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The global status of significant infectious diseases of farmed shrimp

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Abstract

A discussion of the global status of shrimp diseases might best begin with a review of those diseases that are listed by the International Office des Épizooties (OIE). The OIE is also known as the World Animal Health Organization. The OIE is an international organization formed in 1924 in Europe by 28 countries in an effort to more effectively manage certain diseases of livestock. As of June 2010, the OIE consists of 176 member countries. The OIE was designated in 1995 by the newly formed World Trade Organization as the scientific reference body for animal health as it relates to international trade issues. The OIE has, among its functions, the listing of diseases (terrestrial and aquatic) which may pose risks of being transferred to new regions or nations as a consequence of global trade. Because of their economic importance and their potential for transfer with live or dead crustacean commodities, the OIE listed nine crustacean diseases in 2009. Of these nine listed diseases, six are diseases of penaeid shrimp (five viral and one bacterial); the seventh is a viral disease of *Macrobrachium rosenbergii*, the eighth is a disease of farmed spiny (*Panulirus* spp.) lobsters and it is due to infection by a rickettsial-like bacterium and the ninth listed disease affects freshwater crayfish and it is due to infection by a phycomycetous fungus. Two of the nine diseases were listed by the OIE as "under study" in the 12th Edition of the OIE Aquatic Animal Health Code. These diseases were necrotizing hepatopancreatitis (NHP), a rickettsia-like bacterial disease of penaeid shrimp which was approved for full listing in May 2010 and milky hemolymph disease (MHD) of spiny lobsters, which also has as its etiological agent a rickettsial-like bacterium and which may remain "under study" pending further consideration for full listing or removal from the list. While OIE listing gives these diseases global recognition, especially in relation to trade of crustacean commodities (e.g. live, dead or commodity products made from crustacean hosts for one or more OIE listed disease agents), there are other emerging diseases that are not listed by OIE that are also important locally and in some cases globally, to the global shrimp farming industry. Included in this review are the current OIE listed diseases of penaeid shrimp and several examples of emerging diseases which are of potential importance globally.

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Introduction

Penaeid shrimp aquaculture is an important industry in Asia and the Americas that employs millions of people and provides valuable foreign exchange to many developing nations. While the industry is now dominated by the culture of the Pacific white shrimp, *Litopenaeus vannamei* (the shrimp taxonomy used in this paper is according to Perez Farfante and Kensley 1997), there is also significant culture of black tiger (*Penaeus monodon*) and Indian white shrimp (*Fenneropenaeus indicus*), which contribute significant quantities of shrimp to the global market (FAO 2006, 2009). Since shrimp aquaculture became a significant commercial entity in the 1970s, disease has had a major impact on the industry nearly everywhere it has been developed (Lightner 1996a, 1999; Flegel and Alday-Sanz 1998; Flegel 1997, 2006; Bondad-Reantaso et al. 2001; OIE 2009a, 2009b). Diseases due to viruses, rickettsial-like bacteria, true bacteria, protozoa and fungi have emerged as significant diseases of farmed shrimp (Lightner 1996a). Many of the bacterial, fungal and protozoan which caused diseases are managed using improved culture practices, routine sanitation, chemotherapeutics and, recently, the use of probiotics. Some bacterial diseases of farmed shrimp are increasingly difficult to manage, especially with the restriction of antibiotic use in farmed shrimp intended for certain markets.

The virus diseases have been far more problematic to manage and they have been responsible for the most costly epizootics. Examples of those with the most important socioeconomic impacts include the Taura syndrome and yellow head disease pandemics that began in 1991-92 when these diseases emerged in Ecuador, Thailand, respectively and the white spot disease pandemic that emerged in East Asia at about the same time. The most important diseases of cultured penaeid shrimp, in terms of economic impact, in the Americas (and in Asia) have viral agents as their cause (Flegel and Alday-Sanz 1998; Flegel 1997, 2006; Lightner 1999; Walker and Mohan 2009; OIE 2009a, 2009b). Of significance is that some of the most important diseases (and their etiological agents) were once limited in distribution to either the Western or Eastern Hemisphere (Lightner 1996a, 1996b, 2003a, 2003b; Flegel and Alday-Sanz 1998; OIE 2009b; Walker and Mohan 2009). However, the international movement of live (for aquaculture) and dead (commodity shrimp for reprocessing, direct retail commerce and for use as bait by sport fishermen) shrimp have been implicated or suspected as being responsible for the transfer and establishment of certain pathogens from Asia to the Americas (Lightner 1996b; Durand et al. 2000; AQUIS 2000; Hasson et al. 2006; Walker and Mohan 2009). While frozen commodity shrimp have been implicated as the route by whichwhite spot syndrome virus (WSSV) was moved from Asia to the Americas, TSV was moved in the opposite direction with infected live broodstock from Central America (Nunan et al. 1998a; Tu et al. 1999; Yu and Song

2000; Durand et al. 2000; Tang and Lightner 2005; Walker and Mohan 2009). Because of their socioeconomic importance and their significance to shrimp farming, five of the seven crustacean diseases listed by the World Animal Organization (OIE) in 2009 were virus diseases of shrimp (Fig. 1). Two additional diseases of crustacean, necrotizing hepatopancreatitis (NHP) of shrimp and milky hemolymph disease (MHD) of spiny lobsters were listed in the 12th Edition of the OIE Aquatic Animal Health Code (OIE 2009a) as "under study" and were being considered for full listing by the OIE. Both NHP and MHD have bacterial etiologies. NHP was approved for full listing by the OIE in May 2010 while MHD may remain listed as "under" study pending further consideration for full listing or removal from the list (OIE 2009a; OIE 2010). MHD (also called milky hemolymph syndrome - MHS) is also a disease affecting farmed penaeids and captive-wild crabs. In farmed *P. monodon* in East Asia and Madagascar, MHD has caused significant epizootics recently (Nunan et al. 2010). Should it continue to emerge in that region and spread to other regions, the possible full listing of MHD in spiny lobsters might be expanded to shrimp.

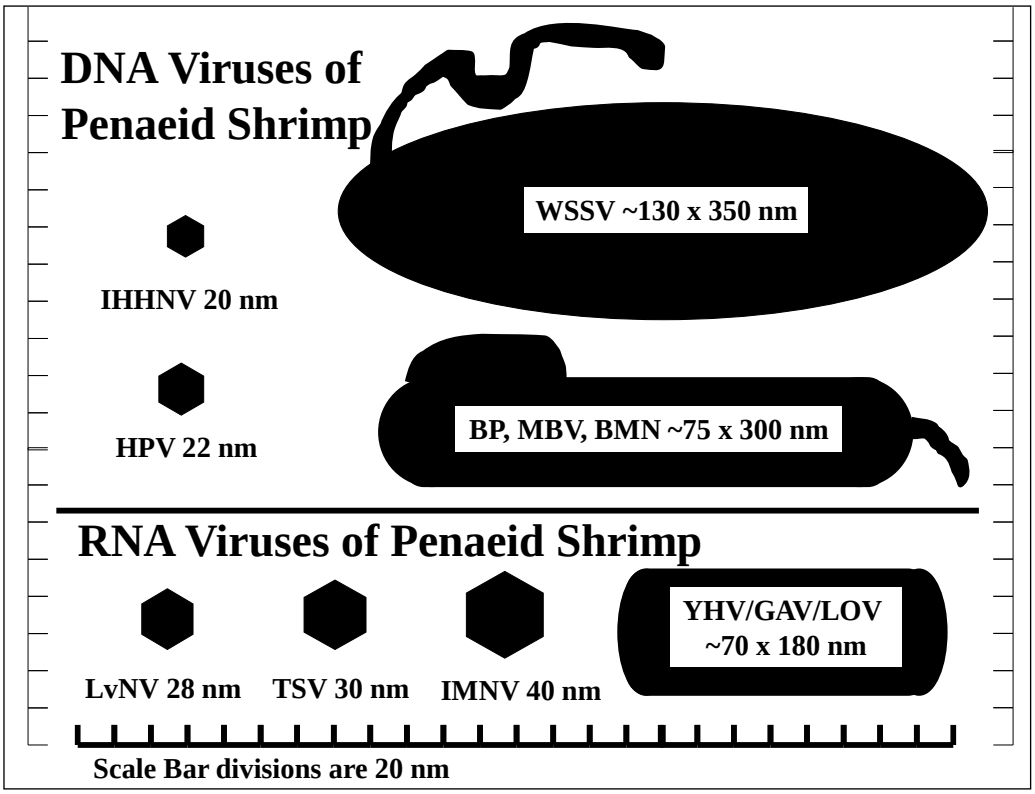

Fig. 1. Schematic of the major viruses of penaeid shrimp. The virions are drawn to scale;scale divisions are 20 nm. See text for definition of the acronym shown for each virus.

As a consequence of the rapid growth and development of the penaeid aquaculture industry, many of the most significant shrimp pathogens were moved from the regions where they initially appeared to new regions even before the "new" pathogen had been recognized, named, proven to cause the "new" disease and before reliable diagnostic methods were developed. The diseases due to the shrimp viruses IHHNV, TSV and WSSV were all transferred with live shrimp stocks from country to country and from one continent to another, well before their etiology was understood and diagnostic methods were available. With some diseases, the introduced pathogen encountered totally naive hosts with little or no innate resistance. The pandemics due to the penaeid viruses WSSV and TSV and to a lesser extent to IHHNV, IMNV and YHV have collectively cost the penaeid shrimp industry billions of dollars in lost crops, jobs and export revenue (Lightner 2003a; Walker and Mohan 2009). The social and economic impacts of the pandemics caused by these pathogens have been profound in countries in which shrimp farming constitutes a significant industry and this has led to the listing of several of the virus diseases of penaeid shrimp by the OIE or World Animal Health Organization (OIE 2009a).

