, and these may not be the case for *P. homari.* For *C. errans*, the juveniles apparently survive for long periods on the sternum, abdomen, and axillae of their non-ovigerous hosts (Kuris 1993). Over time, a crab may accumulate a heavy infestation. Worms are also transmitted sexually from males to females (Wickham et al. 1984), a feature which may serve to amplify infections onto female crabs. For *C. regicides*, newly hatched larvae can reinvade the host from which they hatched (i.e., auto-infection); therefore, worm populations can reach extremely high levels (>600,000 worms per clutch) over a short summer period (Kuris et al. 1991). Contributing to the amplification of *C. regicides* is the fact that prevalence was highest in fjords, particularly those with shallow sills, and these fjord systems have limited water exchange with the open ocean; thus infectious larvae may be retained within the fjord system (Kuris et al. 1991). Interestingly, egg mortality was only correlated with worm intensity over the summer months namely because the worms did not remain on their hosts at high levels over other months (Shields et al. 1990). For *P. homari*, perhaps epidemics do not occur because most lobsters preen their clutches, reducing their infestations, or perhaps lobsters do not inhabit areas with restricted water circulation, or mortality occurs over a shorter time span than when the worms are present.

Stressors in Long Island Sound

The American lobster supports a large fishery in Long Island Sound, USA. Prior to 1999, the lobster industry in New York and Connecticut had annual harvests of 10-11 million pounds, valued at over $40 million (Pearce and Balcom 2005). Several diseases have recently impacted the fisheries and management of the American lobster in these waters. In 1999, the pathogenic amoeba *Neoparamoeba pemaquidensis* emerged in concert with other stressors to decimate the commercially important population in western Long Island Sound (Mullen et al. 2004, 2005, Pearce and Balcom 2005). Mortalities also occurred in many other species such as the blue crab (*Callinectes sapidus*), spider crabs (*Libinia* spp.), rock crab (*Cancer irroratus*) and the horseshoe crab (*Limulus polyphemus*). However, the pathogenic amoeba was not linked to mortalities in any species other than *H. americanus* although it must be noted that other host species were not examined. At about the same time, lobsters from eastern Long Island Sound and Block Island Sound were experiencing an outbreak of epizootic shell disease (Castro and Angell 2000). In 2002, lobsters suffering from calcinosis, a physiological response to temperature stress, were also evident within central areas of Long Island Sound (Dove et al. 2004); and at about the same time, ~50% of the lobsters in western portions of the sound were diagnosed with blindness (Maniscalco and Shields 2006, Magel et al. 2009). In addition, another emergent pathogen, *Vibrio fluvialis* was implicated in lobster mortalities off the coast of Maine (Tall et al. 2003).

The emergent disease issues in Long Island Sound appeared to be related to environmental changes, such as increased bottom temperatures during summers, the general effects of eutrophication (Pearce and Balcom 2005), and intoxication from anthropogenic substances (Zulkowsky et al. 2005). Virtually all of these stressors can lead to an immunologically compromised animal (Paterson and Stewart 1974, DeGuise et al. 2004), with weakened host defenses, which can lead to secondary infection. What made these situations especially problematic was that the observed decline in lobster health and viability may have causes that were linked to a convergence of more than one factor. Moreover, these diseases are indicators of environmental change and anthropogenic degradation, and their emergence may have implications for additional regulation of industrial chemical discharges into marine and estuarine environments.

For example, from 1997 to the present, epizootic shell disease has affected lobsters from Naragansett Bay and Block Island Sound off Rhode Island (Castro and Angell 2000, Castro et al. 2005, Landers 2005, Powell et al. 2005), and from Buzzards Bay north to Cape Cod Bay off Massachusetts (Glenn and Pugh 2005). In addition, the incidence of epizootic shell disease has markedly increased, with prevalence ranging from 25-65% of the lobster population, particularly in ovigerous females (Castro et al. 2005, Glenn and Pugh 2005, Howell et al. 2005, Landers 2005). Epizootic shell disease appears to reduce both the quantity and quality of commercial lobster landings (Cobb and Castro 2006). Heavily infected animals are not marketable due to gross external pathology, which in severe cases presents as the nearly complete erosion of the dorsal carapace and claws of the lobster.

