Special topic review: Viruses, biosecurity and specific pathogen-free stocks in shrimp aquaculture

J.M. Lotz

The greatest threat to the future of world shrimp aquaculture is disease, in particular the virulent untreatable viruses, infectious hypodermal and haematopoietic necrosis virus (IHHNV), taura syndrome virus (TSV), yellow head virus (YHV), and white spot syndrome virus (WSSV). To overcome these hazards, the industry of the future must be based on: (i) specific pathogen-free and genetically improved shrimp stocks; (ii) biosecure systems including enclosed, reduced water-exchange/increased water-reuse culture systems; (iii) biosecure management practices; and (iv) co-operative industry-wide disease control strategies. Specific pathogen-free shrimp are those that are known to be free of specified pathogens and such stocks will ensure that seed shrimp are not the conduit for introduction of pathogens and that if pathogens are encountered the stocks will not be severely affected. Commercially acceptable biosecure culture systems that are under cover and use recirculated sea water will need to be developed for shrimp production. Adherence to operating protocols that incorporate strict biosecurity practices, including restricted access and disinfection strategies, will need to become standard. Co-operative efforts will include: early warning surveillance; co-ordination of harvest and water exchange schedules of contaminated ponds; processor co-operation to ensure that processing wastes are not threats; quick response to outbreaks.

Key words: Aquaculture, biosecurity, pathogen, shrimp, specific-pathogen-free, viruses.

Introduction

Although aquaculture of marine shrimp dates back to ancient times, the practice was primarily incidental to brackish water fish culture until the 1970s. Stimulated by advances in shrimp reproduction and hatchery technologies, rapid development of shrimp farming culminated in the explosive growth observed during the 1980s. In 1980, culture contributed 44 tonnes or about 3% of the world’s supply of shrimp, but by the end of the decade shrimp farm production had eroded to over 500,000 tonnes and accounted for almost 30% of the world’s supply (Rosenberry 1989). In the 1990s production of cultured shrimp increased less dramatically and over the past several years production has plateaued at around 700,000 tonnes per annum (Rosenberry 1995; Figure 1). The stagnation of growth has continued despite an ever increasing number of hectares used for production.

The reason for the languishing growth in production is related to the increasing number of mass mortalities in pond culture throughout the world. In 1985 Taiwan experienced a drop in production of nearly 70%. During the period 1987–1990 the highly successful pond culture of Penaeus stylirostris in northwestern Mexico began a dramatic decline (Lightner et al. 1992). In 1992, Ecuador began to experience a rash of mass mortalities in grow-out ponds resulting in a drop in production. China ceased being the largest exporter of cultured shrimp in 1993 and by 1995 had become a net importer. Other major shrimp farming countries such as Thailand, the Philippines, Indonesia, and India have recently been affected by dramatically lowered survival rates. These episodes of shrimp mortality may be attributable to one of four viral disease agents: infectious hypodermal and hematopoietic necrosis virus (IHHNV) (Lightner et al. 1983a, 1983b; Bonami et al. 1990), Taura syndrome virus (TSV) (Brock et al. 1995; Hasson et al. 1995; Lightner et al. 1995), yellow head virus (YHV) (Boonyaratpalin et al. 1993; Flegel et al. 1995; Wongteerasupaya et al.; 1995a), or white spot syndrome virus (WSSV) (Takahashi et al. 1994; Wongteerasupaya et al. 1995b; Lightner 1996).

The Category 1 Shrimp Viruses

Lotz et al. (1995) categorized shrimp pathogens into one of three types based on their pathogenicity and danger to the shrimp-farming industry: category 3 (C-3) pathogens are those that have minimal impact; category 2 (C-2)
pathogens pose a somewhat greater threat and may affect productivity by reducing growth or lowering survival; category 1 (C-1) pathogens are the most dangerous, they cause mass mortalities and they may pose a threat to the survival of the industry in a geographical region. Not only are the C-1 pathogens highly pathogenic but they are also untreatable (Table 1); these are of the greatest concern to the shrimp-farming industry. Four shrimp viruses are presently recognized to be C-1 pathogens: IHHNV and TSV are primarily problems in the Americas and YHV and WSSV are problems in Asia. However, given the unregulated movement of live seed, broodstock and frozen and raw products it is likely that the four agents will become cosmopolitan in the future.

**Infectious Hypodermal and Hematopoietic Necrosis Virus**

IHHNV was the first Category 1 shrimp pathogen to be documented. It was first observed during 1980–1981 mass mortalities of *P. stylirostris* in intensive shrimp culture operations in the Hawaiian Islands, USA (Lightner *et al.* 1983a, 1983b). The virus particles are 22 nm in diameter, contain single-stranded DNA; the agent belongs to the family Paroviridae (Bonami *et al.* 1990).

**Course of Infection.** IHHNV causes infectious hypodermal and hematopoietic necrosis disease of *P. stylirostris* and is associated with runt deformity syndrome in *P. vannamei*. IHHNV is highly pathogenic to *P. stylirostris* and has been responsible for considerable losses to high-density aquaculture of this shrimp in Hawaii, and has virtually eliminated the once highly successful pond culture of *P. stylirostris* in northwestern Mexico (Lightner *et al.* 1992). Mortality rate for *P. stylirostris* 1–5 g in size is typically 85–95% over 10–20 days. Because of IHHNV *P. stylirostris* gave way to *P. vannamei* as the species of choice in the Americas, and so IHHNV never became the cosmopolitan scourge of aquaculture that it otherwise might have been.

