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Diseases of American lobsters (Homarus americanus): A review

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ABSTRACT

The American lobster fishery is a significant economic driver in coastal communities of North America. Increasingly, the impacts of infectious disease are recognized as important components and factors in the population ecology and subsequent management of the lobster fishery. Both environmental and anthropogenic factors impact marine diseases. The review herein highlights aspects of several important bacterial, fungal and protistan diseases, including gaffkemia, shell disease, vibriosis, disease caused by species of *Lagenidium*, *Haliphthoros* and *Fusarium*, paramoebiasis and Bumper Car disease. As the global environment continues to change, these diseases could more severely affect both wild caught and impounded lobsters.

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1. Introduction

The American lobster *Homarus americanus* is a major economic factor in the coastal communities of North America. In the five Atlantic Canadian provinces of New Brunswick, Newfoundland Labrador, Nova Scotia, Prince Edward Island and Quebec, ~53,370 metric tonnes of lobster were harvested in 2006, with landed value of ~\$633 million (Fisheries and Oceans Canada, 2008). The majority (59%) is harvested in Nova Scotia. In the Atlantic states of Connecticut, Delaware, Maine, Massachusetts, New Hampshire, New Jersey, New York, Rhode Island and Virginia, ~42,000 metric tonnes were landed, with value of \$~395 million. The majority (78%) is harvested in Maine (National Marine Fisheries Service, 2008). Although

the American lobster fishery has traditionally been, and continues to be been significant, there are few recent reviews of health and disease (see Cawthorn, 2005a; Brock and Lightner, 1990; Meyers, 1990; Shields et al., 2006; Stewart, 1980 as examples). There are major challenges associated with evaluating infectious and noninfectious diseases in marine environments. The recent review of Harvell et al. (2004) proposes major research priorities to address these issues: (1) development of diagnostic tools to identify pathogens and to determine their origin and spread; (2) development of rapid response capabilities to identify, monitor and manage disease outbreaks; (3) determination of the life cycles, longevity and host range of various stages of pathogens; (4) evaluation of the role of environmental and anthropogenic factors in disease outbreaks; and (5) development of forecasting models of disease in aquatic hosts, sensitive to environmental (climatic) factors. These are issues which can be significant impediments to understanding the etiology and ecology of marine pathogens.

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For lobsters, a major symposium recently focused on their uses as model organisms for the study of behavior, ecology and fisheries (Factor et al., 2006) Shell disease of lobsters was evaluated as the model system of disease in large decapods (see Factor et al., 2006). Surprisingly, there are relatively few diseases in American lobsters. However, gaffkemia, shell disease, neoparamoebiasis, and bumper car disease, as presented in this review, may have important roles in the natural mortality of lobsters. The complexity of outbreaks of these diseases highlight both the highly dynamic internal and external environments of lobsters. The review focuses on bacterial, fungal and protist pathogens, because of their economic importance to various segments of the lobster fishery. Human-induced changes of our global environment impact arboreal, terrestrial and aquatic habitats. Therefore, it is important to ask whether epidemics are increasing among marine animals because of global warming and increasing pollution, or do these observations reflect our concern with our environment (Harvell et al., 1999; Ward and Lafferty, 2004).

2. Bacterial diseases

2.1. Gaffkemia – Aerococcus viridans

Gaffkemia is one of the most important and well described infectious diseases of American lobsters, primarily as a disease of impounded lobsters (see reviews by Sindermann, 1990; Stewart, 1984, 1993). The bacterium and resulting disease were first described in a holding facility in Maine (Snieszko and Taylor, 1947). Although the focus herein is on American lobsters in North American waters, gaffkemia also occurs in the European lobster *Homarus gammarus* (Gjerde, 1984; Stewart, 1993). Translocation of the etiologic agent likely occurred to Europe by movement and release of American lobsters for commercial purposes; accidental release of infected lobsters and dumping of infected offal are means whereby the bacterium could also be established (Alderman, 1996).

The causative agent. Aerococcus viridans (var.) homari is a freeliving, gram positive, tetrad-forming coccus, leading to systemic disease in homarid lobsters (Stewart, 1980). The bacterium cannot cross intact lobster integument and does not survive gastric acids; consequently invasion of the host occurs via wounds or punctures of the cuticle. The organism survives well in the benthic environment (i.e. mud) and in holding facilities (i.e. biofilm of tanks, piping), in its freeliving form outside the host. A very few organisms (i.e. 10 per kg body weight) of virulent strains of A. viridans will rapidly cause fatal disease, at the appropriate environmental temperatures. Infection progressing to disease is temperature dependent, with death occurring in 180 days at 3 °C to 2 days at 20 °C. No deaths occurred at 1 °C during an observation period of 250 days (Stewart, 1984, 1993). Recently Battison et al. (2004), using a field isolate of A. viridans var. homari, demonstrated that survival of lobsters was related to both experimental temperature and size of inoculum. Apparently several factors within the triad of host, pathogen, and environment affect survivability of lobsters and alterations in total hemocyte counts during the course of gaffkemia. Stressors include strain of bacterium, handling, temperature changes, and trauma. There are decapod reservoirs of infection of A. viridans in the marine environment: however, homarid lobsters are most susceptible to infection and disease (Brock and Lightner, 1990).

