Minireview

Diseases of spiny lobsters: A review

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A R T I C L E  I N F O

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Jasus
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A B S T R A C T

Spiny lobsters have few reported pathogens, parasites and symbionts. However, they do have a diverse fauna comprised of a pathogenic virus, several bacteria, protozoans, helminths and even symbiotic crustaceans. A few idiopathic syndromes have also been reported, but these appear correlated with lobsters held in poor conditions. Fungal and bacterial pathogens present significant threats for rearing spiny lobsters in aquaculture settings, but only one pathogen, Panulirus argus virus 1, is thought to have damaged a fishery for a spiny lobster. No doubt others will emerge as lobsters are brought into aquaculture settings and as fishing pressure intensifies with stocks become more susceptible to anthropogenic stressors.

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1. Introduction

Spiny lobsters in the genera *Jasus*, *Panulirus*, and *Palinurus* support worldwide fisheries with annual landings of over 70,000 m (Booth, 2006; Phillips and Melville-Smith, 2006). There are surprisingly few diseases reported from these lobsters given the size and value of their fisheries. However, recent attempts to culture lobsters have failed because of mortalities associated with potential agents, most notably bacterial infections (Kittaka, 1997; Bourne et al., 2004, 2006). That, plus the emergence of a pathogenic virus in the Caribbean spiny lobster (Shields and Behringer, 2004), highlights the potential damage that can be caused by unrecognized pathogens. I review the microbial diseases, parasites, and symbionts of the spiny lobsters, *Panulirus spp.*, *Palinurus spp.*, and *Jasus* spp. For earlier reviews of the diseases of spiny lobsters see Evans and Brock (1994) and Evans et al. (2000). For a broader perspective on the diseases of clawed and spiny lobsters, see Shields et al., (2006). Several overviews and syntheses of crustacean diseases are also available (Couch, 1983; Johnson, 1983; Overstreet, 1983; Brock and Lightner, 1990; Sindermann, 1990; Meyers, 1990; Shields and Overstreet, 2007), as well as those presented in this issue of the Journal of Invertebrate Pathology.

2. *Panulirus argus* virus 1 (PaV1)

PaV1 is the first naturally occurring pathogenic virus found infecting a lobster. As its name implies, it infects *P. argus*. The virus has a tropism for mesodermal cells, namely certain hemocytes (hyalinocytes and semi-granulocytes), soft connective tissue cells, some hematopoietic tissues, and fixed phagocytes (Shields and Behringer, 2004; Li et al., 2008). PaV1 is currently an unclassified virus, but it shares several morphological features with the Herpesviridae and the Iridoviridae (Shields and Behringer, 2004). For example, it is an unenveloped, icosahedral, DNA virus with a nucleocapsid approximately 182 nm in size that develops within the nucleus of host cells. The Herpesviridae are icosahedral, but they are enveloped and have a conserved size of approximately 125 nm. The Iridoviridae are icosahedral and unenveloped, but they are usually formed within the cytoplasm, whereas PaV1 is formed within the nucleus.

Lobsters heavily infected with PaV1 are often lethargic or slow. Their hemolymph does not clot and is milky in color, from cellular debris and exudates (Shields and Behringer, 2004). PaV1 initially infects the fixed phagocytes in the hepatopancreas (Li et al., 2008). These cells line the hepatic arterioles and, as implied by their name, they phagocytize non-self particles, including virons of PaV1. The infected fixed phagocytes lyse, and the virus spreads to spongy connective tissues as well as hemocytes. In late stages of the infection, virtually all of the spongy connective tissue cells become infected, with some apparently proliferating around the site of the infection, virtually all of the spongy connective tissue cells as well as hemocytes. In late stages of infection, virtually all of the spongy connective tissue cells become infected, with some apparently proliferating around the site of the infection, virtually all of the spongy connective tissue cells as well as hemocytes. In late stages of infection, virtually all of the spongy connective tissue cells become infected, with some apparently proliferating around the site of the infection, virtually all of the spongy connective tissue cells as well as hemocytes. In late stages of infection, virtually all of the spongy connective tissue cells become infected, with some apparently proliferating around the site of the infection, virtually all of the spongy connective tissue cells as well as hemocytes. Under the light microscope, the infected hemocytes have concurrently bacterial infections (Shields and Behringer, 2004, Li et al., 2008). PaV1 has been shown to induce a remarkable behavior in healthy, uninfected lobsters. Such lobsters have the ability to detect and avoid diseased lobsters (Behringer et al., 2006). This is the first example of avoidance of diseased individuals by healthy animals other than in humans. Moreover, the avoidance behavior occurs around the time infected lobsters become infectious! This has important consequences for the denning behavior of *P. argus*, because the animals are normally gregarious and the ability to detect and avoid disease may serve to limit the spread of the disease in the host population (Behringer et al., 2006). The nature of the avoidance is likely chemical detection as spiny lobsters have an exquisite sense of smell. PaV1 has been transmitted to uninfected lobsters via inoculation, ingestion, contact and through seawater (Butler et al., 2008). Contact and water-borne transmission are apparently the primary modes of transmission in nature. Lobsters inoculated with infected hemolymph can develop acute infections and die within 30–90 days, but some take much longer to show signs of infection. Lobsters infected more naturally, via contact or water-borne transmission, die within 200 days, with mortality highly correlated with smaller sized animals. Infection declines significantly with lobster size. In lobsters exposed via contact, 63% of animals <25 mm carapace length (CL) became infected, as opposed to 33% of those 30–40 mm CL, and 11% of the large juveniles (40–50 mm CL), respectively (Butler et al., 2008). Transmission coefficients used in mass-balance models were calculated for the different modes of infection. Contact transmission among the smallest juveniles had the highest transmission coefficient; that for water-borne transmission was about five times lower. Thus, the virus appears to be transmitted by direct contact among small juveniles and over short distances through the water column.

In short-term mark-recapture studies, overtly diseased lobsters had a lower survival rate than animals without disease (Behringer et al., 2008). In the laboratory, lightly infected lobsters showed no significant differences in movement compared to uninfected animals, but as the disease progressed, infected lobsters were much less active than healthy animals. The physiological state of intermolt lobsters from the field was assessed using hemolymph serum proteins as a general indicator of health. Overtly diseased lobsters had significantly lower hemolymph protein concentrations than presumably uninfected lobsters, by as much as 4–5 mg/dl protein levels (Behringer et al., 2008). Interestingly, starvation did not increase the susceptibility of animals to viral infection. Given that lightly infected animals were still highly active, they were thought to facilitate dispersal of the virus to new habitats.