World Animal Health Organization (OIE) and Listed Diseases

The World Animal Health Organization (or Organization des Epizooties or OIE) was founded in 1924 in response to the need to control Rinderpest, which first occurred in Europe as a consequence of transfer of zebus being shipped from India to Brazil through a seaport in Belgium. In 1924, there were 28 member countries. By 2010, OIE was composed of 176 member countries. In 1955, the World Trade Organization (WTO) was created to replace the GATT (General Agreement on Trade & Tariffs) that was established in 1947 to facilitate international trade following World War II. WTO's SPS (Sanitary and Phytosanitary Measures) Agreement recognized the OIE as the leading international standards-setting organization for animal health and animal diseases that are transmissible to humans (zoonosis) (OIE 2009c).

The OIE has three principal functions, which are: 1) to inform members of the occurrence and course of animal diseases throughout the world and of means of controlling these diseases; 2) to coordinate international research devoted to the surveillance and control of animal diseases; and 3) to promote the harmonization of health regulations for trade in animals and animal products among members. Among the components of the OIE are four specialists' commissions that work to help the OIE meet these three functions. The Biological Standards Commission has as part of its mandate the responsibility to establish and approve methods for diagnosing OIE listed terrestrial animal (mammal, bird and bee) diseases and for testing of biologics (vaccines) for disease control purposes. The Aquatic Animal Health Standards

Commission (AAHSC) has similar functions for finfish, mollusks, crustaceans and amphibians. These methods are published as the Manual of Diagnostic Tests and Vaccines for Terrestrial Animals and the Manual of Diagnostic Tests for Aquatic Animals. The diagnostic manuals are published at 3-4 year intervals. Another commission, the Terrestrial Animal Health Standards Commission and the AAHSC, annually produce and publish the Terrestrial Animal Health Code and the Aquatic Animal Health Code, respectively, to ensure that these codes reflect current scientific information on OIE listed diseases. The fourth OIE commission, the Scientific Commission for Animal Diseases, identifies the most appropriate strategies and measures for disease prevention and control. This commission also reviews requests for official OIE "disease-free status" for four major diseases of terrestrial food animals (FMD, Rinderpest, CBPP and BSE) and it reviews and publishes in the OIE Bulletin "self" declarations for "disease-free status" from member countries (OIE 2009d).

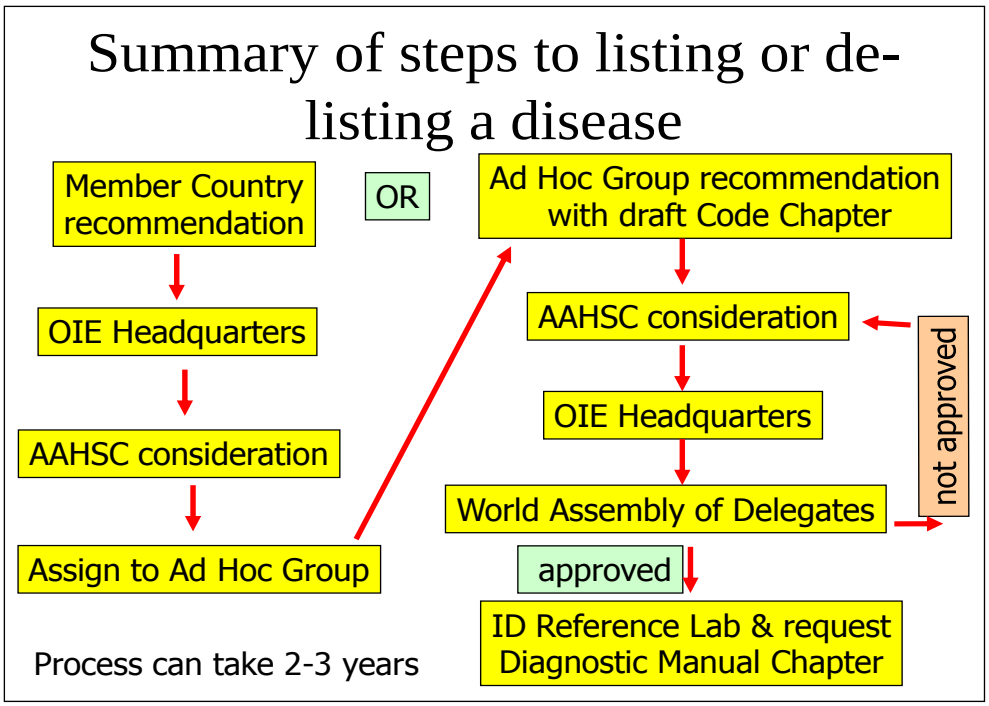

Fig. 2. A summary of the steps to listing (or de-listing) a disease by the OIE. The process begins with a recommendation that a disease be listed that may come from a Member Country, from an ad hoc group of experts appointed by the Aquatic Animal Health Standards Commission (AAHSC), from an OIE Reference Laboratory, or from OIE. The process can take 2-3 years and the disease in question becomes listed (or de-listed) when the recommendation is approved by the World Assembly of Delegates.

The OIE specialist commissions also have the mandate of ensuring that listed diseases reflect current scientific information. For the AAHSC, this means that it has the responsibility of either adding or deleting listed diseases depending on the most

current and available scientific information. To do this, the AAHSC forms ad hoc groups (AHGs) of specialists that assess the available information for disease(s) under consideration to determine if the criteria for listing is as defined in Articles 1.2.2.1 or 1.2.2.2 of Chapter 1.2.2 of the Aquatic Animal Health Code (OIE 2009a). The AHGs provide recommendations to the AAHSC for consideration and when the Commission agrees with an AHG's recommendations, it recommends to the OIE Central Bureau and to the International Commission (composed of delegates from all OIE member countries) that a disease be listed (Fig. 2). In the case of those diseases which no longer justify being listed by OIE (e.g. it no longer meets the criteria of Article 1.2.2.1 of the Aquatic Code), the AHG and the AHHSC can recommend to the OIE Central Bureau and to the International Commission that one or more specific diseases be removed from the list. With this background stated about the functions of the OIE and its listed diseases, the list of diseases (and de-listed diseases) of Crustaceans for 2009-2010 is given in Table 1 (OIE 2009d).

Table 1. OIE listed crustacean diseases for 2009-2010 and those de-listed in 2009 (OIE 2009a; OIE 2010)

Disease Name	Agent	Agent Classification & Type
Taura syndrome (TS)	Taura syndrome virus (TSV)	*Dicistroviridae*; ssRNA
White spot disease (WSD)	White spot syndrome virus (WSSV)	*Nimaviridae*; dsDNA
Yellowhead disease (YHD)	Yellowhead virus (YHV) & gill associated virus (GAV)	*Roniviridae*; ssRNA
Infectious hypodermal & hematopoietic necrosis (IHHN)	IHHN virus (IHHNV)	*Parvoviridae*; ssDNA
Infectious myonecrosis (IMN)[1]	IMN virus (IMNV)	*Totiviridae*; dsRNA
White Tail Disease (WTD)[1]	WTD virus (MrNV)	*Nodaviridae*; ssRNA
Tetrahedral baculovirosis[2]	*Baculovirus penaei* (BP)	*Baculoviridae*; dsDNA
Spherical baculovirosis[2]	Monodon baculovirus (MBV)	*Baculoviridae*; dsDNA
Necrotizing hepatopancreatitis (NHP)[4]	NHP-bacterial (NHP-B)	Alpha proteobacteria
Crayfish plague	*Aphanomyces astaci*	Phycomycete fungus
Milky hemolymph disease (MHD) of spiny lobsters[3]	MHD rickettsial-like bacteria	Rickettsial-like bacteria

[1] Listed by OIE, May 2007; [2] De-listed by OIE in May 2009; [3] Listed as "under study" by OIE; [4] Listed by the OIE in May 2010.

The 2009-2010 list of crustacean diseases listed as notifiable to the OIE consisted of five virus diseases of penaeid shrimp (TS, WSD, YHD, IHHN and IMN), one virus disease of freshwater prawns (WTD) and one fungus disease of freshwater crayfish. Two virus diseases of penaeid shrimp (tetrahedral baculovirosis and spherical baculovirosis) were de-listed by the OIE in May 2009 because control of both diseases

progressed to the point where, for the purposes of international trade, neither disease fully met the listing criteria in Chapter 1.2.2 of the Aquatic Animal Code (OIE 2008a, 2009a). Of the two bacterial diseases (necrotizing hepatopancreatitis – NHP, a bacterial disease of penaeid shrimp and milky hemolymph disease of spiny [*Panulirus* spp.] lobsters) that were listed as "under study" for possible future listing by the OIE, NHP was approved by the OIE for full listing in May 2010 (OIE 2010) (Table 1).

This paper provides a brief review of the current status of the virus diseases due to TSV, WSSV, IHHNV, IMNV and YHV in the Americas in terms of their biology, history, distribution, production impacts and diagnostic methods. There is little new information on baculovirus-caused diseases that is not well covered in other reviews (Lightner 1996a; Bondad-Reantaso et al. 2001; Flegel 2006; OIE 2006b; Walker and Mohan 2009). Hence, not included in this review will be information on the baculovirus caused diseases caused by monodon baculovirus (MBV), *Baculovirus penaei* (BP) and baculoviral midgut gland necrosis (BMN). Also reviewed is some of the current information of the bacterial diseases necrotizing hepatopancreatitis (NHP), milky hemolymph syndrome/disease (MHS or MHD) and Streptococcosis.

Taura Syndrome and Taura Syndrome Virus (TSV)

Biology of the agent

Taura syndrome is caused by Taura syndrome virus (TSV), a small, simple RNA virus. The virion is a 32 nm diameter, nonenveloped icosahedron with a buoyant density of 1.338 $g \cdot mL^{-1}$ (Table 1; Fig. 1). The genome of TSV consists of a linear, positive-sense single-stranded RNA of 10, 205 nucleotides. The TSV genome contains two large open reading frames (ORFs). ORF 1 contains the sequence motifs for nonstructural proteins, such as helicase, protease and RNA-dependent RNA polymerase. ORF 2 contains the sequences for TSV structural proteins, including the three major capsid proteins CP1, CP2 and CP3 (40, 55 and 24 kDa, respectively) (Bonami et al. 1997; Mari et al. 1998; Mari et al. 2002; Robles-Sikisaka et al. 2001). The virus replicates in the cytoplasm of host cells. Based on its characteristics, TSV has been assigned by the International Committee on Taxonomy of Viruses (ICTV) to the newly created genus *Cripavirus* in the new family *Dicistroviridae* (in the "superfamily" of picornaviruses) (Mayo 2002a, 2000b).

