Major diseases of cultured Pacific White Shrimp (*Litopenaeus vannamei)*

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Aquaculture of *Litopenaeus vannamei* has been recently permitted in India. This is undertaken using seed produced from Specific Pathogen Free (SPF) brood stock of the shrimp imported from selected companies overseas after they underwent require quarantine and disease screening process that determines them to be free from specified pathogens of concern to aquaculturists. SPF shrimp are expected to be free from the viral pathogens which are known to cause major losses to shrimp aquaculture, for various reasons including pathogenic viruses such as WSSV, YHV, TSV, IHHNV, BPV and HPV. SPF refers only to the present pathogen status for specific pathogens and not to pathogen resistance or future pathogen status. SPF means that these animals will not suffer from diseases caused by specified diseases for which the animal is declared 'free' when cultured under 'strict' biosecurity. However, it does not guarantee against these shrimp getting infected with unknown pathogens or known pathogens which are not screened. Further, the SPF shrimp are not resistant to pathogens and these shrimp can become infected by any pathogen that they encounter during culture.

Focal Points at a Glance

In the context of the upsurge of farming of white Pacific shrimp *(P. vannamei)*, the authors enlighten us on the diseases afflicting this shrimp under farming and their control so as to take precautions while farming vannamei shrimp.

More than 20 viruses have been identified that are known to infect penaeid shrimp including *L. vannamei*. The OIE now lists five viral diseases and one bacterial disease (necrotising hepatopancreatitis) of penaeid shrimp in the Aquatic Animal Health Code of Office International des Epizooties (OIE, 2010) which are considered to be transmissible and of significant socio-economic importance. These viral diseases include white spot disease (WSD), yellow head disease (YHD), Taura syndrome (TS), infectious myonecrosis (IMN) and infectious hypodermal and haematopoietic necrosis (IHHN) (OIE, 2010). All OIE member countries are obliged to report these diseases so that a disease spread can be monitored and legislation instituted to prevent disease spread. IMN and TS are so far not reported from India. Although YHV has been reported from India in one instance in farmed black tiger shrimp (using histopathological techniques), its economic impact was negligible. All these diseases are known to cause considerable loss to farmed *L. vannamei*. In this article, a brief outline on these important diseases, the causative agents, symptoms, mode of transmission, possible methods of prevention and control are provided for the information of the shrimp farmers who have already taken up or are likely to take up *L. vannamei* culture in India.

I. *Important viral diseases reported that can infect L.vannamei*

1. *White Spot Disease (WSD)*

This virus is the most serious threat facing the shrimp farming industry. WSSV was first reported in farmed *P. japonicus* from Japan in 1992/93, but was thought to have been imported with live infected PL from Mainland China. WSD has been identified from crustaceans in China, Japan, Korea, South-East Asia, South Asia, the Indian sub -continent, the ☞

Mediterranean, the Middle East, and the Americas. All penaeid shrimp species are highly susceptible to infection, often resulting in high mortality. Crabs, crayfish, freshwater prawns, spiny lobsters and clawed lobsters are susceptible to infection, but morbidity and mortality as a consequence of infection is highly variable. Prevalence of WSSV is reported as highly variable, from <1% in infected wild populations to up to 100% in captive populations.

What is the causative agent of WSD?: WSD is caused by one of the largest animal viruses. WSSV is a large double-stranded DNA virus of 120-150 by 270-290 nm size, assigned to a new virus family, whispoviridae. WSSV had several names such as Chinese baculovirus (CBV), White spot syndrome baculovirus complex (WSBV), Shrimp Explosive Epidermic Disease (SEED), Penaeid Rod-shaped DNA Virus (PRDV), Japan's Rod-shaped Nuclear Virus (RV-PJ) of *P. japonicus* and Thailand's Systemic Ectodermal and Mesodermal Baculovirus (SEMBV) of *P. monodon*. WSSV can infect a wide range of aquatic crustaceans including marine, brackish and freshwater penaeids, crabs and crayfish. All decapod crustaceans from marine and brackish or freshwater sources are susceptible host species.