The discovery of PaV1 coincided with a decline in the landings of *P. argus* from the Florida Keys fishery (Shields and Behringer, 2004). Whether the virus caused this decline or not remains to
be determined, but there is no doubt that the agent is widespread and pathogenic to lobsters in their early benthic juvenile stage. Lobsters infected with PaV1 have been found in several places around the Caribbean Sea, including the Florida Keys, US Virgin Islands, Mexico, and Belize (Butler et al., 2008; Huchin-Mian et al., 2008, 2009). PaV1 is widespread in the Florida Keys and Florida Bay, particularly in the shallow juvenile nurseries. In field studies, the mean prevalence was 7%, with local “hot spots” of up to 30% (Shields and Behringer, 2004). The size predilection for the virus stands out in the field studies because prevalence was 16% among the smallest juveniles (<20 mm CL), whereas it was 5% among the largest juveniles (>40 mm CL); in field surveys of adults, <1% of adults were visibly infected. Off of Puerto Morelos and the Chinchorro Bank, Mexico, prevalence was as high as 10.9% in juvenile lobsters, but there was no apparent change in host density with increased prevalence of disease. (Lozano-Álvarez et al., 2008). The differences in the nursery habitat between the Florida Keys and the area around Puerto Morelos are large enough to warrant further attention to density issues and how habitat restrictions potentially alter the host–pathogen association.

PaV1 appears to be specific to *P. argus* (Butler et al., 2008). In infection trials, three potential alternate hosts (the spotted lobster, *P. guttatus*, the stone crab, *Menippe mercenaria*, and the channel crab, *Mithrax spinosissimus*) were inoculated with hemolymph from an infected Caribbean spiny lobster. After 80 days, none of the three alternate hosts showed signs of disease using histology. This is particularly interesting because the spotted lobster, *P. guttatus*, is a close congener with *P. argus* (Ptacek et al., 2001), and its lack of observable disease suggests that PaV1 is highly specific to *P. argus*.

The recent emergence of PaV1 and its potential association with a decline in the spiny lobster fishery in the Florida Keys is cause for some concern. Several viruses have severely damaged the shrimp aquaculture and fishery industries (Flegel, 1997; Lightner and Redman, 1998); therefore, programs should be developed to monitor for PaV1 in populations of the spiny lobster from around the Caribbean Sea to establish baselines and to document its potential effect on artisanal and regional fisheries. Infections have been identified in two culture facilities in Florida (Shields, unpubl. data), and we know that it occurs in several countries around the Caribbean Sea. Given that lobsters and lobster tails are shipped internationally (see Huchin-Mian et al., 2009), plus the fact that nascent aquaculture ventures have been attempting to culture spiny lobsters from enzootic areas, lobsters should be screened for the virus as well as other microbial infections to control for infections as well as to prevent their potential spread to other areas (see Table 1).

### 3. White spot syndrome virus

Spiny lobsters are not naturally infected with the white spot syndrome virus (WSSV), but with epidemics of WSSV occurring in native shrimp stocks, it seems inevitable that it will spread into new host species. Indeed, at least 42 crustaceans, including three species of *Panulirus*, can serve as experimental reservoir hosts for WSSV (see Supamattaya et al., 1998; Rajendran et al., 1999; Mustthaq et al., 2006). In experimental infections, a DNA probe was used to detect WSSV in the tissues of *P. versicolour* and *P. penicillatus* (Chang et al., 1998). Lobsters survived for at least 70 days, with the virus present in their tissues, but they were not examined histologically for signs of disease. That is, the lobsters were infected but were not obviously diseased. PCR has also been used with a virus-specific primer set to detect WSSV in several species of lobster (*P. versicolour*, *P. penicillatus*, *P. ornatus* and *P. longipes*) fed infected...
Table 1
Parasites and symbionts naturally occurring in spiny lobsters, including the tissues in which they are found.

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Host</th>
<th>Tissue</th>
<th>Key reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaV1</td>
<td>P. argus</td>
<td>Connective tissues, hemocytes</td>
<td>Shields and Behringer (2004)</td>
</tr>
<tr>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Aerococcus viridans</td>
<td>H. americanus, H. gammarus, P. argus</td>
<td>Systemic</td>
<td>Sniezko and Taylor (1947), Rabin (1965), Stewart et al. (1966), Stewart et al. (1980), Bobes et al. (1988)</td>
</tr>
<tr>
<td>Several genera</td>
<td>P. argus, P. ornatus</td>
<td>External</td>
<td>Porter et al. (2001), Bourne et al. (2006), Payne et al. (2007)</td>
</tr>
<tr>
<td>V. alginitolyticus</td>
<td>Panulirus homarus</td>
<td>Systemic</td>
<td>Silva dos Fernandes Vieira et al. (1987)</td>
</tr>
<tr>
<td>Leucothrix mucor</td>
<td>Several genera</td>
<td>Carapace, eggs</td>
<td></td>
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<tr>
<td>Fungi*</td>
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<td></td>
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<tr>
<td>Arkoselis panulirata</td>
<td>P. japonicus</td>
<td>Larva</td>
<td>Kitanchareon and Hatai (1995)</td>
</tr>
<tr>
<td>Haliphthoros sp.</td>
<td>P. argus, J. verreaux</td>
<td>Pueruli, small juveniles</td>
<td>Fisher et al. (1975), Diggles (2001)</td>
</tr>
<tr>
<td>Fusarium solani</td>
<td>P. cygnum</td>
<td>Cuticle</td>
<td>McAlister and Baxter (1983)</td>
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<tr>
<td>Didymaria palinuri</td>
<td>P. elephas</td>
<td>Gills</td>
<td>Sordi (1958)</td>
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<tr>
<td>Ramalina branchiales</td>
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<tr>
<td>Protozoans</td>
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<tr>
<td>Panulirus spp.</td>
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<tr>
<td>Helminths</td>
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<tr>
<td>Turbellaria</td>
<td>Panulirus spp.</td>
<td>On mouthparts</td>
<td>Shields (unpubl. data)</td>
</tr>
<tr>
<td>Thulakotrema genitale</td>
<td>P. cygnum</td>
<td>Gonad</td>
<td>Deblay et al. (1991)</td>
</tr>
<tr>
<td>Cynotonocarpus soleriis</td>
<td>P. argus</td>
<td>Abdominal muscle</td>
<td>Gomez del Prado-Rosas et al. (2003)</td>
</tr>
<tr>
<td>Tetraphyllid cestode</td>
<td>Panulirus spp.</td>
<td>Foregut</td>
<td>Shields (unpubl. data)</td>
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<tr>
<td>Carcinonemertes</td>
<td>P. interruptus</td>
<td>Eggs</td>
<td>Shields and Kuris (1990)</td>
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<tr>
<td>wickhami</td>
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<tr>
<td>Carcinonemertes</td>
<td>P. cygnum</td>
<td>Eggs</td>
<td>Campbell et al., (1989)</td>
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<tr>
<td>australiensis</td>
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<tr>
<td>Carcinonemertes sp.</td>
<td>J. edwardsii</td>
<td>Eggs</td>
<td>Frusher and Shields (unpubl. data)</td>
</tr>
<tr>
<td>Crustaceans</td>
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<tr>
<td>Choniomyzon panuliri</td>
<td>Panulirus spp.</td>
<td>Eggs</td>
<td>Pillai (1962)</td>
</tr>
<tr>
<td>Paramphiascopis sp.</td>
<td>J. edwardsii, J. verreaux</td>
<td>Gills</td>
<td>Booth (pers. comm.)</td>
</tr>
<tr>
<td>Parapleustes commensalis</td>
<td>P. interruptus</td>
<td>Eggs</td>
<td>Shoemaker (1952)</td>
</tr>
<tr>
<td>Gitanopsis iseebi</td>
<td>P. japonicus</td>
<td>Branchial chamber</td>
<td>Yamato (1993)</td>
</tr>
<tr>
<td>Octolasmis californiana</td>
<td>P. interruptus</td>
<td>Gills, carapace</td>
<td>Newman (1960)</td>
</tr>
<tr>
<td>Octolasmis ssp.</td>
<td>P. polyphagus</td>
<td>Gills, carapace</td>
<td>Jeffries et al. (1962)</td>
</tr>
<tr>
<td>Trilasmis fissum</td>
<td>P. japonicus, P. penicillatus</td>
<td>Mouthparts</td>
<td>Bowers (1968)</td>
</tr>
<tr>
<td>Balanomorphs</td>
<td>P. argus</td>
<td>Carapace</td>
<td>Eldred (1962)</td>
</tr>
</tbody>
</table>