The etiology and pathogenicity of the disease remains to be determined. However, it is partially non-infectious, caused by environmental changes, and partly an infectious one, caused by chitinoclastic bacteria

(Chistoserdov et al. 2005). The dynamics of infection are partly known, in that infected lobsters can molt successfully, but they can become rapidly reinfected (Castro and Angell 2000). The disease syndrome is more abundant in the fall, after animals have molted.

Epizootic disease has not been shown to be horizontally transmitted to healthy lobsters in laboratory experiments (Chistoserdov et al. 2005a, b). It is associated with bacterial species other than *Vibrio* spp., such as members of the Flavobacteriaceae, but there is no apparent relationship with bacteria other than with chitinoclastic forms (Chistoserdov et al. 2005a, b). It is histologically different from classical shell disease because of the formation of pseudomembranes under the large friable areas of necrotic cuticle (Smolowitz et al. 2005a, b). Classical shell disease can be transmitted horizontally to healthy lobsters (Bullis 1989), as observed in many lobster holding facilities (Getchell 1987, Geddes et al. 2003).

Long Island Sound and Buzzards Bay have high levels of environmental contaminants, including PCBs, pesticides, metals, and PAHs (e.g., http://www.longislandsoundstudy.net/pubs/facts/fact10.pdf, http://www.buzzardsbay.org/toxicact.htm). Indeed, a major oil spill off Naragansett Bay, Rhode Island, killed an estimated 7 million lobsters, immediately prior to occurrence of epizootic shell disease from that region (North Cape oil spill fact sheet, http://www.dem.ri.gov/news/2006/cape/factsgen.pdf).

Further, Biggers and Laufer (2004) and Laufer et al. (2005a) found that lobsters with epizootic shell disease had higher levels of alkylphenols than did healthy lobsters, and that bottom sediments had higher than normal levels of these compounds in areas where diseased lobsters resided. Four alkylphenols related to Bisphenol A have been found in lobster tissues, some at relatively high levels. The alkylphenols (2-t-butyl-4-(dimethylbenzyl)phenol; 2,4-bis-(dimethylbenzyl)phenol; 2,6-bis-(t-butyl)-4-(dimethylbenzyl)phenol; and 2,4-bis-(dimethylbenzyl)-6-t-butylphenol) have been used as antioxidants and surfactants in industrial applications, and one (2,6-bis-(t-butyl)-4-(dimethylbenzyl)phenol-MON-0585) has been tested as a pesticide because of it is a Juvenile Hormone analog. Bisphenol A is a known endocrine disruptor and the alkylphenols found in lobsters are known analogs for Juvenile Hormone that can disrupt molting activity (Biggers and Laufer 2004). Lobsters from Long Island Sound had over 21.0 µg/ml of 2,6-bis-(t-butyl)-4-(dimethylbenzyl)phenol in their hemolymph compared to <0.5 µg/ml in lobsters from nearby Vineyard Sound (Biggers and Laufers 2004). Ovigerous lobsters with epizootic shell disease also have increased ecdysone levels, >10 times higher than unaffected ovigerous animals, and the increased ecdysone may induce abnormal molting activity in these animals during a sensitive period (Laufer et al. 2005b). Much remains to be done to identify the stressors involved in this syndrome, but the relatively narrow confinement of diseased animals to Rhode Island, eastern Connecticut and southern Massachussetts suggests a pivotal role for a contaminant.