What are the symptoms of WSD?: WSSV affects organs of ectodermal and mesodermal origin, including the cuticular epithelium, connective tissue, nervous tissues, muscle, lymphoid organ and haematopoietic tissues. The virus also severely damages the stomach, gills, antennal gland, heart and eyes. During later stages of infection, these organs are destroyed and many cells are lysed. The shrimp then show reddish discolouration of the hepatopancreas and characteristic 1-2 mm diameter white spots on their carapace, appendages and inside surfaces of the body. Affected shrimp show lethargic behaviour. Cumulative mortality typically reaches 100 percent within two to seven days of infection.

How WSD is diagnosed?: It can be visually diagnosed through the presence of the characteristic white spots, which can be seen in advanced stage of infection. However, white spots may not be always present in early stages of infection in shrimp. WSSV can be detected by using Polymerase Chain Reaction (PCR), or with probes for dot-blot and *in situ* hybridisation (ISH) tests. PCR detection efficiency can be increased by exposure to stressful conditions (e.g. eye-stalk ablation, spawning, moulting, changes in salinity, temperature or pH, and during plankton blooms). WSSV can be confirmed histologically (particularly in asymptomatic carriers) by the presence of large numbers of Cowdrey A-type nuclear inclusions and hypertrophied nuclei in H&E-stained sectioned tissues, or simply by rapid fixation and staining of gill tissue.

How WSD is transmitted?: The mode of transmission of WSD is believed to be through exports of live PL and infected broodstock. The infection can be transmitted vertically and horizontally by cannibalism, predation, etc. and by water-borne routes. Dead and moribund animals can be a source of disease transmission. However, some studies have shown that disinfection of water supplies and the washing and/or disinfection of the eggs and nauplius is reported to be successful in preventing its transmission from positive broodstock to their larvae. It is generally believed that the virus sticks to the outside of the egg, and if it gains entry to the egg, it is rendered infertile and will not hatch. Using proper testing and disinfection protocols, vertical transmission can be prevented in the hatchery. The presence of WSSV in a pond does not always lead to disaster. Outbreaks are usually triggered from latent carriers by some environmental changes, probably related to osmotic stress through changes in salinity or hardness or rapid temperature changes. Fluctuations in temperature have been shown to induce mortalities of infected shrimp.

How can WSD be prevented/ controlled?: Broodstock should be PCR screened before breeding. The brooder's PLs should also be PCR screened before stocking into ponds, as this has been proven to result in a higher percentage of good harvests. Washing and disinfection of eggs and nauplii has also been shown to prevent vertical transmission of WSSV from infected broodstock to larval stages. Feeding of broodstock with fresh crab and other crustaceans should be avoided. Polyculture techniques with mildly carnivorous fish species (such as Tilapia sp.) has also proven effective at limiting the virulence of WSSV in ponds, as the fish can eat infected carriers before they become available to the live shrimp. The white spot virus only remains viable in water for 3-4 days, so disinfection of water used for changes and fine screening is effective in preventing transmission. Formalin treatment (70 ppm) has been shown to prevent transmission and not cause any harm to shrimp. In addition, all effluent from farms or processing plants should be disinfected with formalin or chlorine prior to discharge.

2. *Infectious Hypodermal and Haematopoietic Necrosis (IHHN)*

IHHN was first discovered in *L. vannamei* and *P. stylirostris* in the Americas in the year 1981. However, it was thought to have been introduced along with live *P. monodon* from Asia. IHHNV has probably existed for some time in Asia without detection due to its insignificant effects on *P. monodon*, the major cultured species in Asia. Recent studies have revealed geographic variations in IHHNV isolates, and suggested that the Philippines was the source of the original infection in Hawaii, and subsequently in most shrimp farming areas of Latin America. Large-scale epizootics were responsible for multi-million dollar losses in *L. vannamei* culture in the Americas during the 1990s.

What is the causative agent of IHHN?: IHHNV is caused by a small (20-22 nm) single-stranded DNA-containing parvovirus.