* The Phylum Fungi no longer includes the fungus-like Phycometes and other classical forms that are now placed in a separate Kingdom, the Chromista.

shrimp (Wang et al., 1998). The virus was detectable in all of the lobsters, but none developed disease. Lobsters (Panulirus homarus) inoculated with WSSV-infected shrimp tissues died, presumably from the infection (Rajendran et al., 1999; Musthaq et al., 2006), but the question is unresolved because animals fed infected tissues did not develop infections (Musthaq et al., 2006), and controls consisting of uninfected tissue homogenates were not used. However, in the latter study, all of the lobsters injected with the virus died over 160 days. It is surprising that no other viruses are known from lobsters. Shrimp have at least 20 and the blue crab, Callinectes sapidus, has at least eight pathogenic viruses (Lightner and Redman, 1998; Shields and Overstreet, 2007). Several shrimp viruses have spread as pandemics through the Americas and Asia with catastrophic results for shrimp aquaculture (Flegel, 1997; Lightner and Redman, 1998). Perhaps more viruses are known from shrimp because they are intensively cultured; hence, there are more opportunities for outbreaks under crowded conditions. Nonetheless, clawed and spiny lobsters support large fishing industries in which diseased animals would be observed or recognized. Perhaps the paucity of viruses in lobsters is simply a reflection of the fact they are only now being investigated for intensive aquaculture, and that most of the focus on lobsters has been on their fisheries and not on the more susceptible larval and juvenile stages.

4. Gaffkemia – Aerococcus viridans

Gaffkemia, or red-tail disease, is a serious disease of clawed lobsters, primarily Homarus americanus, but also H. gammarus. Outbreaks usually occur in holding facilities; it is not common in natural populations of lobsters (Stewart et al., 1966; Keith et al., 1992; Lavallée et al., 2001). Aerococcus viridans, a Gram-positive bacterium, is the causative agent of gaffkemia, and it is a common soil microbe with many variants. The variant that is pathogenic to lobsters is known as A. viridans var. homari. The pathogen can be spread via shipment and holding of diseased clawed lobsters; therefore, further risk assessment should be initiated in areas where there could be likely introductions of contaminated water or discarded lobsters. Aerococcus viridans reportedly occurs naturally in P. argus from Cuba. Bobes et al. (1988) isolated and cultured A. viridans from six
animals that presented signs of disease. The animals had reddish discolored exoskeletons, lethargy, pink hemolymph and coagulop athies. The bacterium was susceptible to 10 of 13 antibiotics in culture (Bobes et al., 1988). While Koch’s postulates were not attempted, this finding has further implications for the transportation of spiny lobsters. Aerococcus viridans var. homari has been isolated from other crustaceans from Massachusetts (Rabin and Hughes, 1968) and the Gulf of Mexico (Lizuzo et al., 1965). While other crustaceans can carry the bacterium, clawed lobsters are apparently the only hosts in which it is highly pathogenic (Cornick and Stewart, 1975).

The route of transmission of Aerococcus viridans is through damage to the host exoskeleton, not through intact cuticle or ingestion (Stewart et al., 1969). The bacterium is not part of the natural microflora of lobsters (Stewart, 1975). It has been isolated from lobster holding facilities; and the isolation of the agent usually indicates the presence of diseased lobsters (Goggins and Hurst, 1960; Kellog et al., 1974; Stewart, 1980). Unfortunately, it can remain viable in the benthos for long periods where it can act as a reservoir of infection (Stewart, 1984). Aerococcus viridans has also been documented in muds adjacent to a holding facility for clawed lobsters in Anaheim Bay, California, and was likely transported there through diseased clawed lobsters (Kellog et al., 1974).

Gaffkemia can be experimentally induced in P. interruptus, but the lethal dose is higher than that for H. americanus (Sacchiro et al., 1974; Steenbergen and Sacchiro, 1974). That is, spiny lobsters show a high degree of resistance to experimental infections (Sacchiro and Steenbergen, 1974). Because most studies of gaffkemia have been carried out in the clawed lobsters, there will be no further treatment of it here. For reviews of gaffkemia, see Stewart (1980) and Brock and Lightner (1990).

5. Shell disease

Shell disease, or shell disease syndrome, is a progressive chitinolyis and necrosis (bioerosion) of the exoskeleton of crustaceans (Rosen, 1970). Virtually all crustaceans are susceptible to shell disease, but it is most notable in large decapods. The syndrome first appears as small pits or erosions in the cuticle and progresses to large eroded lesions (Rosen, 1970; Getchell, 1989; Noga et al., 1994). The bacterial form, which is referred to as classical, or enzootic, shell disease, was first described from the American lobster (Hess, 1937). Recently, an epizootic form of the syndrome has emerged in American lobsters from Rhode Island (see Castro et al., 2006), for review. This new form of the disease has not been reported from palinurid lobsters. For comprehensive reviews of shell disease in crabs and lobsters see Shields et al. (2006) and Shields and Overstreet (2007).