In *P. vannamei*, IHHNV causes runt deformity syndrome, which is a more insidious malady than infectious hypodermal and hematopoietic necrosis disease in *P. stylirostris*. Runt deformity syndrome results in stunted growth of many individuals and therefore affects the quality of the grow-out product (Kalagayan *et al.* 1991). Although IHHNV has been responsible for direct economic loses to *P. vannamei* culture, it is important because it was targeted for eradication in the USA by the US Marine Shrimp Farming Program (Lotz 1992), and was the focus for development of the first specific pathogen-free program in shrimp aquaculture. The US-MSFP is a US Department of Agriculture supported programme designed to accelerate the development of shrimp aquaculture in the USA (Dill *et al.* 1994).

**Mode of Transmission.** In the laboratory, transmission of infections can be achieved by injection of cell-free extract of infected shrimp, by ingestion of infected material, or

---

**Table 1. Potential causative agents of mass mortalities in growout ponds.**

| Shrimp species       | Untreatable pathogens                                      | Treatable pathogens                      |
|----------------------|------------------------------------------------------------|------------------------------------------|
| *Penaeus vannamei*   | Taura syndrome virus                                       | Necrotizing hepatopancreatitis           |
|                      | Yellow head virus                                           | Vibriois                                 |
|                      | White spot syndrome virus                                   |                                          |
| *Penaeus stylirostris* | Infectious hypodermal and hematopoietic necrosis virus   | Vibriois                                 |
|                      | Yellow head virus                                           |                                          |
|                      | White spot syndrome virus                                   |                                          |
| *Penaeus monodon*    | Yellow head virus                                           | Vibriois                                 |
|                      | White spot syndrome virus                                   |                                          |
| *Penaeus japonicus*  | White spot syndrome virus                                   | Vibriois                                 |
| *Penaeus indicus*    | White spot syndrome virus                                   | Vibriois                                 |
| *Penaeus chinensis*  | White spot syndrome virus                                   | Vibriois                                 |
by exposure to effluent from aquaria housing infected shrimp (Lightner *et al.* 1983b). The mode of transmission outside the laboratory is likely to be by ingestion of dead infected shrimp or by contact with water containing infected animals. Furthermore, there is evidence that vertical transmission of IHHNV occurs from the mother to offspring, either through viral particles being shed at the time of spawning and then ingested by larvae at first feeding, or directly by transmission to larvae by passage of virus in oocytes. In our laboratory we have detected the virus in tissue sections treated with BS4.5, a gene probe for IHHNV developed by Don Lightner (D. Lightner, University of Arizona, Tucson, AZ, USA, personal communication). Virus can be detected in ovarian tissues surrounding oocytes as well as in developing oocytes (J.M. Lotz, unpublished work). Size (age) at exposure is a factor in the susceptibility and pathogenicity of IHHNV in *P. stylirostris*. Bell & Lightner (1987) found that animals 1–5 g in size were much more likely to succumb to infection than were 14 g subadults.

**Reservoir Hosts.** Only shrimp of the genus *Penaeus* are known to carry IHHNV (Lightner 1996).

**Occurrence in Wild Populations.** The distribution of IHHNV in shrimp prior to the advent of aquaculture is unknown and so the origin of the virus is obscure. IHHNV is widely distributed in aquaculture facilities throughout Asia and the Americas but its distribution among wild stocks is much less well documented. Lotz *et al.* (1991) and Lightner *et al.* (1992) report that wild stocks of shrimp carrying IHHNV infections occur throughout the eastern Pacific Ocean. IHHNV is also widely distributed among culture facilities in Asia (Lightner 1996) and probably occurs in some Asian wild shrimp stocks although no direct evidence for this is available. It is clear from the work of Lightner *et al.* (1992), that IHHNV was introduced from aquaculture into wild stocks of *P. stylirostris* in the Gulf of California along western Mexico. This suggests that the virus may have been introduced into the wild from aquaculture throughout most of its distribution, and it suggests that aquaculture is a major mode for viral dissemination among native populations. The effect of IHHNV on wild stocks is largely unknown, although Lightner *et al.* (1992) suggest that its introduction was associated with a decline in shrimp landings in the Gulf of California.

**Taura Syndrome Virus**

TSV was recognized in 1994 by Jim Brock of the Hawaii Aquaculture Development Program (Brock *et al.* 1995) as the causative agent of Taura syndrome, a disease that had been observed in Ecuador since 1992. TSV, a 30–32 nm, icosahedral virus particle containing single-stranded RNA, is provisionally categorized as a picornovirus (Lightner 1996).

**Course of Infection.** Taura syndrome is the most important disease of farmed penaeid shrimp in the western hemisphere. It is a rapidly progressing disease marked by extensive mortalities that become evident 25–35 days after a shrimp pond is stocked with post-larvae (Brock *et al.* 1995). Elevated death rates last only a few days but commonly reach 25% per day and leave a mere 5–25% of the shrimp alive. Survivors carry latent infections for at least a year and probably for life (J.M. Lotz, unpublished work). It does not appear that larger (older) animals are more resistant to mortality induced by TSV particularly if animals are exposed to the same dose on a per gram basis (J.M. Lotz, unpublished work).

