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Viral disease emergence in shrimp aquaculture: origins, impact and the effectiveness of health management strategies

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Abstract

Shrimp aquaculture has grown rapidly over several decades to become a major global industry that serves the increasing consumer demand for seafood and has contributed significantly to socio-economic development in many poor coastal communities. However, the ecological disturbances and changes in patterns of trade associated with the development of shrimp farming have presented many of the pre-conditions for the emergence and spread of disease. Shrimp are displaced from their natural environments, provided artificial or alternative feeds, stocked in high density, exposed to stress through changes in water quality and are transported nationally and internationally, either live or as frozen product. These practices have provided opportunities for increased pathogenicity of existing infections, exposure to new pathogens, and the rapid transmission and transboundary spread of disease. Not surprisingly, a succession of new viral diseases has devastated the production and livelihoods of farmers and their sustaining communities. This review examines the major viral pathogens of farmed shrimp, the likely reasons for their emergence and spread, and the consequences for the structure and operation of the shrimp farming industry. In addition, this review discusses the health management strategies that have been introduced to combat the major pathogens and the reasons that disease continues to have an impact, particularly on poor, small-holder farmers in Asia.

Key words: disease emergence, health management, shrimp, small-scale farmers, virus.

Introduction

Over the past three decades, shrimp farming in Asia has expanded rapidly from a very low base of traditional, low-density polyculture to become a vibrant export industry currently valued at more than $US8 billion (FAO 2006). Industry growth, fuelled by commercial investment, government support and expanding global commodity markets, has been a major contributor to socio-economic development in Asia, particularly in poor coastal communities. However, the rapid expansion of the industry has also been a source of significant environmental and sociological disturbances associated with changes in land use, the ecology of aquatic species and patterns of global trade. Not surprisingly, a major consequence of these disturbances has been the emergence and spread of disease. Commencing in the late 1980s, a succession of previously unknown diseases emerged in farmed shrimp, in both Asia and the Americas, and spread rapidly across international boundaries to impact very significantly on production (Fig. 1). Since 1994, it has been estimated that annual losses globally as a result of disease, primarily caused by viral pathogens, have been as high as $US3 billion (Lundin 1995; Lightner 2003). Although there has been a marked recovery in recent years, disease remains a major concern for the sustainability and profitability of the industry. This review will discuss the nature of these viral pathogens, the likely reasons for their emergence and spread, and the consequences of pandemic disease, both good and

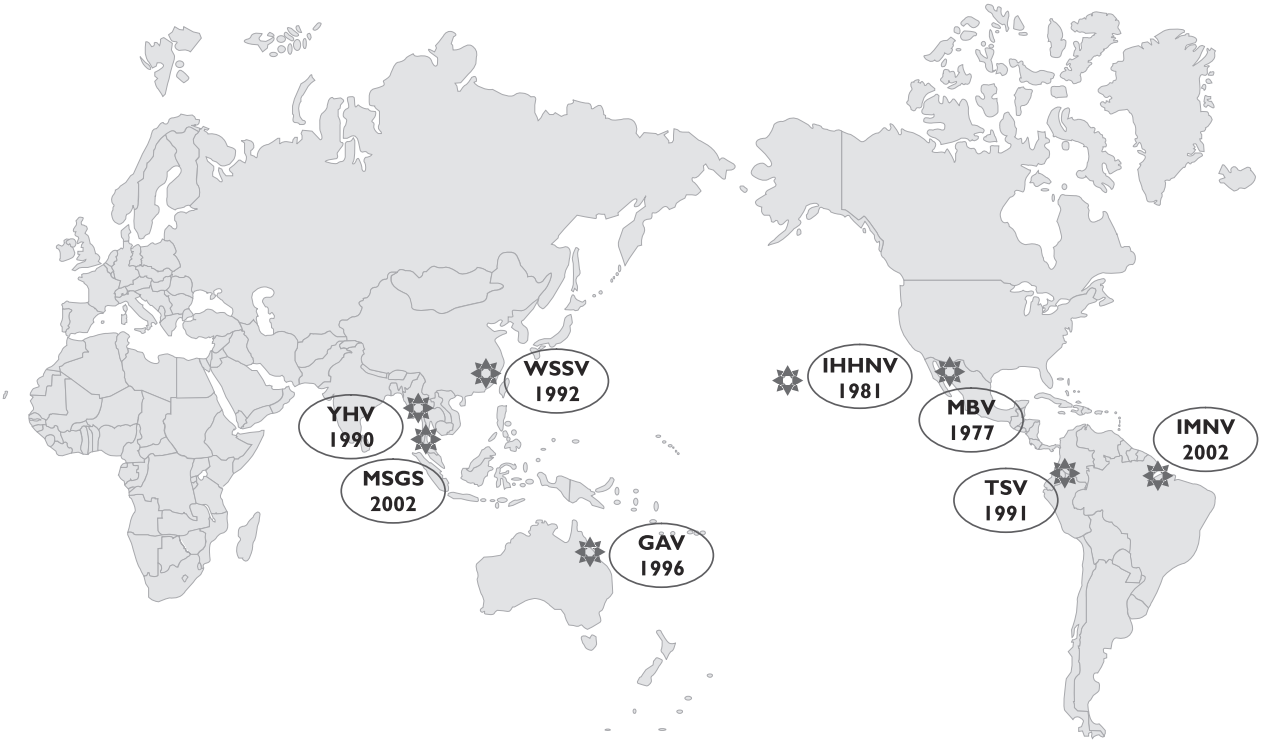

Figure 1 History of the emergence of the major pathogens of farmed shrimp. GAV, gill-associated virus; IHHNV, infectious hypodermal and haematopoietic necrosis virus; IMNV, infectious myonecrosis virus; MBV, monodon baculovirus; MSGS, monodon slow growth syndrome; TSV, Taura syndrome virus; WSSV, white spot syndrome virus; YHV, yellow head virus.

bad, for the structure and operation of the shrimp farming industry. The review will also discuss health management strategies that have been introduced to combat the major pathogens and the reasons that disease continues to have an impact, particularly on poor, small-holder farmers in Asia.

Major pathogens of farmed shrimp

More than 20 viruses have been reported to infect marine shrimp. Many have not been associated with clinical signs of disease and some have been observed only by electron microscopy and are poorly characterised (Table 1). Seven viral pathogens of marine shrimp are currently listed by the World Organization for Animal Health (OIE) as causing notifiable aquatic animal diseases and two are under study for listing (OIE 2008). By international agreement, diseases listed by the OIE should be reported by member countries and are subject to specified health measures that are intended to limit disease spread and assure sanitary safety of international trade in aquatic animals and their products. Six of the seven OIE-listed marine shrimp viruses have been reported to occur in Asia.

White spot syndrome virus

White spot syndrome virus (WSSV) is by far the most devastating pathogen of farmed shrimp. It infects all cultured penaeids and has been responsible for much of the economic impact of disease on production globally. White spot disease (WSD) was first reported in June 1992 in cultured kuruma shrimp (*Penaeus japonicus* Bate, 1888) in the Fujian Province of China and in nearby Taiwan (Zhan *et al.* 1998; Jiang 2001). The disease spread rapidly north and south along the coast of China, affecting each of the major production species (*P. japonicus*, *Penaeus monodon* Fabricius, 1798 and *Penaeus chinensis* Osbeck, 1765). From March 1993, outbreaks were reported in several prefectures in Japan, commencing in farms that had imported *P. japonicus* juveniles from China (Nakano *et al.* 1994). The disease also first appeared in Korea in 1993 (Park *et al.* 1998) and by 1994 had spread to most shrimp-farming countries throughout South and South-East Asia. The first recorded outbreak of WSSV in the Americas was at a farm in Texas in November 1995. A nearby processing plant that imported frozen shrimp from Asia was considered to be the likely source (Lightner *et al.* 1997). In 1997 and 1998, WSSV

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Table 1 Major viruses infecting marine shrimp

Virus	Abbreviation	Family‡	Genus‡	Genome	Known distribution
White spot syndrome virus†	WSSV	*Nimaviridae*	*Whispovirus*	dsDNA	Asia, Americas
Yellow head virus†	YHV	*Roniviridae*	*Okavirus*	(+) ssRNA	Asia, Central America
Gill-associated virus	GAV	*Roniviridae*	*Okavirus*	(+) ssRNA	Australia, Asia, Pacific
Taura syndrome virus†	TSV	*Dicistroviridae*	Unassigned	(+) ssRNA	Americas, Asia
Infectious myonecrosis virus†	IMNV	*Totiviridae*	Unassigned	(+) ssRNA	South America, Asia
Baculovirus penaei†	BP	*Baculoviridae*	Unassigned	dsDNA	Asia
Monodon baculovirus†	MBV	*Baculoviridae*	*Nucleopolyhedrosisvirus*	dsDNA	Asia, Australia, Africa, Americas
Infectious hypoderma and haematopoietic necrosis virus†	IHHNV	*Parvoviridae*	*Brevidensovirus*	ssDNA	Asia, Australia, Africa, Americas, Pacific
Hepatopancreatic parvovirus	HPV	*Parvoviridae*	*Brevidensovirus*	ssDNA	Asia, Australia, Africa, Americas
Mourilyan virus	MoV	Bunavirus-like	Unassigned	(–) ssRNA	Australia, Asia
Laem Singh virus	LSNV	Leuteovirus-like	Unassigned	(+) ssRNA	Asia
Spawner-isolated mortality virus	SMV	*Parvoviridae*	Unassigned	ssDNA	Australia, Asia
Baculoviral midgut gland necrosis virus	BMNV	*Baculoviridae*	Unassigned	dsDNA	Asia, Australia
Lymphoid organ vacuolization virus	LOVV	Togavirus-like?	Unassigned	(+) ssRNA?	Americas

†OIE (2008) listed.
‡International Committee on Taxonomy of Viruses.

was detected in wild shrimp stocks in South Carolina and in bait prawns in Texas. The major epizootic of WSD in the Americas commenced in Nicaragua, Honduras and Guatemala in mid-January 1999. Subsequently, the disease was reported in Panama in March, Ecuador in May and had reached Peru by October 1999 (Alday de Graindorge 2000). It is now enzootic in all shrimp-farming regions throughout much of Central and South America. The original source of emergence of WSSV in East Asia is not known, but it does not appear to be a natural shrimp pathogen and may have been introduced to shrimp broodstock via an unusual source of live or frozen feed.

White spot disease commonly results in 80–100% mortality within 5–10 days of the first appearance of clinical signs (Chou *et al.* 1995). Infected shrimp display reddish-pinkish discolouration, appear lethargic, cease feeding and congregate at the pond edges. White spots are commonly observed under the cuticle of diseased shrimp, but are not pathognomonic, and similar signs can occur as a result of bacterial infection (Takahashi *et al.* 1994; Goarant *et al.* 2000; Wang *et al.* 2000b). White spot syndrome virus infects cells of ectodermal and mesodermal origin and the most characteristic histological lesion is the appearance of eosinophilic Cowdry A-type inclusions in hypertrophied nuclei with marginated chromatin that become lightly basophilic late in infection (Wongteerasupaya *et al.* 1995b; Chang *et al.* 1996). The virus replicates and is assembled in the nucleus of cells and is not occluded as a result of infection (Wang & Chang 2000).