Classical shell disease is caused by a number of chitinoclastic, Gram-negative bacteria. Vibrios (Vibrio vulnificus, V. paraahaemolyticus, V. alginolyticus) are the most common bacteria associated with shell disease, but other species have also been isolated from the lesions, including Shewanella spp. and Aeromonas hydrophila (Geddes et al., 2003; Porter et al., 2001; Reuter et al., 1999). Both V. alginolyticus and a V. harveyi-like bacterium were isolated from the hemolymph and lesions occurring on P. homarus reared in the laboratory (Abraham et al., 1996). Using the 16s and 23s rDNA intergene regions, Porter et al. (2001) examined the bacterial fauna of lobsters with and without classical shell disease. DNA fingerprinting found no specific etiological agent associated with the lesions, but bacteria in several genera were identified, including Vibrio, Pseudoalteromonas, Pseudomonas and Shewanella. The native bacterial flora was thought to be responsible for shell disease lesions in spiny lobsters (Porter et al., 2001), and many of these are indeed chitinoclastic.

Classical shell disease requires a portal of entry through the epicuticle, and mechanical abrasion is the usual mode of transmission. Shell disease starts as small pits or “burn” marks in the cuticle. The lesions occur on the sternum or legs of American lobsters (Rosen, 1967; Johnson, 1983), but they are more commonly found on the uropods of spiny lobsters (Porter et al., 2001; Geddes et al., 2003). As the pits coalesce, they develop into large, brown or black, irregularly shaped lesions (Rosen, 1967). The lesions can penetrate through the cuticle into underlying tissues of the lobster, but they rarely do so; instead they develop laterally with further erosion of the cuticle (Overstreet, 1978; Johnson, 1983). The exposed cuticle is weakened and brittle, and discolored from the deposition of melanin (Johnson, 1983; Smolowitz et al., 1992). Melanization and cellular infiltration occur as part of the host response to shell disease in American lobsters (Smolowitz et al., 1992, Smolowitz et al., 2005). Pseudomembrane formation can occur in particularly severe cases of shell disease, particularly in epizoic shell disease (Smolowitz et al., 2005). It is no doubt the same process in spiny lobsters. Individuals usually overcome the disease by molting (Rosen, 1967; Castro and Angell, 2000), but animals that molt frequently can presumably succumb to infection.

In spiny lobsters, P. argus, Panulirus cygnus and Jasus edwardsii, another form of shell disease occurs. It is known as “tail fan necrosis”, an aptly named syndrome because the uropod and telson appear melanized and partly or fully eroded away (Porter et al., 2001; Geddes et al., 2003). In clawed lobsters, shell disease lesions are more frequently seen on the ventral side of the claws, tail and carapace (Estrella, 1991). Shell disease is normally found at low (<1%) levels in healthy wild populations of crustaceans. Iversen and Beardsley (1976) examined 9000 P. guttatus, and found only two infected individuals. They cite several unpublished reports indicating a very low prevalence of shell disease in P. argus.

Classical shell disease is often associated with pollution (Gopalan and Young, 1975; Young and Pearce, 1975; Couch, 1983; Morado et al., 1988; Gemperline et al., 1992; Weinstein et al., 1992; Ziskowski et al., 1996), and spiny lobsters require relatively pristine water quality parameters; therefore, shell disease is not usually found at a high prevalence in palinurid lobsters. However, clawed lobsters held in crowded pounds with poor water quality can develop fatal shell disease (Taylor, 1948, in Sinderman, 1989). Therefore, in culture systems, efforts should be directed at maintaining good water quality and reducing crowding effects. Lobsters with moderate to severe forms of shell disease are not esthetically pleasing and are not used in the “live food” trade. Indeed, epizoic shell disease has had an economic impact on the clawed lobster fishery off Rhode Island, because the cuticle of affected animals is severely compromised making the animals unsuitable for live trade (Castro et al., 2006).

Antibiotics have been used to treat shrimp and lobsters with shell disease, but none are currently registered for use in crustacean aquaculture in the USA. Some such as furanace may be used in other countries. Juvenile lobsters have been successfully treated with Malachite green (Fisher et al., 1976a,b, 1978). Shrimp have been treated with Malachite green, formalin, or antibiotic baths (penicillin–streptomycin, furanace, erythromycin, oxolinic acid) (Tareen, 1982; Brock, 1983; El-Gamal et al., 1986). Contaminated aquaria should be disinfected with bleach solutions, and it is a good practice to do this prior to any laboratory culture. Animals with severe lesions should be destroyed to prevent further spread in impoundments.

6. Vibriosis

Vibriosis is an infection by any species of Vibrio. Vibrios are ubiquitous in the marine environment, and several species cause
serious diseases to invertebrates, fishes and even humans. Lobsters, in turn, can be exposed to several pathogenic species of Vibrio. Vibrio alginolyticus, V. harveyi, V. parahaemolyticus and V. anguillarum are facultative, opportunistic pathogens that have been implicated in disease in clawed and spiny lobsters (Bowser et al., 1981; Brinkley et al., 1976; Jawahar et al., 1996). Vibrios can also contaminate seafood products including lobsters (Wong et al., 1999); and there are many cases of human food poisoning from improper handling and preparation of marine crustaceans. Lobsters should be properly cooked prior to eating or freezing, and all surfaces used in the preparation of lobsters should be cleaned and disinfected afterwards.

Vibrio infections can cause serious disease in spiny lobsters, but few outbreaks have been reported. This is probably because spiny lobsters are usually kept in short-term culture, but not long-term culture where there would be more opportunity for disease under crowded conditions. However, outbreaks are known to occur in larval cultures. Indeed, vibriosis may be the single most important impediment to successful larval culture (Bourne et al., 2004). Cultures of phyllosoma larvae of Sagmarius jesus verreauxii experienced significant mortalities due to a luminescent strain of V. harveyi in New Zealand (Diggles et al., 2000). Infected larvae were opaque and had small red spots throughout the body. The hepatopancreas was atrophied and the hepatopancreatic tubules had numerous bacterial plaques. Mortalities occurred over 4 weeks. The bacterium is an opportunistic pathogen and attacked injured larvae more than uninjured ones. Two drugs, sulfadimidine and trimethoprim, were found to enhance survival the lobster larvae (Diggles et al., 2000).

The dynamics of bacterial flora have been studied in cultures of larval spiny lobsters. Larval cultures of Panulirus ornatus had a high microbial diversity, with representatives from many bacterial taxa (Bourne et al., 2006; Payne et al., 2007). Denaturing gradient gel electrophoresis (DGGE) was used to study diversity in larval cultures (Bourne et al., 2004; Payne et al., 2007) as well as biofilms in culture aquaria (Bourne et al., 2006). There were few changes in the bacterial flora in relation to mortality. In fact, bacterial densities decreased significantly over time and had no relationship with mortality (Bourne et al., 2004). However, larval mortality was high, reaching 100% in 13–24 days. Vibrios were visualized and enumerated using a fluorescence in situ hybridization (FISH) technique (Webster et al., 2006). Vibrios increased both on and within phyllosoma larvae starting about seven to ten days after hatching, and reached high levels 18 days after hatching. Two isolates of V. harveyi were used in a challenge study and one strain caused more mortality than the controls (Bourne et al., 2006). Several species of Vibrio and other bacteria have been identified from cultures of the phyllosoma of J. edwardsii (Handlinger et al., 1999). An isolate of V. harveyi was obtained from the diseased gut of an infected larva. More research is needed in this area because it is not clear how larval nutrition, host defenses, temperature, culture stress and disease agents interact in culture systems.