TSV is a particularly virulent pathogen of *P. vannamei*, the most commonly cultured shrimp in the Americas (Brock *et al.* 1995; Hasson *et al.* 1995; Brock *et al.* 1995) found TSV to be less virulent to *P. stylirostris*; Overstreet *et al.* (1997) found TSV to be lethal to *P. setiferus*. In addition, although Overstreet *et al.* (1997) found little evidence of pathogenicity, they demonstrated that TSV could infect *P. aztecus* and *P. duorarum*.

**Transmission.** Like IHHNV, TSV can be transmitted by injection of cell-free extract of infected shrimp, by ingestion of infected shrimp tissue and by contact with water containing infected animals (Brock *et al.* 1995; J.M. Lotz, unpublished work). Unlike IHHNV, TSV appears not to be transmitted vertically through the oocytes of the female to the nauplii. Lightner (1996) reported that TSV is concentrated in the lymphoid organ of Taura syndrome survivors and, in our laboratory, we have reproduced infected broodstock whose offspring remained TSV-free after more than 9 months. Neither age nor size appear to have any influence on the susceptibility to infection with TSV (Overstreet *et al.* 1997; J.M. Lotz, unpublished work).

**Reservoir Hosts.** No organisms other than shrimp of the genus *Penaeus* have been shown to harbour active infections of TSV.

**Occurrence in Wild Populations.** TSV is established in the aquaculture industries of many countries of Central and South America and outbreaks have been confirmed in Mexico and the USA (Lightner 1996). In addition, there is evidence that TSV has become established in natural populations of *P. vannamei* in Central America, Mexico, and Ecuador and probably elsewhere (Lightner 1996). As with IHHNV it is unknown where TSV originated but aquaculture has played a major role in its dissemination throughout the Americas. The effects on wild stocks are unknown.
**Yellow Head Virus**

YHV was first observed in Thailand aquaculture in 1990 but was probably responsible for problems in the culture of *P. monodon* much earlier (Chantankachookin et al. 1993). The virus is an enveloped, rod-shaped, single-stranded RNA pathogen with approximate dimensions of 45 nm by 175 nm (Lightner 1996). YHV is probably a rhabdovirus, paramyxovirus, or coronavirus (Wongteerasupaya et al. 1995a; Flegel et al. 1996).

**Course of Infection.** YHV causes a disease that is of most significance to the culture of *P. monodon* in Asia. Culture ponds are affected about 50–70 days after stocking when animals are 5–15 g in size. Animals initially cease feeding and mortalities soon begin. Deaths reach massive proportions (up to 50% mortality per day) and cumulative mortalities run to nearly 100% 3–5 days after onset (Chantankachookin et al. 1993).

In the laboratory, YHV causes mortalities in American penaeids (Lu et al. 1994; Lightner 1996) and one outbreak of YHV in cultured *P. setiferus* has been detected in the USA (Lightner 1996). Either age or size appears to influence the impact of yellow head disease; younger shrimp are less susceptible than older individuals. Post-larvae are not affected in pond culture and Lightner (1996) reports that post-larvae of the American penaeids are less susceptible to mortalities than are larger juveniles.

**Transmission.** In the laboratory, YHV has been transmitted by feeding infected carcases and by inoculation with cell-free extract from affected shrimp. In addition, Flegel et al. (1995) report that virus can remain infectious in water for more than 72 h. Vertical transmission of YHV has not been investigated.

**Reservoir Hosts.** The shrimp *Palaemon styliferus* and *Acetes* sp. have been shown to act as reservoirs of YHV (Chantankachookin et al. 1993; Flegel et al. 1995). The penaeids *P. merguensis* and *Metapenaeus ensis* have been shown to act as asymptomatic carriers (Chantankachookin et al. 1993).

**Occurrence in Wild Populations.** The distribution of YHV in wild populations is not known. It would, however, be surprising if it were not present in wild populations of *P. monodon* in areas of south-east Asia where YHV is very common in culture. It may also be present in other penaeids as well as susceptible non-penaeid shrimp species in those areas.

**White Spot Syndrome Virus**

WSSV is a complex of nominal non-occluded baculoviruses that may in fact represent a single viral agent (Lightner 1996; Lo et al. 1996). The enveloped virions are rod-shaped with a size range of 70–150 by 250–380 nm. The pathogen was first reported in 1992 and 1993 from China, Japan, Taiwan, and northern Thailand, and it subsequently spread into southern Thailand, India, and Indonesia.

**Course of Infection.** WSSV is highly pathogenic to all species of the genus *Penaeus* for which there are data but the disease is particularly widespread among the Asian species including *P. monodon, P. chinensis, P. japonicus,* and *P. indicus.* Shrimp in affected ponds become anorectic and within 1–2 days mass mortalities ensue. By 3–10 days following onset of mass mortalities, deaths typically reach levels greater than 80% and usually 100% of the population succumbs (Nakano et al. 1994; Kasornchandra et al. 1996; Lightner 1996).

