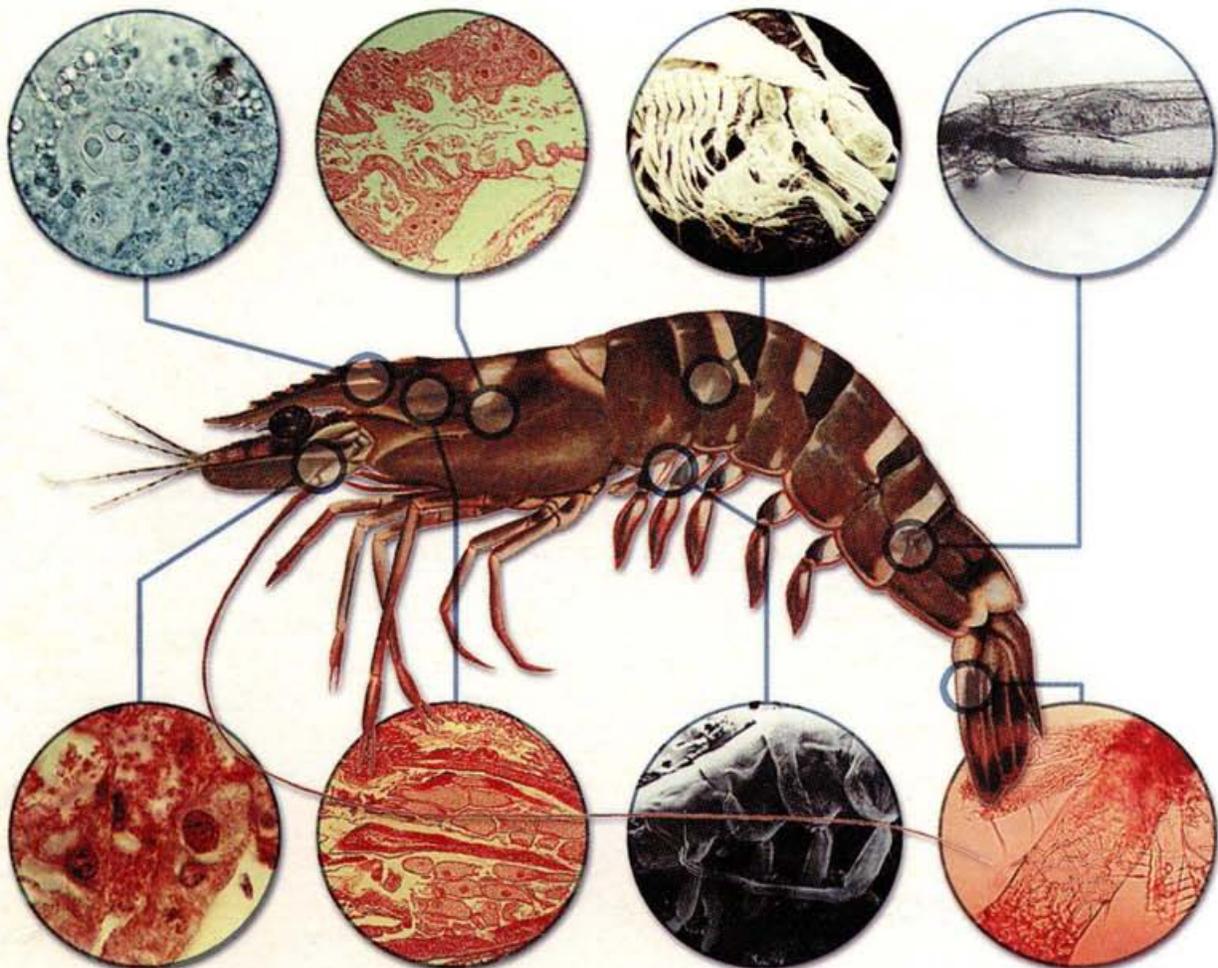


Diseases of Penaeid Shrimps in the Philippines

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AQUACULTURE DEPARTMENT
SOUTHEAST ASIAN FISHERIES DEVELOPMENT CENTER
Tigbauan, Iloilo, Philippines



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Second Edition
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Foreword

Aquaculture is one of the fastest growing food producing sectors of the world. However, disease outbreaks are increasingly recognized as significant constraints to aquaculture production and trade affecting both economic and social development in many countries.

Within the shrimp culture sector, the number of diseases has expanded steadily with expansion and intensification of large-scale commercial cultivation. Shrimp disease is considered as the single most limiting factor to successful commercial production. There is therefore a pressing need to develop and implement practical solutions to this problem.

Several countries in the Southeast Asian region have already begun to take real and meaningful strides towards this end. The Southeast Asian Fisheries Development Center, Aquaculture Department (SEAFDEC/AQD) in its pursuit for responsible aquaculture has come up with a revision of the manual *Diseases of Penaeid Shrimps in the Philippines*, to include newly discovered diseases. We hope that this manual will be of considerable help to shrimp farmers in identifying the disease and lead to prevention or early disease diagnosis and control.



ROLANDO R. PLATON, Ph.D.
Chief, SEAFDEC Aquaculture Department

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Introduction

Cultured shrimp is a major export commodity of the Philippines. Because of its high yield, monoculture of shrimps in semi-intensive and intensive systems has been the preferred production method. However, this approach led to the proliferation of diseases resulting in growth retardation, physical deformity, reduced fecundity, physiological malfunction, and mortality. Even if they are outrightly rejected, diseased shrimps command a lower price in the market.

Diseases of penaeid shrimps may be caused by living agents like viruses, bacteria, fungi, and epicommensals and parasites. Moreover, stress from nutritional deficiency in the shrimp, exposure to toxic substances, and advance rearing water parameters may also lead to disease. To sustain the production of good quality shrimps, there is a need to understand disease and the factors that contribute to its development and outbreak. Although many solutions to disease problems need long-term study and research, early detection and diagnosis are crucial factors to well-timed and prompt control. Therefore, the shrimp farmer must be familiar with potential shrimp disease problems that may occur.

It has been ten years since the first edition of this manual. Most of the disease that were originally presented are included because of their continued adverse consequence on shrimp culture systems. In addition two viral diseases: white spot virus disease and yellow-head virus disease are included. Both viruses have negative impacts on shrimp survival and, therefore, of economic significance to the farmer. The devastating luminous bacterial disease, which was the scourge of the shrimp hatchery industry in the 1980s, found its way into pond culture systems causing massive losses of stocks and disruption of a majority of pond operations in key shrimp growing areas in the country. The continued appearance of new shrimp diseases locally and globally has increased awareness in the aquaculture community of the importance of diseases. Major efforts at disease surveillance, containment and eradication are primary factors to prevent its spread.

This manual aims to provide information on diseases observed among the three major species of shrimps cultured in the country: *Penaeus monodon*, *P. merguensis*, and *P. indicus*. It includes the common name of the disease, causative agent, species affected, stages affected, gross signs, effects on the host, and methods of prevention and treatment. Although not all diseases included have been photographed to show gross signs for effective pond-side diagnosis, simple diagnosis using a microscope, can be performed on site. Also included are diagrams to help readers recognize

the specific organs where histological sections in the figures are located. Appendices, a Glossary and a Reference List are included for detailed information about the diseases.

In cases where diagnosis has been established and chemical treatment is indicated, it is necessary to test the tolerance of a small number of shrimps to the chemical. Water parameters vary from place to place and what may be effective and tolerated in one place may not be true for another. Moreover, the indiscriminate use of drugs must be avoided because of the danger of developing drug-resistant strains of the pathogen. Since, many of the drugs available in the market are used for treatment of human diseases and thus would consequently lead to public health problems.

The threat of viral and bacterial diseases as well as the deterioration of culture environments and water source continue to make shrimp culture challenging. The emergence of new shrimp diseases will continue with intensification in shrimp culture. Fortunately, much of the historical data is there for us to learn from.