Clinically, there are no obvious signs in infected lobsters, other than weakness or lethargy and a spread-eagle posture which are apparent in later stages of the disease, and which are not pathogonomic for gaffkemia. Lobsters rapidly become anorexic after infection. The bacteria multiply rapidly in the hepatopancreas then in the heart; the pathogen only develops in the hemolymph much

later in the infection (Stewart and Arie, 1973). However, hemolymph is an excellent growth medium for A. viridians. Pink discoloration of the ventral abdomen and hemolymph can develop, hence the name "red tail" disease (Snieszko and Taylor, 1947). Most bacteria multiply readily in fixed and circulating phagocytes, with no major ill effects on the organisms. There is no bacterial toxin produced. Death likely results from metabolic incapacity resulting from dysfunction of the hepatopancreas. Additionally, the clotting mechanism is impaired, and it is associated with marked hemocytopenia. Infected lobsters can become exsanguinated, especially in end-stage disease (Stewart et al., 1969). Johnson et al. (1981) concluded that gaffkemia manifests as a non-toxic bacteremia; although hemocytes, including phagocytes are affected, there are no significant tissue lesions. The pattern of total hemocyte counts indicates an inflammatory-like response develops during course of the disease (Battison et al., 2004).

Early infections with A. viridans can be detected in lobsters with various culture methods (i.e. presumptive phenylethyalcohol broth test) and examination of hemolymph smears (Stewart et al., 1966). However, culture methodology typically requires 4-7 days for complete analyses. Heavy infections can be detected by direct Gram stain of hemolymph smears or the co-agglutination technique of Saxegaard and Hastein (1978). Newer diagnostic tools include a polymerase chain reaction/oligonucleotide probe which is highly sensitive and specific to detection of A. viridans (Grant et al., 1992). However, none of the strains tested were A. viridans var. homari. This relatively rapid (5 h) technique is primarily used for human disease investigations. An indirect fluorescent antibody technique, developed to detect A. viridans from lobsters, is a major advance (Marks et al., 1992). The antiserum was prepared from strains of A. var. homari. The method is specific and rapid: in light infections which require bacterial incubation, the time required is only 48 h; in heavy infections, with application directly to hemolymph smears, the time required is only 2 h. The technique was critically evaluated using traditional bacteriological procedures in a large number of (>1000) lobsters with a presumptive diagnosis of gaffkemia. This approach has significant utility for large scale surveys targeting A. viridans in wild lobster populations (Marks et al., 1992).

Although A. viridans var homari is sensitive to several antibiotics (Brock and Lightner, 1990) and disinfectants (Stewart and Cornick, 1967), the only therapeutant approved for use in Canada is oxytetracycline. The product is incorporated into feed for prevention and treatment of gaffkemia in impounded lobsters (Syndel, 2008). However, during outbreaks of disease, affected lobsters are anorexic and unlikely to consume adequate quantities of medicated feed. The withdrawal period is a minimum of 30 days at 4 °C. Major challenges associated with use of oxytetracycline in the marine environment include persistence in sediments, bioaccumulation by bivalves or other crustaceans, and development of bacterial resistance (Burridge, 2003). Oxytetracycline sensitivity in humans is also a concern (see for example www.drugs.com/cons/oxytetracycline); the 'greener' the fishery and its products, the better is public perception and acceptance. Although several disinfectants were effective experimentally, Stewart and Cornick (1967) indicated that field testing was necessary. Additionally, there are newer disinfectants available which should be evaluated for use in lobster holding facilities.

An alternative to chemotherapy is utilization of immunogens derived from virulent strains of *A. viridans* (Stewart, 1984). Low levels of protection were induced with formalin-killed bacteria, and high levels were induced with a vancomycin/live bacteria combination in laboratory experiments (Stewart and Zwicker, 1974). Subsequently, Keith et al. (1992) demonstrated the efficacy of these types of vaccines in both experimental and field trials, including the effectiveness of immersion vaccination. A newer Download English Version:

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