Physicochemical and more recent molecular studies of TSV suggest that a single strain of the virus was present in the initial TSV pandemic in the Americas, but that new strains, which differ in host range and virulence, are emerging (Yu and Song 2000; Zarain-Herzberg and Ascencio-Valle 2001; Erickson et al. 2002; Chang et al.

2004; Tang and Lightner 2005; Nielsen et al. 2005; Wertheim et al. 2009). Curiously, the strain of TSV in the early diagnostic cases from the Americas reacts with the monoclonal antibody MAb1A1, but more recently, emerged genetic variants of TSV from the Americas do not react with the antibody in Western blot transfers or in IHC (immuno-histochemistry) preparations with paraffin embedded TSV infected tissues (Erickson et al. 2002; Erickson et al. 2005). MAb1A1 reacts with CP2, which is the most variable of the three TSV capsid proteins. Genotyping of TSV has been based on variations in the sequence of CP2 (Tang and Lightner 2005; Wertheim et al. 2009). Comparison of the cDNA sequence of TSV CP2 from approximately 80 TSV isolates in the author's collection shows that there are currently four distinct strains (or genetic variants) of the virus (Tang and Lightner 2005; Wertheim et al. 2009).

History, hosts and geographic distribution of Taura syndrome (TS)

TSV emerged from an unknown source in Ecuador in 1991. The disease was recognized as a major new disease of farmed *L. vannamei* by early 1992 and it was named 'Taura syndrome' (Jimenez 1992; Lightner 1996a, 2003a, 2003b; Brock et al. 1995, Brock 1997; Lightner et al. 1995). The viral etiology of TS was confirmed in 1994 and the virus was named Taura syndrome virus (TSV) (Hasson et al. 1995). In the interest of supporting litigation brought by a group of Ecuadorian shrimp farmers against several international pesticide companies, whose products had been implicated as the cause of a toxicity syndrome they called 'Taura syndrome', Intriago et al. (1997) and Jimenez et al. (2000) reported on the epizootiology of the disease in Ecuador and suggested that TSV be assigned the synonym 'infectious cuticular epithelial necrosis virus (ICENV)' to distinguish it from the 'Taura syndrome' with a putative toxic etiology (Jimenez et al. 2000).

The principal host for TSV is the Pacific white shrimp, *L. vannamei*, although other species can be infected and present the disease (Aguirre Guzman and Valle 2000; Hasson et al. 1995, 1999a, 1999b; Lightner 1999; Overstreet et al. 1997; Robles-Sikisaka et al. 2001; Srisuvan et al. 2006). Cumulative mortalities due to TSV epizootics have ranged from 40 to >90% in cultured populations of postlarval (PL), juvenile and subadult *L. vannamei*. TS is best known as a disease of nursery- or grow-out-phase in *L. vannamei* that occurs within ~14 to 40 days after stocking postlarvae into grow-out ponds or tanks; hence, shrimp with TS are typically small juveniles of ~0.05 g to <5 g. Larger shrimp may also be affected, especially if they are not exposed to the virus until they are larger juveniles or subadults. Survivors of TSV infections may carry the virus for life (Brock et al. 1995, 1997; Hasson et al. 1999a, 1999b; Lightner 1996a, 1996b; Lotz 1997b). In regions where the virus is enzootic in farmed stocks, the prevalence of chronic phase TSV infections has been found in various

surveys to range from 0 to 100% (Brock 1997; Jimenez et al. 2000; Laramore 1997). TSV can also infect other Western Hemisphere penaeid species (i.e. *L. stylirostris, L. setiferus* and *L. schmitti*), sometimes resulting in disease and mortalities in PL or early juvenile stages, but also in asymptomatic persistent infections (Brock et al. 1997; Overstreet et al. 1997). Other Western Hemisphere penaeids (*Farfantepenaeus aztecus* and *Fa. duorarum*) and Eastern Hemisphere penaeids (*Fenneropenaeus chinensis*, *P. monodon* and *Marsupenaeus japonicus*) have been experimentally infected with TSV, but appear not to develop clinical disease (Brock et al. 1997; Overstreet et al. 1997; Flegel 2006).

Transmission of TSV may be by horizontal or vertical routes. Horizontal transmission by cannibalism, or by contaminated water, has been demonstrated (Brock, 1997; Hasson et al. 1995; Lightner 1996a, 1996b; Lotz 1997b; Overstreet et al. 1997; White et al. 2002). Vertical transmission from infected adult broodstock to their offspring is strongly suspected but has not been experimentally confirmed (Lightner 1996a, 2003a).

By 1994, when the viral etiology of TS had been established, the virus had been moved with live shrimp transfers to many of the shrimp growing countries of the Americas (Brock et al. 1995; Hasson et al. 1995, 1999a; Bonami et al. 1997; Lightner 1996a, 1996b, 2003a). While wild postlarvae with TSV infections were reported found near shrimp farms with ongoing TSV epizootics (Lightner et al. 1995), TSV infections in wild shrimp have not been further documented, suggesting that TSV does not have a discernable impact on wild populations of shrimp (Brock 1997). In 1998, TSV was documented in Taiwan in infected stocks of *L. vannamei*, introduced for aquaculture purposes (Tu et al. 1999; Yu and Song 2000). Within a few years, TSV had been disseminated to most of the shrimp farming countries in SE Asia with trans-boundary movements of TSV infected *L. vannamei* (Phalitakul et al. 2006; OIE 2009b).

In addition to being moved from country to country with live shrimp, there is also evidence that TSV has the potential of being transferred in frozen TSV-infected shrimp products.

TSV has been found in frozen commodity shrimp (*L. vannamei*) products in samples from markets in the USA that originated in Latin America and Southeast Asia. Improper disposal of wastes (liquid and solid, i.e. peeled shells, heads, intestinal tracts, etc.) from value-added reprocessing of TSV-infected shrimp at coastal locations may provide a source of TSV that may contaminate wild or farmed stocks near the point of the waste stream discharge (Lightner 1996b; Nunan et al. 2004; OIE 2009b).

Shrimp eating birds and insects may be important factors in the transmission of TSV within shrimp farms and among shrimp farms within a geographical zone or region (OIE 2009b). TSV has been demonstrated to remain infectious in the feces of sea gulls that have ingested infected shrimp carcasses (Garza et al. 1997; Lightner 1999). The virus was demonstrated to remain infectious for up to 48 h (after ingestion of TSV infected shrimp carcasses) in the feces of wild or captive sea gulls (*Larus atricilla*) and chickens (*Gallus domesticus*, used as a laboratory surrogate for all shrimp-eating birds). These findings implicate birds as being an important mechanical vector for the transmission of the virus within affected farms or farming regions (Vanpatten et al. 2004). There is some evidence that flying aquatic insects, such as the water boatman (*Trichocorixa reticulata* [*Corixidae*]) that feed on shrimp carcasses can also serve as mechanical vectors of TSV (Brock 1997; Lightner et al. 1995, 1996b).

Taura syndrome gross signs in susceptible host species

Gross signs of Taura syndrome have been documented in all life stages (i.e. postlarvae, juveniles and adults) of *L. vannamei* except in eggs, zygotes and larvae (Brock and Main 1994; Lightner 1996a). Following experimental or natural infection, Taura syndrome has three distinct phases: acute, transition and chronic (Brock 1997; Hasson et al. 1999b). In disease outbreaks at farms the onset of mortality is often sudden and massive, with moribund shrimp coming to the pond surface or edges where large numbers of shrimp eating birds may be attracted to the dead and dying shrimp. Moribund shrimp in the acute phase of the disease typically present a pink to reddish coloration due to expansion of red cuticular chromatophores (especially in the tail fan), are off feed and with empty guts and they are lethargic. The acute phase is rapid in individual shrimp, probably lasting less than 24 h, but the acute phase in a shrimp farm pond may last for several days in an affected population (Brock 1997; Garza et al. 1997; Hasson et al. 1999b). As implied by its name, the transition (or recovery in some publications) phase of Taura syndrome is that phase of the disease when affected shrimp may resolve the lesions due to TSV infection that developed in the acute phase. Shrimp in the transition phase typically present randomly distributed variably sized melanized lesions in or under the cuticle (exoskeleton). Those shrimp that successfully resolve the acute phase TSV lesions and survive the next molting process typically appear normal. Death due to TSV infection during the three phases most often occurs in the acute phase, probably due to osmotic failure. In the transition phase of the disease, death may also occur due to osmotic failure as a consequence of widespread destruction of the cuticular epithelium and two localized or systemic infections by opportunistic bacteria (Lightner 1996a; Brock 1997). Shrimp in the chronic phase of Taura syndrome may carry the virus for life as a persistent infection (Lotz 1997b; Hasson et al. 1999b). While persistently infected *L. vannamei* may appear and behave

normally, they show slightly less tolerance to low salinity stress than uninfected shrimp (Lotz et al. 2005). Some members of populations of *L. vannamei* or *L. stylirostris* that survive TSV infections and/or epizootics may carry the virus for life and, although not documented, pass the virus on to their progeny by vertical transmission (Hasson et al. 1999a, 1999b; OIE 2009b).

Diagnostic methods

Available methods for diagnosing infection by TSV include routine histology, in situ hybridization (ISH) with specific cDNA probes, antibody-based methods and cDNA amplification methods using standard and real-time PCR after a reverse transcription step (RT-PCR) to convert the viral ssRNA genome to cDNA (OIE 2009b). Of the available methods, RT-PCR is recommended in the OIE Manual of Diagnostic Tests for Aquatic Animals ("Aquatic Manual"; OIE 2009b) for disease surveillance and screening purposes. For TSV, disease surveillance/screening information can be applied to determine presence, absence or prevalence of infection in wild or cultured populations, in commodity products made from susceptible hosts and to support declarations of disease freedom (OIE 2009a, 2009b).