Paramoeba outbreaks in the blue crab

Paramoeba perniciosa causes “gray crab” disease in the blue crab. The sternums of heavily infected and dead crabs become discolored with a characteristic gray color (Sprague and Beckett 1966, Johnson 1977). Outbreaks of *P. perniciosa* are focal and are typically noticed in shedding facilities when a large number of crabs become moribund and die. Around Chesapeake Bay, mortal-

ities occur in shedding facilities from May to June and in the dredge fishery from October to February (Couch 1983). During epizootics, prevalence ranged from 17 to 35% in premolt juvenile crabs at shedding facilities on Chincoteague Bay (Sawyer 1969, Sprague et al. 1969). Newman and Ward (1973) assessed mortality at 30% per month from Chincoteague Bay. Prevalence reached >20% during winter dredge samples from Chincoteague Bay and prevalence ranged from 3 to 13% in lower Chesapeake Bay (Couch 1983). After peak mortality events, prevalence dropped to 8% in trawled crabs (Sawyer 1969). Such declines in prevalence probably reflect host mortality and not a seasonal reduction of disease or any increase in host resistance.

Heavily infected crabs are sluggish and die shortly after capture (Johnson 1977). Crabs with light and moderate infections often exhibit no overt sign of disease. Infected premolt crabs die shortly after molting, presumably because the increased metabolic demands of molting, causes additional stress to the diseased animal.

The mode of transmission of *P. perniciosa* remains unknown. Transmission experiments with the amoeba have been inconclusive (Johnson 1977, Couch 1983). Cannibalism may spread the disease when infected lethargic and moribund crabs are eaten by conspecifics (Johnson 1977). Infections, however, have not been experimentally established by feeding infected tissues to naïve hosts nor by inoculating them with infected hemolymph (Newman and Ward 1973, Johnson 1977, Couch 1983). The fact that infected crabs are found in high salinity waters, coupled with the fact that blue crabs are osmoregulators, suggest that cannibalism plays little role in transmission; otherwise, infections would be sustained at low and moderate salinities. Because mortalities peak in late spring, Newman and Ward (1973) and Couch (1983) speculated that transmission may occur during ecdysis or in post molt when the carapace is soft.

Paramoeba perniciosa infects blue crabs from Long Island Sound south to the Atlantic coast of Florida (Newman and Ward 1973, Johnson 1977) and the ameba is relatively common in the coastal bays of the Delmarva Peninsula in Delaware, Maryland, Virginia and south to Georgia (Sprague and Beckett 1966, Sawyer 1969, Messick 2002). It has not been reported from the Gulf of Mexico (Overstreet 1978, Messick 2002). The disease exhibits a strong summer peak in prevalence (Newman and Ward 1973, Johnson 1977), but infections can persist into winter (Couch 1983). Amebae apparently overwinter in crabs (Johnson 1977), but a histological study of overwintering crabs is needed to confirm this. Temperature and seasonality play important roles in outbreaks of amebic infections in other species. *Paramoeba invadens* occurs in epizootics in green sea urchins, *Strongylocentrotus droebachiensis*, which have been correlated with increased water temperatures (Scheibling and Hennigar 1997, Scheibling et al. 2010), and *P. pemaquidensis* (now *Neoparamoeba pemaquidensis*) causes disease in cultured salmonids (Kent et al. 1988, Roubal et al. 1989) as well as lobsters (Mullen et al. 2004).

The blue crab is the primary host for *P. perniciosa*, but the green crab (*Carcinus maenas*), Jonah crab (*Cancer borealis*), the lesser blue crab (*Callinectes similis*), and American lobster have also been reported as hosts (Sawyer 1976 as cited in Sawyer and MacLean 1978, Campbell 1984, Messick 2002). The paucity of reports of disease from these hosts suggest that they are not important reservoirs for the ameba *Paramoeba perniciosa* infections in blue crabs

appear to have a strong association with seaside coastal bays and lagoons. This type of "landscape" association with a disease has been noted for other parasitic diseases of crustaceans, namely *Hematodinium* infections in snow crabs and blue crabs (Meyers et al. 1987, Shields 1994, Messick and Shields 2000, Shields et al. 2005), and rhizocephalan infections in king crabs (Sloan 1984).