Vibrio alginolyticus, V. parahaemolyticus and a V. anguillarum-like species were isolated from P. argus and P. laevicauda from commercial landings in Brazil. Prevalence varied from 2.8% in V. anguillarum to as high as 45.7% in P. laevicauda (Silva dos Fernandes Vieira et al., 1987). Unfortunately, the disease status of the lobsters was not reported. Vibrio alginolyticus and a V. harveyi-like bacterium were isolated from shell disease lesions and hemolymph of juvenile and adult P. homarus from India (Hameed, 1994). In inoculation trials, the Vibrios became systemic and induced shell disease-like lesions in larvae of Penaeus indicus and P. homarus (Hameed, 1994). Several tissues showed histopathological alterations. In a separate study by Abraham et al. (1996), mortality reached 100% in P. homarus injected with 10^8 bacterial cells, but there was no mortality in lobsters inoculated with 10^7 bacterial cells. I have obtained similar results in inoculation trials with Vibrios in J. edwardsii (Shields, unpubl. data).

Vibrios are frequently isolated from the hemolymph of crustaceans; and the fact is that crustacean hemolymph is not necessarily sterile. In a debate in the 1970s, Bang (1970), Colwell et al. (1975) and Tubishat et al. (1975) found a high prevalence of bacteria, particularly Vibrios in the hemolymph of apparently healthy blue crabs. These were thought to have been acquired through abrasions or other wounds in the cuticle that occurred because of the rough handling of crabs en route to markets (Johnson, 1976). Indeed, crabs are handled roughly during capture and transport to processing plants (Shields, pers. obs.). However, in a definitive study, species of Vibrio were shown to occur in the hemolymph and various other tissues of freshly caught, unstrressed crabs (Davis and Sizemore, 1982). Nonetheless, we rarely isolate bacteria from the hemolymph of spiny lobsters. Thus, while it is possible that spiny lobsters can carry background levels of septicaemia, there are few reports of such in adults, nor have I observed many bacterial isolates in routine health examinations of P. argus and J. edwardsii (Shields, unpubl. data).

7. Fouling bacteria

Filamentous bacteria are ubiquitous on the eggs of marine crustaceans, and spiny lobsters are no exception (Johnson et al., 1971; Bland and Brock, 1973). Leucothrix mucor is a common filamentous species that has been observed on or isolated from cultured larvae of the American lobster (Nilson et al., 1975; Dale and Blom, 1988). It and other fouling agents are common fouling organisms in larval culture of spiny lobsters (e.g., Kittaka, 1997; Bourne et al., 2004, 2006; Payne et al., 2007). Leucothrix mucor is very common in seawater systems. It is a saprophyte of dead algae. I have observed it on crustacean eggs and limbs, including amphipods, crabs and lobsters. Filamentous bacteria can be difficult to control because they are so common and because they grow readily in the organically enriched environments encountered in culture systems. Heavy infections are thought to contribute to larval mortality. Control requires careful site selection and system design, good water quality parameters and strict attention to nutrition. Outbreaks of L. mucor have been controlled with antibiotics (Steenbergen and Schapiro, 1976; Fisher et al., 1976a,b; Sadusky and Bullis, 1994).

8. Water molds and Fungi

The “lower fungi” have been reclassified as protists in the Kingdom Chromista (Cavalier-Smith, 1993). The Oomycota include the Oomycetes and Phycomycetes after Margulis et al. (1990). They are now referred to as water molds. The true Fungi include Deuteromycetes, Ascomycetes and Basidomycetes and a few other classes of “higher” fungi.

9. Oomycetes

A few oomycetes are known from spiny adult lobsters. These typically infect the eggs or larvae, but a few infect adult lobsters (e.g., Alderman, 1973). Atkinsonia panulirata is an oomycete described from phyllosoma of P. japonicus (Kitancharoen and Hatai, 1995). Haliphthoros mildfordensis has been implicated in mortalities in aquaculture facilities for postlarval spiny and clawed lobsters (Fisher et al., 1975, 1978; Nilson et al., 1975, 1980). An outbreak of Haliphthoros sp. caused mortalities in a grow-out facility in New Zealand (Diggles, 2001). The disease infected the pueruli and juveniles of J. edwardsii, which exhibited morbidity, including lethargy and loss of appetite. Only lobsters less than 30 mm CL became infected. Perhaps this was due to larger animals having a thicker or more resil-
ient cuticle. Antibiotic treatment using Malachite green and formalin was successful in preventing the spread of disease. The outbreak was associated with poor water quality (Diggles, 2001).

Melanization of infected tissues is a common defense against Haliphthoros and other mold and fungal infections in crustaceans. Therefore, black or brown spots are commonly seen in necropsies involving mold infections. The quinones and melanin, which are activated by the phenoloxidase pathway, cause these black spots.

Prophenoloxidase occurs naturally in the cuticle and its activated enzyme, phenoloxidase, is activated through a well known cellular process involving hemocytes. Melanin is highly toxic to many microbial pathogens (for review, see Jiravanichpaisal et al., 2006).

Maintaining animals in conditions of excellent water quality is the preferred method to control water mold outbreaks in cultures of lobsters. Ozonated or dechlorinated seawater should be used in cultures. Water changes to maintain low organic loads and low ammonium levels is also critical. Furanace and other antimold drugs are not approved for use in aquaculture by the Food and Drug Administration in the USA and in several other countries.

10. Fusarium solani

A deuteromycete fungus, F. solani, causes disease in cultured crustaceans. This species and other unidentified species have caused disease in shrimp (Lightner and Fontaine, 1975), spiny lobsters (McAleer and Baxter, 1983) and clawed lobsters (Alderman, 1981). Black lesions resembling shell disease occur on the cuticle of infected hosts. An outbreak of F. solani was reported from P. cygnus off Western Australia (McAleer and Baxter, 1983). The lesions occurred on the abdomen, uropods, telson and pereopods. The fungus was isolated from lobsters and from seawater samples in areas with diseased lobsters. Environmental factors were thought to have facilitated the outbreak, but they were not identified. Given the location of the lesions around the uropods and telson, water quality was likely an issue.

The pathology of Fusarium infections has been studied in shrimp and clawed lobsters. The fungus penetrates through the cuticle, which causes the formation of lesions (Lightner and Fontaine, 1975; Fisher et al., 1978). The lesions often become melanized through the phenoloxidase pathway. Lesions containing cellular aggregates, necrotic tissues, and fungal hyphae can be observed in histology. The hyphae of Fusarium produce diagnostic micro- and macroconidia (Lightner and Fontaine, 1975).