White Spot Disease and White Spot Syndrome Virus (WSSV)

Biology of the agent

The causative agent of white spot disease (WSD) is white spot syndrome virus (WSSV). WSSV is a very large, enveloped, double-stranded DNA (dsDNA) virus with a density of approximately 1.2 $g \cdot mL^{-1}$. WSSV was recently assigned by the ICTV to its own new genus, *Whispovirus* and family, *Nimaviridae* (Table 1; Fig. 1) (Mayo 2002a, 2002b). Virions are large (80-120 x 250-380 nm), rod-shaped to elliptical and with a trilaminar envelope (Wang et al. 1995; Durand et al. 1997; Inouye et al. 1994, 1996; Kanchanaphum et al. 1998; Nadala et al. 1998; van Hulten et al. 2001; Vlak et al. 2005; Greenwood et al. 2005). Negatively stained virions purified from shrimp hemolymph show unique, tail-like appendages (Wang et al. 1995; Fauquet et al. 2005). The virions are generated in hypertrophied nuclei of infected cells without the production of occlusion bodies (Lo et al. 1997). In initial reports, WSSV was described as a non-occluded baculovirus, but WSSV DNA sequence analysis has shown that it is not related to the baculoviruses (van Hulten et al. 2001; Yang et al. 2001). The size of the WSSV genome has been differently reported for different isolates: 305,107 bp (GenBank Accession No. AF332093), 292,967 bp (GenBank Accession No. AF369029) and 307,287 bp (GenBank Accession No. AF440570) for viruses isolated from the People's Republic of China, Thailand and Taipei, China, respectively. The

sequences of these three isolates are almost identical, with the size differences being due mostly to several small insertions and one large (~12 kbp) deletion. In accordance with a genome size of ~300 kb, a total of 531 putative open reading frames (ORFs) were identified by sequence analysis, among which 181 ORFs are likely to encode functional proteins. Thirty-six of these 181 ORFs have been identified by screening and sequencing a WSSV cDNA library, or have already been reported to encode functional proteins, many of which show little homology to proteins from other viruses (OIE 2009b).

Temperature was found to have a profound effect on the expression of disease in WSSV infected *L. vannamei* (Vidal et al. 2001; Granja et al. 2003). These authors found that at temperatures above 32 °C, WSD did not develop in WSSV infected *L. vannamei*. However, when the same shrimp were cooled to 25 °C, the disease would quickly develop with 100% mortality. Subsequent studies demonstrated that the hyperthermic phenomenon also occurred in other penaeids (Guan et al. 2003). Recent work has shown that replication of WSSV is significantly reduced or stopped under hyperthermic conditions (Du et al. 2006). These findings have helped to explain why WSD epizootics occur most often in the cooler seasons in most shrimp farming regions. In the Americas, that information has helped shrimp farmers manage WSD by avoiding stocking in the cool season and in some countries like Ecuador and Peru, growing shrimp year-round in temperature controlled greenhouses.

History and geographic distribution of white spot disease

WSSV has a wide host range among decapod crustaceans (Lo et al. 1996; Lo and Kou 1998; Flegel 1997; Flegel and Alday-Sanz 1998) and is potentially lethal to most of the commercially cultivated penaeid shrimp species (OIE 2009b). WSD, caused by WSSV, emerged in East Asia in 1992-93 and was quickly dispersed with infected seed and broodstock across the Asian continent to Southeast Asia and India where it caused a major pandemic and continues to cause significant losses in some regions. In Japan, WSD outbreaks were first reported from farmed *Marsupenaeus japonicus* in 1993 (Inouye et al. 1994, 1996; Nakano et al. 1994) and the causative agent was named penaeid rod-shaped DNA virus (PRDV) or rod-shaped nuclear virus of *M. japonicus* (RV-PJ). Later, outbreaks of viral disease with similar gross signs caused by similar rod-shaped viruses were reported from elsewhere in Asia and other names were applied: hypodermal and hematopoietic necrosis baculovirus (HHNBV) in the People's Republic of China (Huang et al. 1995a, 1995b); white spot baculovirus (WSBV) and PmNOBIII in Taipei China (Chou et al. 1995, Lo et al. 1996); and systemic ectodermal and mesodermal baculovirus (SEMBV) or PmNOBII in Thailand (Wongteerasupaya et al. 1995). The virus from the People's Republic of China has also been called Chinese

baculovirus (CBV) (Lu et al. 1997). Shrimp exhibiting the gross signs and histopathology of WSD have also been reported from Korea (Kim et al. 1998), India (Karunasagar et al. 1998), the Philippines and the USA (Lightner 1996a, 1996b; Durand et al. 2000). WSSV has even reached shrimp farms in southeastern Europe (1997) and the Middle East (1999) via live shrimp movements and Australia and Spain with introductions of frozen infected shrimp, which were used as fresh food for broodstock (OIE 2006b; Stentiford and Lightner "in press").

Beginning in 1999, WSD had a severe impact on the shrimp farming industries of both Central and South America (GAA 1999a, 1999b; Durand et al. 2000; Vidal et al. 2001; Lightner 2003a, 2003b; OIE 2009b). Despite the absence of evidence of live shrimp introductions from Asia to the Americas, WSSV was diagnosed at several sites between 1995-1997 in captive wild shrimp and crayfish and in cultured domesticated shrimp stocks in the eastern and southeastern U.S. (Nunan et al. 1998b; Durand et al. 2000; Lightner et al. 2001). Early in 1999, WSSV was diagnosed as the cause of serious epizootics in Central American shrimp farms. By mid to late 1999, WSSV was causing major losses in Ecuador (then among the world's top producers of farmed shrimp) and by 2000-2001, export of shrimp from Ecuador was down nearly 70% from pre-WSSV levels (Rosenberry 2001, 2003; Lightner 2003a). Although the documentation is sketchy, WSSV has been found in wild shrimp stocks in the Americas (Nunan et al. 2001; Chapman et al. 2004). In the US, the virus was successfully eradicated from shrimp farms and except for two confirmed outbreaks at an isolated shrimp farm on the Island of Kauai, Hawaii in 2004 and 2008 (CEI 2004; Ostrowski 2004; USMSFP 2008), WSSV has not been reported from farmed shrimp stocks grown in other regions of the USA since 1997. However, its sporadic detection in wild shrimp stocks (Pacific Coast of Panama, Gulf of Mexico and SE Atlantic states) (Nunan et al. 2001; Chapman et al. 2004; Hasson et al. 2006) suggests that WSSV has become established in wild penaeid shrimp stocks in coastal waters of the eastern Pacific and southeastern U.S. and the Gulf of Mexico, or that it continues to be introduced. Introduction is possibly from wastes (peeled shells, etc.) from value-added reprocessing of imported shrimp in coastal packing plants, or from infected shrimp used as bait by sport fishermen. It has been proposed that the introductions of WSSV to the Americas were the result of importation of frozen shrimp products from WSSV-affected areas of Asia and the value-added reprocessing of those frozen shrimp for the US market in coastal processing plants (Nunan et al. 1998b; Durand et al. 2000; Lightner et al. 2001; Lightner 2003a), or are possibly due to the use of imported frozen WSSV-infected shrimp as bait by sport fishermen (Hasson et al. 2006). WSSV also reached Spain and Australia in 2000-2001. In both cases, successful containment and eradication were reported and for both events the importation and use of infected frozen shrimp as a fresh feed for broodstock were implicated as the route of introduction (OIE 2003; Lightner 2003a). Regardless of

where they were obtained, isolates of WSSV have shown little genetic or biological variation, suggesting that the virus emerged and was spread from a single source (OIE 2003, 2006b, 2009b).

WSD gross signs and histopathology in Litopenaeus vannamei

The gross signs, histopathology and diagnostic procedures (antibody-based and molecular) for WSSV infections have been thoroughly reviewed since the disease emerged (Lightner 1996a; Flegel 1997, 2006; Lightner and Redman 1998a, 1998b; Lightner 1999; Loh et al. 1997; Lo and Kou 1998; Greenwood et al. 2005; OIE 2006b, 2009b). The reader is referred to these reviews for additional details on the disease in Asia and elsewhere that are not included in the present review.

Litopanaeus vannamei acutely affected with WSD are reported to show a rapid reduction in food consumption, become lethargic and have a loose cuticle with some showing characteristic white spots 0.5 to 2.0 mm in diameter, which are most apparent on the inside surface of the carapace but may be present anywhere on the inner surface of the exoskeleton. The white spots represent abnormal deposits of calcium salts by the WSSV-infected cuticular epithelium. In many cases, moribund shrimp with WSD display a pink to reddish-brown coloration, due to expansion of the cuticular chromatophores and few, if any, white spots. Populations of shrimp showing these signs display high mortality rates with cumulative mortalities reaching 100% within 3 to 10 days of the onset of clinical signs (Lightner 1996a; OIE 2006b, 2009b).

Diagnostic methods

Available methods for diagnosing infection by WSSV include routine histology, in situ hybridization (ISH) with specific DNA probes, antibody-based methods and DNA amplification methods using standard PCR and real-time PCR (OIE 2009b). Of the available methods, a nested PCR is recommended in the OIE Manual of Diagnostic Tests for Aquatic Animals (“Aquatic Manual”; OIE 2009b) for disease surveillance and screening purposes. For WSSV, disease surveillance/screening information can be applied to determining presence, absence or prevalence of infection in wild or cultured populations, in commodity products made from susceptible hosts and to support declarations of disease freedom (OIE 2009a, 2009b).

Infectious Hypodermal and Hematopoietic Necrosis and IHHNVirus

Biology of the agent

Infectious hypodermal and hematopoietic necrosis disease (IHHN) is caused by IHHN virus, which is the smallest of the known penaeid shrimp viruses. The IHHN virion is a 22 nm diameter, nonenveloped icosahedron (Table 1; Fig. 1) with a density of 1.40 $g \cdot mL^{-1}$ in CsCl. Its genome is linear single-stranded DNA of 4.1 kb in length. Because of its characteristics, IHHNV has been classified as a member of the *Parvoviridae* and a probable member genus *Brevidensovirus* (Bonami et al. 1990; Bonami and Lightner 1991; Mari et al. 1993; Nunan et al. 2000; Shike et al. 2000).