Insidious effects of rhizocephalans

Rhizocephalans are bizarre, highly modified barnacles that cause interesting pathologies to their hosts, including castration, feminization, anecdysis, or a cessation of molting, stunting, and mortality. They are cryptic and masquerade as the egg mass of their host. While their effects can be insidious to individuals, the range in prevalence of some species is elevated enough to cause direct effects on the population biology of their hosts.

Several species of commercially important crustaceans are infected by rhizocephalans. *Briarosaccus callosus* infects lithodid king crabs in boreal, Antarctic and Arctic waters. High prevalences, 50% to 75%, have been reported in king crabs in fjords off British Columbia (Sloan et al. 1984) and southeastern Alaska (Hawkes et al. 1986). Prevalence was about 20% in *Paralomis granulosus* in the deepwater canyons around South Georgia Island (Watters 1998). Infections of *Sacculina granifera* in *Portunus pelagicus* can reach prevalences of 40% or more (Phillips and Cannon 1978, Weng 1987, Shields and Wood 1993). Infections of *Loxothylacus texanus* in the American blue crab, *C. sapidus*, range in prevalence from 30 to 70% in lagoons and embayments around the Gulf of Mexico (Christmas 1969, Ragan and Matherne 1974, Wardle and Tirpak 1991, Alvarez and Calderon 1996, Lazaro-Chaves et al. 1996). *Sacculina carcini* can reach a prevalence over 90% in the green crab, *C. maenas* (Stentiford and Feist 2005), and local patchiness in prevalence is a relatively common phenomenon with this species (Heath 1971, Stentiford and Feist 2005) as well as other species (Tolley et al. 2006, Sloan et al. 2010). *Sylon hippolytes* occurs on a variety of shrimp and can reach high prevalences, but these parasites have received comparatively little attention (Bower and Boutillier 1988).

Many rhizocephalan infections stunt the growth and molting of their hosts. This is typically the result of interference with the hormonal regulation of molting in the hosts, causing anecdysis. When the prevalence is high, a large proportion of the host population can become stunted, including blue crabs (*C. sapidus*) and lithodid crabs (Overstreet 1978, Over-street et al. 1983, Hawkes et al. 1986, Höeg 1995). Stunted animals do not enter the fishery and they may accumu-late in the fishing grounds as "shorts" (Hawkes et al. 1986, Meyers 1990). Re-turned "shorts" may artificially increase the prevalence of the parasite and serve as foci for transmission to new hosts (Hawkes et al. 1986, Meyers, 1990). The potential for ac-cumulation of shorts in a fished population has led to the suggestion to actively overfish isolated populations, which would remove the parasites (Hawkes et al. 1986, Kuris and Lafferty 1992, Shukalyuk et al. 2005), but this practice has not been implemented.

Rhizocephalan infections can have other insidious, indirect effects on their hosts and host populations. Indirect effects of infestation at the population level may include castration, sterile matings, loss of fecundity, homosexual matings, and competition with phenotypically identical parasites (Shields and Wood 1993). The potential for compe-

tition between castrated crabs (phenotypically crabs, but genetically reproductive parasites) and uninfected crabs has not been explored and could prove interesting in terms of the ecology of the parasite as well as the behavioral ecology of the host-parasite association.