**Transmission.** In the laboratory WSSV has been transmitted by feeding infected carcases and by inoculation with cell-free extract from affected shrimp. Flegel et al. (1996) report that WSSV can be carried by contaminated water. Neither age nor size appear to be important for susceptibility of *P. japonicus* to infection; it affects animals in the range 0.015–25 g (Nakano et al. 1994). Vertical transmission of WSSV has not been investigated.

**Reservoir Hosts.** In addition to infecting all species of the genus *Penaeus* that have been tested, WSSV has been reported to infect a wide range of non-penaeid crustaceans (Flegel et al. 1996).

**Occurrence in Wild Populations.** The distribution of WSSV in wild populations is not known. However, it is likely to be found in wild penaeids of Asian countries having the most severe aquaculture epidemics of the disease. It may also be present in other penaeids as well as susceptible crustaceans in those areas.

**The Virus Problem**

It is quite clear that C-1 pathogens threaten the survival of aquaculture industries throughout the world; the dissemination of C-1 pathogens to aquaculture is being effected by transport of live shrimp for culture (Momayama et al. 1994) and through the shipment of frozen products carrying infectious pathogens (Lightner 1996). However, what is often less well appreciated, is the threat that arises from the unintentional introduction or widespread enhancement of these viruses in wild stocks of shrimp. In aquaculture areas that are presently affected by these pathogens, large amounts of virus enter native waters during routine water exchange and during emergency drain harvests (Chantankachookin et al. 1993). If these viruses become established in wild stocks of shrimp, those stocks can become reservoirs for infecting...
pond-reared shrimp through contaminated water exchange, stocking of infected wild post-larvae or from seed produced from infected wild spawners. In addition to the impact on aquaculture, the viruses could be the factor that alone or in combination with environmental degradation might result in a reduction of wild harvest of the affected species.

The Solution

For shrimp aquaculture to overcome the disease threats and to resume growth in production will require a new blueprint for shrimp farming industries throughout the world. The industry of the future will be based on: (i) specific pathogen-free and genetically improved shrimp stocks; (ii) biosecure systems including enclosed, reduced water-exchange and increased water-reuse culture systems; (iii) biosecure management practices; and (iv) co-operative industry-wide disease control strategies.

A Specific Pathogen-free Based Industry

A specific pathogen-free based shrimp aquaculture industry relies upon a supply of animals that are free of specified significant pathogens. Such industries comprise three components or streams, the ‘specific pathogen-free’ stream, the ‘high health’ stream, and the ‘commodity production’ stream (Lotz et al. 1995). The specific pathogen-free stream is focused on a few high-security isolation research and development facilities called nucleus breeding centres. The long term goal of the breeding centres is to develop and provide new strains of genetically improved shrimp to the industry. In addition to genetic improvement, the centres are the source of specific pathogen-free post-larvae for the rest of the industry.

From the high-security breeding centres, small numbers of seed shrimp of known genetic background flow into medium-security facilities of the high health stream of the industry. The high health stream is the portion of the industry that provides large numbers of young seed shrimp to the farmers. Three production operations are found in this stream: (i) broodstock multiplication stations that acquire post-larvae from a breeding centre and then produce breeder animals; (ii) shrimp reproduction facilities that reproduce the breeders; and (iii) larval rearing facilities that rear larval shrimp to the post-larval stage for stocking into ponds for growing commodity shrimp.

The two phrases ‘specific pathogen free’ and ‘high health’ imply firstly, that those facilities so designated have programs in place to prevent the introduction of pathogens and to monitor for outbreaks of pathogens, and secondly, that they can provide documentation of such programs and the results of monitoring. The difference between the two phrases is that the high health stream is somewhat lower in security because there is a greater amount of human traffic and movement of animals associated with the commercial production of seed shrimp than with a breeding program. Unfortunately, the two phrases have occasionally been misunderstood to imply not only freedom from specific pathogens but also to imply that the animals are in some way resistant to the pathogens. This is an unjustified conclusion: specific pathogen free and high health animals are simply free of the specific pathogens and assured to be so to varying degrees, whether a particular shrimp strain or species is resistant to a specific pathogen is independent of its present pathogen status; these phrases refer only to present pathogen status and to neither pathogen resistance nor future pathogen status.

Some individuals have suggested that the way to deal with the most devastating disease agents is to employ shrimp species that are resistant regardless of their pathogen status or to develop resistant strains of the species of choice. Even if resistant carriers, i.e. survivors of epidemics, could be bred and used in aquaculture there would always be the danger that the resident virus would mutate into a pathogenic strain. Although the concern of pathogenic strain development is relevant to all resident pathogens, the danger is particularly pertinent to YHV and TSV both of which are RNA viruses. RNA viruses are notorious for rapid mutation and evolution (Steinhauer & Holland 1987).

The development of specific pathogen-free and resistant strains should be a long term goal of breeding programs and these characteristics should be pursued vigorously. However, such an activity is fraught with problems, and it is unlikely that breeding programs will ever result in shrimp strains that are unaffected by any disease organisms: unfortunately, resistant strains and species often provide only short term remedies. For example in the 1980s many farmers in the western hemisphere switched from P. stylirostris to P. vannamei to remove the immediate threat from IHNV; however, IHNV was still a problem in P. vannamei seed production where it caused runt deformity syndrome. Now TSV, which is devastating to P. vannamei but is apparently less virulent in P. stylirostris (Brock et al. 1995) is causing farmers to consider switching back to P. stylirostris. Such a change would probably expose farmers to losses from IHNV. At present no penaeid shrimp species are known to be resistant to WSSV.