Viral Diseases

**Monodon Baculovirus
(MBV) Disease**

**Infectious Hypodermal and
Hematopoietic Necrosis
Virus (IHHNV) Disease**

**Hepatopancreatic Parvo-
like Virus (HPV) Disease**

**White Spot Syndrome
Virus (WSSV) Disease**

**Yellow Head Virus (YHV)
Disease**

Common name: **MONODON BACULOVIRUS (MBV) DISEASE**

Causative agent: *Penaeus monodon*-type baculovirus (75 - 300 nanomicros)

Species affected: *Penaeus monodon*, *P. merguensis*

Stages affected: Mysis, postlarvae, juveniles, adults

Gross signs:

- Affected shrimps exhibit pale-bluish gray to dark blue-black coloration
- Sluggish and inactive swimming movements
- Loss of appetite
- Retarded growth
- Increased growth of benthic diatoms and filamentous bacteria may cause fouling on the exoskeleton/gills
- Infected pond-reared shrimps at 45 days of culture (DOC) stocked at 4 to 100 per m² manifested slow growth rates and pale yellow to reddish brown hepatopancreas
- High incidence of bacterial infections expressed as localized “shell disease”
- Significant mortalities can occur during stress and crowding

Effects on host:

- Causes destruction of the hepatopancreas and lining of the digestive tract
- Spherical, occlusion bodies fill up enlarged nuclei of hepatopancreatic cells (Figure 1a and b) and discharged into the lumen (Figure 1c) after cells have been destroyed. This maybe followed by necrosis with secondary bacterial infection.
- PL 3 is the earliest stage found infected with MBV. However, experimental waterborne inoculation of MBV to mysis 2 (M 2), postlarvae 3 (PL 3), PL 6, PL 9 and PL 11 resulted in MBV infections within 12 days post-inoculation.
- Incidence rate of MBV reported was 20-100%
- Accumulated mortality of 70% was observed among *P. monodon* juveniles cultured in raceways and tanks

Preventive methods:

- Use MBV-free postlarvae
- Eggs washed with ozone-disinfected seawater yielded 68% survival of PL 7 compared with untreated ones yielding 31% survival
- Reduce stress by good husbandry practices and good nutrition
- Experimental feeding of shrimp juveniles (0.2 - 0.3 g) using feeds with 100 ppm phosphated ascorbic acid (MAP) for 92 days showed that shrimps with initially moderate to severe infection of MBV displayed improvement in the histological profile of the hepatopancreas and the absence of MBV occlusion bodies in the cells
- Destroy infected shrimp juveniles by burning or burying in pits lined with lime
- Disinfect rearing facilities (Appendix I)

Treatment: None reported

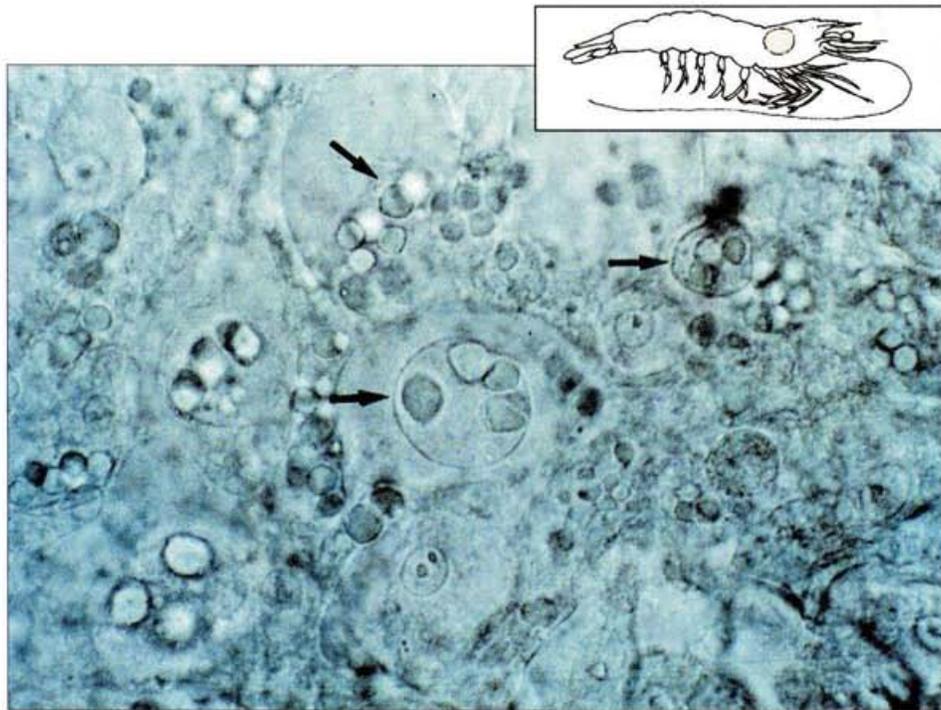


Figure 1a. A squash preparation of the hepatopancreas of postlarval shrimp infected with monodon baculovirus (MBV). The multiple intranuclear occlusion bodies (arrows) are diagnostic for MBV. (Malachite green, 400X).

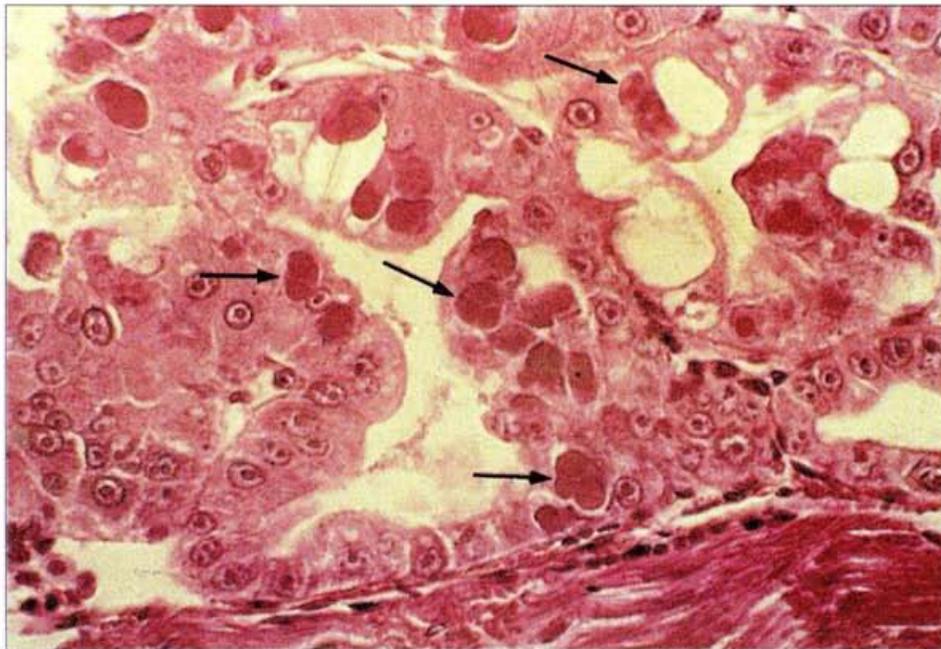


Figure 1b. High magnification of the distal region of the hepatopancreas of a shrimp juvenile heavily infected with monodon baculovirus (MBV) showing numerous multiple occlusion bodies within infected cells (arrows). (Hematoxylin and Eosin (H&E) stain, 200X).

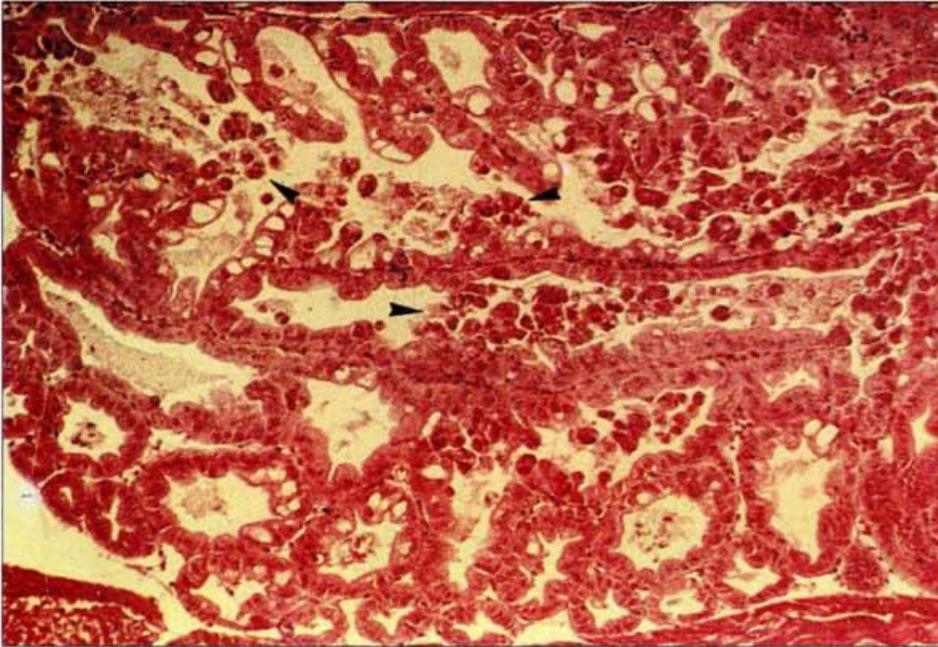


Figure 1c. Low magnification of the sagittal section of the hepatopancreas of a shrimp juvenile heavily infected with monodon baculovirus (MBV). Note the numerous multiple occlusion bodies within infected cells and the lumen (arrowheads). (H & E, 100X).