Several environmental factors facilitate outbreaks of rhizocephalans. High prevalences of *Briarosaccus callosus* have been associated with turbid fjords in British Columbia and Alaska, particularly those with shallow sills (Sloan et al. 1984, Hawkes et al. 1986). The fjord water either has a long residence time within the system or it becomes entrained allowing the transmissive cyprid larva of the parasite to more readily find and infect new hosts than in more open oceanic systems. Higher prevalences may co-occur with turbid waters, which may reduce the ability of crabs to clean their gills, which are the initial sites of infection (Hawkes et al. 1986). Shallow lagoons and embayments with long residence times are also thought to facilitate high prevalences of rhizocephalans and *Hematodinium* infections in portunid crabs (Alvarez and Calderon 1995, Shields and Overstreet 2007). Confined habitats may limit the dispersal of the parasite larvae, limit their dilution, and lead to high prevalences (Kuris and Lafferty 1992). Other environmental factors such as temperature, seasonality, salinity, depth and turbulence are known to influence the prevalences of rhizocephalans in non-commercial species (Ritchie and Høeg 1981, Høeg 1995, Høeg et al. 2006).

A model of the parasitic castrator *Hemioniscus balani* and its barnacle hosts suggest that rhizocephalans can regulate their host populations (Blower and Roughgarden 1989 a,b). Other models have examined how castrators can affect their host populations when their ecological connections are within hydrodynamic or demographic closed systems (Sloan et al. 1984, Kuris and Lafferty 1992). These models indicate that rhizocephalans can reach a high prevalence in their host populations when the model conditions simulate a closed circulation system with internal recruitment. Therefore, an outbreak of rhizocephalans in a commercially fished population of crabs or shrimps could have significant potential to damage the fishery by altering the population structure of the fished population.

Bitter crab disease by *Hematodinium*

In the early 1980s, large numbers of Tanner crabs (*Chionoecetes bairdi*) from fjords in southeastern Alaska developed an unusual condition which made them taste bitter and unmarketable. The condition was called bitter crab syndrome or bitter crab disease and was caused by a parasitic dinoflagellate, *Hematodinium* sp. (Meyers et al. 1987, 1990). At that time, estimated losses to the industry were >$250,000, and these losses were centered in the fjords of southeastern Alaska (Meyers et al. 1987). Infected crabs represented up to a third of the commercial fishery in southeastern Alaska (Meyers et al. 1990). Outbreaks of *Hematodinium* spp. have since been reported from a number of commercially important crabs and lobsters (Stentiford and Shields 2005).

Two outbreaks of *Hematodinium* sp. have been documented in snow crabs, *C. opilio*, from Newfoundland. Taylor and Khan (1995) initially reported the parasite at low levels during the early 1990's; however, in later surveys its prevalence increased significantly in the coastal bays (Pestal et al. 2003, Shields et al. 2005, 2007). The first outbreak in snow crabs occurred in Bonavista and Conception Bays in 1999 and 2000 and mostly affected female and juvenile crabs (Shields et al. 2005). From

2003 through 2005, another epidemic occurred, but it primarily affected large-clawed mature males, with a prevalence of up to 35% (Shields et al. 2007). This latter epidemic was associated with a 1ºC increase in bottom temperature, which caused an apparent five-fold increase in molting activity in larger crabs. Because crabs are infected shortly after molting, the large number of newly molted, susceptible animals fueled the epidemic over the three year period (Shields et al. 2007). Interestingly, female and juvenile crabs had similar prevalences to those in the earlier outbreak. There was no outbreak in Bonavista Bay during 2003 through 2005 and that bay apparently has a shorter residence time compared to Conception Bay, which has a shallower sill and a longer residence time (DeYoung and Sanderson 1995).

In the late 1980's, an outbreak of *Hematodinium* sp. was partially documented in the velvet crab (*Necora puber*) fishery off Brittany, France (Wilhelm and Mialhe 1996). The relatively small fishery suffered a catastrophic loss due to the parasite. Infections of the same or a related parasite have occurred in *Maia squinado* and *C. pagurus* from Brittany and the English Channel, but the prevalence in these hosts remains undetermined (Latrouite et al. 1988, Stentiford et al. 2002).