Two other deuteromycetes, Didymaria palmieri and F. (Ramularia) brachialis were isolated from moribund P. elephas and H. gammarus from the Mediterranean Sea (Sordi, 1958), but no attempts were made to infect healthy lobsters. It is not clear if the fungi were primary pathogens or opportunists. Little data were reported from these infected animals.

11. Peritrich ciliates

Peritrich and suctorian ciliates are often found as commensals on the external surfaces and embryos of crustaceans. Heavy infestations often indicate poor water quality or the presence of a disease. They are not usually pathogenic but heavy infections can impose a respiratory debt to the host (Schuwerack et al., 2001). They can be quite abundant in larval cultures of spiny lobsters (e.g., Handliger et al., 1999; Bourne et al., 2004). Their presence on crustacean embryos generally indicates that a female host is not feeding well or is not preening or otherwise taking care of herself. Several studies have found peritrichs in the genera Vorticella and Zoanthinium, suctorian ciliates Acinetla and Ephelota, several cyanobacteria and diatoms in the clutches of clawed lobsters (Dannevig, 1928, 1937; Nilson et al., 1975; Harper and Talbot, 1984; Dale and Blom, 1988).

Vorticella sp., Navicula sp., L. mucor and other organisms fouled the larvae of J. edwardsii (Kittaka, 1997). Fouled phyllosoma larvae died, but their cause of death was not documented. Interestingly, other fouling agents, including Saprolegnia sp., hindered fouling by the ciliates. Chilodonella-like protists and Epistyris spp. have also been reported as fouling agents (Handliger et al., 1999; Bourne et al., 2004). Wescodey and Malachite green have been used to control Vorticella infestations on clawed lobsters (Boghen, 1982). Baths of formalin, oxytracycline, erythromycin and streptomycin were used to control fouling organisms on P. ornatus (Bourne et al., 2004). Weak formalin solutions have been used to effect pathogen control in many culture situations, including ciliate infections in fishes.

12. Microsporidia

The Microsporidia is a phylum of small, intracellular and intranuclear parasites. The phylum is named after the small, highly refractory spore produced as the transmissive stage. All members of the phylum have an extrusible polar tube that is used to inject the sporoplasm into a new host cell. The Microsporidia were once considered a phylum within the Protozoa, but they are now considered to be closely related to the true Fungi (for review, see Lee et al., 2008). Some species, such as Nosema apis, can survive in their cadavers for very long periods.

Microsporidia are very rare in lobsters. A single infection was reported from P. argus from Florida (Bach and Beardsley, 1976), but no details were given. More recently, Kiryu et al., 2009 found two lobsters with Amesnon-like microsporidiosis from Florida. The microsporidians are aptly named as those in the lobster were 1.4 × 1.0 μm in size (Kiryu et al., 2009). In Australia, at least one species of Amesnon sp. is known to infect spiny lobsters, P. cygnus and P. ornatus (Dennis and Munday, 1994; Owens and Glazebrook, 1988). In both systems, infections occur in the muscles. They cause a distinctive liquefactive and coagulative necrosis of the muscles, with heavy infections causing the muscle fibers to turn creamy white in color. A similar condition in crabs and shrimp is known as “cotton” disease. The flesh of these animals when cooked has a consistency like that of cotton; and it is very unappetizing (Shields, pers. obs.). Very little is known about infections in lobsters. In blue crabs, Amesnon michaelsi infections are transmitted via cannibalism (Overstreet, 1978), but their rarity in lobsters suggests that other pathways are involved or that lobsters may be accidental hosts. Microsporidia are known to damage prawn aquaculture (Flegel et al., 1992; Hudson et al., 2001) so there is a precedent for their potential infection of lobster populations. However, in field surveys from the Florida Keys that involved several hundred animals, there was no obvious histological infection by any microsporidian (Shields, unpubl. data). Given the live market for lobsters and their shipment to international markets, the potential transport of microspores in infected hosts should be investigated as a risk factor, albeit many microsporidians show high host specificity, which could lower the risk to other hosts.

13. Helminths

Few platyhelminthes reportedly use lobsters as hosts. There are no published reports of turbellarians on spiny lobsters, but I have observed them on the mouthparts and gills of several species of Panulirus, from the Great Barrier Reef (Shields, unpub. data). Turbellarians have been reported from the egg clutches of crabs (Kuris et al., 1991), and it would not be surprising to find them in the clutches of other crustaceans, including lobsters. There are a few
reports of digenetic trematodes from palinurid lobsters, but there are no published reports of cestodes from them other than my anecdotal observations below.

14. Digenean trematode infections

Lobsters serve as the second intermediate host to digenetic trematodes. They bear the metacercaria, or the encysted stage. The metacercarial cysts are usually quite small (<1 mm) and can be detected using microscopy. As the name implies, the microphal- lid trematodes are known for their small phallus. Several species use crustaceans as intermediate hosts. While the metacercaria of most trematodes are difficult to identify to the species level, the microphalld trematodes develop sexual characteristics as metacercariae making it possible to identify and describe them. *Thulakiotrema

* genitale is a microphallid found encysted as metacercairae in the gonads of *Panulirus cycnus* (Deblock et al., 1991). The prevalence in Western Australia was quite high, ranging from 47% to 87%. The vertebrate definitive host is unknown, but it is likely to be a fish. *Cymatocarpus solearis* (= *C. undulatus*) is a brachycocellid tremato- mode. Metacercariae infect the abdominal muscles of *P. argus* (Gómez del Prado-Rosas et al., 2003). The cysts of *C. solearis* are large enough to see by eye (1.5 mm in size). The parasite was rel- atively abundant in lobsters, with a prevalence of 35.8% and a mean intensity of 26 cysts per host. The loggerhead turtle *Caretta caretta* serves as the definitive host, where it lives in the intestines (Linton, 1910; Caballero, 1959). Light infections of metacercariae probably do not result in significant pathology, albeit in other systems, particularly those involving the nervous systems of their hosts, they can cause alterations to host behaviors.

15. Cestoda

Lobsters may serve as an intermediate host for cestodes, bearing the metacestode, which is usually encysted in the connective tissues. Metacestodes vary in size, some are microscopic whereas others can be quite large. I have observed metacestodes of a tetra- phyllidean cestode in the foreguts of *Panulirus* spp. and *Scyllarides* sp. from the Great Barrier Reef (Shields, unpubl. data). The tetraphylinean cestodes use eelworms as definitive hosts. Tetraphyllidean metacestodes cannot be identified to species unless fed to and recovered from an eelworm host. This can be diffi- cult to do in the laboratory, but it is possible (see Sakanari and Moser, 1989). There are no published records of metacestodes from lobsters. This is intriguing because they no doubt serve as interme- diate hosts for other parasites that use eelworms, other fishes, and turtles as definitive hosts.