Common name: **INFECTIOUS HYPODERMAL AND HEMATOPOIETIC NECROSIS VIRUS (IHHNV) DISEASE**

Causative agent: Parvovirus (20 ~ 22 nanomicros)

Species affected: *Penaeus monodon*

Stages affected: Postlarvae, juveniles, adults

Gross signs:

- Shrimps show erratic swimming behavior, rising slowly to the water surface, hanging and rolling over until the ventral side is up
- Shrimps become weak and lose their appetite for food. They repeat the process of rising to the surface and sinking until they die usually within 4-12 hours.
- Decreased preening and delayed molting
- Acutely affected shrimps develop white opaque abdominal muscles, bluish to distinctly blue cuticular color often with mottled buff to tan pigment patches in the hypodermis and very soft cuticles

Effects on host:

- Affects the epidermis, lining of foregut and hindgut, nerve cord and nerve ganglia, hematopoietic organ, antennal gland, connective tissues, muscles, heart, gonad, mandibular organ, and hemocytes of shrimp
- Induces the development of eosinophilic inclusion bodies (Figure 2a) in the affected cells in the early acute stage of the disease, followed by necrosis and inflammation of target tissues
- Presence of the virus can cause death of the cells of the cuticle, blood-forming tissues and connective tissues of the shrimp which leads to abnormal metabolism and, eventually, mortality
- Inclusion bodies are common early in acute infections, later decreasing in number followed by necrosis and inflammation of target tissues
- Poor resistance to stress; susceptibility to secondary infection
- Mortality rates of above 90% were observed among penaeid juveniles in intensive culture systems
- Has been linked to the runt deformity syndrome (RDS)
- Some survivors of epizootic may carry the virus for life

Preventive methods:

- Use IHHNV-free stocks
- If the disease agent is suspected among cultured shrimp stocks, destroy exposed shrimps and disinfect contaminated premises
- Strict hygiene
- Disinfection of ponds and inlet canals
- Use of only dry commercial feeds
- Pasteurize fresh feed at 60°C for 15 min

6 Diseases of Penaeid Shrimps in the Philippines

- Fine screening of inlet water
- Refrain from exchanging water for at least three days after infected ponds have been discharged
- Ban importation of non-indigenous shrimps
- Compliance with the Hazard Analysis and Critical Control Point (HACCP) programs for product safety

Treatment: None reported

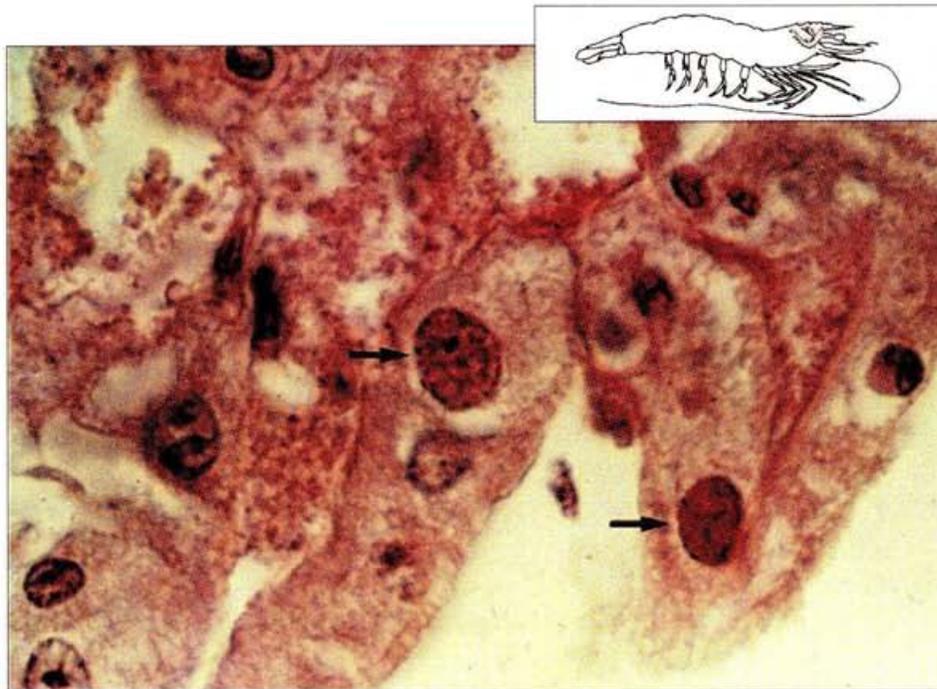


Figure 2a. Histological section of the antennal gland of juvenile shrimp infected with infectious hypodermal and hematopoietic necrosis virus (IHHNV) showing eosinophilic intranuclear inclusion bodies (arrows). (H & E, 400X).

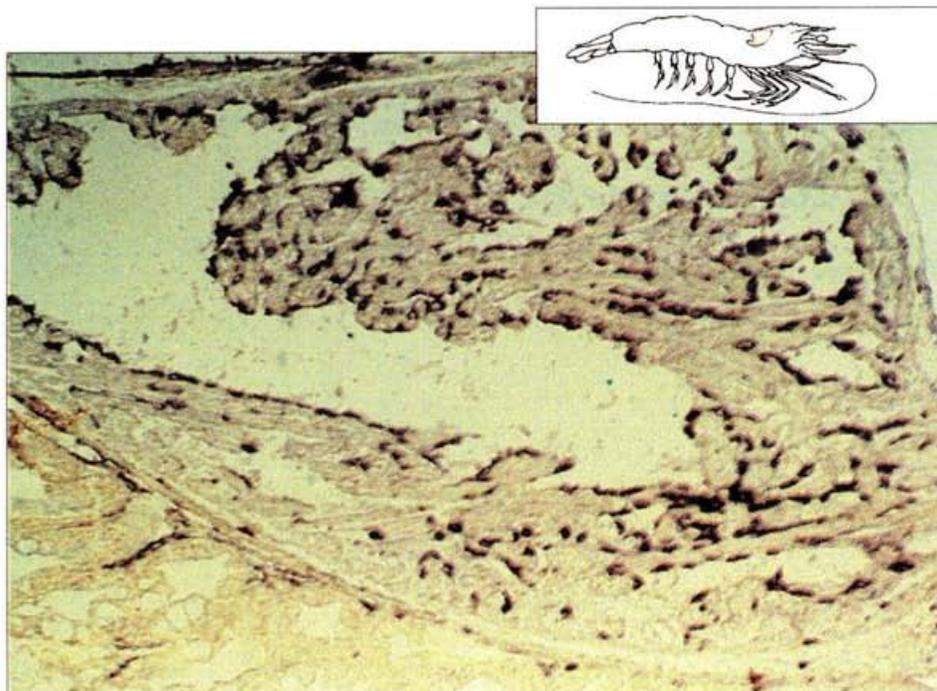


Figure 2b. Sagittal section of the heart of *Penaeus monodon* juvenile stained with ShrimProbe (Diagxotics, Inc.). Note abundant probe-positive cells (blue color). (100X).