In the late 1980's and early 1990's, outbreaks of *Hematodinium* were also documented in the Norway lobster fishery off western Scotland (Field et al. 1992). Losses to the fishery were estimated at £2-4 million annually (Field et al. 1992). Outbreaks occur seasonally in the fishery and prevalences can reach 70% during the height of the outbreaks, which occur in winter months (Field et al. 1998, Stentiford et al. 2001). As with other *Hematodinium* infections, the highest prevalences of the disease occurred in and around the Scottish fjords (Field et al. 1992, 1998, Stentiford et al. 2001). As with snow crabs, Norway lobsters are apparently in-fected after molting (Stentiford et al. 2001). In years that there is synchrony in the molting activity of males and females, prevalences show a peak in infections. In years when molting is asynchronous between males and females, there is a lower 'plateau' in prevalence.

Outbreaks of *Hematodinium* are common in the American blue crab and show a distinct autumnal periodicity (Messick and Shields 2000). In Virginia and Maryland, periodic summer and autumn mortalities are cryptic, but losses may exceed $500,000 per year in nonepidemic years (Stentiford and Shields 2005). An outbreak in the early 1990's reached a prevalence of 100% and was primarily centered on juvenile crabs (Messick 1994). Similar outbreaks have been reported off the coast of Georgia (USA), particularly in relation to drought (Lee and Frischer 2004). Along the Delmarva Peninsula, out-breaks occur in the shallow coastal bays and lagoons (Messick 1994, Shields 1994, Messick and Shields 2000). Prevalences are generally much lower at the mouth of Chesapeake Bay, which is a more open physiographic environment.

Virtually all outbreaks of *Hematodinium* are associated with distinct physiographic features (Stentiford and Shields 2005), such as fjords or shallow poorly drained bays and lagoons, but key spatial features of these outbreaks have not been adequately described. For example, deep "confined" areas and certain habitat types (mud, sand, gravel, shell) are associated with infections in snow crabs, but the nature of the association remains to be determined (Shields et al. 2005). The fjords of Alaska, British Columbia and Scotland and the shallow bays of the east

coast of America are ideal for the growth and spread of pathogens. These regions possesses several features that facilitate epidemics of *Hematodinium* and other pathogens: (1) relatively "closed" host populations (i.e., those with little immigration and emigration of juveniles and adults; but not necessarily closed to larvae), (2) restricted exchange with the open ocean (i.e., narrow channels with shallow sills, deep confined areas, entrained water masses), (3) stressful conditions for the crab population (i.e., summer and winter thermal stress, high salinity, seasonal hypoxia, intense fishing pressure), and, of course, (4) a pathogen that can amplify rapidly within a population (Shields 1994). Several of the other parasites discussed in other sections of this paper have similar associations: e.g., *Briarosaccus callosus* (Sloan et al. 1984), *Loxothylacus texanus* (Alvarez and Calderon 1995), *Sacculina granifera* (Shields and Wood 1993), *Carcinonemertes regicides* (Kuris et al. 1991) as well as infections of *Hematodinium* (see above).