What are the symptoms of IHHN?: Gross signs of disease are not specific to IHHN, but may include reduced feeding, elevated morbidity and mortality rates, fouling by epicommensals and bluish body colouration. Larvae, PL and broodstock rarely show symptoms. In *L. vannamei*, IHHNV can cause runt deformity syndrome (RDS), which typically results in cuticular deformities (particularly bent rostrums), slow growth, poor food conversion ratio (FCR) and a greater size variation at harvest, contributing substantially to reduction in profits. These effects are typically more pronounced when the shrimp are infected at larval stages. Hence strict hatchery biosecurity including checking of broodstock by PCR, or the use of SPF broodstock, washing and disinfecting of eggs and nauplii is essential in combating this disease. IHHNV typically causes no problems for *P. monodon* since they have developed a tolerance to it over a long period of time, but they may suffer with RDS. *P. merguiensis* and *P. indicus* appear refractory to the IHHNV. However, these species may be life-long carriers of the virus and so could easily pass it on to *L. vannamei*, which typically suffers from RDS when exposed to IHHNV.

How is IHHN diagnosed?: IHHNV can be diagnosed using methods such as DNA probes in dot blot, ISH and PCR techniques as well as histological analysis of H&E-stained sections looking for intracellular, Cowdrey type A inclusion bodies in

ectodermal and mesodermal tissues such as cuticular epithelium, gills, foregut, hind gut, lymphoid organ and connective tissues.

How is IHHN transmitted?: Transmission of IHHN is known to occur rapidly by cannibalism of weak or moribund shrimp. It can also be transmitted through waterborne route and co-habitation. Vertical transmission from broodstock to larvae is common and has been shown to originate from the ovaries of infected females (whilst sperm from infected males was generally virus-free). IHHNV may be also transmitted through vectors such as insects and birds which have been shown to act as mechanical carriers for the disease.

How to prevent / control IHHN?: IHHNV is reported to be highly resistant to all the common methods of disinfection including those of chlorine, lime and formalin. One of the big problems with IHHNV is its eradication in infected facilities. Complete eradication of all stocks, complete disinfection of the culture facility and avoidance of restocking with IHHNV-positive animals may bring down incidences of IHHNV infections.

3. *Taura Syndrome (TS)*

Taura Syndrome was first identified from farms around the Taura River in Ecuador in 1992 and the disease spread rapidly to the whole of Latin and North America within three years. Subsequently, TS was also reported from Asia including Mainland China and Taiwan (from 1999), and in late 2003 in Thailand, probably through the regional and international transfer of live PL and broodstock of *L. vannamei*.

What is the causative agent of Taura Syndrome?: Initial work suggested that TS was caused by a toxic pesticide. However, it is now known that a single or perhaps several very closely related mutant strains of the Taura syndrome virus (TSV) are responsible for the TS. TSV is a single stranded RNA virus of 32 nm size, non-enveloped icosahedrons and more prone to mutations causing more concern.

What are the symptoms of Taura Syndrome in shrimp?: TSV infections occur in juvenile shrimp (0.1-1.5 g body weight) within two to four weeks of stocking ponds and occur largely within the period of a single moult cycle. In the acute phase of the disease, during pre-moult stage, the shrimp are weak, soft-shelled, have empty gut and diffuse expanded chromatophores that appear red, particularly in the tail (hence the common name - red tail disease). Such animals will usually die during moulting (5-95 percent). Adult shrimp are known to be more resistant than juveniles. Shrimp that survive infection show signs of recovery and enter the chronic phase of the disease. Such shrimp show multiple, randomly distributed, irregular, pitted, melanised lesions of the cuticle. These gross lesions will persist, but may be lost during moulting, and the shrimp thereafter appear normal.

How is Taura Syndrome diagnosed?: TS can be diagnosed using standard histological and molecular methods of detection. Specific DNA probes applied to ISH assays with paraffin sections provide the confirmatory diagnosis. Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) assay is commonly used for larger sample sizes and non-lethal sampling for broodstock.

How Taura Syndrome is transmitted?: The mechanism of transmission of TSV can be through infected PL and broodstock. Recently it has been shown that mechanical transfer through insect and avian vectors may be a more likely route of infection. Shrimp-eating seagulls can transmit TSV through their faeces. Hence birds are likely to transmit TSV.

How to prevent / control Taura Syndrome?: Infected stocks must be totally destroyed and the culture facility must be disinfected. The disease can be prevented by avoidance of reintroduction of the virus from nearby facilities, wild shrimp and carriers and stocking with TSV-free PL produced from TSV-free broodstock. Switching of culture to refractory species such as *P. stylirostris* has been suggested. Other methods suggested for controlling the virus include the best management practices (BMPs) and maintenance of optimal environmental conditions, weekly applications of hydrated lime, $Ca(OH)_2$ at 50 kg/ha, polyculture with fish (to consume dying and dead carriers).