White spot syndrome virus is a large, ovaloid, DNA virus (120–150 nm × 270–290 nm) with a lipid envelope that features an unusual tail-like appendage (Wongteerasupaya *et al.* 1995a,b). It is distantly related to other large DNA viruses and has been classified taxonomically as the only member of the genus *Whispovirus* in the newly formed family *Nimaviridae* (Vlak *et al.* 2005). Virions comprise at least 45 structural proteins that are arranged in three morphologically distinct layers (Tsai *et al.* 2004; Li *et al.* 2007). The nucleocapsid, a helical, bacilliform structure (∼70 nm × ∼300 nm) containing the circular, double-stranded DNA (dsDNA) genome and nine proteins, lies at the core of the virions. The nucleocapsid proteins include a basic DNA-binding protein (VP15) and a giant protein (VP664), which forms the stacked ring subunits and appears to protrude through the overlaying tegument (Leu *et al.* 2005; Witteveldt *et al.* 2005). The tegument layer is loosely associated with both the nucleocapsids and the envelope and is composed of at least four structural proteins including the major capsid protein VP26 and the minor proteins VP36A, VP39A and VP95 (Tsai *et al.* 2006; Xie *et al.* 2006). The envelope, which forms the 6–7-nm-thick outer layer, comprises a tri-laminar lipid membrane and at least 28 viral proteins, many of which contain transmembrane domains. The major envelope proteins VP19 and VP28, which protrude from the outer surface of the membrane, have been shown to induce a protective response in shrimp (Witteveldt *et al.* 2004a,b).

The ∼305 kbp WSSV genome encodes ∼180 long, open reading frames (ORF) and nine homologous regions containing direct repeats, inverted repeats and palindromes (van Hulten *et al.* 2001; Yang *et al.* 2001).

Significant variations in genome size have been reported between different isolates, primarily as a result of large sequence deletions (up to 13 kb) in the region between the DNA polymerase and the protein kinase genes (Marks *et al.* 2004). Variations in the genome sequence have been used in molecular epidemiological studies to trace the movement of WSSV and to identify sources of infection (Wongteerasupaya *et al.* 2003; Dieu *et al.* 2004; Hoa *et al.* 2005; Musthaq *et al.* 2006). There is also evidence that different isolates vary significantly in virulence (Marks *et al.* 2005) and that passaging of WSSV in different hosts can alter both pathogenicity and the sequences of tandem repeat regions (Waikhom *et al.* 2006). Nevertheless, the overall nucleotide sequence identity between isolates is >99% (Marks *et al.* 2004) and variations do not appear to affect the performance of the polymerase chain reaction (PCR) tests commonly used for WSSV detection (Kiatpathomchai *et al.* 2005).

White spot syndrome virus has a very broad host range in decapod crustaceans. In marine shrimp, the virus occurs commonly as a low-level persistent infection in the absence of clinical signs, but rapid increases in viral load, precipitated by physiological stress, salinity change or lower temperatures, can lead to disease and mass mortalities in ponds (Peng *et al.* 1998; Vidal *et al.* 2001; Du *et al.* 2006; Granja *et al.* 2006; Liu *et al.* 2006). There is evidence that WSSV replicates most efficiently at 23–28°C (Guan *et al.* 2003; Du *et al.* 2006; Granja *et al.* 2006; Reyes *et al.* 2007) and that high water temperatures prevent the onset of disease (Rahman *et al.* 2006). The virus also survives for longer periods in seawater at lower temperatures (Momoyama *et al.* 1998) and seawater temperatures <30°C are conducive to higher prevalence of WSSV infection in wild shrimp populations (Rodriguez *et al.* 2003; Withyachumnarnkul *et al.* 2003). All farmed marine shrimp species are susceptible to WSD. Other decapod crustaceans, although universally susceptible to infection, may not develop clinical signs (Jiravanichpaisal *et al.* 2001). Indeed, some crab and crayfish species have been reported to carry very high viral loads in the absence of disease and may be important reservoirs, sustaining the virus in the natural environment and representing potential sources of infection in shrimp ponds. Other invertebrates, such as polychaetes, bivalves, rotifers, artemia, copepods and some insect larvae, as well as microalgae, can accumulate high levels of viable WSSV in the absence of demonstrated virus replication and may act as mechanical vectors of infection (Yan *et al.* 2004, 2007; Liu *et al.* 2007).

White spot syndrome virus infection can be transmitted either horizontally or vertically. Horizontal infection of shrimp and other crustaceans has been demonstrated experimentally by exposure to infected water or by ingestion of infected tissue (Chou *et al.* 1998; Kanchanaphum *et al.* 1998; Supamattaya *et al.* 1998; Corbel *et al.* 2001; Soto *et al.* 2001). Ingestion appears to be the more effective means of transmission in shrimp (Soto & Lotz 2001). Vertical transmission from infected broodstock to early life stages appears to be a common source of infection. Although there is evidence of WSSV in the muscles and connective tissues of gonads, infection of sperm has not been reported. Oogonia and oocytes may be positive by histology or *in situ* hybridisation, but mature eggs are not (Lo *et al.* 1997; Mohan *et al.* 1997). This suggests that the virus in developing oocytes does not survive maturation and that transovarial transmission is unlikely. Rather, WSSV appears to be transmitted transovum by surface contamination of the egg (Lo *et al.* 1997; Lo & Kou 1998). Both horizontal and vertical transmission of WSSV infection can occur in the absence of disease.

Yellow head virus

Yellow head disease was first reported in farmed black tiger shrimp (*P. monodon*) in central Thailand in 1990 and it remains the most virulent of the shrimp viruses, commonly causing total crop loss within 3–5 days of the first appearance of visible signs of the disease in a pond (Limsuwan 1991; Boonyaratpalin *et al.* 1993). From central Thailand, the disease spread to southern areas on the eastern and western coasts of the Gulf of Thailand, reaching the far south by February 1992 (Chantanachookin *et al.* 1993). Yellow head disease or yellow head virus (YHV) has since been reported from many shrimp-farming countries in Asia, including India, Indonesia, Malaysia, the Philippines, Sri Lanka, Vietnam and Taiwan (Mohan *et al.* 1998; Wang & Chang 2000; WB, NACA, WWF, FAO 2001; Natividad *et al.* 2002). There is also recent evidence of the presence of YHV in *Penaeus vannamei* Boone, 1931 and *Penaeus stylirostris* Stimpson, 1874 shrimp from the north-west coast of Mexico (de la Rosa-Velez *et al.* 2006). Several yellow-head-like viruses have also been reported. Gill-associated virus (GAV) is a closely related virus that has been associated with a disease called mid-crop mortality syndrome (MCMS) in *P. monodon* shrimp in Australia (Spann *et al.* 1997; Munday & Owens 1998; Cowley *et al.* 1999; Callinan & Jiang 2003; Callinan *et al.* 2003). Yellow-head-related viruses have also been reported in *P. monodon* from Thailand and in *P. japonicus* from Taiwan (Wang *et al.* 1996; Soowannayan *et al.* 2003). A survey of shrimp samples from Mozambique, India, Thailand, Malaysia, Indonesia, the Philippines, Vietnam, Taiwan and Australia has revealed that YHV is one of at least six genetic lineages (genotypes) of related viruses that occur commonly in farmed *P. monodon* throughout the Indo-Pacific region (Wijegoonawardane *et al.* 2008a). However, only YHV (genotype one) has been detected in shrimp

with typical signs of yellow head disease. Of the other genotypes, only GAV (genotype two) has been associated with any form of disease. The original source of YHV infection in Thai shrimp in 1990 has not been determined, but it has been suggested that it may have previously been responsible for similar serious epizootics in Indonesia, Malaysia, China and the Philippines and possibly for the crash of the *P. monodon* industry in Taiwan in 1986–1987 (Lightner 1996b). The close genetic relationship of YHV to other genotypes in the YHV complex suggests that it is most likely a natural infection of wild penaeid or palemonid shrimp (Wijegoonawardane *et al.* 2008a).

Yellow head disease affects shrimp in early to late juvenile stages and is characterised by rapid feed consumption followed by a cessation of feeding and the congregation of disoriented and moribund shrimp at the pond edge, with rapidly accelerating mortalities. The disease derives its name from the bleached or pale-yellow appearance of the cephalothorax, resulting from discolouration of the underlying hepatopancreas, but this condition is not always apparent (Chantanachookin *et al.* 1993; Flegel *et al.* 1995). Disease associated with GAV infection is far less severe, with mortalities developing more slowly and rarely reaching 100% and, although moribund shrimp commonly develop a reddish appearance, the pale discolouration typical of yellow head disease does not occur. Yellow head virus is also far more virulent that GAV ($>10^6$-fold by LD_{50}) in experimental infections in *P. monodon* (Nusra Sittidilokratna and Peter Walker, unpublished data, 2007). Yellow head virus infects tissues of ectodermal and mesodermal origin and causes severe necrosis, particularly in the lymphoid organ and gills, with prominent nuclear pyknosis and heterokaryosis and densely basophilic spherical cytoplasmic inclusions evident in histological sections (Chantanachookin *et al.* 1993; Lu *et al.* 1995). Similar histological lesions are seen in moribund shrimp infected with GAV. Yellow head virus and GAV replicate in the cytoplasm in which long filamentous pre-nucleocapsids are abundant, and virions bud into cytoplasmic vesicles in densely packed paracrystalline arrays for egress at the cytoplasmic membrane (Boonyaratpalin *et al.* 1993; Chantanachookin *et al.* 1993; Spann *et al.* 1997).

The YHV is a rod-shaped, enveloped positive-sense single-stranded RNA (ssRNA) virus (40–60 nm × ~150–200 nm) with a helical nucleocapsid and prominent knob-like surface projections (Chantanachookin *et al.* 1993; Wongteerasupaya *et al.* 1995a,b; Nadala *et al.* 1997; Tang & Lightner 1999). The virus is distantly related to other large ssRNA viruses infecting vertebrates and fish, including the coronaviruses, toroviruses, arteriviruses and bafniviruses, and, together with GAV, YHV is currently classified as the species *Gill-associated virus* in the genus *Okavirus*, family *Roniviridae*, order *Nidovirales* (Walker *et al.* 2005; Schultze *et al.* 2006). A roni-like virus has also been described recently and associated with mortalities in the freshwater crab (*Eriochier sinensis* Milne-Edwards, 1854) in China, but its relationship to YHV has not yet been determined (Zhang & Bonami 2007). The YHV virions contain three major structural proteins. The nucleocapsid contains the ssRNA genome and the nucleoprotein (p20), which encapsidates and protects the RNA. The envelope comprises a tri-laminar lipid membrane and two glycoproteins, gp116 and gp64, which penetrate the membrane to form the spike-like projections on the virion surface. Glycoprotein gp116 has been identified as the target for virus-neutralising antibodies *in vitro* and *in vivo* (Assavalapsakul *et al.* 2005; Sittidilokratna *et al.* 2009). There is evidence that suggests that gp116 may be preferentially suppressed in persistently infected shrimp that survive YHV infection (Chaivisuthangkura *et al.* 2008). Glycoprotein gp116 must perform a critical function in viral infection, but the domain structure has not been defined and the role of gp64 is presently unexplored. There is also evidence that a small triple-membrane-spanning protein (gp22) is a very minor component of YHV virions (Nusra Sittidilokratna and Peter Walker, unpublished data, 2007).