Hosts, history and geographic distribution of IHHN disease

The disease IHHN and later its causative agent, IHHNV, was first described as the cause of acute epizootics and mass mortalities (> 90%) in juvenile and subadult *L. stylirostris* farmed in super-intensive raceway systems in Hawaii (Brock et al. 1983; Lightner 1983, 1988; Lightner et al. 1983a, 1983b; Brock and Lightner 1990). Shortly after its discovery in *L. stylirostris*, the virus was found in *L. vannamei* being cultured at the same facility in Hawaii and these *L. vannamei* were also shown to be generally asymptomatic carriers of the virus (Lightner et al. 1983b; Bell and Lightner 1984). Some members of populations of *L. stylirostris* and *L. vannamei* that survive IHHNV infections and/or epizootics may carry the virus for life and pass the virus on to their progeny and other populations by horizontal and vertical transmission (Bell and Lightner 1984; Lightner 1996a; Morales-Covarrubias et al. 1999, Morales-Covarrubias and Chavez-Sanchez 1999; Motte et al. 2003). IHHNV has been demonstrated by PCR (Motte et al. 2003) and by in situ hybridization with IHHNV specific probes to be vertically transmitted in the oocytes (Lightner 2011).

A few years after it was reported that *L. vannamei* can be infected with IHHNV and not cause significant mortalities (Lightner et al. 1983b; Bell and Lightner 1984), IHHNV was shown to be the cause of 'runt deformity syndrome' (RDS) in *L. vannamei* (Kalagayan et al. 1991). With RDS, affected shrimp present irregular, reduced growth, cuticular deformities and generally no remarkable elevation in mortality (Kalagayan et al. 1991; Browdy et al. 1993; Bray et al. 1994; Brock and Main 1994; Lightner 1996a). Hence, the economic and production impacts of IHHNV infection in *L. vannamei* are due to reduced and irregular growth and small count size shrimp at harvest, not to elevated mortality (OIE 2006b, 2009b; Lightner 2011). To mitigate this effect, several strategies have been used. With one strategy, selected lines of *L. stylirostris* were

developed that were not only resistant to IHHN disease but also refractory to infection (Tang et al. 2000; Dhar et al. 2001). IHHNV-free lines of *L. vannamei* were also developed as SPF (specific pathogen-free) lines and these stocks were the first developed in the SPF stock development program (Wyban et al. 1992; Pruder et al. 1995; Moss et al. 2002; Lightner et al. 2009a; Moss and Moss 2009).

After its initial discovery in cultured shrimp in Hawaii in 1981, IHHNV was subsequently found to be widely distributed in cultured shrimp in the Americas and in wild shrimp collected along the Pacific coast from Peru to Mexico. As of 2006, the only country in the Americas, which can claim to have IHHNV-free zones or compartments (OIE 2009a; Lightner et al. 2009a) is the United States. This was achieved with the development and use of SPF shrimp stocks (Pruder et al. 1995; Lightner et al. 2009a; Moss and Moss 2009). The introduction of IHHNV into shrimp farms in northwestern Mexico and wild shrimp stocks in Mexico's Gulf of California during the late 1980's and early 1990's resulted not only in significant losses in farmed *L. stylirostris*, but also contributed to the collapse of the northern Gulf of California'swild fishery for *L. stylirostris* that began in 1990 (Lightner et al. 1992a; Martinez-Cordova 1992; Lightner 1996b; Pantoja et al. 1999; Morales-Covarrubias and Chavez-Sanchez 1999; Morales-Covarrubias et al. 1999; Walker and Mohan 2009). A decade later, the *L. stylirostris* fishery of the northern Gulf of California had recovered sufficiently to support commercial fishing, but the prevalence of IHHNV infection in adult *L. stylirostris* collected from the northern Gulf fishery has remained high (80% to 100% females and 60% in males)(Morales-Covarrubias et al. 1999; Morales-Covarrubias and Chavez-Sanchez 1999). *L. stylirostris* collected from the Gulf remain unsuitable for aquaculture because they carry IHHNV and consequently, these stocks do not survive well in culture tanks or ponds.

IHHNV has been found to be widely distributed in wild and cultured *P.monodon* in East and Southeast Asia where it does not seem to cause production losses (Flegel 1997, 2006; Primavera and Quinitio 2000; Tang et al. 2003; Chayanburakul et al. 2005; Withyachumnarnkul et al. 2006). Molecular studies show considerable variation among Asian isolates of the virus (Tang et al. 2003; Krabsetsve et al. 2004; Tang and Lightner 2006), while little variation was found in isolates from the Americas (Tang and Lightner 2002). All isolates of IHHNV from the Americas are nearly identical with IHHNV from the Philippines. This finding, along with other aspects of its history and epidemiology of IHHN in the Americas, suggests that IHHNV was introduced from the Philippines, perhaps with live *P. monodon* that were imported in the early 1970's as a candidate aquaculture species during the very early development of shrimp farming in the Americas (Lightner 1996b; Tang and Lightner 2002; Tang et al. 2003).

In addition to the Americas/Philippines genotype of IHHNV, three other genetic variants of the virus have been documented. For the purposes of this review, the IHHNV genotype from the Americas/Philippines genotype will be designated as IHHNV-I; the variant from Southeast Asia will be designated IHHNV-II; and the IHHNV variant from East Africa, Madagascar and Mauritius and Australia will be referred to as IHHNV-III. The first two genotypes (IHHNV-I and IHHNV-II) are infectious to the representative penaeids, *L. vannamei* and *P. monodon*, while the latter genetic variant has been demonstrated to be not infectious to these species (Tang et al. 2003; Krabsetsve et al. 2004; Tang and Lightner 2006). The apparent reason for the lack of infectivity of the IHHNV-III genotype was recently explained by the discovery that the DNA fragment represented by IHHNV-III is incorporated into the genome of some genetically distinct populations of *P. monodon* in the Indo-Pacific region (Duda and Palumbi 1999; Tang and Lightner 2006; Tang et al. 2007).

IHHN gross signs in L. stylirostris

IHHNV often causes an acute disease with very high mortalities in juveniles of this species. Vertically infected larvae and early PL do not become diseased, but in approximately 35-day-old or older juveniles, gross signs of the disease may be observed, followed by mass mortalities. In horizontally infected juveniles, the incubation period and severity of the disease is somewhat size and/or age dependent, with young juveniles always being the most severely affected. Infected adults seldom show signs of the disease or mortalities (Bell and Lightner 1984, 1987; Brock and Lightner 1990; Lightner 1996a). Gross signs are not IHHN specific, but juvenile *L. stylirostris* with acute IHHN show a marked reduction in food consumption, followed by changes in behavior and appearance. Juvenile shrimp at this stage of infection often have white or buff-colored spots (which differ in appearance and location from the white spots that sometimes occur in shrimp with WSSV infections) in the cuticular epidermis, especially at the junction of the tergal plates of the abdomen, giving such shrimp a mottled appearance. This mottling later fades in moribund *L. stylirostris* as such individuals become more bluish. In *L. stylirostris* and in *P. monodon* with terminal phase IHHNV infections, moribund shrimp are often distinctly bluish in color, with opaque abdominal musculature (Lightner et al. 1983; Brock and Lightner 1990; Lightner 1996a).

IHHN gross signs in L. vannamei

The chronic disease, RDS, occurs in this species as a result of IHHNV infection. The severity and prevalence of RDS in infected populations of juvenile or older *L. vannamei* may be related to infection during the larval or early PL stages (Kalagayan et al. 1991; Motte et al. 2003). RDS has also been reported in cultured

stocks of *L. stylirostris* and *P. monodon* (Lightner 1996a; Primavera and Quinitio 2000). Juvenile shrimp with RDS may display a bent (45° to 90° bend to left or right) or otherwise deformed rostrum, a deformed 6th abdominal segment, wrinkled antennal flagella, cuticular roughness, 'bubble-heads' and other cuticular deformities. Populations of juvenile shrimp with RDS display disparate growth with a wide distribution of sizes and many smaller than expected ('runted') shrimp. The coefficient of variation (CV = the standard deviation divided by the mean of different size groups within a population) for populations with RDS is typically greater than 30% and may approach 90%, while IHHNV-free (and thus RDS-free) populations of juvenile *L. vannamei* and *L. stylirostris* usually show CVs of 10–30% (Kalagayan et al. 1991; Browdy et al. 1993; Brock and Main 1994; Carr et al. 1996; Lightner 1996a; Motte et al. 2003).

Diagnostic methods

The available methods for diagnosing infection by IHHNV include routine histology, in situ hybridization (ISH) with specific DNA probes and DNA amplification methods using standard PCR and real-time PCR (Nunan et al. 2000; Tang and Lightner 2001; OIE 2009b). Of the available methods, PCR is recommended in the OIE Manual of Diagnostic Tests for Aquatic Animals ("Aquatic Manual"; OIE 2009b) for disease surveillance and screening purposes. For IHHNV, disease surveillance/screening information can be applied to determining presence, absence or prevalence of infection in wild or cultured populations, in commodity products made from susceptible hosts and to support declarations of disease freedom (OIE 2009a, 2009b).

IHHN disease was recently found to be unique among the shrimp virus diseases when IHHNV related sequences were found in the genome of some stocks of *P. monodon* from the Indian Ocean (Tang and Lightner 2006). ISH and qPCR (real-time) studies of PCR positive *P. monodon* with this genome integrated form of IHHNV showed no signs of virus replication in the normal target tissues for the virus. Further, shrimp to shrimp transmission studies showed no evidence of this IHHN Type III agent being transmissible. In order to distinguish infectious forms of IHHNV (Types I and II) from the genome integrated form of IHHNV (Types III) in *P. monodon*, a PCR method was developed that produces a PCR product that bridges and hence includes, portions of the IHHNV sequence and a segment of the adjacent *P. monodon* genome (Tang et al. 2007; OIE 2009b). The lessons learned from applying PCR detection methods to IHHNV and the resulting false positive tests for the virus in some stocks of *P. monodon* underscore the importance of confirming unexpected positive and/or negative PCR results for IHHNV with a second primer set, or by the use of another diagnostic method (i.e. real time PCR, bioassay, ISH).