4. *Yellow Head Disease (YHD)*

Yellow head disease was the first major viral disease that caused extensive losses to shrimp farms in Thailand during 1990-91. YHD outbreaks have been reported in the black tiger prawn and the white Pacific shrimp. YHD has been reported in China,Taipei, India, Indonesia, Malaysia, the Philippines, Sri Lanka, Thailand and Vietnam. Outbreaks of YHD with heavy mortalities have been reported in farmed black tiger shrimp and Pacific white shrimp. It is reported to be highly prevalent (>50%) in sampled farmed and wild populations in Australia, Asia, East Africa and Mexico.

What is the causative agent of YHD?: YHD is caused by yellow head virus (YHV), and its related gill-associated virus (GAV). YHV is rod-shaped enveloped virus of 40-60 nm by 150-200 nm size, containing single stranded RNA.

What are the symptoms of YHV?: YHV affects tissues of ectodermal and mesodermal origin tissues such as lymphoid organ, haemocytes, haematopoietic tissue, gill lamellae and spongy connective tissue of the subcutis, gut, antennal gland, gonads, nerve tracts and ganglia. YHV principally affects pond reared juvenile stages of 5 -15 g. Affected shrimp typically feed voraciously for two to three days and then stop feeding abruptly and are seen swimming near the periphery of the pond. YHV infections can cause swollen and light yellow coloured hepatopancreas in infected shrimp, and has a general pale appearance, before dying within a few hours. YHD can cause up to 100% mortality in infected *P. monodon* ponds within 3-5 days of the first appearance of clinical signs. GAV has been reported to be associated with mortalities of up to 80% in *P. monodon* ponds in Australia.

How YHV is diagnosed?: Yellow head virus can be detected by RT-PCR or with a probe designed for dot-blot and ISH tests. It can also be diagnosed histologically in moribund shrimp by the presence of intensely basophilic inclusions, most easily in H&E stained sections of stomach or gill tissue, or simply by rapid fixation and staining of gill tissue and microscopic examination.

How YHV is transmitted?: The primary mechanism of spread of YHV in pond culture appears to be through water and mechanical means or from infected crustacean carriers. YHV is reported to remain viable in aerated seawater for up to three days. However, the most serious threat is latent or asymptomatic carriers, from which the virus can spread either by ingestion or cohabitation. Other shrimp such as *P. merguiensis, P. indicus, Metapenaeus ensis, Palaemon styliferus* and *Acetes* spp. may become infected and act as carriers having latent infections, while others such as *Euphausia superba* may die upon infection. Other crustaceans, such as *Macrobrachium rosenbergii* and many crab species and *Artemia* appear to be refractive to YHV.

Infected broodstock can pass on the virus to larvae in the maturation/hatchery facilities if thorough disinfection protocols are not strictly adhered to.

How YHD can be prevented /controlled?: Although YHD is not causing much loss at present, methods of YHV eradication in ponds are much the same as for other viruses and involve BMPs that include pond preparation by disinfection and elimination of carriers, chlorination (30 ppm active ingredient) of reservoir water, filtering inlet water with fine screens, avoidance of live feeds, maintenance of stable environmental conditions, disinfection of YHV infected ponds before discharge, and monitoring (by PCR) and production of virus free broodstock and PL for pond stocking.

6. *Infectious Myonecrosis (IMN)*

Infectious myonecrosis is an emerging *L. vannamei* disease, first detected in Brazil during 2004, and then in Indonesia in 2006. To date, IMN has been detected in East Java, Bali, and West Nusa Tenggara provinces. The principal host species in which IMNV is known to cause significant disease outbreaks and mortalities is *L. vannamei*.

What is the causative agent of IMN?: IMN is caused by a putative totivirus. IMNV particles are icosahedral in shape and 40 nm in diameter.