The 26 662 nt YHV genome is polyadenylated and contains four long ORF (Sittidilokratna *et al.* 2008). ORF1a and ORF1b overlap and encode non-structural enzymes involved in replication and transcription. ORF1b is expressed only as an extension of ORF1a as a result of a ribosomal frame-shift at a predicted pseudoknot structure in the overlap region (Sittidilokratna *et al.* 2002). ORF1a encodes papain-like and cysteine-like proteases that are required for post-translational auto-processing of the large pp1a and pp1ab polyproteins (Sittidilokratna *et al.* 2008). ORF1b encodes several replication enzymes, including helicase, polymerase, exonuclease, endonuclease and methyl transferase domains (Cowley *et al.* 2000a; Sittidilokratna *et al.* 2002). ORF2 encodes the p20 nucleoprotein (Sittidilokratna *et al.* 2006). ORF3 encodes a long polyprotein (pp3) that is processed post-translationally to generate the envelope glycoproteins gp116 and gp64 and the small triple-membrane-spanning protein gp22 of unknown function (Jitrapakdee *et al.* 2003; Nusra Sittidilokratna and Peter Walker, unpublished data, 2007). Downstream of ORF3, the long 3′ untranslated region contains a fifth small ORF (ORF4) that does not appear to be functional (Sittidilokratna *et al.* 2008).

The 26 235 nt GAV genome is very similar in organisation to that of YHV, but there is some evidence that ORF4 (which is somewhat longer than the ORF4 in YHV) may be functional (Cowley & Walker 2002, 2008). The overall nucleotide sequence identity between the YHV and

GAV genomes is ~79%, varying from ~74% in ORF3 to ~82% in ORF1b (Sittidilokratna *et al.* 2008). There is a similar level of sequence identity between YHV and each of the other four genotypes, but genotypes three and six are more closely related to GAV than genotypes four and five (Wijegoonawardane *et al.* 2008a). Analysis of a short region in ORF1b has indicated that nucleotide sequence variation between isolates within a genotype is generally within 2–3%, except for genotype five, which is more diverse (Wijegoonawardane *et al.* 2008a). The molecular basis of the differences in pathogenicity and virulence among the genotypes is not known. The level of diversity between the genotypes is sufficient to require careful design of PCR tests to ensure the required level of test specificity. Polymerase chain reaction (PCR) tests have been described for the genotype-specific detection of YHV, detection and differentiation of YHV and GAV, and group-specific detection of each of the known genotypes in the YHV complex (Wongteerasupaya *et al.* 1997; Cowley *et al.* 2004; Wijegoonawardane *et al.* 2008b). However, recent observations indicate that genetic recombination occurs with high frequency between some YHV genotypes in natural populations of shrimp, suggesting that genotype assignments and the association of genotype with virulence is not straightforward (Wijegoonawardane *et al.* 2009).

Although yellow head disease outbreaks have been reported only in *P. monodon*, natural and/or experimental YHV infection has been reported in several other commonly farmed species of penaeid shrimp, including *P. vannamei*, *P. stylirostris*, *Penaeus merguiensis* De Man, 1888, *Penaeus setiferus* Linnaeus, 1767, *Penaeus aztecus* Ives, 1891 and *Penaeus duorarum* Burkenroad, 1939 (Chantanachookin *et al.* 1993; Lu *et al.* 1994; Lightner *et al.* 1998; de la Rosa-Velez *et al.* 2006). A yellow-head-like virus has also been reported in *P. japonicus* in Taiwan (Wang *et al.* 1996). There is evidence of natural and/or experimental YHV infection in wild penaeid and palemonid shrimp and krill (*Acetes* sp.) found within ponds or nearby canals (Flegel *et al.* 1997b; Longyant *et al.* 2006). However, the severity of disease varies, with some susceptible shrimp displaying mild signs and surviving up to 30 days after experimental infection (Longyant *et al.* 2005). Freshwater palemonid shrimp (*Machrobrachium* spp.) appear to be resistant to infection, although a mild infection has been established experimentally in one species (*Machrobrachium sintangese* De Man, 1879) (Longyant *et al.* 2005). A survey of 16 species of crabs collected from the vicinity of shrimp farms in Thailand detected no evidence of either natural infection or experimental susceptibility (Longyant *et al.* 2006). Other genotypes in the YHV complex have been detected almost exclusively in *P. monodon*. The prevalence of GAV in wild and farmed populations of *P. monodon* in eastern Australia approaches 100% (Cowley *et al.* 2000b; Walker *et al.* 2001). *Penaeus merguiensis*, *P. japonicus* and *P. esculentus* Haswell, 1879 are also susceptible to experimental GAV infection, but pathogenicity varies (Spann *et al.* 2000). Although *P. esculentus* co-cultivated with *P. monodon* have been reported to be infected, there is no evidence that GAV infection occurs commonly in any species other than *P. monodon*. All other genotypes have been detected exclusively in *P. monodon*, but a survey of other potential hosts has not been conducted (Wijegoonawardane *et al.* 2008a). The evidence to date suggests that penaeid shrimp are the natural hosts of YHV and other viruses in the YHV complex, and that low-level, persistent infections occur commonly in healthy *P. monodon* populations throughout the Indo-Pacific region.

Yellow head virus and GAV can be transmitted horizontally by injection, immersion or ingestion of infected shrimp tissue and by co-habitation with infected shrimp (Lu *et al.* 1994; Flegel *et al.* 1995; Lightner *et al.* 1998; Walker *et al.* 2001; Longyant *et al.* 2006). In shrimp that survive experimental challenge, the infection can persist for up to 30 days following recovery, and perhaps for life. Recent evidence suggests that preferential suppression of expression of envelope glycoprotein gp116 is associated with viral persistence (Chaivisuthangkura *et al.* 2008). There is no direct evidence that YHV is transmitted vertically. However, vertical transmission of GAV has been demonstrated experimentally (Cowley *et al.* 2002). High levels of GAV infection have been detected in spermatophores, seminal fluid, vas deferens and mature ovarian tissue (Walker *et al.* 2001; Callinan *et al.* 2003). Infection can originate from either the male or female parent, and the virus is most likely to be transmitted transovum on the egg surface (Cowley *et al.* 2002). The high prevalence of infection in healthy *P. monodon* postlarvae throughout the Indo-Pacific region suggests that viruses in the YHV complex are maintained in a cycle of low-level, life-long chronic infections by vertical transmission and persistence (Walker *et al.* 2001; Spann *et al.* 2003). Virus amplification and associated disease outbreaks appear to occur as a result of physiological stress induced by poor water quality or other environmental factors (Flegel *et al.* 1997a; de la Vega *et al.* 2004). The much higher virulence of YHV compared with GAV and other genotypes ($>10^6$-fold by LD_{50}) ensures that the threshold of infection required for disease is far more easily obtained (Nusra Sittidilokratna and Peter Walker, unpublished data, 2008).

Taura syndrome virus

Taura syndrome first emerged in farmed white Pacific shrimp (*P. vannamei*) near the mouth of the Taura River

in Ecuador in June 1992 (Jiminez 1992). It was originally attributed to the use of chemical fungicides in nearby banana plantations, but was subsequently shown to be of viral aetiology (Hasson *et al.* 1995). By 1996, Taura syndrome virus (TSV) had spread to Peru and north-eastern Brazil, throughout the Pacific and Caribbean coasts of Central America, and to Florida and Texas in the USA (Lightner 1996a). It had also spread to *P. vannamei* breeding programs in Hawaii by 1994, but was subsequently eradicated (Brock *et al.* 1995; Brock 1997). The rapid spread of the virus has been attributed to international trade in postlarvae and broodstock. In 1998, TSV spread to Taiwan in *P. vannamei* broodstock imported from Central and South America (Tu *et al.* 1999; Yu & Song 2000). By 2004, following the dramatic amplification of *P. vannamei* production, TSV had become endemic in most major shrimp-farming countries in East and South-East Asia, including China, Korea, Thailand, Myanmar, Indonesia and Vietnam (Sunarto *et al.* 2004; Van 2004; Nielsen *et al.* 2005; Do *et al.* 2006). The impact of Taura syndrome on the shrimp-farming industry in the Americas was estimated to be $US1–2 billion up to 2001 (Lightner 2003). The impact in Asia is yet to be systematically evaluated. The original source of TSV in Ecuador is unknown, but its apparent absence prior to 1992 and the radiant nature of its spread indicate that it is unlikely to be a natural infection of shrimp. Taura syndrome virus may well have crossed species from another invertebrate host in the farming environment.

Taura syndrome usually occurs in juvenile *P. vannamei* (0.1–5 g) within 14–40 days of stocking in nursery or grow-out ponds, but it can also occur in postlarvae and adult shrimp (Lightner *et al.* 1995; Lightner 1996b). Acute, transition and chronic phases of TSV infection have been described (Hasson *et al.* 1999a). Diseased shrimp in the acute phase typically display reddish colouration, particularly in the tail fan and pleopods, as a result of expansion of the red chromatophores. Individuals have soft shells and an empty gut and usually die during moulting. Surviving shrimp entering the transition (recovery) phase display irregularly shaped melanised lesions on the cephalothorax, tail and appendages as a result of haemocyte accumulation, but may otherwise appear and behave normally. During the acute phase, cumulative mortalities may be as high as 95% (Brock 1997). Following moulting and recovery, shrimp enter the chronic phase in which they remain persistently infected in the absence of obvious clinical signs. Although there is evidence that TSV may be cleared from shrimp during the chronic phase, it may continue for at least 8–12 months, and possibly for life (Hasson *et al.* 1999b). Taura syndrome virus infects tissues of ectodermal and mesodermal origin, particularly the cuticular epithelium, subcuticular connective tissue and striated muscle, haematopoietic tissue, the lymphoid organ and the antennal gland (Lightner *et al.* 1995; Hasson *et al.* 1999a,b). The virus replicates in the cytoplasm, with evidence of TSV RNA in association with proliferating membranes and cytoplasmic vesicles (Srisuvan *et al.* 2006b).

Taura syndrome virus is a small, non-enveloped, positive-sense ssRNA virus with icosahedral architecture (31–32 nm diameter). It is similar to small insect viruses in the genus *Cripavirus* of the family *Dicistroviridae* (e.g. cricket paralysis virus, drosophila C virus) and is currently classified as an unassigned species (*Taura syndrome virus*) in this family (Christian *et al.* 2005). The TSV virions comprise the RNA genome, three major capsid proteins, VP1 (55 kDa), VP2 (40 kDa) and VP3 (24 kDa), and a minor protein VP0 (58 kDa) (Bonami *et al.* 1997; Mari *et al.* 2002). The functions of the individual capsid proteins are not yet known, but there is evidence that VP1 (also called CP2) binds to the laminin receptor/p40 protein, which is a common receptor for several arthropod-borne RNA viruses (Senapin & Phongdara 2006). The 10 205 nt TSV RNA genome is polyadenylated and contains two long ORF flanked by long 5′ and 3′ untranslated regions and a long (207 nt) intergenic region (Mari *et al.* 2002). ORF1 encodes non-structural proteins, including protease, helicase and RNA-dependent RNA polymerase elements, and a domain that is homologous to IAP (inhibitor of apoptosis) sequences of animals and some other viruses. ORF2 encodes a long polyprotein from which the capsid proteins are generated by proteolytic cleavage. Translation of ORF2 appears to be from a single genome-length messenger RNA (mRNA) and is mediated by an internal ribosomal entry site (IRES) at a predicted stem-loop structure in the long intergenic region (Robles-Sikisaka *et al.* 2001; Mari *et al.* 2002). Genetic and antigenic variations have been reported among TSV isolates in the major capsid protein precursor encoded in ORF2. At least two distinct lineages of TSV have been identified in shrimp from the Americas and variations at a locus in VP1 have been attributed to the failure of a standard diagnostic monoclonal antibody to detect some isolates (Erickson *et al.* 2002, 2005; Robles-Sikisaka *et al.* 2002). Isolates from Asia also display variation in ORF2 sequences, but cluster separately from isolates from the Americas, possibly as two separate clades representing the early isolates from Taiwan and later isolates from Thailand, China, Myanmar and Korea (Chang *et al.* 2004; Nielsen *et al.* 2005; Do *et al.* 2006). This suggests that introductions of TSV to Asia from the Americas have occurred relatively infrequently and that the virus has been dispersed within Asia from locally established breeding facilities. A recent isolate from Belize may represent a third genetic lineage (Srisuvan *et al.*

2005; Lightner *et al.* 2007). There is also evidence of variations in virulence between TSV strains (Erickson *et al.* 2002, 2005) and of adaptation to growth in other shrimp species (Erickson *et al.* 2002; Chang *et al.* 2004).