Although disease resistance is an important breeding goal of nucleus breeding centers, their overall and most important role is to produce strains of shrimp that are domesticated and genetically improved for aquaculture.

Biosecurity in shrimp aquaculture
The only way that shrimp aquaculture can become a sustainable portion of agriculture is to focus on domesticated shrimp and reduce reliance on wild stocks. The success of terrestrial agriculture has been tied to the development of domesticated plants and animals: domesticated strains, even those not resistant to all pathogens, will have growth and behavioural characteristics that make them more suitable for aquaculture than their wild counterparts.

A specific pathogen-free based industry is designed to ensure that the post-larval seed shrimp going into commodity pond production are free of particular pathogens: the commodity production stream is the most vulnerable to pathogen outbreaks. This vulnerability can be attributed to several factors: (i) the stream is composed of open ponds which are large targets for pathogen contamination; (ii) the ponds are exposed to large quantities of untreated water during filling and water exchange; (iii) commodity production farms often have large amounts of traffic associated with stocking, maintaining, and harvesting ponds that can transport pathogens; (iv) the large amount of seed that is necessary to stock a commodity production farm can carry pathogens.

Biosecurity

The term biosecurity refers to practices that reduce the probability of pathogen introduction and subsequent spread from one place to another. The first consideration in biosecurity is for the possible carriers of contamination. These include: (i) infected hosts; (ii) non-host biological carriers; and (iii) inanimate objects contaminated with pathogens.

The four C-1 pathogens described above are likely to be transmitted via infected seed. However, there are others sources of infection with YHV and WSSV; non-seed reservoir hosts exist and these pathogens can be introduced to ponds by such hosts during pond filling. Alternatively, these viruses may survive if the ponds are not completely dried between crops.

Non-host biological carriers include predators that pick up infected shrimp and carry them elsewhere e.g. birds which are often seen foraging over ponds containing weakened and dying shrimp; they carry infected shrimp to nearby ponds, that may be uninfective, where they drop them or have them dislodged by other birds. In addition, birds, rodents, and insects that feed on shrimp offal from heading and peeling operations could carry contamination; if shrimp offal is improperly treated and disposed of uncooked or unburied, infectious material may be conveyed from the dumping area to the farm; if uncooked shrimp waste or reservoir hosts are used as fresh feed or incorporated into moist artificial feed, pathogens could be transmitted.

Predators may pass viable infectious agents, that have survived passage through the digestive tract, in their faeces: transmission of some insect viral pathogens in bird faeces has been documented (Andreadis 1987). Similarly, birds and flying insects could carry shrimp pathogens in their intestines after ingestion of infected shrimp. Faecal transmission results in much wider dissemination of virus than carrying infected shrimp pieces by mouth: birds migrate hundreds of miles in a few days and they may carry viable viruses in their intestines for that long.

The major non-living conveyor of pathogens is water. Pathogens may be present in the incoming water because of the presence of natural hosts, effluent from a contaminated farm, or effluent from operations that are heading and peeling infected shrimp.

Perhaps the most important route of pathogen admissance is through human traffic that carries contamination on clothing, vehicles, buckets, nets or other materials.

The ideal biosecure culture facility design is covered ponds or tanks using recirculating sea water systems. To date, completely closed recirculating culture systems have proved to be productive at commercial levels only for *P. vannamei* maturation and reproduction (Lotz & Ogle 1994). Although maturation and larval rearing facilities have not completely adopted closed systems, they are typically covered and in the USA and other countries many employ up to 75% recirculation per day and often use biofiltration.

Commercial scale, covered, entirely recirculating water systems have not yet proved to be effective for the grow-out of commodity shrimp. A commercial scale grow-out system, the design of which came close to being completely biosecure, was that developed by the University of Arizona and implemented by Marine Culture Enterprises in Hawaii for the culture of *P. stylirostris* at high densities (Moore & Brand 1993). The system was covered, but it used water recirculation only for aeration and employed a daily water exchange of 280% late in the grow-out cycle. Although the system used large amounts of untreated water, the source of the water was not surface open sea water, but a shallow subsurface well-field; well water is generally more biosecure than open sea water (Lotz et al. 1995). Nonetheless, the operation failed, not because the system was unproductive, but because of contamination by a C-1 pathogen, IHHNV. Moore & Brand (1993) attributed the outbreak to the failure to follow consistently the biosecurity practices that had been set up. However, it is possible that the shallow well was too shallow because effluent waste water from the facilities was subsequently shown to seep back into it (Moore & Brand 1993) and could have been the source of the pathogen contamination.
The culture of penaeid shrimp without exchanging water is developing. Ogle & Lotz (1992) report promising results on the growth of P. vannamei in an experimental system having total water re-use. Hopkins et al. (1995) demonstrate that pond culture of P. vannamei with low or no water exchange is feasible and may be attained in the near future. A no-water-exchange culture would solve two great disease problems in aquaculture: contamination of farms from incoming water and contamination of source waters by effluent from farms with pathogen outbreaks. This an area of shrimp culture in need of much rapid research and development.