What are the symptoms of IMN?: Juveniles and sub-adults of *L. vannamei*, farmed in marine or low salinity brackishwater, appear to be the most severely affected by IMN disease. The principal target tissues for IMNV include the striated muscles (skeletal and less often cardiac), connective tissues, haemocytes, and the lymphoid organ parenchymal cells. IMN disease causes significant mortality in grow out ponds and is characterised by acute onset of gross signs including focal to extensive whitish necrotic areas in the striated muscle, especially of the distal abdominal segments and the tail fan, which may become necrotic and reddened similar to the colour of cooked shrimp. Severely affected shrimp become moribund and mortalities can be instantaneously high and continue for several days. Mortalities from IMN range from 40 to 70% in cultivated *L. vannamei*, and FCR of infected populations increase from normal values of ~ 1.5 to 4.0 or higher.

How IMNV is diagnosed?: IMN can be confirmed by histopathology, using routine haematoxylin–eosin (H&E) stained paraffin sections and demonstrating characteristic coagulative necrosis of striated skeletal muscle fibers, often with marked oedema among affected muscle fibers. IMN may be also rapidly diagnosed using a nested RT-PCR method which provides a rapid, sensitive and specific test to detect IMNV in penaeid shrimp. Published methods available are ISH and nested RT-PCR and real-time RT-PCR for the molecular detection of IMNV.

How is IMNV Transmitted?: IMNV has been demonstrated to be transmitted through cannibalism. Transmission *via* water and vertical transmission from broodstock (transovarium or by contamination of the spawn eggs) to progeny is also likely to occur. IMNV may also be transmitted among farms by faeces of seabirds or shrimp carcasses. Outbreaks of IMN with sudden high mortalities may follow stressful events such as capture by cast-net, feeding, sudden changes in salinity or temperature, etc., in early juvenile, juvenile, or adult *L. vannamei* in regions where IMNV is enzootic.

How IMN can be prevented /controlled?: Stocking only pre-screened broodstock and/or their spawned eggs/nauplii and discarding those that test positive for the IMN virus by RT-PCR. Fallowing and restocking of affected farms or entire culture regions with IMNV-free stocks of *L. vannamei* may help in preventing its recurrence. No effective therapeutants have been reported for IMN.

7. *Penaeus vannamei* Nodavirus Infection

The disease was first reported in cultured *Penaeus vannamei* in Belize in 2004 and in Guayas Province, Ecuador in 2006.

What is the causative agent?: The causative agent is a positive single stranded RNA virus, named as *Penaeus vannamei* nodavirus (*Pv*NV) and is related to *Mr*NV belonging to Nodaviridae.

What are the symptoms?: The RNA virus has been detected in adult shrimps. Affected shrimps exhibit clinical signs, white, opaque lesions in the tail muscle that cause muscle necrosis. Histological examination reveals multifocal necrosis and haemocytic fibrosis in the skeletal muscle and basophilic, intra-cytoplasmic inclusions in striated muscle, lymphoid organ and connective tissues.

How is the disease diagnosed?: ISH method and a nested RT-PCR assay and histopathology specific for *P. vannamei* nodavirus (*Pv*NV) have been developed. Also commercial IQ2000 RT - PCR diagnostic kit is available for routine screening.

How is the disease transmitted?: It appears to affect survival of shrimp in grow out ponds. The disease is associated with environmental stress, such as crowding and high temperature. When the shrimps are stocked at a high density (>50 m^{-2}), or when the temperature is >32°C, survival decreases by 40% in *Pv*NV-infected ponds. 50% production loss was estimated due to *Pv*NV in 2004 from the infected farm in Belize.

How to prevent / control PvNV?: The horizontal spread of *Pv*NV from infected farm to other farms could result in significant production losses in infected areas. The shrimp farms should be monitored regularly for having knowledge of the incidence of diseases.

II. *Important bacterial diseases reported*

8. *Necrotising Hepatopancreatitis (NHP)*

This disease is also known as Texas necrotising hepatopancreatitis (TNHP), Texas pond mortality syndrome (TPMS) and Peru necrotising hepatopancreatitis (PNHP). NHP has been reported as an important disease since its first diagnosis in 1985. It has been reported to cause mass mortalities to the tune of 20-90 percent of *L. vannamei* in highly saline commercial grow-out ponds nearly every year since then. By 1993, NHP spread to Ecuador, Guatemala, Honduras, Mexico,and Peru and by 1995, coincided with warm waters with high salinity associated with El Nino, and caused severe mortalities (60-80 percent mortality) of *L. vannamei* and *L. stylirostris* throughout Ecuador. NHP has not yet been reported in Asia, but could cause significant damage were it to be transferred here with untested shrimp introduction.