Although *P. vannamei* is the major host of TSV, experimental infections have demonstrated susceptibility to infection in several other penaeid shrimp species, including *P. stylirostris*, *P. setiferus*, *P. aztecus*, *P. duorarum*, *P. chinensis* and *P. monodon* (Overstreet *et al.* 1997; Srisuvan *et al.* 2005). Natural infections have also been detected in *P. stylirostris*, *P. monodon*, *P. japonicus*, *Metapenaeus ensis* De Haan, 1844 and the freshwater shrimp, *Macrobrachium rozenbergii* De Man, 1879 (Erickson *et al.* 2002; Chang *et al.* 2004; Nielsen *et al.* 2005). However, susceptibility to disease varies. Clinical signs and mortalities have been reported following natural or experimental infection of *P. stylirostris*, *P. schmitti*, *P. setiferus*, *P. monodon* and *M. ensis*. Susceptibility to disease may also vary in selected lines of shrimp and for different strains of TSV (Brock 1997; Overstreet *et al.* 1997; Erickson *et al.* 2002; Chang *et al.* 2004; Nielsen *et al.* 2005; Srisuvan *et al.* 2005, 2006a). Selected lines of TSV-resistant *P. vannamei* have been developed (Argue *et al.* 2002; White *et al.* 2002; Xu *et al.* 2003; Srisuvan *et al.* 2006a).

Taura syndrome virus can be transmitted horizontally by injection, ingestion of infected tissue or exposure to infected shrimp or shrimp carcasses (Brock *et al.* 1995; Hasson *et al.* 1995; Lotz 1997; Lotz *et al.* 2003; Srisuvan *et al.* 2006a). Exposure to chronically infected shrimp can result in TSV transmission in the absence of disease (Lotz *et al.* 2003). Although considered likely to occur, vertical transmission of TSV has not been demonstrated experimentally (Lightner & Redman 1998a; Dhar *et al.* 2004). Mechanical transmission is also considered a likely mechanism for the local geographical spread of TSV infection between ponds or farms. Taura syndrome virus has been detected in the faeces of sea gulls collected from the edges of shrimp ponds during disease outbreaks (Garza *et al.* 1997). There are also reports that salinity-tolerant water boatmen (*Trichocorixa reticulata* Guerin-Meneville, 1857) carry TSV within the gut and may act as mechanical vectors (Lightner 1996a; Brock 1997; Dhar *et al.* 2004).

Infectious hypodermal and haematopoietic necrosis virus

Infectious hypodermal and haematopoietic necrosis (IHHN) first emerged in mid-1981 as a viral disease causing mass mortalities (>90%) in juvenile and sub-adult *P. stylirostris* farmed in super-intensive raceways in Hawaii (Lightner *et al.* 1983a). Infectious hypodermal and haematopoietic necrosis virus (IHHNV) was subsequently detected in apparently healthy *P. vannamei* farmed in the same facility and shown not to cause mortalities or the clinical signs typical of IHHN (Bell & Lightner 1984). However, subsequent investigations demonstrated that IHHNV could cause a condition described as 'runt deformity syndrome' (RDS) in *P. vannamei* in which affected shrimp display cuticular deformities and irregular and slow growth (Kalagayan *et al.* 1991). Economic losses as a result of RDS in *P. vannamei* have been estimated to be 10–50% per crop (Lightner & Redman 1998b). Runt deformity syndrome has also been reported in cultured *P. monodon* infected with IHHNV, but the aetiology was not established (Primavera & Quinitio 2000) and subsequent studies have indicated that IHHNV infection causes no disease in *P. monodon* and has little or no impact on either growth or fecundity (Chayaburakul *et al.* 2005; Withyachumnarnkul *et al.* 2006).

Following the initial detection in Hawaii, IHHNV was found to be widely distributed in *P. stylirostris* and *P. vannamei* throughout shrimp-farming regions of the Americas (Morales-Covarrubias *et al.* 1999; Lightner 2003; Motte *et al.* 2003). It has also been reported to be highly prevalent in wild fisheries on the Pacific coast of the Americas and may have contributed to the collapse of the wild *P. stylirostris* fishery in the northern Gulf of California in 1990 (Lightner 1996a, 2003; Pantoja *et al.* 1999). In addition, IHHNV has been detected in *P. stylirostris* imported to French Polynesia and Guam for aquaculture (Costa *et al.* 1998; Tang & Lightner 2002) and in *P. vannamei* imported to China (Yang *et al.* 2007). In *P. monodon*, IHHNV is endemic in many Asian countries, including the Philippines, Singapore, Malaysia, Indonesia, Thailand and Taiwan (Lightner 1996a; Tang *et al.* 2003a; Flegel *et al.* 2004). Infectious hypodermal and haematopoietic necrosis virus or IHHNV-related DNA sequences have been reported in *P. monodon* from Australia, Mauritius, Madagascar and Tanzania (Owens *et al.* 1992; Krabsetsve *et al.* 2004; Tang & Lightner 2006). There is genetic evidence that *P. monodon* broodstock imported to Hawaii from the Philippines were the source of the IHHNV epizootic in the Americas (Tang & Lightner 2002).

Infectious hypodermal and haematopoietic necrosis virus is a small (22 nm), non-enveloped DNA virus with icosahedral symmetry (Bonami *et al.* 1990). It is closely related to small DNA viruses infecting *Aedes* spp. mosquitoes and is currently classified as the tentative species *Penaeus stylirostris densovirus* in the genus *Brevidensovirus* of the family *Parvoviridae* (Tattersall *et al.* 2005). Virions comprise four capsid proteins of 74, 47, 39 and 37.5 kDa and a ~4.1 kb linear ssDNA genome that is primarily encapsidated in negative polarity (Bonami *et al.* 1990; Mari *et al.* 1993; Shike *et al.* 2000). The genome organisation is similar to other brevidensoviruses, comprising three long ORF of which the left ORF encodes replication

initiator motifs and helicase and NTP-binding domains of the major non-structural protein (NS-1). By analogy with other parvoviruses, the middle ORF may encode the N-terminal region of a second non-structural protein (NS-2) that is expressed through an alternative splicing mechanism. The right ORF appears to encode the capsid proteins (Shike *et al.* 2000; Dhar *et al.* 2007). Significant genetic variation between IHHNV sequences has been observed with differences of up to ~15% over a large (2.9 kb) region of the genome. *Penaeus stylirostris* and *P. vannamei* isolates originating from the Americas and Hawaii between 1982 and 1997 have been shown to cluster tightly (>99.6% similarity) and share a high level of identity (99.8%) with a *P. monodon* isolate collected from the Philippines in 1996 (Tang & Lightner 2002). A second genotype identified in *P. monodon* from Thailand and Taiwan shares 96.2% identity with the Philippine genotype (Tang *et al.* 2003a). Two other genotypes have been identified in healthy *P. monodon*; one was detected in shrimp from Tanzania and the other in shrimp from Madagascar, Mauritius and Australia (Tang *et al.* 2003a; Krabsetsve *et al.* 2004). These genotypes share ~90% identity and differ from the Asian genotypes by 8–14%. The African and Australian genotypes have been shown to be integrated into the host genomic DNA. They have not been associated with histological lesions typical of IHHNV infection and experimental transmission studies suggest that they are not infectious for *P. monodon* or *P. vannamei* (Tang & Lightner 2006). However, other parvoviruses are known to cause latent infections, sometimes involving genome integration, and may be re-activated by environmental factors or infection with unrelated DNA viruses (Tattersall *et al.* 2005; Geoffroy *et al.* 2006). Further work is required to determine the relationship between the integrated and free forms of IHHNV and the potential for reactivation of the African and Australian genotypes (Flegel 2006).

Infectious hypodermal and haematopoietic necrosis virus can be transmitted horizontally by injection, ingestion or exposure to infected water (Lightner *et al.* 1983a; Lotz 1997). Shrimp may be asymptomatic carriers of infection and may remain infected for life (Morales-Covarrubias *et al.* 1999; Lightner 2003). Infectious hypodermal and haematopoietic necrosis virus can also be transmitted vertically from infected females, in which the virus has been detected in ovarian tissue and within developing oocytes, and high levels of infection can cause embryo development to abort (Lotz 1997; Motte *et al.* 2003). Selected lines of IHHNV-resistant *P. stylirostris* have been developed (Weppe *et al.* 1992; Tang *et al.* 2000). There is also evidence of interference between IHHNV and WSSV in co-infections, resulting in delayed or reduced mortalities (Tang *et al.* 2003b; Bonnichon *et al.* 2006).

Infectious myonecrosis virus

Infectious myonecrosis (IMN) is the most recently emergent of the major viral diseases of shrimp. It was first recognised in September 2002 in farmed *P. vannamei* at Pernambuco in the state of Piaui in north-eastern Brazil (Lightner *et al.* 2004). By 2004, infectious myonecrosis virus (IMNV) had spread to other regions of north-eastern Brazil and was subsequently detected in *P. vannamei* collected from an outbreak in the Situbondo District of East Java in Indonesia in May 2006 (Senapin *et al.* 2007). It is likely that the virus was introduced to Indonesia in broodstock imported from Brazil. By April 2007, IMNV had reached *P. vannamei* farming regions in south and north-east Sumatra (Ketut Sugama, pers. comm., 2007).

Infectious myonecrosis virus is a small (40 nm) non-enveloped, non-segmented double-stranded RNA (dsRNA) virus with icolahedral symmetry (Poulos *et al.* 2006). Phylogenetic analysis of RNA-dependent RNA polymerase (RdRp) sequences and aspects of the genome organisation and likely transcription strategy indicate that it is most closely related to members of the genus *Giadiavirus* of the family Totiviridae (Poulos *et al.* 2006; Nibert 2007). Virions appear to contain several structural proteins, including a 106 kDa major capsid protein. The 7560 bp dsRNA genome contains two long ORF. ORF1 contains two 2A-like proteolytic cleavage sites and encodes the coat protein and a dsRNA-binding motif. ORF2 overlaps ORF1 and encodes the RdRp. A −1 ribosomal frame-shift at a slippery GU-rich heptamer appears to be facilitated by a predicted pseudoknot in the ORF1/ORF2 overlap to generate a read-through polyprotein (Nibert 2007).

Experimental infection studies have demonstrated that *P. vannamei*, *P. stylirostris* and *P. monodon* are all susceptible to IMNV infection, but only *P. vannamei* was shown to be susceptible to disease with the isolate used (Tang *et al.* 2005). However, as *P. vannamei* and *P. monodon* are now commonly cultured in the same regions of Indonesia (Agus Sunarto, pers. comm., 2008) there remains a risk of adaptation to natural infection in *P. monodon* and associated disease. Infectious myonecrosis virus primarily infects skeletal muscle, but there is also evidence of infection in the lymphoid organs, hindgut and gills, and the virus has been detected in phagocytic cells within the hepatopancreas and heart (Tang *et al.* 2005). There is currently little other published information on the biology of IMNV infection.