Yellow Head Disease (YHD) and Yellow Head Virus (YHV)

Biology of the agent

The causative agent of yellow head disease (YHD) is yellow head virus (YHV). Six closely related strains of YHV, including gill associated virus (GAV) have been reported which vary greatly in their ability to cause disease (Table 1) (OIE 2006b, 2009b; de la Rosa-Vélez et al. 2006; Cedano-Thomas et al. 2009). YHV is an enveloped, rod-shaped, single stranded RNA virus in the family *Roniviridae* of the order Nidovirales. The density of virions is approximately 1.20 $g \cdot mL^{-1}$ (OIE 2009b; Walker and Mohan 2009). Transmission electron microscopy (TEM) of YHV-infected tissues shows enveloped bacilliform virions. The virions range from approximately 150 nm to 200 nm in length and from 40 nm to 50 nm in diameter and are located within vesicles in the cytoplasm of infected cells and in intercellular spaces. The virions arise from longer, filamentous nucleocapsids (approximately 15 nm x 130-800 nm), which accumulate in the cytoplasm and obtain an envelope by budding at the endoplasmic reticulum into intracellular vesicles. Negatively stained YHV virions show regular arrays of short spikes on the viral envelope (Boonyaratpalin et al. 1993; Chantanachookin et al. 1993; Lightner 1996a).

YHV was originally described mistakenly as a granulosis-like virus (Boonyaratpalin et al. 1993; Chantanachookin et al. 1993), but it was later found to be a single-stranded, positive sense RNA (ssRNA) virus (Tang and Lightner 1999) related to nidoviruses in the Coronaviridae and Arteriviridae (Sittidilokratna et al. 2002). GAV, the Australian strain of YHV, has been recognized as the type species for the new virus genus *Okavirus* in the new family *Roniviridae* (Mayo 2002a, 2002b; OIE 2003, 2009b).

History and geographic distribution of yellow head disease:

Yellow head disease (YHD) was first described in 1991 as an epizootic in Thai shrimp farms (Limuswan 1991) and subsequent outbreaks have been reported from other shrimp farming countries in Asia (OIE 2003, 2009b). A closely related strain of YHV, named gill associated virus (GAV), has been reported from Australian shrimp farms (Walker et al. 2001) and at least six genetic variant of the virus are now recognized (OIE 2006b; OIE 2009b). Laboratory trials have shown that YHV can cause high mortality in representative cultured and wild penaeid species from the Americas (Lu et al. 1994, 1997; Lightner 1999; Pantoja and Lightner 2003). YHD in *P. monodon* is characterized by high and rapid mortality that is sometimes accompanied by the gross signs of yellowing of the cephalothorax and general bleaching of body color from which the disease got its name. Although in laboratory studies American

penaeids challenged with YHV did not develop yellow heads or signs of marked discoloration (Lightner and Redman 1998a, 1998b), infection by YHV is potentially lethal to most of the commercially cultivated penaeid shrimp species (OIE 2009a, 2009b).

YHD in the Americas?

While there are no confirmed reports of YHD outbreaks in the Americas, YHV has been reported from frozen imported commodity shrimp in the United States (Nunan et al. 1998b; Durand et al. 2000) and in some early reports it was incorrectly reported in farmed shrimp from the Americas. The errant reports were based on the presentation of severe necrosis of the lymphoid organ, a lesion once thought to be pathognomonic for YHD (Limuswan 1991; Lightner 1996a; Lightner and Redman 1998a; Pantoja and Lightner 2003). However, the diagnosis of YHV infection in these cases was not confirmed with a second diagnostic method until after the errant reports were published. More recent work has shown that the presumptive histological diagnoses were due to severe infections with white spot virus, which can cause histopathology in the lymphoid organ to mimic that occurring in severe acute YHD (Pantoja and Lightner 2003).

More recently, several well documented studies have documented that that an apparently avirulent genotype of YHV is present in farmed and wild penaeid shrimp in northwest Mexico (de la Rosa-Veléz et al. 2006; Castro-Longoria et al. 2008; Cedano-Thomas et al. 2009; Sánchez-Barajas et al. 2009). In the first of these reports, de la Rosa-Veléz et al. (2006) reported the discovery of suspect cases of YHV infections from shrimp farms along the Pacific coast of Mexico. From 39 samples from 26 randomly chosen shrimp farms, 11 YHV positive samples of *L. vannamei* were found by RT-PCR using primers specific for the virus (Tang and Lightner 1999). Further analysis of selected isolates using primers that amplify other regions of the YHV genome also gave positive results (de la Rosa-Veléz et al. 2006). Of considerable interest in the de la Rosa-Veléz et al. (2006) report was the general absence of notable mortalities in farms with the YHV positive shrimp. However, a laboratory challenge study with the putative YHV agent resulted in 50% mortality in 14 days in the indicator *L. vannamei* used to assay for the presence of YHV (de la Rosa-Veléz et al. 2006).

In subsequent studies on YHV in Mexico, Castro-Longoria (2008) surveyed wild blue shrimp, *Litopenaeus stylirostris* and *L. vannamei* collected from the Gulf of California near areas with significant shrimp farms. The samples of wild *L. vannamei* were negative for YHV, but a few YHV positive specimens were found in the wild *L. stylirostris*. YHV was confirmed to be present by bioassay with healthy *L. vannamei*. The presence of YHV in the challenged shrimp was confirmed by RT-PCR, by

sequencing the amplicons and by sequence alignment with YHV and GAV sequences in GenBank.

Another recent study demonstrated the presence of YHV in inland freshwater aquaculture systems growing *L. vannamei* in Colima in west-central Mexico (Sánchez-Barajas et al. 2009). In this study the prevalence of YHV in the affected farms was 13%, but as was reported in earlier reports of YHV in Mexico, significant mortalities were not observed in the YHV infected shrimp stocks.

In another study run to further confirm that the agent found in affected farms in Mexico was YHV, Cedano-Thomas et al. (2009) has amplified replicative and structural protein encoding regions of the several Mexican YHV isolates and compared the sequences obtained with homologous virus sequences from YHV, GAV and other coronaviruses. The authors found that the Mexican YHV isolates differed slightly from YHV and GAV, but nonetheless were closely related to Asian YHV and GAV (Cedano-Thomas et al. 2009).

The finding of a strain of YHV in Mexico (de la Rosa-Veléz et al. 2006; Castro-Longoria et al. 2008; Cedano-Thomas et al. 2009; Sánchez-Barajas et al. 2009), reflects the ongoing risk of additional introductions of YHV into the Western Hemisphere with imported frozen commodity shrimp from Asia (Nunan et al. 1998a, Durand et al. 2000). In addition, because of the possibility that concurrent WSSV/YHV infections may occur, all YHV suspect samples should be further analyzed by another method (i.e. RT-PCR or ISH with a YHV specific probe) to confirm or rule out the presence of YHV.

Of interest is the observation that YHD has not emerged as a major disease in cultured stocks of *L. vannamei* in East and Southeast Asia where YHV is enzootic and highly prevalent in wild and farmed stocks of *P. monodon* (OIE 2009b; Flegel 2006). According to FAO statistics, the shrimp farming industry in Asia began to switch from culturing *P. monodon* to *L. vannamei* in 1999 and by 2005, more than half of the ~2 million metric tons of production from the region was *L. vannamei* (FAO 2006).Despite the predominance of monocultures of *L. vannamei*, a presumably highly susceptible species to YHV, Flegel (2006) did not report the occurrence of any significant outbreaks of yellow head disease in this species in the Southeast Asian region.

Diagnostic methods

The available methods for diagnosing infection by YHV include routine histology, in situ hybridization (ISH) with specific cDNA probes and DNA amplification after reverse transcription using standard RT-PCR and real-time RT-PCR

(OIE 2009b). Of the available methods, a RT-PCR is recommended in the OIE Aquatic Manual (OIE 2009b) for disease surveillance and screening purposes. For YHV, disease surveillance/screening information can be applied to determining presence, absence, or prevalence of infection in wild or cultured populations, in commodity products made from susceptible hosts and to support declarations of disease freedom (OIE 2009a, 2009b).

Infectious Myonecrosis Disease (IMN) and IMN Virus (IMNV)

Biology of the agent

Infectious myonecrosis disease (IMN) is caused by infectious myonecrosis virus (IMNV). IMNV particles are icosahedral in shape and 40 nm in diameter, with a buoyant density of 1.366 $g \cdot mL^{-1}$ in CsCl (Fig. 1). The genome consists of a single, double-stranded (dsRNA) molecule of 7560 bp. Sequencing of the viral genome reveals two non-overlapping open reading frames (ORFs). ORF 1 encodes a RNA-binding protein and a capsid protein. The coding region of the RNA-binding protein is located in the first half of ORF 1 and contains a dsRNA-binding motif. The second half of ORF 1 encodes a capsid protein with a molecular mass of 106 kDa. ORF 2 encodes a RNA-dependent RNA polymerase (RdRp). Based on these characteristics, IMNV is most similar to members of the *Totiviridae* (Poulos et al. 2006).

History and geographic distribution of IMN disease

Infectious myonecrosis was listed by OIE in 2007 (Table 1) (OIE 2006a). The disease was first described in cultured *L. vannamei* in Northeast Brazil (Lightner 2003a; Lightner et al. 2004). IMN causes significant disease and mortalities in juvenile and subadult pond-reared stocks of *L. vannamei*. In 2003, IMN was reported to have been responsible for millions of dollars in losses in Northeast Brazil and by 2004 losses due to IMN in the affected regions of Brazil were estimated at $20 million (Nunes et al. 2004). More recent estimates for IMN losses from 2002 to 2006 in Brazil exceed $100 million (Brazilian Shrimp Farmers Association, unpublished). In Brazil, outbreaks of the disease seemed to be associated with certain types of environment and physical stresses (i.e. extremes in salinity and temperature, collection by cast net, etc.) and possibly with the use of low quality feeds (Lightner 2003a).