16. Nemertea – *Carcinonemertes* spp. and *Pseudocarcinonemertes*

Two species of *Carcinonemertes* are known from spiny lobsters. They are egg predators and, at least on crabs, they can occur in epi- demics where they damage reproduction on a population level (Wickham, 1979, 1986; Hobbs and Botsford, 1989; Shields and Kuris, 1988; Kuris et al., 1991). *Carcinonemerte* can be difficult to detect on lobsters because they only infest the egg masses and only during reproduction; that is, one must assess their presence when lobsters carry eggs. *Panulirus interruptus* from Southern California are infested by *Carcinonemertes wickhami* (Shields and Kuris, 1990). It is a rela- tively large carcinonemertid worm, 30–50 mm in length. *Panulirus cygnus* from Western Australia are infested by *C. australiensis* (Campbell et al., 1989). It is a relatively small worm for the genus at 7 mm in length. *Jasus edwardsii* from Tasmania is also infested by a carcinonemertid worm, but it has yet to be identified (Frusher and Shields, unpubl. data). It is a larger, more robust species than *C. australiensis*.

Both *C. wickhami* and *C. australiensis* are monostyliferous hoplo- nemertes; that is, they each bear a single stylet on the proboscis armature. They are dioecious, and use internal fertilization for reproduction. Other members of the Carcinonemertidae can be hermaphroditic (Roe, 1986; Shields, 2001). The eggs are extruded into a mucilaginous egg strand, which adheres to a seta on the pleo- opod within the clutch. The egg strands can also be used to indicate the presence of the worms. The biology and ecology of these nem- eretes is unknown. Comparative studies on the life history of these species would be very interesting.

Clawed lobsters bear a nemaertean that is unlike the carcinonemertid. It is known as *Pseudocarcinonemertes homari* and it probably belongs in the family Tetrastemminidae. Charmantier et al., (1991) used freshwater baths to rid clawed lobsters of infestations. Freshwater killed worms after only 4 min. Freshwater baths and Malachite green have been used to control infestations of *C. errans* on Cancer magister (Wickham, 1988). The fact that carcinonemert- ids occur in epidemics on crab species indicates that these worms have the potential to occur in outbreaks on lobsters. Care should be taken in transporting ovigerous lobsters to new locations to avoid the introduction of their worms and other parasites. Broodstock should also be examined prior to establishing colonies of lobsters.

17. Miscellaneous metazoan symbionts

As yet, spiny lobsters are not known to be infested with any member of the Cyclophora. *Symbion pandora* and *S. americanus* are the only described members of the phylum Cyclophora (Obst et al., 2006). The phylum is highly specialized with both *S. pandora* and *S. americanus* living on the mouthparts of the Norway lobster and American lobster, respectively (Funch and Kristensen, 1995; Obst et al., 2006). However, I have observed rotifers and rotifer-like animals on the mouthparts and gills of spiny lobsters from the Great Barrier Reef, and given what is now known about the Cyclophora, these organisms may be of further scientific interest.

18. Nicothoidae – parasitic copepods

Nicothoid copepods are a family of highly specialized symbionts that live on other crustaceans. Nicothoidae were once in the family Choniostomatidae (see Boxshall and Lincoln, 1983). The copepods live in the egg clutches or marsupia of their hosts. They are egg predators and in some cases they are very similar to host eggs; in fact, they are considered egg mimics (Hansen, 1897; Bowman and Kornicker, 1967). *Choniomyzon panuliri* is a nicothoid copepod that infests the clutches of *Panulirus* spp. (Pillai, 1962). Several spe- cies of *Panulirus* from the Great Barrier Reef, Australia, have a sim- ilar species in their egg clutches (Shields, unpubl. data). The larval stages of species of *Choniomyzon* occur in the gill chambers of non-ovigerous crab hosts, but this has not been examined for *Choniomyzon*. The feeding habits of at least one species of Nicothoi- dae can cause significant damage to the fecundity of individual hosts (Shields and Wood, 1993).

Two other copepods have been reported from spiny lobsters. The harpacticoid copepod *Parampblascopis* sp. is apparently symbiotic in the gills of male and female *J. edwardsi* and *S. verreauxii* (Booth, personal communication). Members of this genus apparently occur in symbiotic relationships with other invertebrates (Soyer, 1973; Hicks, 1986). Another harpacticoid copepod occurs in the gills of *Panulirus* spp. from the Great Barrier Reef, Australia. It is similar to *Sunaris* (Shields, personal observation).
19. Amphipods

Spiny lobsters often have amphipods living in close association with them, either on their egg clutches or within their branchial chambers. The relationship is likely commensal, but those found on the egg clutches are probably predatory on the eggs of their hosts. *Parapeluses commensalis* occurs on the pleopods and egg clutches of *P. interruptus* and *Cancer anthonyi* from Southern California (*Shoemaker, 1952; Shields pers. observation*) and *Paralithodes californiensis* (*Wicksten, 1982*). It ate crab eggs in experimental systems (*Shields, unpubl. data*). *Ischyrocerus* sp. infests the egg clutches of *Paralithodes camtschaticus* (*Kuris et al., 1991*). It was correlated with significant egg mortality on that host; hence, the potential of amphipods as egg predators on lobsters. *Gitanopsis isebei* has been reported from the branchial chamber of *Panulirus japonicus* (*Yamato, 1993*). Other members in the family of amphipod hosts are commensals; therefore, it is likely to be a commensal. *Isaea elmhirsti* lives as a commensal on the mouthparts of *H. gammarus* (*McGrath, 1981, in Costello et al. (1990)*). Amphipods are highly motile and they can swim away from a host when it is captured; therefore, some of these relationships may be hard to study. Symbiotic amphipods warrant attention, particularly in studies of fecundity or reproductive behaviors or when establishing broodstock for nascent culture studies.

20. Fouling organisms

A plethora of organisms can foul the exoskeleton of lobsters and other crustaceans. Some are opportunists that simply require a hard surface. Others are obligate symbionts. Some are also useful as indicators of the health of their host. The gooseneck barnacles, *Octlasmis* spp., use a number of decapod crustaceans as hosts, including panulirid and scyllarid lobsters (*Jeffries et al., 1982*). These barnacles live on the exoskeleton and within the gill chambers of their hosts. They are highly adapted to the life history patterns of their hosts. One species, *O. bullata*, had a prevalence of 48% on *Panulirus polyphagus* off Singapore (*Jeffries et al., 1982*). No other species were found on the lobster, but other species were recorded from a number of different decapods from the area. *Octolasmis angulata* has been recorded from *P. versicolor* from Western Australia (*Jones, 2004*). *Octolasmis californiana* was first reported from the California spiny lobster, *P. interruptus* (*Newman, 1960*), but it is known to occur on several decapods (*Newman and Abbott, 1980*). Another lepadomorph barnacle, *Trilasmis fission hawaiensis* is specific to *P. japonicus* and *P. penicillatus* where it lives on the setae of the mouthparts (*Bowers, 1968*). This relationship is quite intriguing and indicative of the specialization that can occur in barnacles.