Penaeus monodon-type baculovirus

Monodon baculovirus (MBV), also called spherical baculovirus, was first observed in 1977 in adult *P. monodon* that

were laboratory reared in Mexico from postlarvae imported from Taiwan (Lightner & Redman 1981; Lightner *et al.* 1983b). The virus was soon found to occur commonly in *P. monodon* populations from Taiwan, the Philippines and French Polynesia (Lightner *et al.* 1983b) and has subsequently been shown to be enzootic in *P. monodon* throughout its geographical range from east Africa and Madagascar in the west, through the Middle East and the Indian subcontinent, South-East and East Asia, to Australia and islands of the South Pacific. It has also been reported in many countries to which *P. monodon* shrimp have been introduced, including West Africa, the Mediterranean, Tahiti, Hawaii, North and South America and the Caribbean (Lightner 1996b; OIE 2006). No systematic study of MBV strains has been conducted, but there is anecdotal evidence for the existence of different types and it has recently been reported that PCR tests designed to detect isolates from East Asia and South-East Asia do not detect African strains (OIE 2006). Monodon baculovirus or MBV-like viruses have been described in several other penaeid shrimp species, including *Penaeus merguiensis*, *Penaeus plebejus* Hess, 1865 and *Metapenaeus bennettae* Racek & Dall, 1965 from Australia (Lester *et al.* 1987; Doubrovsky *et al.* 1988; Spann & Lester 1996), *Penaeus indicus* Milne-Edwards, 1837 from Vietnam (Hao *et al.* 1999), *Penaeus penicillatus* Alock, 1905 from Taiwan (Chen *et al.* 1989a) and *P. indicus*, *Metapenaeus monoceros* Fabricius, 1798 and *Metapenaeus elegans* De Man, 1907 from India (Vijayan *et al.* 1995; Manivannan *et al.* 2004). However, attempts to infect *P. californiensis*, *P. stylirostris* and *P. japonicus* by experimental exposure *per os* have not been successful (Lightner & Redman 1981; Fukuda *et al.* 1988) and observations of infection in species other than *P. monodon* have relied primarily on histology, electron microscopy and/or positive PCR reactions. A more detailed molecular analysis, together with bioassays, should be conducted to establish with more confidence the susceptible host range of MBV.

Monodon baculovirus can cause high mortalities in larvae (protozoea and mysis) and early postlarval stages (Natividad & Lightner 1992a). There are varied reports of the effect of MBV on the survival of juvenile and adult stages. Although MBV was implicated in the collapse of the *P. monodon* culture industry in Taiwan in 1987/1988 (Chen *et al.* 1989b; Lin 1989) and as the cause of mortalities in pond-reared juveniles in Indonesia and Malaysia in the mid-1980s (Nash *et al.* 1988), concomitant bacterial, parasitic or viral infections are now thought to be the likely cause of disease (Lightner *et al.* 1987; Chen *et al.* 1989a). Persistent infections in juvenile and adult stages occur commonly and appear to be tolerated without apparent signs of disease (Chen *et al.* 1989a; Fegan *et al.* 1991; Natividad & Lightner 1992b). Environmental or other stress has been identified as a significant factor in disease associated with MBV (Lightner *et al.* 1983b; Chen *et al.* 1989a; Fegan *et al.* 1991; OIE 2006). Co-infection with other viruses, such as HPV, IHHNV and WSSV, has also been reported to occur commonly (Manivannan *et al.* 2002; Chayaburakul *et al.* 2004; Oanh *et al.* 2005; Natividad *et al.* 2006) and multiple infections may result in retarded growth (Chayaburakul *et al.* 2004; Flegel *et al.* 2004).

Monodon baculovirus is a large, bacilliform dsDNA virus that is classified as the tentative species Penaeus monodon nuclear polyhedrosis virus (PemoNPV) in the genus *Nucleopolyhedrovirus* of the family *Baculoviridae* (Theilmann *et al.* 2005). Enveloped virions (~325 nm × 75 nm) mature in the nucleus and contain nucleocapsids (~250–300 × 45–70 nm) of similar shape, but with one conical and one blunt end (Lightner *et al.* 1983b; Mari *et al.* 1993; Vickers *et al.* 2000). Naked nucleocapsids of similar dimensions are also detected in the cytoplasm and attached to the nuclear envelope, most likely in traverse from the plasma membrane following adsorption and endocytosis of the infecting virions (Johnson & Lightner 1988; Vickers *et al.* 2000). In the late stages of infection, virions in the nucleus are observed within spherical inclusion bodies comprising a paracrystalline network of small polyhedrin subunits, each ~20 nm in diameter (Lightner *et al.* 1983b; Mari *et al.* 1993). The spherical shape of these inclusion bodies is the origin of the alternative common name of MBV (i.e. spherical baculovirus), although by scanning electorn microscopy they appear polyhedral (Flegel 2006). The 58 kDa polyhedron protein displays a high level of amino acid sequence identity with the polyhedron protein of *Autographa californica* MNPV (AcMNPV), the type species of the genus *Nucleopolyhedrovirus* (Chang *et al.* 1993; Lu *et al.* 1993; Mari *et al.* 1993). There are no published data available on the polypeptide composition of MBV virions or on the molecular characterisation of the viral DNA.

Monodon baculovirus can be transmitted by co-habitation, feeding or exposure to homogenates of infected tissue (Lightner & Redman 1981; Natividad & Lightner 1992a; Paynter *et al.* 1992). All life stages, except eggs and nauplii, are susceptible to MBV infection. Infection occurs in the epithelial cells of the hepatopancreatic tubules and anterior midgut from which virus particles and inclusion bodies are released to enter the intestinal tract via the hepatopancreatic lumen (Lightner *et al.* 1983b; Johnson & Lightner 1988; Chen *et al.* 1989a). As for other baculoviruses, faecal–oral transmission is the most likely route of infection, with polyhedron providing environmental protection of virus released in inclusion bodies. Monodon baculovirus can be transmitted from broodstock to progeny, but there is no evidence of transovarial transmission and the evidence indicates that

infection occurs by faecal contamination of eggs during spawning (Chen *et al.* 1992; OIE 2006). Good husbandry practices, including washing of fertilised eggs or nauplii with filtered seawater, formalin and iodophores, can be effective in breaking the transmission cycle (Chen *et al.* 1992).

Factors contributing to disease emergence in shrimp aquaculture

The ecology of viral disease emergence

The emergence and spread of new or previously quiescent viral diseases is an increasingly common phenomenon that has drawn serious attention in recent years. Much of this interest has been stimulated by the emergence of a range of human diseases of animal origin, such as HIV-AIDS, Ebola and SARS, and recognition of the alarming prospect that human-to-human transmission of avian influenza could precipitate a devastating global pandemic (Webby & Webster 2003). However, there are many other examples of emerging viral diseases in humans, animals and plants, and there is now a growing understanding of the factors that cause disease emergence based on an appreciation of viruses as an integral part of the ecosystem (Morse 1993, 1995; Brown 1997; Mayer 2000).

Viruses are obligate parasites that normally exist in a biological cycle that involves a stable ecological association with one or more hosts (Wilcox & Gubler 2005). A virus must achieve efficient replication and progressive transmission of infection in an environment that is constantly changing because of individual differences in host genetics and the immune response of the host. These environmental variations are met by the virus by ongoing competitive selection of the most efficient mutants with suitable variant phenotypes, or through an inherent capacity for evasive behaviour (Holland 2006). In this way, viruses achieve a dynamic equilibrium that sustains a natural ecological balance with their host(s). This equilibrium does not a priori require the induction of pathology or disease. Many viruses infecting humans, animals or plants do not normally cause disease. Indeed, the absence of pathology or mortality may provide the virus with a better opportunity for replication and efficient ongoing transmission. Covert infections without disease commonly occur for viruses that have established a stable ecological niche (Hyatt *et al.* 2004; Walker 2004).

New diseases usually emerge as a consequence of a major shift in the environment of the virus that upsets this natural balance and leads to significant changes in the biology of infection (Morse 1993; Daszak *et al.* 2001; Hyatt *et al.* 2004). Such disturbances usually result, directly or indirectly, from anthropogenic influences on the ecosystem. Human social and industrial activities that have been implicated in disease emergence include increased urbanisation, the globalisation of trade, cultural and behavioural changes, the population of new environments, climate change, and the establishment and growth of new industries or industrial practices (Brown 1997; Mayer 2000). Changes of this nature can allow a virus, through necessity or opportunity, to occupy a new niche in which the ecological balance is temporarily lost (Hyatt *et al.* 2004).

There are two fundamentally different mechanisms by which new infectious diseases may emerge as a result of ecological imbalance. The first involves viruses (or other micro-organisms) that have a natural association with a particular host, but do not normally cause disease. Through a shift in the natural ecological balance, a normally benign agent may become pathogenic in its natural host. Factors with the potential to cause such an imbalance include immunosuppression or reduced resistance to disease (induced by environmental stressors, chemicals, radiation or other infections), enhanced replication (induced by a shift in ambient temperature) or modified cell tropism (induced by a different route of exposure). The second mechanism involves a more dramatic change in viral ecology associated with cross-species transmission. This complex process, by which a virus establishes in a new host, comprises four stages: (i) contact between a pathogen and a new, potentially susceptible host; (ii) transmission to and replication of the pathogen in the new host; (iii) sustained transmission of the pathogen between individuals of the new host; and (iv) genetic adaptation of the pathogen to achieve a new ecological balance in the new host (Childs *et al.* 2007). In the case of zoonotic diseases (human diseases derived from animals), the process by which cross-species transmission occurs is termed 'spill-over' (Childs *et al.* 2007). This requires that a virus is capable of completing the replication cycle in the new host and will be critically dependent on the availability of a suitable molecular landscape, including receptors and replication co-factors provided by the new host cell. As the molecular landscape is most similar for closely related species, spill-over is most likely to occur between phylogenetically related organisms. As the process of adaptation to a new host is assisted by genetic variability in the virus through mutation and/or recombination, it is also more likely that RNA viruses and some small DNA viruses with a high intrinsic replication error frequency will be involved in cross-species transmission rather than large DNA viruses (Holland 1993). These likely trends are confirmed by the recent history of disease emergence involving cross-species transmissions (Morse 1993).

Disease emergence in shrimp aquaculture

Shrimp aquaculture is an important seafood industry that has created new employment and contributed significantly to socio-economic development in many countries in Asia and the Americas. However, large-scale shrimp aquaculture also represents a change in human activity with the potential for very significant impacts on the environment and the ecology of pathogens. The displacement of shrimp from their natural offshore or estuarine environments to terrestrial, earthen ponds provides opportunities for exposure to pathogens that they may not naturally encounter. The use of artificial or alternative live feeds presents risks of inadvertent cross-species transmission of contaminating pathogens. The culture conditions can often be stressful, particularly when water quality is poor or variable, compromising the natural defences of the shrimp and their capacity to contain infection. High stocking densities facilitate rapid transmission of infection and disease (Soto & Lotz 2001). Aquaculture has also led to a large and rapidly growing trade in live aquatic animals and fresh or frozen shrimp product, providing ready pathways for the rapid transboundary spread of disease (Lightner 1996b; Renault 1996; Yoshimitsu 1996). The emergence and spread of a growing array of shrimp diseases is a natural and predictable consequence of these practices.