What is the causative agent of NHP?: Necrotising hepatopancreatitis is caused by obligate intracellular Rickettsia-like bacterium, a member of the order - Proteobacteria. It is Gram-negative, pleomorphic, rod-shaped or helical-shaped bacterium.

What are the symptoms of NHP?: Affected shrimp are lethargic, anorexic with empty gut and show epibiotic fouling. Exoskeleton becomes soft and show abdominal muscle atrophy. Affected ponds have increased FCR and growth of affected shrimp is retarded. The hepatopancreas becomes watery with white or black streaks. Mortality rates reach up to 90% within 30 days of the

appearance of clinical signs.

How NHP is diagnosed?: NHP can be diagnosed by demonstration of lipid droplets and melanisation of hepatopancreas by microscopic examination of wet mount of preparations. It may be confirmed by histopathological examination showing atrophy and the presence of granulomata in the hepatopancreas, and haemocyte aggregations around the haepatopancreatic tubules. Intra-cytoplasmic Rickettsia-like bacteria may be prominently seen in the cytoplasm. Molecular diagnostic tools such as ISH, dot blot hybridisation, and PCR for specific -Proteobacterial DNA are also available.

How is NHP transmitted?: NHP could be transmitted horizontally with infected PL.

How NHP can be prevented /controlled?: Stocking only pre-screened broodstock and/or their spawned eggs/nauplii and discarding those that test positive. Maintaining optimal environmental parameters using BMPs will be useful in preventing NHP.

9. *Vibriosis*

Vibriosis is one of the major backterial diseases accountable for mortality of cultured shrimp worldwide. *Vibrio* species are commonly distributed in culture facilities throughout the globe. Vibrio-related infections, though they occur frequently in hatcheries, also occur in cultured shrimp and outbreaks may occur when environmental factors trigger the rapid multiplication of bacteria. This is a severe systemic bacterial disease.

What is the causative agent of Vibriosis?: Vibriosis is caused by gram-negative bacteria of the family Vibrionaceae such as *V. harveyi, V. vulnificus, V. parahaemolyticus, V. alginolyticus*

What are the symptoms of Vibriosis ?: The affected shrimps are lethargic, show abnormal swimming behaviour, loss of appetite, red discolouration, necrosis of the sub cuticular tissue in the tail and appendages region. In severely affected shrimp, the gill covers appear flared up and eroded and extensively melanised black blisters can be seen on the carapace/abdomen. There will be high mortalities in PLs and young juveniles. Moribund shrimp appear hypoxic and often come to the pond surface or edge. Presence of luminescence shrimp in rearing waters will be observed due to luminescent bacterial infections.

How is Vibriosis diagnosed?: Cuticle, hepatopancreas (midgut gland), lymphoid organ, antennal gland, heart and striated muscle are commonly affected in shrimp Vibriosis. Based on gross signs and confirmed by isolation of bacterial pathogen from haemolymph and water sample by standard microbiological methods vibriosis is diagnosed.

How Vibriosis is transmitted?: *Vibrio* species are widely distributed in culture facilities. *Vibrio*-related infections frequently occur in hatcheries and in pond reared shrimp species and a major problem for PLs in ponds. *vibrio* species are opportunistic pathogens and in heavily stocked commercial systems, *Vibrio*-related disease can progress rapidly. Stressful environment conditions like poor water and nutrition quality, improper handling, overcrowding and parasites act as predisposing factors for disease occurrence.

How to prevent / control Vibriosis?: Vibriosis can be prevented through improved management practices, feed quality, and water source purity and maintaining good water quality and increasing water exchange with good quality seawater. It can be controlled with shrimp feeds fortified with antibiotics (after ascertaining *in vitro* sensitivity of the pathogen), e.g. feeds containing oxytetracycline @ 1.5g/kg, fed at 2-10% of body weight for 10-14 days along with proper water and pond management. Sufficient withdrawl period (about 25-30 days) should be allowed for the antibiotic to become inactive or harmless. Environmental stress which favours disease outbreaks should be kept at minimum.