There are few reports of balanomorph, or acorn barnacles, on spiny lobsters. However, *Chelonobia patula* and a few other species sometimes infest them. *Chelonobia patula* is a host generalist, found on turtles, drift wood, decapods and other hard surfaces. It will settle on virtually any hard surface. Species of Paralepas occur on spiny lobsters from Australia (*Jones, 2003*). Several species of balanomorph barnacles have been found on spiny lobsters *P. argus* (*Eldred, 1962*). Heavy infestations of acorn barnacles may indicate that the host lobster is not in good health and may be suffering an underlying infection. Such hosts should receive priority for health examinations for rare infections.

Bryozoans, hydroids, serpulorbid molluscs, bivalve molluscs and many other animals can be found as fouling organisms on the shells of spiny lobsters, but there are few records of their occurrence. In horseshoe crabs and true crabs, the fouling community has been used to address ecological questions on succession (*Key et al., 1996, 1997, 1999*). I have seen fewer fouling organisms on lobsters than on crabs, perhaps because many crab species have terminal molts whereas lobsters often do not.

21. Disease syndromes

21.1. Turgid lobster syndrome

Turgid lobster syndrome is an unusual condition that occurs in *J. edwardsii* from New Zealand (*Diggles, 1999*) and *Panulirus ornatus* from Australia (*Evans, pers. communication*). The etiology of the syndrome is unknown, but the condition is easy to identify because the thin arthrodial membranes between the tergites swell from fluid pressure, causing the animals to become stiff and lifeless. Affected animals have difficulty moving so they stop feeding and become lethargic. It occurs in short-term aquaculture facilities and is probably the result of poor water quality, but that remains to be determined. Up to 50% of the affected animals can die (*Diggles, 1999*). Animals can be bled with a syringe to relieve fluid pressure, and those treated this way often survive.

A similar condition was described in lobsters from Japan (*Wada et al., 1994*). Lobsters were lethargic and had visible swelling of the arthrodial membranes. Histology revealed an inflammation of the myocardium as well as infiltration of hemocytes into affected cardiac muscles. Interestingly, the changes to the heart were visible as white nodules in the heart. The etiology of the syndrome was not determined, but it was not a microbical agent. Both wild and laboratory-held animals showed signs of the syndrome.

21.2. Pink lobster syndrome

An unusual syndrome occurs in *P. cygnus* from Western Australia. The condition is known as pink lobster syndrome and the flesh of affected animals becomes pink or orange (*see Shields et al., 2006*). Affected lobsters usually die from the syndrome. They show signs of lethargy and have a bitter flavor, making them unpalatable. Very little information is available on this syndrome, but it appears to occur in holding facilities as well as in nature. This condition should be further documented to determine if it is caused by an infectious or non-infectious agent.

21.3. Mass mortalities

Several mass mortalities have been documented in spiny lobster populations. An interesting mortality event was recorded from a tagging study that was undertaken to estimate the size of the population of *P. ornatus* at Yule Island, Papua New Guinea (*Dennis et al., 1992*). The mortality of adult lobsters was high, ~95%, during the fishing period; however, only a third of the mortality was attributed to fishing pressure, the rest was thought to be due to physiological stress from reproduction and migration, but disease agents were not examined. These events are different from those that alter habitat, thereby forcing migrations such as observed with hypoxia events. Lobster mortalities are relatively common along the coast of South Africa (*see Cockcroft, 2001*). Hypoxia caused by harmful algal blooms develops in deep waters and forces lobsters, *Jasus lalandii*, to move into extremely shallow littoral areas, which are subject to rapid tidal changes. Lobsters cannot escape exposure to air, and when caught in the intertidal zone, they die in mass strandings. Ecological disturbances to the Florida Keys have altered the habitats of the juvenile lobsters causing them to move into less impacted areas (*Butler et al., 1995*). The potential for increased exposure to pathogens has not been studied in these.
systems, but the emergence of PaV1 shortly after these events is very intriguing.

22. Conclusions

Spiny lobsters have few reported parasites, diseases and symbi- 

ons. Several species of spiny lobster have supported fisheries for 

many years, so one would expect their diseases to be well docu-

mented in their fished populations. However, this is not the case. This is primarily because fisheries focus on animals of legal size, which are typically adults. In my experience, diseases are more common and more serious in postlarval and early juvenile stages, a segment of the population that is not sampled by the fishery. Further, it is likely that new emerging diseases will become significant issues in nascent aquaculture industries for spiny lobsters. In fact, several microbial pathogens are known to be problematical to cul-

tures of larval and juvenile clawed lobsters (see Fisher et al., 1976a,b, Nilsson et al., 1975, 1976; Brock and Lightner, 1990). These include bacterial (Vibrio spp., L. mucor), fungal (Lagenidium spp.) and protozoal (peritrich ciliates) agents, all of which can be diffi-

cult to control in closed systems. No doubt more symbiotic and disease agents will emerge as fisheries become more intensively managed and aquaculture production force animals into high den-

sity, stressful conditions.

The presence of emerging pathogens and disease outbreaks are often signs that a population is under significant stress. Identifying the stressors that ultimately lead to or contribute to outbreaks can be very difficult. Such factors as degrading water quality, seasonal stressors, but in the case of the 1999 mortality, experiments indi-


cated that lobsters cannot tolerate combinations of severe hypoxic 

conditions, poor water quality and temperature stress (Dove et al., 

2004, 2005; Draxler et al., 2005a,b; Robohm et al., 2005). Thus, it is 

important to understand both the proximate and ultimate causes 

give rise to emerging pathogens to determine their potential threat to lobster fisheries.

At present, the virus PaV1 in P. argus represents the most sig-

nificant pathogen to any spiny lobster. It infects the smallest 

juveniles and, therefore, would normally go unnoticed in popula-

tions. However, fisher men in the Caribbean spiny lobster fishery use juveniles as social attractants (bat) in fished traps and 

lethargic diseased lobsters would be discarded. Thus, fishermen 

can spread diseases among them, or at least the virus, into new fish-

ing grounds (Shields and Behringer, 2004). While contact trans-

mission is the most efficient mode for the spread of PaV1, 

water-borne transmission can also occur, albeit at a much lower efficiency (Butler et al., 2008). However, healthy lobsters can de-

ect and avoid diseased animals, which makes contact transmis-

sion less likely to occur in nature (Behringer et al., 2006). The 

ramifications of transmission to the spread of PaV1 is unclear, 

but it is clear that the virus can threaten the valuable Caribbean 

fisheries (Shields and Behringer, 2004). Recent emerging dis-

eases, such as PaV1 in P. argus and Hematodinium in the Norway 

lobster (e.g. Field et al., 1992), highlight the fact that we know 

very little about the pathogens in our lobster stocks.

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