Shrimp pathogens can be considered to fall into two categories: (i) those for which shrimp are the natural host in which they have existed in for, perhaps, millions of years; and (ii) those that have been introduced to shrimp as a result of a recent cross-species transmission. Of the major viral pathogens of marine shrimp, WSSV, TSV and IMNV have almost certainly emerged through cross-species transmission. Each of these viral pathogens appears to have spread from a single focal origin in Asia or South America and had not been detected in farmed or wild shrimp prior to disease emergence. Each also appears to represent a single evolving genetic lineage that reflects its focal origin. However, neither the original sources of infection nor the mechanism of first exposure have been determined conclusively for these important pathogens.

White spot syndrome virus has a very broad susceptible host range in decapod crustaceans and is now detected commonly in wild shrimp and crabs in many locations where shrimp are farmed. However, reports of WSSV in crustaceans outside of shrimp-farming regions are rare and the virus has not been detected in surveys of crustaceans in Australia where there is no WSSV or WSD in farmed shrimp (East *et al.* 2004). The apparent absence of widespread WSSV infection in wild decapod crustaceans prior to the emergence of WSD in 1992, despite their susceptibility, suggests that the original source was a non-decapod invertebrate from which the virus has 'crossed' and adapted to shrimp and other crustaceans. Alternatively, WSSV may have been introduced to shrimp from a decapod host that is relatively isolated from other susceptible species and in which disease does not occur. There is evidence of the rapid evolution of WSSV since its emergence in shrimp and some strains contain large deletions of the DNA sequence, indicating that these regions of the genome are non-essential for efficient replication and transmission in shrimp (Dieu *et al.* 2004; Marks *et al.* 2005). The high rate of evolution and the severity of disease are characteristics often displayed by infectious agents that have undergone a temporary loss of ecological balance as a result of cross-species transmission (Steinhauer & Holland 1987).

The mechanism of yellow head disease emergence in farmed shrimp is less clear. Like WSSV, YHV was not known prior to its appearance in Thailand in 1990 (Pasharawipas *et al.* 1997). However, YHV has a far more limited susceptible host range than WSSV, primarily infecting penaeid and palemonid shrimp, and it is now known that YHV is only one of six closely related genotypes in the yellow head complex that occur commonly in healthy *P. monodon* throughout its natural distribution in Africa, Asia, Australia and the Pacific (Wijegoonawardane *et al.* 2008a). Each of the other genotypes is either non-pathogenic or far less virulent that YHV and there is evidence that some genotypes cluster geographically, suggesting the evolution of separate lineages in geographical isolation prior to aquaculture-associated shrimp translocations. This suggests that *P. monodon* is very likely to be the natural host of other genotypes in this complex and may also be the natural host of YHV. If so, the explosive emergence of yellow head disease in Thailand in 1990 is perplexing. One possibility is that a mutation or other genetic process created a highly virulent strain in a formerly non-pathogenic virus. However, this would imply that non-virulent strains of the virulent YHV genotype should also continue to exist in *P. monodon* in Thailand and there has been no evidence of this to date. Another possibility is that yellow head disease has emerged in *P. monodon* as a result of cross-species transmission from another similar shrimp species, perhaps a metapenaeid, in which it does not cause disease. If so, the natural host may be an important reservoir of YHV infection and a continuing source of spill-over into farmed shrimp. Further investigation of the genetic basis of virulence and the susceptibility of other shrimp species may unravel the obscure origins of yellow head disease.

Infectious hypodermal and haematopoietic necrosis virus is almost certainly a natural inhabitant of *P. monodon* in which it does not appear to cause disease. It occurs at high prevalence throughout the natural geographical range of *P. monodon* and at least one genotype appears to have integrated into the host genome

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(Tang & Lightner 2006). There is also evidence of geographical clustering of IHHNV genotypes in Africa and Australia (Tang *et al.* 2003a; Krabsetsve *et al.* 2004). However, the history of disease emergence for IHHNV is also associated with cross-species transmission, in this case as a result of exposure to Western Hemisphere penaeid shrimp species (*P. stylirostris* and *P. vannamei*) in which it does cause disease. Other viruses, such as MBV, Mourilyan virus (MoV) and hepatopancreatic parvovirus (HPV), also appear to be natural infections of penaeid shrimp (Flegel 2006), although there is some evidence that both HPV and IHHNV may have distant evolutionary origins in insects (Roekring *et al.* 2002). Shrimp viruses usually cause low-level unapparent infections in their natural host and are commonly transmitted vertically. Disease emergence is usually the result of stress, high stocking density or other aspects of the culture system that disturb the natural cycle of infection.

Future disease emergence risks

Aquaculture practices provide abundant opportunities for the emergence and spread of disease and it is highly likely that new diseases will continue to emerge with significant impacts on production. The risk of future disease emergence is directly related to the opportunities created for shrimp to encounter new pathogens as the industry continues to expand and evolve. Live or frozen feeds, particularly those of crustacean or other invertebrate origin, present a particularly high risk of successful pathogen transfer because of the potentially high dose, the route of exposure and the higher likelihood of trans-species adaptation. Polyculture of shrimp with other aquatic species, particularly invertebrates, also presents a high risk of pathogen transfer. The risk of exposure to new pathogens from natural sources in the environment will increase as shrimp aquaculture expands into new locations. It is perhaps not surprising that three of the major pathogens (WSSV, YHV and TSV) first emerged in countries that were leaders in shrimp culture development. Although the source of IMNV remains obscure, it is also not surprising that this virus first appeared at a time that the shrimp culture industry in Brazil was in a phase of rapid expansion. Africa now looms as a new and exciting opportunity for aquaculture development, but it is also the site of rich biodiversity and has been the source of emergence and re-emergence of many important animal and human pathogens. The expansion of shrimp aquaculture into Africa may well provide opportunities for cross-species transmission that will precipitate the emergence and spread of a new array of virulent shrimp pathogens.

As poikilotherms, marine invertebrates are highly sensitive to changes in ambient water temperatures and global warming and associated extreme weather events are likely to impact on many aspects of their ecology and the emergence and spread of disease (Marcogliese 2008). Climate change may also influence the locations and distribution of suitable farming sites, rendering some sites vulnerable to rising sea levels and severe weather, and placing other areas within the suitable climatic zone for efficient production. It could be argued that, for viruses such as WSSV, higher ambient water temperatures will reduce disease risks (Rahman *et al.* 2006). However, the disruptive environmental effect of rising global temperatures will undoubtedly present new opportunities for the emergence of new pathogens and lead to a generally greater risk of disease.

Impacts of disease in shrimp aquaculture

Socio-economic impacts

The emergence and spread of disease in shrimp aquaculture has caused economic impacts on a scale rivalling that of many of the major emerging diseases of livestock and humans. It was estimated in 1996 that annual disease-related losses in shrimp farming globally were ~US$3000 million or 40% of the total production capacity of the industry (Israngkura & Sae-Hae 2002). The most devastating impacts have followed the first emergence of each of the major pathogens, which was marked either by a pause in industry expansion or a dramatic drop in the level of production (Fig. 2). In Taiwan in 1987/1988, an epizootic attributed to MBV, but likely to have been a combination of disease and environmental degradation, saw a 70% fall in production and near-total collapse of the industry (Lightner 1996a). It has been estimated that the combined economic losses as a result of IHHNV and TSV on wild and cultured shrimp fisheries in the Americas, from first emergence to 2001, totalled US$1.5–3.0 billion (Hasson *et al.* 1999a; Lightner 2003) In China in 1993, the emergence of WSSV resulted in an estimated production loss of US$420 million in a single season (Wei 2002). In Thailand, annual production losses peaked at US$650 million in 1994, falling marginally to US$600 million in 1997 (Timothy Flegel, pers. comm., 1998). Production losses of a similar magnitude have occurred in many shrimp-farming countries in Asia (Walker 2004; Bondad-Reantaso *et al.* 2005) and it has been estimated that the impact of WSSV throughout Asia in the first 10 years after its emergence in 1992 was of the order of US$4–6 billion (Lightner 2003). In the Americas, the emergence of WSSV in 1999–2000 also saw a rapid decline in production with immediate losses estimated at more than US$1 billion. The industry in many countries has never fully recovered (Lightner 2003).

Although losses resulting from disease have subsided since the period of peak impact during the 1990s, WSSV

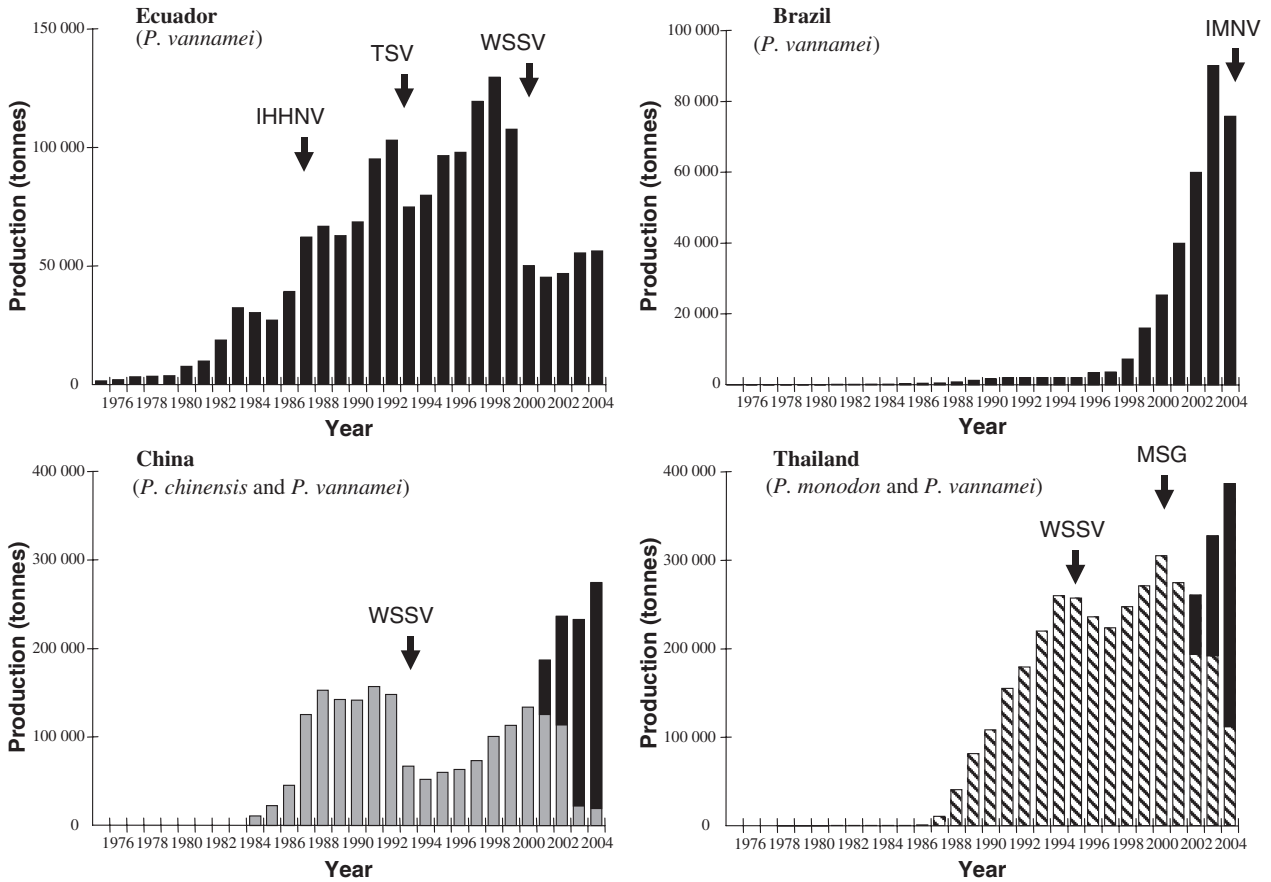

Figure 2 Effect of disease emergence on farmed shrimp production in countries in South America and Asia. IHHNV, infectious hypodermal and haematopoietic necrosis virus; IMNV, infectious myonecrosis virus; MSG, monodon slow growth; TSV, Taura syndrome virus; WSSV, white spot syndrome virus. ■, *P. vannamei*; ▒, *P. chinensis*; ▨, *P. monodon*.

and other newly emerging pathogens continue to impact severely, particularly on the small-holder farmers that comprise much of the industry in major producing countries such as Vietnam, Indonesia and India. Lost production as a result of disease impacts on the income and food security of small-holder farmers, as well as job security for workers on larger farms and in hatcheries, feed mills and processing plants (Bondad-Reantaso *et al.* 2005). As shrimp aquaculture supplies seafood markets in the USA, Europe and Japan, disease also impacts directly on export revenue with flow-on impacts on socio-economic development in the sustaining local communities in tropical coastal regions of many developing countries in Asia and the Americas.