Common name: **HEPATOPANCREATIC PARVO-LIKE VIRUS (HPV) DISEASE**

Causative agent: Parvovirus (22 ~ 24 nanomicros)

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Postlarvae, juveniles, adults

Gross signs:

- Retarded growth
- Loss of appetite
- Benthic diatoms, protozoans such as *Zoothamnium* sp., and filamentous bacteria may cause fouling on the exoskeleton
- Occasional white opaque areas on the tail/abdominal muscles

Effects on host:

- Enlargement of the hepatopancreatic nuclei with development of a prominent occlusion body (Figure 3)
- Affects the distal tubules of the hepatopancreas causing death of cells and shrinkage of the organ
- Damage in this organ can cause abnormal metabolism and, eventually, death
- Postlarvae (PL1 - PL19) from three hatcheries in Iloilo showed prevalence rates of 7.8 to 26.4%
- Mortalities among *P. merguensis* may reach as high as 50% within 4-8 weeks of disease onset

Preventive methods:

- Use HPV-free stocks
- Destroy infected stocks

Treatment: None reported

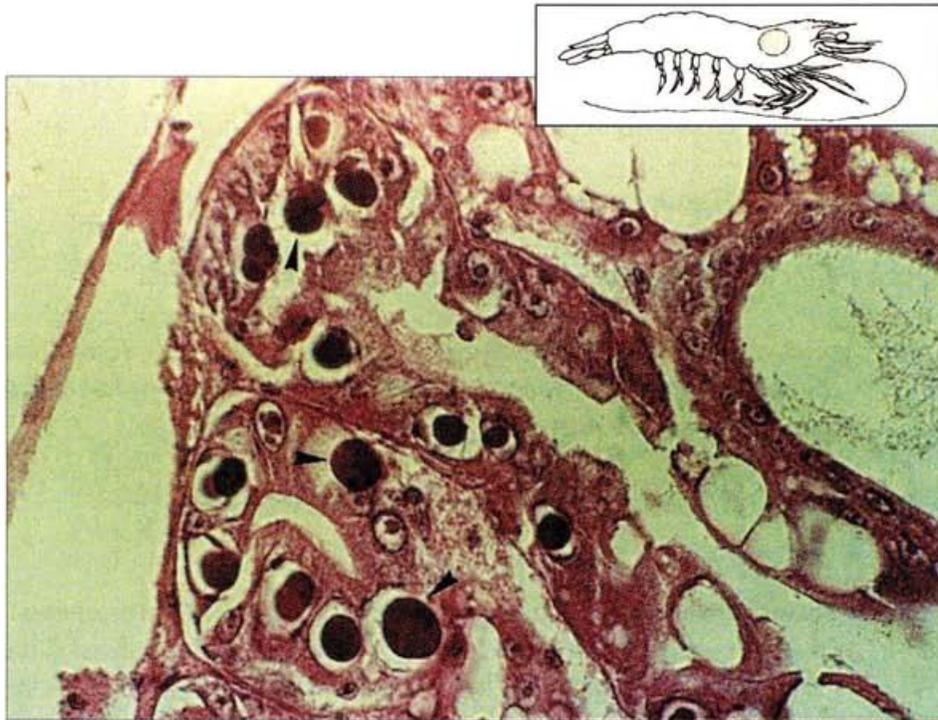


Figure 3. Histological section of the hepatopancreas of juvenile shrimp with heavy hepatopancreatic parvo-like virus (HPV) infection showing single inclusion bodies (arrowheads) within infected nuclei. (H & E, 200X).

Common name: **WHITE SPOT SYNDROME VIRUS (WSSV) DISEASE, WHITE SPOT VIRUS (WSV) DISEASE, WHITE SPOT BACULOVIRUS (WSBV), SYSTEMIC ECTODERMAL AND MESODERMAL BACULO-LIKE VIRUS (SEMBV)**

Causative agent: Baculovirus (100-140 X 270-420 nanomicros)

Species affected: *Penaeus monodon*, *P. indicus*, *P. merguensis*

Other species affected: *P. semisulcatus*, *P. setiferus*, *P. chinensis*, *Litopenaeus stylirostris*, *L. vannamei*, *Scylla serrata*, *Charybdis feriatus*, *Portunus pelagicus*, *Aschetes sp.*, *P. sanguinolentus*

Stages affected: Mysis, postlarvae, juveniles, broodstock

Gross signs:

- Presence of distinct white cuticular spots (0.5 - 3mm in diameter) most apparent at the exoskeleton and epidermis of diseased shrimp about 2 days after onset
- The white spots start at the carapace and 5th and 6th abdominal segments which later affect the entire body shell
- Moribund shrimp display red discoloration and have loose cuticle
- Surface swimming and gathering at pond dikes with broken antennae
- Reduction in food consumption and empty gut
- Rapid onset and high mortalities in 3 to 10 days of up to 100% with dead shrimps at the pond dikes/pond bottom

Effects on host:

- Affects a wide host range and targets various tissues (pleopods, gills, hemolymph, stomach, abdominal muscle, gonads, midgut, heart, periopods, lymphoid organ, integument, nervous tissue and the hepatopancreas) resulting in massive systemic infection
- Presence of inclusions within the nuclei of affected cells (Figure 4a)
- Shrimps 4-15g are particularly susceptible but the disease may occur from mysis to broodstock
- Pre-moulting shrimps are usually affected
- *Penaeus indicus* suffers earlier and greater losses compared to *P. monodon*. Crabs, krill and other shrimps are viral reservoirs
- Pandemic epizootic occur in extensive, semi-intensive and intensive culture systems regardless of water quality and salinities

Preventive methods:

- Use WSV-free stocks
- Feeding with peptidoglycan (PG) at 0.2 mg/kg body weight/day for 2-3 months
- Stress test PL with 100 ppm formalin for 30 min with aeration
- Destroy infected stocks

- Strict hygiene
- Disinfection of ponds and inlet canals
- Use of only dry commercial feeds
- Pasteurize fresh feed at 60°C for 15 min
- Fine screening of inlet water
- Refrain from exchanging water for at least three days after infected ponds have been discharged
- Ban importation of non-indigenous shrimps
- Compliance with the Hazard Analysis and Critical Control Point (HACCP) programs for product safety
- Fucoidan (an extract from the seaweed *Cladosiphon okamuranus*) application at 60 - 100 mg/kg shrimp/day for 15 days

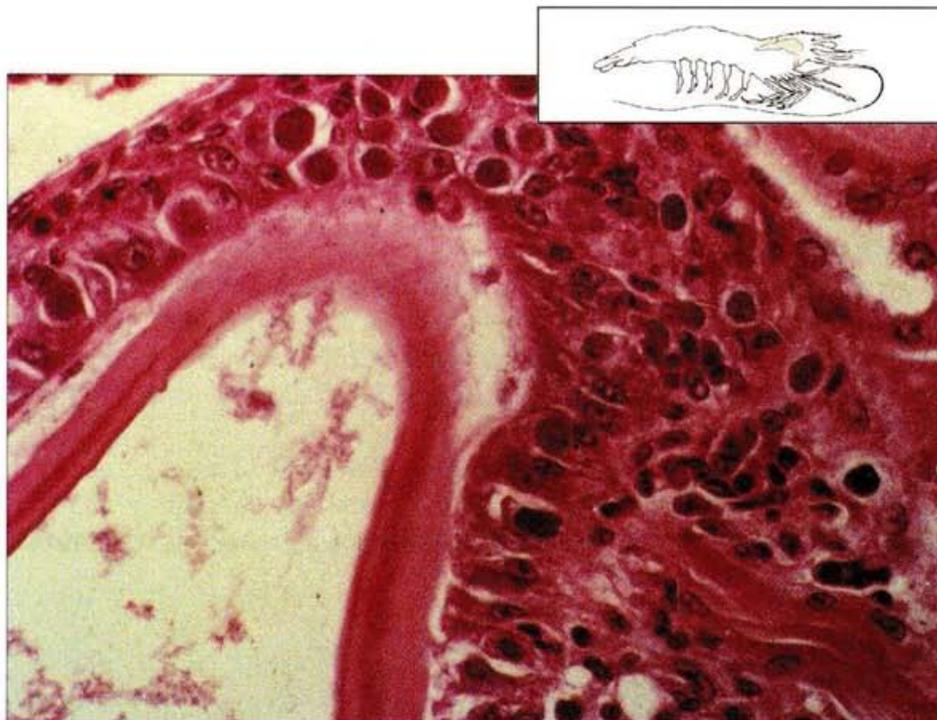


Figure 4a. Histological section of the stomach of shrimp with white spot infection showing intranuclear inclusions in hypertrophied nuclei. (H & E, 200X).

Common name: **YELLOW HEAD VIRUS (YHV) DISEASE**

Causative agent: Rhabdovirus (40-50 X 150-170 nanomicros)

Species affected: *Penaeus monodon*, *P. merguensis*, *P. setiferus*, *Acetes* spp.