III. *Important parasitic diseases reported*

10. *Cotton Shrimp Disease*

Cotton shrimp disease, also known as milk shrimp disease, microsporidian disease, nosema disease, etc is a parasite infecting abdominal muscle that gives a "cottony" appearance to infected shrimp.

What is the causative agent?: Microsporadia such as Agmosoma (*Thelohania*), Ameson (*Nosema*) and Pleistophora

What are the symptoms?: The parasite principally infects and replaces the muscle of the shrimp but can be found in other organs. Muscle of affected shrimps have cooked appearance of muscles. Severely affected shrimp appears bluish black or purplish-mauve discolouration with opaque white muscle and/or gonads. Agmosoma infects the gonads, heart and haemolymph vessels, gills, hepatopancreas and midgut, producing enlarged opaque white gonads, and often multiple whitish tumour-like swellings in the gills and in the subcuticular tissues of the cuticle and appendages. Microsporidians infecting shrimp replace host tissues with spores. Infected individuals are prone to loss by predators and poor survival following capture and handling. Infection of the gonads renders affected individuals sterile, and interestingly may cause feminisation of infected male shrimp.

How is the disease diagnosed?: This is one based on gross signs and symptoms and microscopic demonstration of developmental stages of microsporadia in the affected tissues. Light microscopic examination of unstained wet-mounts or impression smears from affected tissues and demonstration of masses of refractive spores of ~1 to 8 µm in size. Giemsa-stained or acid-fast stained impression smears or histological sections from infected muscles, gonads, or other suspect lesions will disclose multitudes of microsporidian spores, whose characteristics are used to classify the parasite. Gene probes and PCR for some species are available.

How is the disease transmitted?: Oral feeding of microsporidian infected shrimp resulted in infection in Experimental studies.

How to prevent / control the disease?: Microsporidians seem to be ubiquitous in wild penaeid populations. These parasites occasionally are the cause of serious disease. Shrimp with advanced infections are unmarketable, or sterile in the case of gonad infections in broodstock. Affected animals should be destroyed and buried with lime, away from the farms. After harvesting, the pond bottom should be thoroughly dried to kill the spores of the microsporadia. Control is unknown.

IV. *Diseases of unknown etiology*

11. Bamboo-Shaped Disease

Recently, *L. vannamei* farmers in Thailand, Malaysia and Indonesia have observed abnormal body shape in grow-out ponds. The disease was tentatively named bamboo-shaped disease (BSD).

What is the causative agent of BSD?: Investigations have indicated that BSD is☞

caused by a new virus. The non-enveloped viral particles measure about 20-22 nm size, icosahedral in shape. These viral particles are widely found in the cytoplasm of connective tissue, muscles, tegumental gland, gills, including the optic lobe, brain, thoracic ganglia, abdominal ganglia and ventral nerve cord. The virus attacks cells that support normal function of the neural structures of the shrimp and a disruption of normal functions of nerves supplying muscles causing abnormal development and degeneration of the muscles. Secondary infections caused by IHHNV, LSNV and TSV was also noticed in <20% of the shrimp.

What are the symptoms of BSD?: The incidence was about 1-5% (in some ponds >60%) of the shrimp in the affected ponds. abdominal segments of affected shrimp are swollen, bent laterally in a zigzag pattern and sometimes accompanied by opaque muscles However, these shrimp ponds do not suffer significant mortality.

Histopathology of BSD mainly showed muscle necrosis and degeneration, with haemocytic infiltration, especially in the abdominal segments.

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Further reading

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Fig1: Pacific white shrimp farm that experienced disease

Fig.2: Dead Pacific white shrimp seen near edges of shrimp pond

Fig.3: Pacific white shrimp dead due to disease, thrown near shrimp ponds

Fig.4: Emergency harvest of Pacific white shrimp due to disease in a shrimp farm

Fig.5: Normal Pacific white shrimp collected form diseased shrimp pond

Fig.6: Dead shrimp collected during emergency harvest of Pacific white shrimp due to disease in a shrimp farm

Healthy *L.vannamei* shrimp

L.vannamei with white spot disease