Environmental impacts

Assessing the environmental impacts of aquatic animal diseases is a complex and difficult task and rigorous scientific assessments have rarely been conducted. Arthur and Subasinghe (2002) noted that potential impacts of aquatic animal diseases on wild populations and biodiversity could include changes in predator and prey populations, changes in host abundance, reduction in intra-specific genetic variation, local exterpations and extinctions. However, despite the widespread occurrence of disease in shrimp ponds and evidence that major pathogens occur at relatively high prevalence in wild stocks, there is little available evidence that viral diseases have led to a decline in wild shrimp populations in Asia. This may be because of a combination of low population densities and the likely absence of extreme environment stressors that promote disease emergence in the farming system. It may also reflect a lack of objective data. Nevertheless, there is circumstantial evidence that IHHNV has impacted on populations of wild shrimp in the Americas, with *P. stylirostris* fisheries landings in the Gulf of Mexico falling to the lowest on record following the introduction of the virus in 1987 (Morales-Covarrubias *et al.* 1999), and the risks of serious environmental consequences of known or newly emerging viral diseases should not be underestimated.

The indirect environmental impacts of shrimp disease are more clearly evident. Successive crop failures have led to the abandonment of affected ponds and the relocation of farming operations to new areas, compounding the problem of coastal zone and mangrove habitat degradation (Dierberg & Kiattisimkul 1996; Paez-Osuna 2001). Abandoned ponds are rarely re-habilitated for farming and either remain idle or are converted to non-agricultural land uses, such as housing or manufacturing (Szuster 2006). Crop failures in disease-prone coastal areas have also led to the development of low-salinity husbandry practices, resulting in the conversion of irrigated paddy fields and the expansion of shrimp farming into inland areas. There have been concerns that this practice could lead to soil salinization, water pollution and increased competition between agriculture and aquaculture for water resources (Miller *et al.* 1999; Pongnak 1999; Ali 2006). In Thailand, such environmental concerns have led to a national ban of low-salinity shrimp farming in inland areas (Szuster 2006).

Difficulties in maintaining reliable production of the major endemic farmed species in Asia have driven a major shift to exotic species. The white Pacific shrimp (*P. vanammei*), which is native to the west coast of the Americas, has been bred in captivity for many generations from stocks that are free of most known pathogens. Since 1998–1999, white Pacific shrimp have been used for large-scale commercial production in Asia, rapidly becoming the major production species (Fig. 2). In 2004, it was estimated that 28 000 specific-pathogen-free (SPF) *P. vannamei* broodstock were exported from the hatcheries in Hawaii to Asia each month, translating to a possible 6 billion nauplii and 3 billion postlarve (Briggs *et al.* 2004). In 2006, *P. vannamei* production in Asia reached 1.81 million tonnes or 64.8% of total farmed shrimp production (FAO 2008). This may well represent the largest trans-continental relocation of a single species in the history of the planet. Although *P. vannamei* are now commonly recovered off the coast of several Asian countries, and concerns have been expressed about their impact on native habitats (Arthington & Bluhdorn 1996; Szuster 2006), there has been little evidence to date of displacement of endemic species and the environmental consequences of this mass translocation remain uncertain.

Another potential indirect environmental impact is the use of antibiotics, disinfectants and other chemicals to prevent disease or to treat shrimp during disease outbreaks (Boyd & Massaut 1999; Paez-Osuna 2001; Holmstrom *et al.* 2003; Cabello 2006; Biao & Kaijin 2007). A study conducted in 2000 indicated that 74% of shrimp farmers along the Thai coast used antibiotics in shrimp pond management. At least 13 different antibiotics were in use, mostly as prophylactics (Holmstrom *et al.* 2003). Subsequent attention to chemical residue testing and bans by importing countries has significantly reduced chemical use in shrimp-farming operations. Although disinfectants, such as iodophores and hypochlorite, can be used to inactivate viruses contaminating surfaces (Danner & Merrill 2006), there are no commercially available prophylactics or treatments that have been shown to be effective against the major viral pathogens in infected shrimp.

Systemic improvements in response to disease

The impacts of disease on shrimp production and the environment, and the recognition that disease risks are inextricably linked to environmental conditions, have led to systemic improvements in the structure and regulated operation of the industry by international agencies, governments and farmers. Since the mid-1990s, when disease impacts were most severe, there have been significant technological improvements, including sensitive pathogen screening tests and breeding programs for SPF and specific-pathogen-resistant (SPR) stock, and improvements to operational practices, such as structured biosecurity in hatchery and farming systems, and the adoption of Hazard Analysis and Critical Control Points (HACCP) principles and traceability and certification procedures throughout the production cycle (Jahncke *et al.* 2001; Lightner 2005). There has also been improved communication and cooperation between industry, environmental groups and governments, leading to international agreements on trading practices, increased industry regulation and the development of codes of conduct for sustainable development (Bondad-Reantaso *et al.* 2005; FAO, NACA, UNEP, WB, WWF 2006). However, disease continues to impact significantly on production, particularly on smallholder farmers, and health, environmental and product safety issues remain the highest priorities for the industry globally.

Current disease management strategies and their effectiveness

Principles of disease management in shrimp aquaculture

As invertebrates, shrimp lack the key components of the vertebrate adaptive immune response (e.g. immunoglobulins, major-histocompatibility-complex (MHC) antigens, T-cell receptors) that provide a versatile mechanism for natural protection and allow for conventional vaccination against viruses (Arala-Chaves & Sequeira 2000; Johnson *et al.* 2008). Strategies for health management in shrimp aquaculture are therefore based primarily on the principles of pathogen exclusion and the avoidance of environmental conditions that induce stress, stimulate viral replication or facilitate disease transmission. Effective

pathogen exclusion practises require attention to all points in the shrimp production cycle, from spawning to harvest, at which viruses may be encountered or recycled into the environment (Fegan & Clifford 2001). For *P. vannamei* and *P. stylirostris*, the availability of SPF and SPR stock, bred in captivity under biosecure conditions, has provided a source of nauplii and postlarvae from which the major pathogens have been excluded (Lightner 2005). However, for most farmed shrimp species, including *P. monodon*, SPF stock are not widely available and broodstock continue to be sourced from the wild. As many of the viral pathogens of concern are common in wild stocks, stringent viral screening of broodstock and/or seed is an essential pathogen exclusion practice. On-farm biosecurity includes pond preparation by drying and sunlight exposure to eliminate residual pathogens and aquatic crustaceans in soil, filtration and disinfection of pond water prior to stocking and prior to exchange during grow-out, careful monitoring and control of water quality, fencing to exclude potential carrier crustaceans, netting to exclude birds that may feed on and distribute moribund shrimp, physical separation of pond implements, and treatment of effluents to prevent discharge of contaminated pond water into the environment (Fegan & Clifford 2001; Vanpatten *et al.* 2004; Subasinghe 2005). Monitoring of shrimp during grow-out for early signs of disease, such as lethargy, gill fouling, unusual swimming behaviour, feeding patterns or colouration, and regular histological examination for evidence of infection is also commonly used to varying degrees by farmers.

Disease management for medium–large semi-intensive farms

The implementation of effective disease management by medium–large semi-intensive farmers has impacted very significantly on productivity and delivered a range of other benefits to farmers and the community. These include less reliance on wild fisheries as a source of broodstock, better management of land and water usage and treatment of effluents, decreased use of chemicals and antibiotics, and improved quality of record keeping from stocking to market. Biosecurity is now an integral aspect of farm and hatchery management for many medium–large farms and has largely been responsible for the recovery of the industry that has seen the volume of farmed shrimp production increase more than threefold from 2000 to 2006 (FAO 2006). Although this has been driven by the availability of SPF *P. vannamei* in China, Thailand, Indonesia and Vietnam, successful farming operations using SPF stock have been underpinned by, and continue to rely on, a more stringent approach to biosecurity.

Disease management for poorer small-holder farmers

For low-income, small-holder farmers (<1 ha holding), who constitute a very significant sector of the industry in some Asian countries, limited education and lack of resources to implement comprehensive disease management practices are major barriers to achieving reliable production (Padiyar 2009). White spot syndrome virus continues to impact severely on small-holder shrimp farmers and the importation of non-SPF *P. vannamei* from the western hemisphere has seen the arrival of new viral pathogens, such as TSV and IMNV. These are now causing significant production losses in several countries and have the potential for further geographical spread and to infect other shrimp species (Chang *et al.* 2004; Srisuvan *et al.* 2005; Tang *et al.* 2005; Phalitakul *et al.* 2006; Senapin *et al.* 2007). Furthermore, although SPF *P. vannamei* offer some advantage as a source of high-quality seed, they remain susceptible to infection by WSSV, YHV, TSV and other pathogens that are endemic throughout extensive farming systems in Asia.

In view of the importance of aquaculture in maintaining food security and sustaining livelihoods in poor rural and coastal communities, there has been growing international attention to the development and implementation of practical, affordable and effective measures to reduce disease and environmental impacts for small-holder farmers. 'Better Management Practices' (BMP) have been developed by international teams of specialists in conjunction with industry and government. These BMP are based on published scientific information on the biology of infection, epidemiological studies to identify disease risk factors, empirical observations and successful industry practices, and provincial, national and international regulatory requirements (Subasinghe 2005; Mohan *et al.* 2008). In alignment with the International Principles for Responsible Shrimp Farming (FAO, NACA, UNEP, WB, WWF 2006), BMP are designed to support small producers to: (i) increase efficiency and productivity by reducing the risk of shrimp health problems; (ii) reduce or mitigate the impacts of farming on the environment; (iii) improve food safety and the quality of shrimp farm product; and (iv) improve the social benefits from shrimp farming and its social acceptability and sustainability (Mohan *et al.* 2008). Better Management Practices have been developed to provide guidance at all stages of the production cycle from hatchery operation to pond bottom preparation and water management prior to stocking, seed selection and stocking, and post-stocking management of ponds (Subasinghe & Bondad-Reantaso 2006; ADB, ACIAR, AwF, BRR, DKP, FAO, GTZ, IFC, MMAF, NACA, WWF 2007). Better Management Practices are often voluntary practices, but can also be used as a basis for local

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regulations or certification programmes. A critical aspect of the introduction of BMP has been the role of farmer groups and their effective linkage to the public sector (Mohan *et al.* 2008). The provision of science-based information to farmer groups through effective networking and communication is an important key to success (Corsin *et al.* 2007). The successful application of BMP in the shrimp-farming sector in Asia has led to the development of BMP for small-scale farming of other aquatic species.