Other emerging diseases

This review has focused on a few salient diseases of fished crabs and lobsters. However, several epidemics of viruses have occurred in shrimp farms and these have spread to become catastrophic pandemics in shrimp fisheries. The viruses responsible for these outbreaks (e.g., white spot syndrome virus, yellow head virus, Taura virus, gill associated virus, baculoviruses) have been well reviewed (Lightner 2005, Flegel 2006, Walker and Mohan 2009). I will not review them here. There are also documented epi-zootics of oomycete pathogens in freshwater and marine copepods (Redfield and Vincent 1979, Burns 1985) and parasitic ciliates in euphausiids (Capriulo et al. 1991, Gomez-Guiterrez et al. 2003) that may have serious implications for production in these ecologically important host populations. The latter ay have repercussions to pelagic food webs. There is an expansive literature on crayfish plague, or krebspest and its effects on the European crayfish, or noble crayfish (*Astacus astacus*). Crayfish plague is caused by the pathogenic oomycete, *Aphanomyces astaci.* The pathogen fulminated into a widespread pandemic in Europe during the 1860's, and it was the first disease outbreak that was documented in a crustacean. The pathogen was introduced to northern Italy in the early 1860's, probably from contaminated ballast water in ships sailing from ports in the U.S.A. or Canada (Edgerton et al. 2002). From there, the plague rapidly spread northward and was reported within France in 1875, Germany in 1880, Russia in 1890, Finland in 1893, and then Sweden and Norway. In Europe, *A. astaci* decimated native stocks of crayfish, particularly *Astacus astacus,* which is a delicacy in Northern Europe. The disease has since been reported from Spain in 1958, Greece in 1982, the United Kingdom in 1981, and Turkey in 1984 as well as Ireland in 1987 (Holdich and Reeve 1991, Edgerton et al. 2002). It has also spread through the introduction of infected signal crayfish, *Pacifastacus leniusculus,* from the U.S.A. during attempts to revive the crayfish industry in Northern Europe (Edgerton et al. 2002). Many of the studies on this pathogen have focused on host immunology or control of the infection. For recent reviews of crayfish diseases, see Edgerton et al. (2004) and Oidtmann et al. (2002).

Conclusions.

This review has concentrated on significant diseases that principally affect commercial fisheries of crabs and lobsters. Because of the nature of commercial fisheries, i.e., harvest as opposed to culture, there is the

perception that little can be done to limit the effects of pathogens on fished populations. Actually, that perception may be false. Few if any models use disease data (e.g. – prevalence, distribution) to estimate effects or otherwise manage a fishery. Early modeling attempts have asked whether diseases could be managed in fished populations by "fishing out" the affected population through increased fishing activity (Kuris and Lafferty 1994). The model indicated that in systems with closed recruitment, such as might be expected in fjords or entrained water masses, such an approach might work. Other models have indicated that pathogens, such as parasitic castrators, may exert significant population level effects on their hosts, i.e., regulation or declines of their crustacean host populations (Blower and Roughgarden 1989a, b). The development of spatial models for systems with disease has great potential because such models can incorporate biological data and integrate it over different spatial scales. With such mod-els, 'at risk' populations can be identified, especially where the structure (e.g. sex- or size-bias) of a population makes it susceptible to epizootics, or the impact(s) of the disease can be examined in relation to changes in fishing practices. Spatial models of disease have not been applied to crustacean fisheries, but they may be very useful in future studies of transmission and population-level effects on both the host and the pathogen.

In several instances, disease issues are more common in juveniles rather than adults. For example, species of *Hematodinium* have a predilection for juvenile hosts (Stentiford and Shields 2005). While adult hosts become infected, they generally have lower prevalences than juveniles except during epidemics. Other pathogens such as *Panulirus argus* Virus 1 and White Spot Syndrome Virus also have a predilection for juveniles (Shields and Behringer 2004, Flegel 2006). Juvenile crustaceans molt more frequently than adults, and those disease agents that are transmitted via molting would have an increased opportunity to infect juvenile hosts during this stressful time. However, not all of these pathogens use molting as the portal of entry. Some such as WSSV are spread via cannibalism (Lightner and Redman 1998, Lighnter 2005) and others such as PaV1 and WSSV can be spread via waterborne transmission (Butler et al. 2008). The reason for their increased prevalence in juvenile hosts is not clear. However, what is clear is that the industries for these crustaceans focus on the adults and not on the juveniles; hence, the agents of disease are often ignored or not thought to be important to recruitment. Nonetheless, pathogens such as PaV1, *Hematodinium* sp., *Aerococcus viridans* and *Anophryoides haemophila* may seriously affect their hosts and may play a role in the population dynamics of host stocks. They will become increasingly important pathogens with the advent of increased climatic variability associated with global warming (Shields et al. 2007); hence, it is important that we understand basic elements of their life history, transmission and pathogenicity prior to their emergence in larger outbreaks and epidemics.

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