Although IMN seemed confined to the Northeast of Brazil, the disease spread to Southeast Asia and was reported from Indonesia in May 2006 (Wilkinson 2006). Because of the ever increasing importance of *L. vannamei* in the Asia-Pacific and the large scale trans-boundary movement and culture of the species, IMNV was considered

important for the region and it was added in January 2006 to the NACA/FAO/OIE (Asian Region) Quarterly Aquatic Animal Disease Report list for the purpose of surveillance and reporting.

Gross signs of IMNV

IMN presents as a disease in *L. vannamei* with an acute onset of gross signs and elevated mortalities, but it progresses with a more chronic course accompanied by persistent moderate mortalities. Affected shrimp present focal to extensive white necrotic areas in the striated muscle, especially of the distal abdominal segments and tail fan. These may become necrotic and reddened in some individual shrimp. These signs may have a sudden onset following stresses (e.g. capture by cast-net, feeding, sudden changes in temperature or salinity, etc.). Severely affected moribund shrimp may have been feeding just before the onset of stress and will have a full gut. Such severely affected shrimp become moribund and mortalities can be instantaneously high and continue for several days. Exposure of the paired lymphoid organs (LO) by simple dissection will show that the paired LO are hypertrophic to twice or more its normal size (Poulos et al. 2006; OIE 2009b).

Diagnostic methods

The available methods for diagnosing infection by IMNV include routine histology, in situ hybridization (ISH) with specific cDNA probes and DNA amplification after reverse transcription using standard RT-PCR and real-time RT-PCR (Tang et al. 2005; Poulos et al. 2006; Poulos and Lightner 2006; Andrade et al. 2007; OIE 2009b). Of the available methods, a RT-PCR is recommended in the OIE Aquatic Manual (OIE 2009b) for disease surveillance and screening purposes. For IMNV, disease surveillance/screening information can be applied to determining presence, absence, or prevalence of infection in wild or cultured populations, in commodity products made from susceptible hosts and to support declarations of disease freedom (OIE 2009a, 2009b).

Necrotizing Hepatopancreatitis (NHP)

Biology of the agent and gross signs of the disease

NHP disease is caused by an as yet uncharacterized, obligate intracellular rickettsial-like bacteria, which for the purpose of this review, will be referred to as NHP-B (Loy et al. 1996a, 1996b). The NHP organism is classified as an α-Proteobacterium based on sequence of analysis of the cloned 16S rDNA. Phylogenetic

analyses also indicate this bacterium is closely related to bacterial endosymbionts of the protozoa, *Caedibacter caryophila* and *Holospora obtusa*. NHP-B is a gram-negative, dimorphic, intracellular rickettsial-like organism that occurs free within the cytoplasm of infected hepatopancreatic cells. The predominant form is a rod-shaped rickettsial-like organism (0.25 x 0.9 µm), whereas the helical form (0.25x 2-3.5µm) possesses eight flagella at the basal apex. The target tissue is the hepatopancreas with NHP-B infection reported in all hepatopancreatic cell types (Krol et al. 1991; Frelier et al. 1992; Lightner et al. 1992b; Lightner and Redman 1994; Lightner 1996a). NHP-B is transmitted by the horizontal route via contaminated water (shed in feces) and/or per os (cannibalism) (Frelier et al. 1993; Vincent et al. 2004; Vincent and Lotz 2005). An intermediate vector/reservoir is suspected, but not demonstrated (OIE 2006c).

History and geographic distribution of NHP disease

NHP disease was first reported from Texas (Johnson 1990; Krol et al. 1991; Frelier et al. 1992), but it was subsequently found in most Western Hemisphere countries where penaeid shrimp are farmed (Lightner 1996a; OIE 2006c). The disease has been reported in the late postlarval, juvenile and adult stages of various penaeid species (including *L. vannamei, L. stylirostris, L. setiferus, Farfantepenaeus aztecus* and *F. californiensus*). NHP disease tends to occur during the dry season when both water temperature and salinity are elevated. Lengthy periods of elevated water temperature (29-35 °C) and salinity (20-40%) are associated with overt clinical disease (Lightner 1996a; OIE 2006c). Countries with regions having seasonal dry periods when both salinity and water temperature are high and where NHP disease has been documented include the United States (Texas), Mexico (Sonora and Sinaloa), Panama, Belize, Guatemala, Columbia, Ecuador, Nicaragua, Costa Rica, Brazil, Peru, Venezuela (OIE 2006c) and Eritrea (Lightner, unpublished data).

Despite the introduction of *L. vannamei* from the Americas to Southeast Asia and the consequent development of that species as the dominant shrimp species farmed there (FAO 2006), NHP disease has not emerged as a significant disease in the region. Other than an unconfirmed, but official, report from Vietnam (AGDAFF-NACA 2007), NHP has not been reported from Southeast Asian farms where *L. vannamei* is cultured. Perhaps, the necessary environmental conditions (e.g. an extended dry season in which temperature and salinity are elevated (>30 ºC and >30%) for NHP infections to produce disease are not present in Southeast Asia. However, such conditions are more typical in East Africa, Madagascar, the Middle East and West India, where *L. vannamei* is being introduced.

An outbreak of NHP occurred in the East African nation of Eritrea in 2004 in an introduced stock of *L. vannamei* imported from Mexico, which was co-infected with TSV (Wertheim et al. 2009). In Eritrea, the environmental conditions of high water temperature and high salinity were ideal for NHP disease to be expressed and it (with TSV) contributed to high mortalities in the *L. vannamei* and *P. monodon* stocks being cultured at the importing facility (Lightner, unpublished data). Because of the potential for NHP to be introduced with NHP-B infected stocks of *L. vannamei* into the semiarid regions bordering the Western Indian Ocean, the disease is listed as "under study" by the OIE in 2009 and it was fully listed by the OIE in May 2010 (Table 1; OIE 2008a, 2009a).

Gross signs, pathology and diagnostic methods

The clinical signs displayed by NHP-B infected shrimp are nonspecific in nature and characterized by lethargy, reduced feed intake, decreased growth rate, softened shell and an atrophied hepatopancreas. On pond-side examination, infected shrimp display empty midguts with increased superficial epicommensal cuticular fouling and/or opportunistic infections (i.e. black spots) that are also often present. Pond mortality rates of up to 95% have been reported in untreated shrimp populations (OIE 2006c).

Microscopic examination of unstained tissue squashes prepared from suspect hepatopancreata may show reduced lipid and dark melanized necrotic tubules. Histologically, NHP-B infected tubular epithelial cells will be hypertrophied with a generalized basophilic intracytoplasmic granularity due to the presence of numerous pleomorphic intracytoplasmic rickettsial-like organisms (=NHP-B). Three stages of infection have been described and defined in histological studies. In early NHP-B infections, scattered tubules with cytoplasmic rickettsial-like organisms can be detected free within the cytoplasm of the tubule epithelial cells. In affected tubules, resorptive (R), fibrillar (F) and/or secretory (B) cells may be infected (Bell and Lightner 1988). In more advanced infections the cytoplasm of most tubule epithelial cells is filled with NHP-B and affected tubules show increasing levels of tubular epithelial cell hypertrophy and desquamation of NHP-B laden cells, as well as marked lipid depletion, tubular necrosis, interstitial hemocytic infiltrates and melanization of hemocyte encapsulated tubules. Some shrimp that survive the first two stages of NHP-B infection present marked atrophy of the hepatopancreas. Most tubules show reduced height of the epithelial cells and some atrophied tubule epithelia appearing squamous. In such shrimp with such chronic NHP-B infections, intertubular hemocytic inflammation is also reduced and there are little or no stored lipid droplets (Frelier et al. 1992; Lightner 1996a; Morales-Covarrubias et al. 2006; OIE 2006c).

Three levels of examination procedures can be used for NHP diagnosis or surveillance/screening. During periods of elevated water temperature and/or salinity, pond-side examinations of statistically significant numbers of shrimp can be conducted to detect and select suspect shrimp (e.g. demonstrating elevated mortality, reduced feed intake, empty midguts, soft shells and atrophy of the hepatopancreas) for further diagnostic testing. When NHP disease is suspected, histopathologic examination of routine H&E stained paraffin sections may be used to demonstrate pathogonmonic lesions in NHP-B infected shrimp. Varying degrees of hepatopancreatic inflammation/necrosis will be present in affected shrimp with the presence of intracytoplasmic organisms that may be confirmed with special stains (e.g. Steiner's & Steiner's stain), ISH with DNA probes, or IHC with specific antibodies to NHP-B (Frelier et al. 1992; Lightner 1996a; Loy and Frelier 1996c; Morales-Covarrubias et al. 2006; OIE 2006c; Bradley-Dunlop et al. 2004). PCR methods are available and may be most suitable for surveillance/screening purposes (Loy and Frelier 1996c; Loy et al. 1996a, 1996b). A rapid, pond-side, antibody-based diagnostic test is being developed that may be useful in early detection and management of NHP disease (Houghton et al. 2009).

Milky Hemolymph Syndrome (MHS)

Biology of the agent and gross signs of the disease

Milky hemolymph disease (MHD) was recently assigned as the name for a serious disease of farmed spiny (*Panilurus* spp.) lobsters in Vietnam (Lightner et al. 2008; OIE 2008a, 2008b), which is caused by overwhelming systemic infections by an unclassified rickettsial-like bacteria (RLB). While MHD is a descriptive name for this disease in spiny lobsters, the name milky hemolymph syndrome (MHS) has been adopted to include MHD of spiny lobsters and similar RLB-caused diseases in penaeid shrimp and crabs (Nunan et al. 2010). In MHS the hemolymph of these affected decapods becomes increasingly turbid and turns "milky" in appearance in severely affected individuals. The turbidity of the hemolymph is due to the massive numbers of RLB circulating in the hemolymph of affected individuals. Generally, the more moribund an affected animal is, the more turbid ("milky" appearing) its hemolymph. MHD of farmed spiny lobsters is currently listed as "under study" while it is being considered for full listing as a notifiable disease by the OIE (OIE 2008a; OIE 2009a).