Although the liberal use of disinfectants is likely to decrease the probability of a pathogen outbreak, little is known about the effects of particular disinfecting agents on specific shrimp viruses. LeBlanc & Overstreet (1991a) have shown that u.v. light can kill the virus Baculovirus penaei effectively. Pasteurization (heating to 60 or 65 °C for 30 min) will kill Baculovirus penaei (LeBlanc & Overstreet 1991a) but not IHHNV which must be subjected to 80 °C (Al-Mazrooei 1995). LeBlanc & Overstreet (1991b) have determined that a chlorine concentration of 200 mg/l for 1 h is effective in inactivating Baculovirus penaei. Pratampipat et al. (1996) have shown that the minimum concentration of formalin necessary to inactivate WSSV is 70 mg/l. Many viruses cannot withstand drying; LeBlanc & Overstreet found that dessication for 48 h inactivated Baculovirus penaei.

Co-operative Disease Controls Strategies

An overall plan for the control of dangerous pathogens will necessarily include routine monitoring of all farms to detect a pathogen as soon it occurs within a region. Ideally, a co-operative program involving farmers, aquatic health care specialists, university scientists, and government agencies should be developed to track important pathogens. Such a network would result in early warning for farmers of the movement of a dangerous pathogen into a region. In the USA, the Centers for Epidemiology and Animal Health have been developed to perform such a function for terrestrial livestock production. On a world-wide scale the Office International des Epizooties fulfills a similar role and includes aspects of aquatic animal health.

In areas where water exchange or emergency harvest of contaminated ponds are allowed, farmers should agree to notify neighbours of such events. Neighbouring farms should not exchange water while a contaminated one is exchanging water or harvesting ponds. To reduce the likelihood of contaminating the water source, peeling and heading operations should agree to disinfect waste or not dispose of waste in open waters at all. Proper treatment (e.g. cooking) and disposal (e.g. burial of offal from peeling and heading operations) should be assured.

In a culture industry that is attempting to employ biosecure culture practices, rapid response to a C-1 outbreak is necessary to prevent spread to other modules, farms, or the natural environment. It should be realized that once a C-1 pathogen outbreak occurs, the farm and its ponds become amplifiers of the pathogens. The farm may then become a source of the pathogen and may facilitate its rapid dissemination.

The exact requirements for dealing with an outbreak of a C-1 pathogen will depend upon local laws, the proximity and density of farms surrounding the outbreak, and whether the pathogen is exotic or native to the region. However, ensuring that an infected farm will not be the nidus for spread to other farms or to susceptible native species may require drastic measures. First, a diagnostic confirmation is necessary. Appropriately prepared samples should be sent to a competent aquatic animal diagnostic laboratory and reported to the proper authorities. Upon confirmation of a C-1 pathogen the farm should be quarantined. All unnecessary traffic to and from the farm should be suspended. Water exchange should be discontinued, ponds should be covered with bird netting, and round-the-clock bird control measures should be implemented.

At a minimum the affected ponds should be depopulated; however, depopulation of the whole farm may be necessary to ensure prevention of disease spread. To depopulate a pond in the safest manner shrimp should be killed, removed, and the carcasses should be incinerated, buried, or cooked. Shrimp can be killed by some means, e.g. chlorination, or an approved insecticide. Removal of shrimp could occur after draining if the water is previously disinfected or if ponds are drained into nearby empty ponds. Water should be disinfected and the disinfectant neutralized prior to discharge. Whether a pond can be safely harvested to salvage a crop will be determined by the specifics of the pathogen (exotic or native), the threat to the environment, the threat to neighbouring farms and local laws. Harvesting by releasing untreated water into the natural surroundings is not recommended under any circumstances.

Outlook

The clearest way to ensure the future growth of shrimp aquaculture is by control and eradication of pathogens from the industries. This will entail co-operation between public and private sectors to develop specific pathogen-free programmes, to advance shrimp genetics research and development, to produce new technologies and management practices for the biosecure culture of shrimp, and to a better understanding of the pathogens.
and their identification, biology, epidemiology, pathology, and control. The long term worldwide pay-offs from a new approach to shrimp aquaculture are enormous, and the long-term costs of not modifying shrimp aquaculture are equally enormous.

Acknowledgements

This work was supported in part by USDA-CSREES Grant Numbers 92-38808-6920 and 96-38808-2580.

References

Al-Mazrooei, A. 1995 Effect of temperature on infectivity and pre-patent period of Infectious Hypodermal and Haematopoietic Necrosis Virus (IHNNV) in Penaeus vannamei. MS Thesis. University of Southern Mississippi, USA.

Andreadis, T.G. 1987 Transmission. In Epizootiology of Insect Diseases, eds Fuxa, J.R. & Tanada, Y. pp. 159-176. New York: John Wiley & Sons.

Bell, T. A. & Lightner, D. V. 1987 IHNN disease of Penaeus stylirostris: effects of shrimp size on disease expression. Journal of Fish Diseases 10, 165-170.

Bonami, J.R., Trumper, J., Mari, J., Brebelin, M. & Lightner, D.V. 1990 Purification and characterization of the infectious hypodermal and haematopoietic necrosis virus of penaeid shrimps. Journal of General Virology 71, 2657-2664.