Stages affected: Juveniles, sub-adults, broodstock

Gross signs:

- Light yellowish, swollen cephalothorax
- Whitish, yellowish or brown gills
- Abnormally high feed intake and rapid growth prior to cessation of feeding and the onset of rapidly accelerating mortality
- Marked reduction in feed consumption
- Moribund shrimp swim slowly near the surface at the edge of the pond
- Acute epizootic in juvenile to sub-adult shrimps about 20 days post-stocking especially during the 50-70 days grow-out culture period
- May be associated with unstable phytoplankton bloom, bad pond bottom, high stocking density or exposure to pesticides

Effects on host:

- Systemic infection associated with virus assembled in the cells of the gills, lymphoid organ, connective tissues, and hemocytes
- Massive necrosis due to the replication of the virus in these cells
- Can cause a total crop loss within 3 to 5 days of onset of clinical signs
- Incubation time is 7-10 days
- Viral extracts in water remain infective up to 72 h
- Reservoirs of infection include *Palaemon styliferus*
- In the Philippines, a recent sampling of 250 shrimps reported positive YHV in 16% of specimens

Preventive methods

- Use YHV-free stocks
- Destroy infected stocks
- Strict hygiene
- Disinfection of ponds and inlet canals
- Use of only dry commercial feeds
- Pasteurize fresh feed at 60C for 15 min
- Fine screening of inlet water
- Refrain from exchanging water for at least three days after infected ponds have been discharged
- Ban importation of non-indigenous shrimps
- Compliance with the Hazard Analysis and Critical Control Point (HACCP) programs for product safety

Treatment: None reported

Bacterial Diseases

**Luminous Bacterial Disease
(Hatchery)**

**Luminous Bacterial Disease
(Grow-out)**

**Shell Disease, Brown/Black
Spot, Black Rot/Erosion,
Blisters, Necrosis of
Appendages**

**Filamentous Bacterial
Disease**

Common name: **LUMINOUS BACTERIAL DISEASE (HATCHERY)**

Causative agent: *Vibrio harveyi*, *V. splendidus*

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Eggs, larvae, postlarvae

Gross signs:

- Larvae become weak and opaque-white
- Heavily infected larvae exhibit a continuous greenish luminescence when observed in total darkness. When viewed under the microscope, the internal tissues of these larvae are densely packed with highly motile bacteria
- Infected larval tissues streaked on nutrient agar medium show luminescent colonies after 18 - 24 hours incubation (Figure 5a)

Effects on host:

- Systemic infections result in mortalities in larvae and postlarvae, reaching up to nearly 100% of affected population
- Route of infection is oral
- Bacteria form plaques on the mouth apparatus of infected larvae (Figure 5b)
- Bacteria colonize the eggs (Figure 5c) which may then serve as sources of infection upon stocking in the rearing tanks

Preventive methods:

- Prevent the entry of luminous bacteria into the hatchery system by using chlorinated water (Appendix II), or ultraviolet-irradiated water, or by employing a series of filtration equipment (sandfilters, filter bags, cartridge filters, 0.45 micron pore-sized microfilter, etc.). Higher levels of chlorine may be used if necessary, but care must be taken to ensure complete dechlorination prior to use
- Use only previously chlorinated water during spawning and rearing to ensure a clean environment for newly hatched and developing larvae
- Remove the mothers from the tanks immediately after spawning and rinse the eggs with chlorinated water to prevent its colonization with luminous bacteria prior to hatching
- Rinse *Artemia* nauplii and other zooplankton before introducing them as food into larval rearing tanks
- Siphon out sediments and debris from the tank bottom since these could serve as substrates for bacterial growth
- Disinfect infected stock before finally discarding them followed by a complete clean-up and disinfection of hatchery paraphernalia after every larval rearing period (Appendix III)

Treatment:

- At the height of infection, water change must be 80-90% replacement daily

Note: The application of chemotherapeutics can be done with limited results as most luminous bacteria have acquired resistance to many chemotherapeutants (please refer to Baticados *et al.* 1990b).

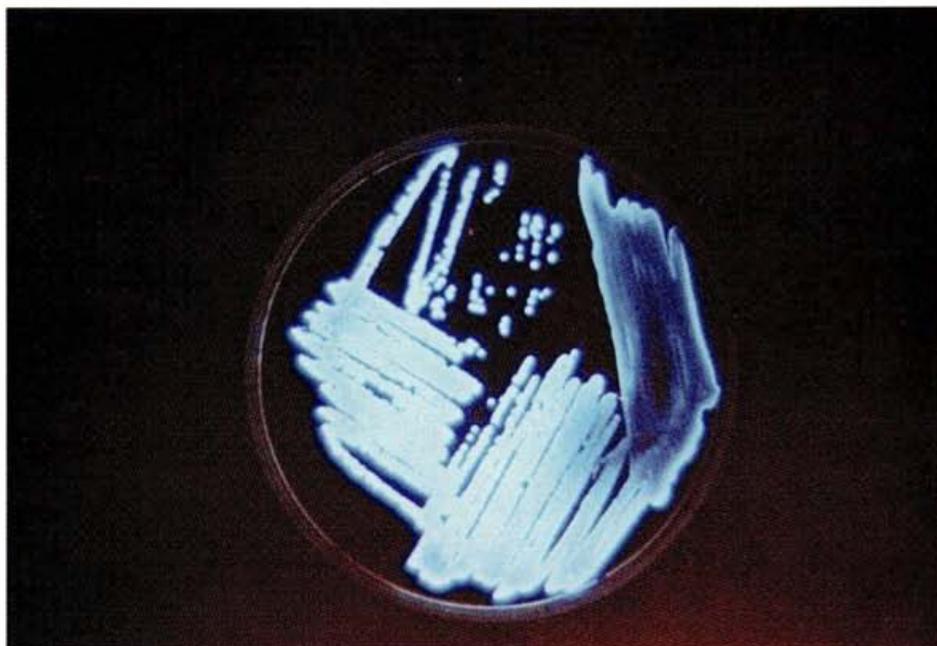


Figure 5a. Nutrient agar culture of the luminous bacterium, *Vibrio harveyi*. Photo taken in total darkness.



Figure 5b. Scanning electron micrograph (SEM) showing bacterial plaques on the mouth of moribund *Penaeus monodon* (mysis 1 stage) after challenge with *Vibrio harveyi* for 48 h (SEM, 4000X).

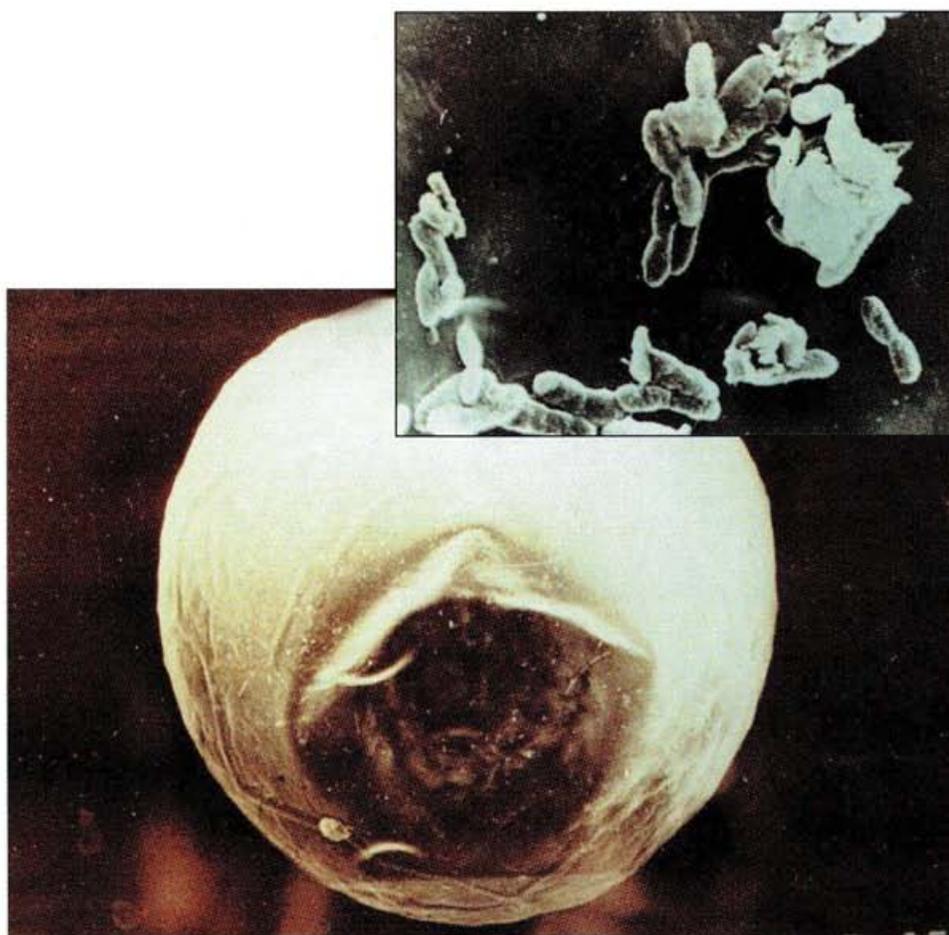


Figure 5c. Scanning electron micrograph of *Penaeus monodon* egg colonized by actively dividing cells (inset) of *Vibrio harveyi*.