The availability of high-quality seed is a critical factor in reducing the risk of crop failure during grow-out. The increasing use of SPF *P. vannamei* broodstock as a source of seed has significantly improved seed quality for medium–large semi-intensive farms, but small-scale farmers continue to rely principally on seed obtained from wild spawners or other sources of non-SPF boodstock. Polymerase chain reaction screening of broodstock, nauplii and/or postlarvae prior to stocking to eliminate the major viral pathogens, such as WSSV, is therefore a critical requirement to reduce the risk of disease during grow-out (Hsu *et al.* 1999; Mushiake *et al.* 1999; Withyachumnarnkul 1999). This has been facilitated by the commercial availability of an increasing number of PCR test kits and the proliferation of government and private PCR service laboratories in shrimp-farming regions. However, recent national proficiency testing surveys in India, Indonesia and Vietnam indicate that less than 50% of the participating laboratories were able to correctly identify standard positive and negative samples (Walker and Gudkovs, unpublished data, 2007). This deficiency is being addressed through measures such as training and the implementation of national registration and accreditation programs for PCR service laboratories. A more widespread adoption of simple, inexpensive and rapid pond-side test formats may also prove to be a very useful adjunct to PCR screening by eliminating heavily infected postlarvae and providing small-scale farmers with more control over seed quality (Sithigorngul *et al.* 2006; Patil *et al.* 2007).

Transboundary pathogen movements

One of the most difficult biosecurity issues facing the shrimp-farming industry is transboundary movement of pathogens through trade in live shrimp (Subasinghe & Bondad-Reantaso 2008). The rapid spread of WSSV and TSV within countries and regions, and between hemispheres, has been attributed primarily to the movement of live broodstock and seed. Shrimp seed (nauplii and postlarvae) are commonly transported freely within countries and there is a prolific international and transcontinental trade in both wild-caught and domesticated shrimp broodstock. Each of the major viral infections of shrimp can occur as low-level persistent infections in apparently healthy stock. Although PCR screening can be an effective method of detection, the vast numbers of animals being moved daily, the inadequacy of international and internal quarantine inspection capabilities, illegal trading in unscreened stock and the substitution of unscreened stock for certified SPF stock all contribute to a very high ongoing risk of transboundary disease spread. The potential for rapid translocation was first realized during the explosive WSSV emergence in Asia between 1992 and 1994, and has been demonstrated most recently by the emergence and spread of IMNV since 2002. The transfer of TSV and IMNV to Asia from the Americas has been a direct consequence of the importation of large numbers of *P. vannamei* broodstock, not all of which have been 'SPF', and the subsequent use of pond-raised progeny in local breeding programs. This has occurred despite the knowledge and costly experience of previous pandemic disease spread.

The transboundary spread of viruses in frozen uncooked shrimp has also been an issue of concern (Durand *et al.* 2000). A total of 4.5 million tonnes of commodity shrimp were traded globally in 2006, ~75% of which was transported from Asia to Europe and the USA (FAO 2006). Several studies have demonstrated the presence of live WSSV and YHV in imported frozen commodity shrimp and bait shrimp (Nunan *et al.* 1998; Durand *et al.* 2000; McColl *et al.* 2004; Hasson *et al.* 2006) and identified risks to both farming and the environment. The Sanitary and Phyto-sanitary (SPS) Agreement of the World Trade Organization provides the basis on which member countries may promote free trade without increasing the risk of the introduction and spread of aquatic animal pathogens. To limit the potential for future disease spread, it is essential that national aquatic animal health policies and strategies provide the basis for the implementation of measures specified in the SPS and associated standards (OIE 2008).

Future prospects

New technologies

There are indications that at least partial solutions to problems of health management in shrimp aquaculture may lie in new technologies. Although conventional vaccination, mediated by antibodies, cytokines and lymphocytes, is not possible because of the lack of a complex adaptive immune system, there is growing evidence that shrimp use a range of mechanisms to respond to viruses and these may be exploited to prevent disease or limit the spread of infection.

The first suggestion of a form of induced anti-viral immunity in shrimp was obtained when shrimp surviving natural or experimental WSSV infection were shown to

remain resistant to experimental WSSV challenge for up to 4 months (Venegas *et al.* 2000; Wu *et al.* 2002). It was subsequently demonstrated that the injection of formalin-inactivated WSSV or recombinant WSSV envelope proteins (VP26, VP28, VP19, VP292 or VP466) can also induce protection against experimental challenge, with VP28 apparently providing the most potent protection (Namikoshi *et al.* 2004; Witteveldt *et al.* 2004a; Vaseeharen *et al.* 2006; Ha *et al.* 2008). Protection can also be induced by oral delivery of formalin-inactivated virus (Bright Singh *et al.* 2005) or recombinant WSSV envelope proteins expressed from bacterial plasmids or baculovirus vectors (Witteveldt *et al.* 2004b; Ha *et al.* 2008). The injection of DNA plasmids expressing envelope proteins VP28 and VP281 also induces protection, but DNA 'vaccines' expressing the nucleocapsid proteins VP15 or VP35 have failed to protect (Rout *et al.* 2007; Kumar *et al.* 2008). Relative protection levels using WSSV envelope proteins delivered directly or expressed from DNA are usually in the order of 50–90%, but immunity is relatively short lived, with a reported duration of approximately 4–7 weeks depending on the formulation. The mechanism by which WSSV proteins induce protection in shrimp is not well understood. Recent evidence suggests that anti-viral phagocytosis in shrimp may be controlled by a Rab GTPase that is upregulated in WSSV-resistant shrimp and forms a complex with β-actin, tropomycin and the WSSV envelope protein VP466 (Wu & Zhang 2007; Wu *et al.* 2008). It has also been suggested that autophagy, a homeostatic mechanism by which cells digest and re-cycle macromolecules and organelles by lysosomal digestion, may be involved (Johnson *et al.* 2008).

RNA interference (RNAi) is another anti-viral strategy that has been shown to have the potential for application in shrimp. RNAi is a mechanism of post-transcriptional gene silencing by which dsRNA, delivered into cells, is digested into small ($\sim$21–23 nt) fragments that form a complex with cellular proteins that recognises, targets and guides the digestion of mRNA containing homologous sequences (Fire 1999; Tijsterman & Plasterk 2004; Su *et al.* 2008). It has been shown that RNAi is active in shrimp and that injection of dsRNA or short interfering (si)RNA homologous to viral mRNA can effectively block WSSV, TSV and YHV infection (Robalino *et al.* 2005; Yodmuang *et al.* 2006; Xu *et al.* 2007). Inhibition of viral infection can be complete when shrimp are challenged within 1 day of the dsRNA treatment by injection, but the long-term durability of the protective effect has not yet been adequately assessed. A non-specific anti-viral immunity has also been induced in shrimp by long dsRNA or siRNA, but targeting to specific viral sequences, particularly essential replication genes, is far more potent (Robalino *et al.* 2004; Westenberg *et al.* 2005).

For each of these potential anti-viral strategies, the core remaining problem is the development of practical and cost-effective methods for application on a commercial scale. For protein-based 'vaccines', the potency of oral delivery suggests that application in shrimp feed may be practicable, but there is a lack of published experimental evidence to demonstrate the efficacy of this approach. The most obvious potential application of RNAi is in the therapeutic treatment of infected broodstock in hatcheries to generate virus-free progeny, either for farm production or domesticated breeding programs. The prolongation of anti-viral effects by microparticle delivery of stable DNA constructs expressing envelope proteins and/or hairpin RNA also requires further exploration.

Development and implementation of national aquatic animal health strategies

The ongoing difficulties in the management of shrimp disease are reflected in other sectors of the aquaculture industry, particularly in Asia, which accounts for >90% of global aquaculture production (FAO 2006). It is now widely recognised that there is a need to address deficiencies in aquatic animal health management and to support sustainable aquaculture development through the implementation of national strategies that parallel those adopted in many countries for terrestrial animal diseases. These include such measures as the identification of national authorities with the responsibility and competence to supervise the implementation of aquatic animal health measures, national lists of reportable diseases, national aquatic animal health committees with the responsibility for coordinating and advising governments on disease issues, national surveillance and disease reporting programs, national contingency programs for dealing with disease emergencies, competent national and regional diagnostic facilities and planned programs for building capability and capacity (Bondad-Reantaso *et al.* 2005). National strategies should be simple, practical and achievable within available resource allocations, and should not only consider farm-level disease management, but also the control of transboundary pathogen movements, management of chemical use, food safety and compliance with regional and international agreements and standards.

Infrastructure development

The intersection of shrimp health and environmental sustainability, and the need to support socio-economic development in poor coastal communities, should also lead governments to consider the need for infrastructure development in small-holder shrimp-farming regions. Commonly, shrimp ponds have proliferated in river

estuaries in an unplanned and unstructured fashion without consideration of the need for appropriate water or land management. Water intake to ponds and effluent discharge are very often drawn from, and returned to, the same shared canal, and there is little cooperation in the event of disease outbreaks to avoid cross-contamination of ponds. White spot syndrome virus is endemic in wild crustaceans in many of these farming systems and, notwithstanding strict adherence to BMP, it is doubtful if reliable production will ever be achieved without substantial investment in infrastructure. As poor small-holder farmers lack the necessary resources, this will require significant investment by governments or corporate benefactors in the interest of poverty alleviation and environmental sustainability.

Attitudinal change

Effective disease management is a shared responsibility in which all stakeholders, from farmers to policy makers, must understand their roles and exercise responsible attitudes and behaviours. Cooperation between industry and government and adherence to policies and practices that serve long-term sustainability and profitability rather than short-term gains will be essential if past disease emergence scenarios and immense socio-economic and environmental impacts are to be avoided in the future. The need for responsible behaviour applies particularly to the core problem of transboundary pathogen dispersal through irresponsible trade in live shrimp. This is a significant challenge as year-to-year profitability and survival are strong drivers of industry attitudes and lack of education and traditional beliefs are widespread in poor coastal communities. Achieving attitudinal change and compliance across this diverse trader sector, often operating beyond national jurisdictions, presents major challenges to policy makers and regulators. Nevertheless, attitudinal change can be achieved through effective communication and an increasing number of peak industry bodies and other farmer groups are engaging with government to assist policy formulation. Creating awareness of the importance of aquatic animal health management and its relevance to sustainable aquaculture development, aquatic animal trade and human food safety will greatly assist in achieving attitudinal changes in key stakeholders.

Conclusion

The pattern of emergence and the global spread of an increasing array of new viral diseases of shrimp is the logical consequence of the massive ecological shift and changes in trade patterns that have accompanied the establishment and growth of a new farming industry. Key aspects of the biology and epidemiology of infection are now known for most of the major pathogens and technological advances have enabled the production of SPF broodstock and provided sensitive detection tools that allow screening of seed to eliminate pathogens. Although new pathogens will almost certainly emerge as the industry continues to expand into new locations and responds to the challenges presented by global warming, we should now be far better prepared to limit impacts and contain the spread of disease. The primary constraints on the effective management of existing or future diseases lie in the will and capacity of governments and industry to respond with appropriate policies and practices and the necessary levels of investment in training and infrastructure development. International cooperation and productive alliances of governments, industry and the community will be required to underpin the goal of profitable and environmentally sustainable aquaculture development.

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