Boonyaratpalin, S., Supamattaya, K., Kasorchanda, J., Direkbusarakom, S., Aekpanithanpong, U. & Chantanachooklin, C. 1993 Non-occluded baculo-like virus, the causative agent of Yellow Head Disease in the black tiger shrimp (Penaeus monodon). Gyo To Kyokushin 28, 103-109.

Brock, J.A., Gore, R., Lightner, D.V. & Hasson, K.W. 1995 An overview on Taura syndrome, an important disease of farmed P. vannamei. In Swimming Trough Troubled Waters, Proceedings of the Special Session on Shrimp Farming, eds Browdy, C.L. & Hopkins, J.S. pp. 84-94. Baton Rouge, LA: The World Aquaculture Society.

Chantanachookin, C., Boonyaratpalin, S., Kasornchandra, J., Direkbusarakom, S., Ekpahanipong, U., Supamataya, K., Srirairaitana, S. & Flegel, T.W. 1993 Histology and ultrastructure reveal a new granulosis-like virus in Penaeus monodon affected by yellow-head disease. Diseases of Aquatic Organisms 7, 145-157.

Dill, J., Mellwain, T., Rowland, W.C., & Pruder, G. 1994 The Gulf Coast Research Laboratory Consortium’s U.S. Marine Shrimp Farming Program. In USMFP 10th Anniversary Review, GCRL Special Publication No. 1, eds Mellwain, T. & Pruder, G. pp. 7-24. Ocean Springs: The Gulf Coast Research Laboratory.

Flegel, T.W., Sriurairatana, S., Wongteerasupaya, C., Boonsaeng, V., Panym, S., Witthayachumnarnkul, B. 1995. Progress in characterization and control of Yellow-head virus of Penaeus monodon. In Swimming Trough Troubled Waters, Proceedings of the Special Session on Shrimp Farming, Aquaculture ’95, eds Browdy, C.L. & Hopkins, J.S. pp. 76-83. Baton Rouge, LA: World Aquaculture Society.

Flegel, T.W., Boonyaratpalin, S., & Witthayachumnarnkul, B. 1996 Current status of research on yellow-head virus and white-spot virus in Thailand. In World Aquaculture ’96. Book of Abstracts. ed Cresswell, R.L. pp. 126-127. Baton Rouge: World Aquaculture Society.

Hasson, K.W., Lightner, D.V., Poulos, B.T., Redman, R.M., White, B.L., Brock, J.A. & Bonami, J.R. 1995 Taura syndrome in Penaeus vannamei: demonstration of a viral etiology. Diseases of Aquatic Organisms 23, 115-126.

Hopkins, J.S., Sandifer, P.A. & Browdy, C.L. 1995 A review of water management regimes which abate the environmental impacts of shrimp farming. In Swimming Trough Troubled Waters, Proceedings of the Special Session on Shrimp Farming, Aquaculture ’95, eds Browdy, C.L. & Hopkins, J.S. pp. 157-166. Baton Rouge, LA: World Aquaculture Society.

Kalagayan, H., Godin, D., Kanna, R., Hagino, G., Sweeney, J. & Wyban, J. 1991 IHNN Virus as an etiological factor in Rust-Deformity syndrome (RDS) of juvenile Penaeus vannamei cultured in Hawaii. Journal of the World Aquaculture Society 22, 235-243.

Kasornchandra, J., Boonyaratpalin, S., Khongpradit, R. & Aekpanithanpong, U. 1996 Mass mortality caused by systemic bacilliform virus in cultured penaeid shrimp, Penaeus monodon, in Thailand. In World Aquaculture ’96. Book of Abstracts. ed Cresswell, R.L. pp. 193-194. Baton Rouge: World Aquaculture Society.

LeBlanc, B.D. & Overstreet, R.M. 1991a Effect of Desiccation, pH, heat, and ultraviolet irradiation on viability of Baculovirus penaei. Journal of Invertebrate Pathology 57, 277-286.

LeBlanc, B.D. & Overstreet, R.M. 1991b Efficacy of calcium hypochlorite as a disinfectant against the shrimp virus Baculovirus penaei. Journal of Aquatic Animal Health 3, 141.

Lightner, D.V. 1996 A Handbook of Shrimp Pathology and Diagnostic Procedures for Diseases of Cultured Penaeid Shrimp. Baton Rouge: World Aquaculture Society.

Lightner, D.V., Redman, R.M. & Bell, T.A. 1983a Infectious hypodermal and haematopoietic necrosis (IHNN), a newly recognized virus disease of penaeid shrimp. Journal of Invertebrate Pathology 42, 62-70.

Lightner, D.V., Redman, R.M., Bell, T.A. & Brock, J.A. 1983b Detection of IHNN virus in Penaeus stylirostris and P. vannamei imported into Hawaii. Journal of the World Mariculture Society 14, 212-225.

Lightner, D.V., Williams, R.R., Bell, T.A., Redman, R.M. & L.A. Perez A. 1992 A collection of case histories documenting the introduction and spread of the virus disease IHNN in penaeid shrimp culture facilities in northwestern Mexico. ICES Marine Sciences Symposium 194, 97-105.

Lightner, D.V., Redman, R.M., Hasson, K.W. & Pantoja, C.R. 1995 Taura syndrome in Penaeus vannamei (Crustacea: Decapoda): gross signs, histopathology and ultrastructure. Diseases of Aquatic Organisms 21, 53-59.