History and geographic distribution of MHD

In addition to its occurrence in cultured spiny lobsters in Vietnam, MHS (inclusive of MHD of spiny lobsters) may have been an overlooked disease of shrimp

for many years. Although not called MHS in the earlier literature (Anderson et al. 1987; Brock 1988; Brock and Lightner 1990), MHS in shrimp may have been reported in farmed *P. monodon* from Malaysia more than 20 years ago. Even earlier, Bonami and Pappalardo (1980) described a very similar systemic RLB-caused disease in wild crabs, *Carcinus mediterraneus*, collected from the Sète region of France. Hence, MHS may be an "old" disease that has been overlooked, or so well controlled with medicated feeds intended for treating other bacterial diseases that it was not rediscovered as a significant disease until recently. More recent reports of MHS have also been from *P. monodon* farmed in east Africa and Madagascar (Nunan et al. 2003a, 2003b) and in wild crabs, *C. maenas*, held in shedding tanks in England (Eddy et al. 2007). Laboratory challenge studies with the RLB from *P. monodon* from Madagascar suggest that the disease had the potential to infect *L. vannamei* (Nunan et al. 2003b).

By histology, MHS is characterized by massive accumulation of very small RLB in cytoplasmic inclusions, primarily in connective tissues, hemocytes and fixed phagocytes. In severely affected individuals, masses of palely basophilic (with H&E stains) RLB fill the hemocoel spaces among and within the tissues. Gram staining of infected tissues, or Gram staining of milky hemolymph smears, shows the RLB agents of MHS to be very small (~0.5 x 1.0 µm) gram negative rods (Lightner et al. 2009b).

Diagnostic methods

MHD of spiny lobsters is considered to be an emerging disease by OIE (OIE 2009a). Hence, there are no OIE recommended methods for its diagnosis or for surveillance and screening. Nonetheless, gross signs (e.g. turbid or milky hemolymph) combined with histological demonstrations of the characteristic lesions of MHS provide a definitive diagnosis for the disease in spiny lobsters, shrimp and crabs.

Molecular methods for MHS are also available. Nunan et al. (2003a) reported on the development of a PCR test for the Madagascar RLB from *P. monodon* using the 16S rDNA gene of the RLB agent. The same strategy was used to develop PCR test methods for the RLB from spiny lobsters (OIE 2008b) and from the European crab (Eddy et al. 2007). From comparisons of the sequence and alignment of the 16 S rDNA PCR products from each of the RLBs that cause MHS in shrimp, crabs and spiny lobsters, it is presumed that the RLB agents of these diseases are related, but not likely to represent a single species (Fig. 3; Nunan et al. 2010).

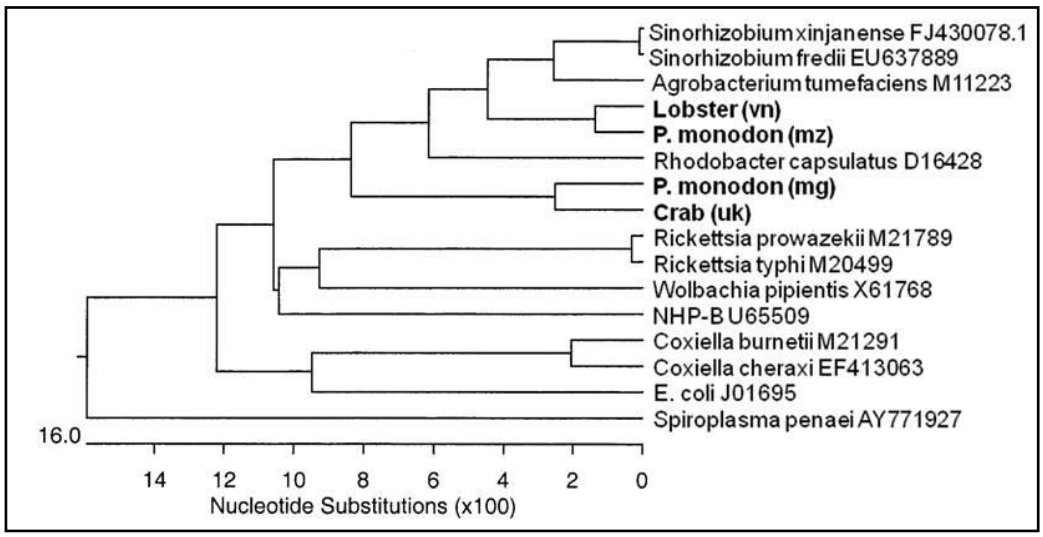

Fig.3. A relatedness plot of the 16S rDNA gene sequence of MHD agents from *P. monodon*, *Panulirus* spp. (spiny lobsters) and *C. maenas* (European crab) compared to the most closely related organisms in GenBank. Included for comparison are NHP-B and *Spiroplasma penaei* from *L. vannamei*.The numbers with most of the isolates are GenBank accession numbers.

Micrococci and Streptococci Diseases

Biology of the agent and gross signs of the disease

Another emerging bacterial disease of shrimp is caused by micrococci, which are probably streptococci based on morphological, biochemical and molecular data. Severe mortalities due toputative *Streptococcus* spp. have occurred in *P. monodon* farmed in the Indo-Pacific (Madagascar and East Africa) and in *L. vannamei* farmed in Central and South Americas and Middle East. *Litopenaeus vannamei* from two significant epizootics due to streptococcus have been processed by the University of Arizona Aquaculture Pathology Laboratory in the past several years. The first was from French Guiana, South America and the second occurred in mid 2008 in several farms culturing *L. vannamei* in Central America (Hasson et al. 2009; Lightner et al. 2009b). Streptococcal disease in shrimp is likely to be due to infections by more than a single species of *Streptococcus*, as the disease has been documented to occur in farms during extended periods of near zero salinity, as well as in farms using seawater of oceanic salinity (Hasson et al. 2009; Lightner et al. 2009b).

The *Streptococcus* spp. that are the putative agents of this disease cause systemic infections in their hosts. The gross signs presented are non-specific and are similar to those induced by both viral and other bacterial diseases. Affected shrimp

become lethargic, anorectic and accumulate at the pond edges, near its surface, or near aerators where dissolved oxygen levels are higher and die there (Lightner et al. 2009b). Histopathological lesions are often remarkable. The most typical presentation of streptococcal infection is a severe acute generalized diffuse necrosis of the lymphoid organ (LO) marked by generalized nuclear pyknosis affecting virtually all LO tubule parenchymal cells, connective tissue cells and circulating hemocytes. Hemocyte accumulations or nodules are sometimes present. Similar, less conspicuous lesions may be present in the heart lumen, gills and diffusely distributed systemically in the connective tissues (Hasson et al. 2009; Lightner et al. 2009b).

Another remarkable feature of these lesions is that while the micrococci may be present in very large numbers in the LO, heart, hemolymph and other tissues, their presence is not easily demonstrated with routine H&E staining. Besides being very small cocci (~1 µm in diameter), these micrococci stain palely basophilic. Their small size and poor staining characteristics with H&E stains make them difficult to demonstrate in low level or developing infections. However, with tissue Gram stains, the putative streptococci stain Gram positive and are prominent in the affected tissues (Hasson et al. 2009; Lightner et al. 2009b).

Disease Management

Until the WSSV pandemic, the penaeid shrimp farming industry in Asia and the Americas remained largely dependent on wild shrimp for stocking its farms and biosecurity was not part of the shrimp farming industry's vocabulary. The farming of essentially all wild shrimp stocks was accomplished by the practice of collection and use of wild seed (postlarvae) for stocking of farms directly, or by the use of captive wild broodstock for the production of seed stock in hatcheries. This dependence has fostered the intensification and spread of the viral diseases in shrimp aquaculture and in wild populations. The shrimp farming industry as a whole has recognized this fact and it has begun to change its farming practices in order to continue to develop, if not survive. While many of the shrimp stocks currently used to stock farms are produced from captive wild broodstock, only those that test negative for WSSV in Asia and WSSV and TSV in the Americas are used to stock biosecure farms. Biosecure production systems (that are designed to exclude potentially infected wild shrimp seed) stocked with shrimp stocks known to be free of the major shrimp pathogens have become a common practice in many shrimp growing regions. A further sign of a maturing industry is its movement towards the expanded development and use of specific pathogen-free (SPF) domesticated shrimp stocks of the most important shrimp species (Pruder et al. 1995; Lotz 1997a, 1997b; Moss et al. 2002; Lightner 2003b, 2005; Lightner et al. 2009a; Lee and O'Bryen 2003). Domesticated lines of *L. vannamei* are

now the dominant shrimp farmed in Asia. The rapid change over of the industry to *L. vannamei* from *P. monodon* was due in large part to disease (FAO 2006). Domesticated stocks made it possible to better manage the health status of the farmed stocks and with specific pathogenfree (SPF) *L. vannamei*, domesticated lines were readily available (Lightner 2005; FAO 2006; Lightner et al. 2009a). These advances have enhanced the technologies used to farm shrimp and have made the industry far more sustainable than it was before the emergence of the virus-caused diseases discussed in the present paper.

The emergence of "new" bacterial diseases, with MHS and streptococcus-caused diseases as examples may be, in part, a consequence of increasing market pressures on the shrimp aquaculture industry to not use antibiotics. However, some of the outbreaks of these diseases occurred for the first time in regions where there was little or no use of antibiotics, or in a new aquacultured species (e.g. *Panulirus* spp. - spiny lobsters).

The dubious history of intercontinental transfer and introduction of the currently OIE listed shrimp virus diseases and the emergence, or re-emergence, of a number of "new" diseases, illustrate the importance to the industry of qualified disease diagnostic laboratories that can do routine diagnostic and surveillance functions.

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