Common name: **LUMINOUS BACTERIAL DISEASE (GROW-OUT)**

Causative agent: *Vibrio harveyi* and other luminescent vibrios

Species affected: *Penaeus monodon*

Stage affected: Juveniles

Gross signs:

- Heavily infected and moribund juveniles are luminescent when observed in the dark
- Infected shrimp swim near the edge of ponds with their heads poised near the water surface
- Occasionally, the region near the hepatopancreas appears dark. These clumps of brownish tissues are melanized tubules of the hepatopancreas (Figure 6)
- Green colonies predominate on thiosulfate citrate bile sucrose (TCBS) agar, a selective culture medium for vibrios

Effects on host:

- Infection results in mass mortality of juveniles especially in the first 45 days of culture
- The hepatopancreas shows atrophy and massive inflammatory response in and around the tubules
- Damaged tubules in the digestive organ become non-functional. This could lead to slow growth among survivors if a significant portion of the organ is affected

Preventive methods:

- Since the onset of mortality is preceded by the dominance of luminous bacteria in the rearing water, monitoring of the bacterial profile using microbial culture media should be done regularly especially in the first 45 days of culture
- Rearing water that is known to harbor a bacterial profile dominated by luminous vibrios should not be used for culture. Avoid prolonged exposure of postlarvae and juveniles to luminous bacterial counts of more than 10² colony-forming-units (cfu) per ml.
- Use reservoirs where settling of sediments, disinfection, conditioning and effective monitoring of bacterial load of the rearing water can be done
- Monitor the bacterial load of postlarvae for stocking. The associated flora should not be dominated by luminous vibrios
- Establish and maintain microbial diversity in the rearing environment through the green water culture system or the application of microbial bioaugmentation agents (commonly called probiotics)

Note: Administration of various antimicrobials had been tried with variable results.

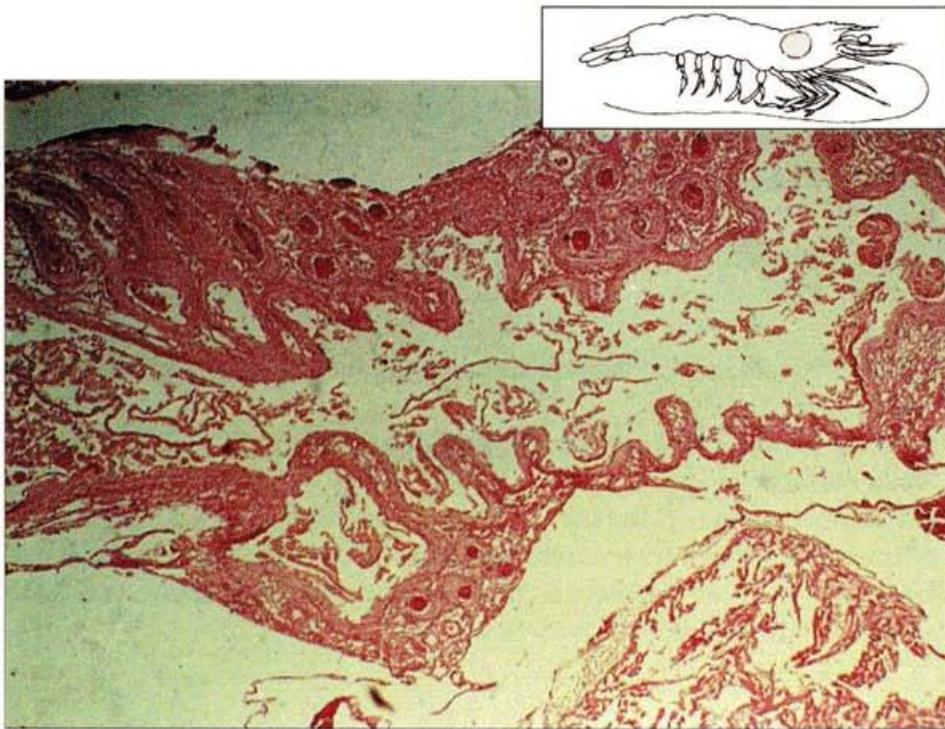


Figure 6. Histological section of a pond-reared *Penaeus monodon* juvenile with luminous bacterial infection. The central region of the hepatopancreas is atrophied with fibrosis and hemocyte infiltration in the intertubular spaces. Most of the tubules are melanized. (H & E, 40X).

Common name: **SHELL DISEASE, BROWN/BLACK SPOT, BLACK ROT/EROSION, BLISTERS, NECROSIS OF APPENDAGES**

Causative agent: Shell-degrading bacteria belonging to *Vibrio*, *Aeromonas* and *Pseudomonas* groups

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Larvae, postlarvae, juveniles, adults

Gross signs:

- Appearance of brownish to black erosion on the carapace, abdominal segments, rostrum, tail, gills, and appendages (Figures 7 a - c)
- Blister containing gelatinous fluid may develop on the carapace and abdominal segment (Figure 7d). The blister may extend to the underside of the lower section of the carapace creating a bulge.
- In larval and post-larval stages, the affected appendage shows a cigarette butt-like appearance (Figure 7a)

Effects on host:

- In larval or postlarval stages, shell disease occurs when there is a build-up of chitinoclastic bacteria on the shell following molting failure or organic fouling of the rearing water. Progressive erosion of these exoskeletal lesions follows upon multiplication of bacterial pathogens in the affected area. The infection may lead to loss of the affected appendage(s), or to the perforation of exoskeleton where localized infection in the underlying musculature may also follow. When these occur, normal locomotion or molting is hampered and may result in shrimp mortality
- The affected shrimp becomes susceptible to cannibalism or dies from stress or energy exhaustion
- In juvenile shrimps, infection may be initiated at sites of punctures or injuries made by either the telson or rostrum, cracks on the abdominal segment from sudden flexure of the shrimp body, or from other damage caused by cannibalism

Preventive methods:

- Maintain good water quality
- Keep organic load of the water at low levels by removing sediments, especially dead shrimps and molted exoskeletons which harbor high numbers of bacteria on the lesions
- Provide adequate diet
- Minimize handling and avoid overcrowding
- Avoid injuries to the exoskeleton of the shrimps to prevent the development of primary portals of entry

Treatment:

- Induce molting as the condition is eliminated upon molting except when underlying tissues are damaged

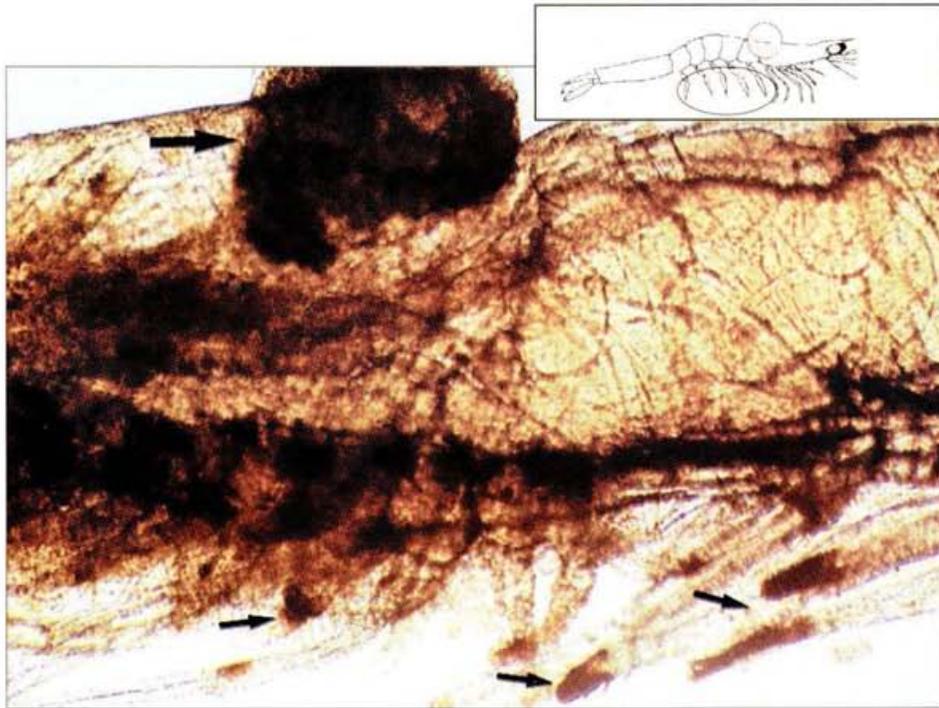


Figure 7a. Wet mount of postlarval *Penaeus monodon* with necrotic appendages and melanized blister on the carapace. (arrows).