Lo, C., Lui, J., Ho, C., Chen, C., Peng, S., Chen, Y., Chou, C., Yeh, S., Huang, C., Chou, H., Wang, C., & Kou, G. 1996. Detection of baculovirus associated with white spot syndrome (WBSV) in penaeid shrimps using polymerase chain reaction. Diseases of Aquatic Organisms 25, 133-141.

Lotz, J.M. 1992 Developing specific-pathogen-free (SPF) animal populations for use in aquaculture: A case study for IHNN virus of penaeid shrimp. In Diseases of Cultured Penaeid Shrimp in Asia and the United States eds Fulk, W. & Main, K.L. pp. 269-283. Honolulu: The Oceanic Institute.

Lotz, J.M. & Ogle, J.T. 1994 Reproduction of the white-legged shrimp Penaeus vannamei in closed and semiclosed recirculating systems. Journal of the World Aquaculture Society 25, 327-345.

Lotz, J.M., Overstreet, R.M., Lightner, D.V. & Redman, R.M. 1991 Occurrence of IHNN virus in penaeid shrimp from wild populations of the eastern Pacific Ocean. Journal of the World Aquaculture Society 22, 37A.
Biosecurity in shrimp aquaculture

Lotz, J.M., Browdy, C.L., Carr, W.H., Frelier, P.F. & Lightner, D.V. 1995 USMSFP suggested procedures and guidelines for assuring the specific pathogen status of shrimp broodstock and seed. In Swimming Trough Troubled Waters, Proceedings of the Special Session on Shrimp Farming, Aquaculture ’95, Browdy, C.L. & Hopkins, J.S. pp. 66–75. Baton Rouge, LA: World Aquaculture Society.

Lu, Y., Tapay, L.M., Loh, P.C. & Brock, J.A. 1994 Infection of the yellow-head bacculo-virus in two species of penaeid shrimp Penaeus stylirostris and P. vannamei. Journal of Fish Diseases 17, 649–656.

Momayama, K., Hiraoka, M., Nakano, H., Koube, H., Inouye, K. & Oseko, N. 1994 Mass mortalities of cultured Kuruma shrimp, Penaeus japonicus, in Japan in 1993: Histopathological study. Fish Pathology 29, 141–148.

Moore, D.W. & Brand, C.W. 1993 The culture of marine shrimp in controlled environmental super-intensive systems. In CRC Handbook of Mariculture, vol. I. Crustacean aquaculture, ed McVey, J.P. pp. 315–358. Boca Raton: CRC Press.

Nakano, H., Koube, H., Umeawa, S., Momoyama, K., Hiraoka, M., Inouye, K. & Oseko, N. 1994 Mass mortalities of cultured Kuruma shrimp, Penaeus japonicus, in Japan in 1993: Epizootiological survey and infection trials. Fish Pathology 29, 135–139.

Ogle, J.T. & Lotz, J.M. 1992 Closed system Culture of P. vannamei. Gulf Research Reports 8, 401–415.

Overstreet, R.M., Lightner, D.V., Hasson, K.W., McLellwan, S., & Lotz, J.M. 1997 Susceptibility to TSV of some penaeid shrimps naive to the Gulf of Mexico and Southeastern U.S. Journal of Invertebrate Pathology 69, 165–176.

Pratanpipat, P., Nithimethachoke, C., Akarajamorn, A., Nash, G., Wityyachumnarnkul, B., Thammats, S. & Lohawattanakul, C. 1996 The efficacy of formalin for disinfection of Systemic Ectodermal and Mesodermal Baculovirus. In World Aquaculture ’96. Book of Abstracts. ed Cresswell, R.L. pp. 318. Baton Rouge: World Aquaculture Society.

Rosenberry, B. 1989 World Shrimp Farming 1989. San Diego: Aquaculture Digest.

Rosenberry, B. 1995 World Shrimp Farming 1995. San Diego: Shrimp News International.

Steinhauer, D.A. & Holland, J.J. 1987 Rapid evolution of RNA viruses. Annual Review of Microbiology 41, 409–433.

Takahashi, Y., Itami, T., Kondo, M., Maeda, M., Fujii, R., Tomonaga, S., Supamattaya K., & Boonyaratpalin, S. 1994 Electron microscopic evidence of bacilliform virus infection in Kuruma shrimp (Penaeus japonicus). Fish Pathology 29, 121–125.

Wongteerasupaya, C., Suriararatana, S., Vickers, J.E., Akrajamorn, A., Boonsaeng, V., Panyim, S., Tassanakajon, A., Wityyachumnarnkul, B. & Flegel, T.W. 1995a Yellow-head virus of Penaeus monodon is an RNA virus. Diseases of Aquatic Organisms 22, 45–50.

Wongteerasupaya, C., Vickers, J.E., Suriararatana, S., Nash, G.L., Akrajamorn, A., Boonsaeng, V., Panyim, S., Tassanakajon, A., Wityyachumnarnkul B. & Flegel, T.W. 1995b A non-occluded, systemic baculovirus that occurs in cells of ectodermal and mesodermal origin and causes high mortality in the black tiger prawn, Penaeus monodon. Diseases of Aquatic Organisms 21, 69–77.