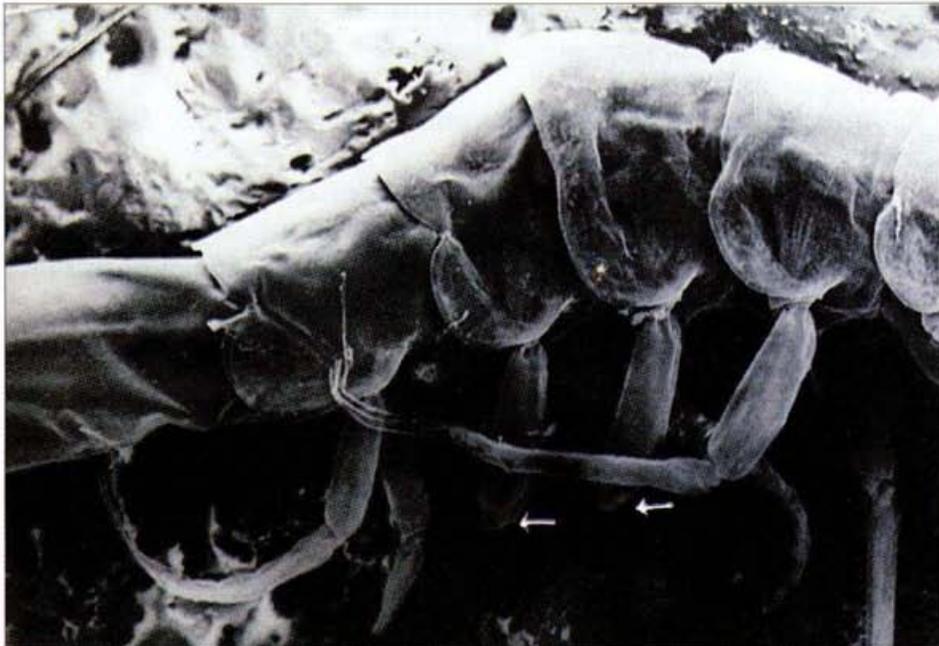


Figure 7b. Scanning electron micrograph showing necrosis of appendages of postlarval *Penaeus monodon* (arrows). (SEM, 94X)



Figure 7c. High magnification view of a necrotic appendage showing a colony of bacteria gradually 'eating away' the affected appendage of a *Penaeus monodon* postlarva. (SEM, 3000X).

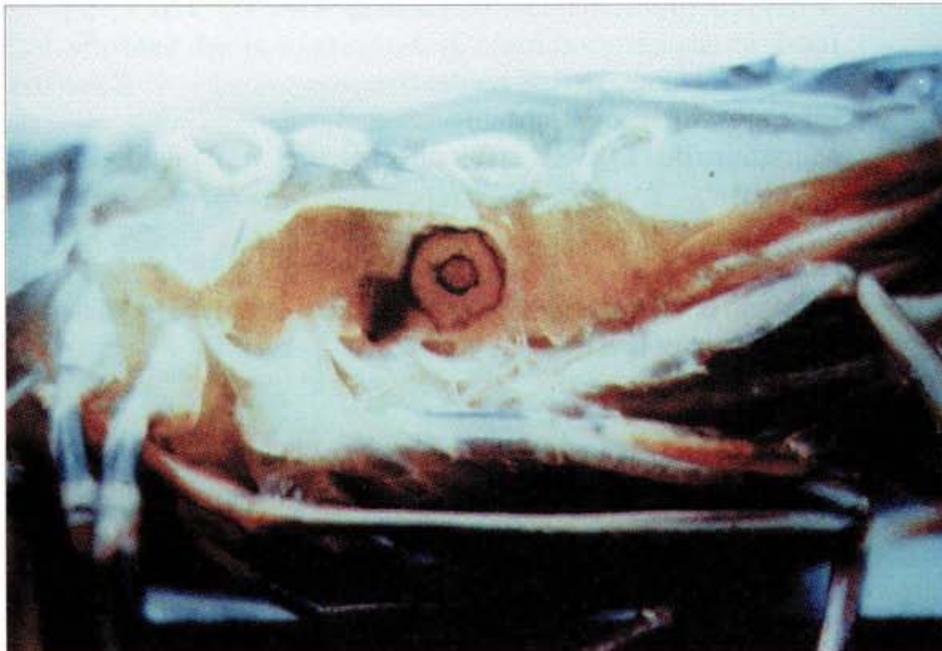


Figure 7d. Shell erosion and black spot on the carapace of adult *Penaeus monodon*.

Common name: **FILAMENTOUS BACTERIAL DISEASE**

Causative agent: *Leucothrix sp.*

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Larvae, postlarvae, juveniles, adults

Gross signs:

- Presence of fine, colorless, thread-like growth on the body surface and gills as seen under a microscope (Figures 8 a - b)

Effects on host:

- Infected eggs show a thick mat of filaments on the surface which may interfere with respiration or hatching
- If found on the gill surface, filamentous bacteria block respiratory surfaces and cause respiratory problems (Figures 8 c - d)
- In larvae and postlarvae, filamentous growth on appendages and body surface may interfere with normal movement and with molting, and may entrap other microorganisms (like fungal spores), which may initiate a new infection
- Larval shrimps are less prone to infestations by filamentous bacteria than post-larval, juvenile, and adult stages due to the rapid succession of molts throughout the different larval stages. Frequent molting does not allow adequate time for the bacteria to accumulate on the exoskeleton
- In larger shrimps, filamentous bacteria on the gills and other body surfaces may result in respiratory distress at the point of attachment. An indirect effect of such filamentous growth on the host is entrapment of algae and debris which interfere with respiration and promote further fouling
- Mortalities due to direct and indirect effects of filamentous bacteria have been reported

Preventive methods:

- Maintain good water quality with optimum dissolved oxygen (>5 ppm) and low organic matter levels

Treatment:

- 0.15 ppm copper (Cutrine-Plus) in 24-h flow through treatments or 0.5 ppm copper in 4 to 6 h static treatments for postlarva 2 or older. For treatment guidelines, see Appendix IV.

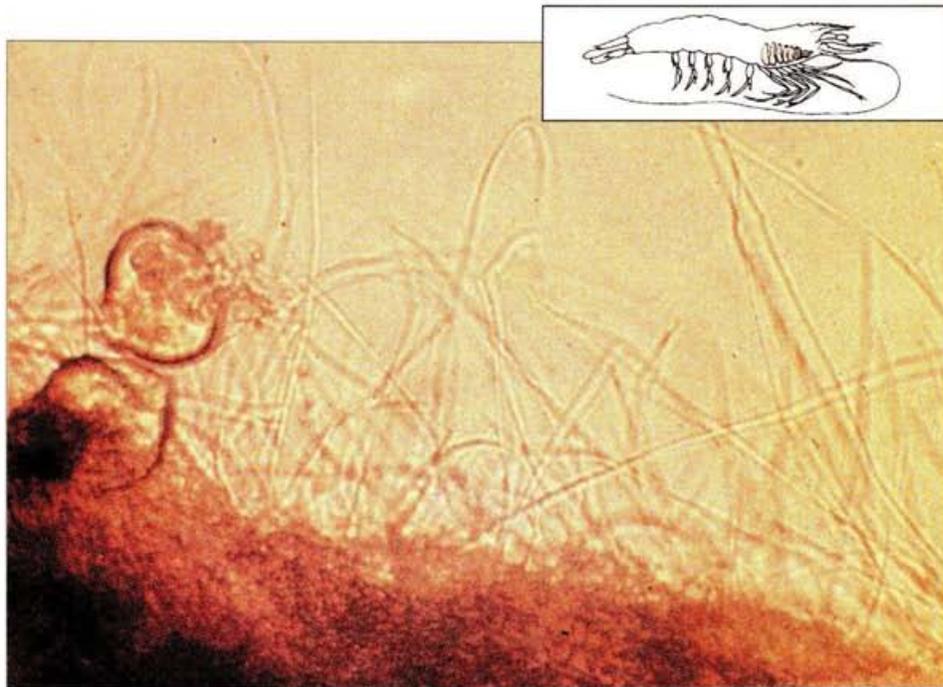


Figure 8a. Strands of filamentous bacterium *Leucothrix* sp. on heavily infested gills of juvenile *Penaeus monodon*. At upper left is the protozoan *Zoothamnium*. (Wet mount, 200X).

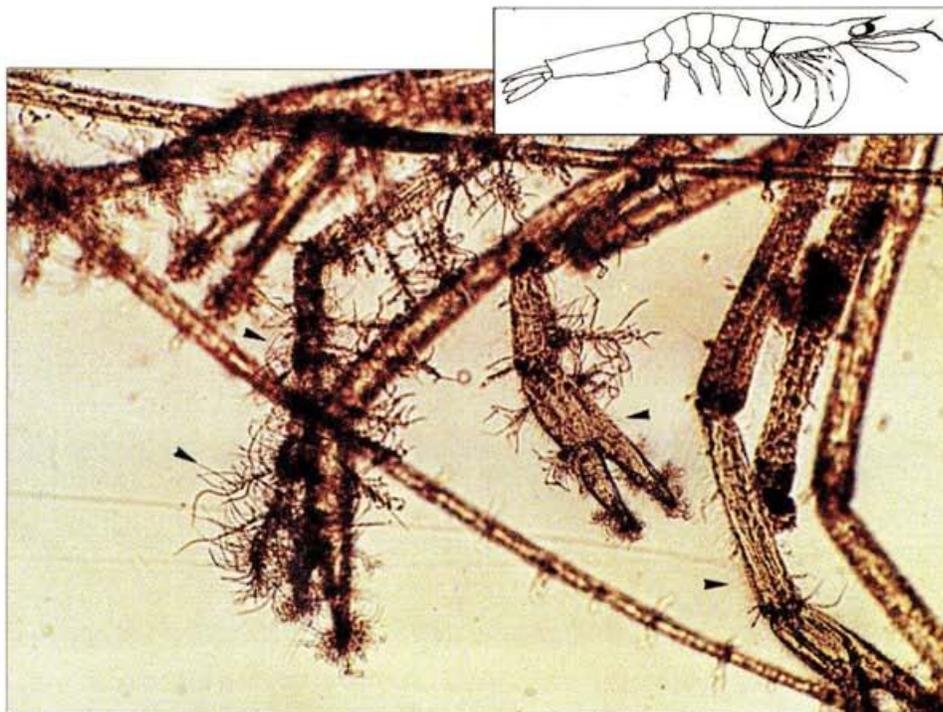


Figure 8b. Wet mount of *Penaeus monodon* postlarva with filamentous bacteria (arrowheads) on the appendages.



Figure 8c. Scanning electron micrograph of the gills of *Penaeus monodon* postlarva showing strands of filamentous bacteria attached to the lamellae. (SEM, 540X).



Figure 8d. 'Rosettes' of bacterial filaments (arrowheads) colonize the lamella and block respiratory surfaces (SEM, 1100X).

Fungal Disease

Larval Mycosis

Common name: **LARVAL MYCOSIS**

Causative agents: *Lagenidium callinectes*, *Lagenidium* sp., *Haliphthoros philippinensis*, and *Sirolopidium* sp. In *Lagenidium*, zoospores are developed in a vesicle which is formed at the end of discharge tube. *Haliphthoros* does not form vesicles but has long discharge tubes, whereas *Sirolopidium* has short discharge tubes and forms no vesicle. These aquatic fungi produce highly motile zoospores that can easily invade other hosts.

Species affected: *Penaeus monodon*

Stages affected: Eggs, larvae, early postlarvae

Gross signs:

- Infected eggs, larvae, and postlarvae appear whitish, become weak, and may eventually die
- Signs, such as presence of fungal filaments and their reproductive structures within infected tissues (Figure 9), are readily apparent when disease is already widespread

Effects on host:

- Heavy mortalities up to 100% within 2 days may occur. The fungal hyphae replace the internal tissues of the shrimp and extend outside the shrimp body to form discharge tubes. Infected eggs do not hatch and larvae lose equilibrium and exhibit respiratory difficulties.

Preventive methods:

- Siphon sediments and dead shrimps
- Reduce stocking density
- Increase water circulation
- Disinfect materials and tank with 100 ppm detergent (Tide*) for 24 h
- Observe rigid water management and sanitation
- Disinfect eggs with 20 ppm detergent for 2 h at least 6 h before hatching; for spawners, use 5 ppm Treflan* for 1 h. (Appendix V)
- In areas where larval mycosis is known to occur, Treflan* or trifluralin may be used at prophylactic levels of 0.1 ppm every 2-3 days
- Dispose infected stocks only after disinfection with 100 ppm detergent
- Regular monitoring of the stock species through microscopic examination is important

Treatment:

- 0.2 ppm Treflan* or trifluralin for 24 h (Appendix VI)

* Mention of brand name does not mean endorsement of the product.

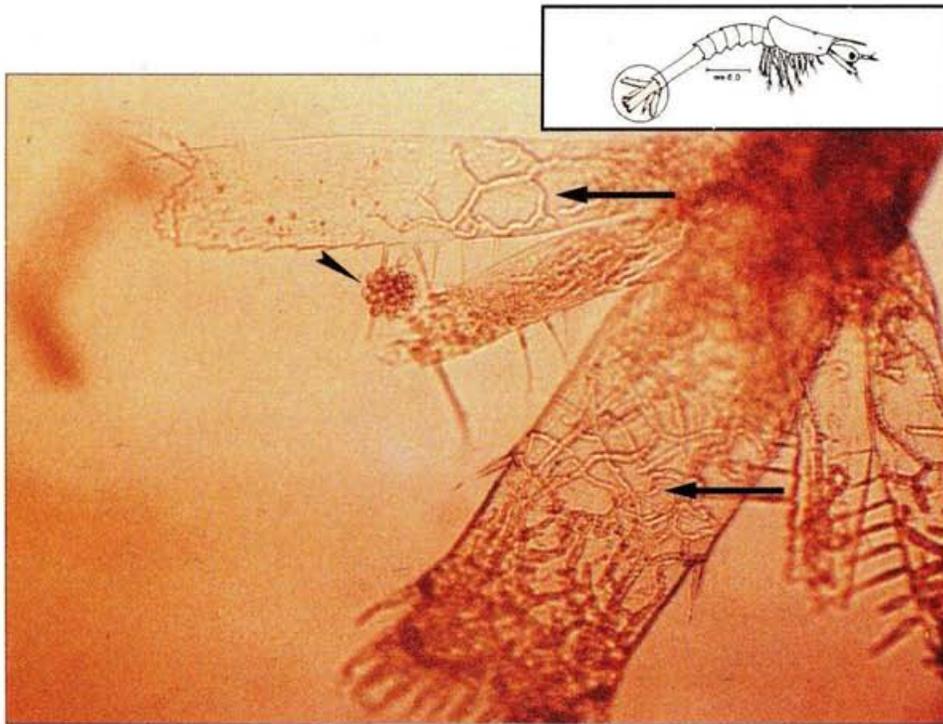


Figure 9. Filaments of *Lagenidium* in the tail of *Penaeus monodon* larva (arrows). Zoospores in a vesicle (arrowhead) are about to be released by the fungus. (Wet mount, 100X).

Protozoan Diseases

Ciliate Infestation

**Microsporidiosis, White
Ovaries, Microsporidian
Infection**

Gregarine Infestation

Common name: **CILIATE INFESTATION**

Causative agents: *Vorticella*, *Epistylis*, *Zoothamnium*, *Acineta*, and *Ephelota*. *Vorticella* is solitary and has a contractile stalk. *Zoothamnium* and *Epistylis* are both colonial, but only the former has a contractile stalk. *Acineta* and *Ephelota* are suctoreans equipped with feeding tubules (Figure 10a)

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Eggs, larvae, postlarvae, juveniles, adults

Gross signs:

- Fuzzy mat on shell and gills of heavily infected juveniles and adults
- Reddish to brownish gills
- Loss of appetite

Effects on host:

- Microscopically, protozoans may be observed attached to any external part of the shrimp
- The protozoans may cause locomotory difficulties when present in large numbers
- Respiratory problems occur when they are present in large numbers on the gills (Figure 10b), particularly at low dissolved oxygen levels

Preventive methods:

- Maintain good water quality
- Avoid high organic load, heavy siltation, turbidity, and low oxygen levels

Treatment:

- Among juveniles in nursery tanks, application of 1.1 ppm chloroquin diphosphate for 2 days was reported to be effective against the ciliates after three treatments
- *Zoothamnium* infestation in adults was reported to be effectively treated with 50-100 ppm formalin for 30 min
- *Epistylis* infestation in juveniles was observed to be eliminated by 30 ppm formalin
- Change water by draining from the pond or tank bottom daily to remove excess feeds, fecal matter, and other organic wastes

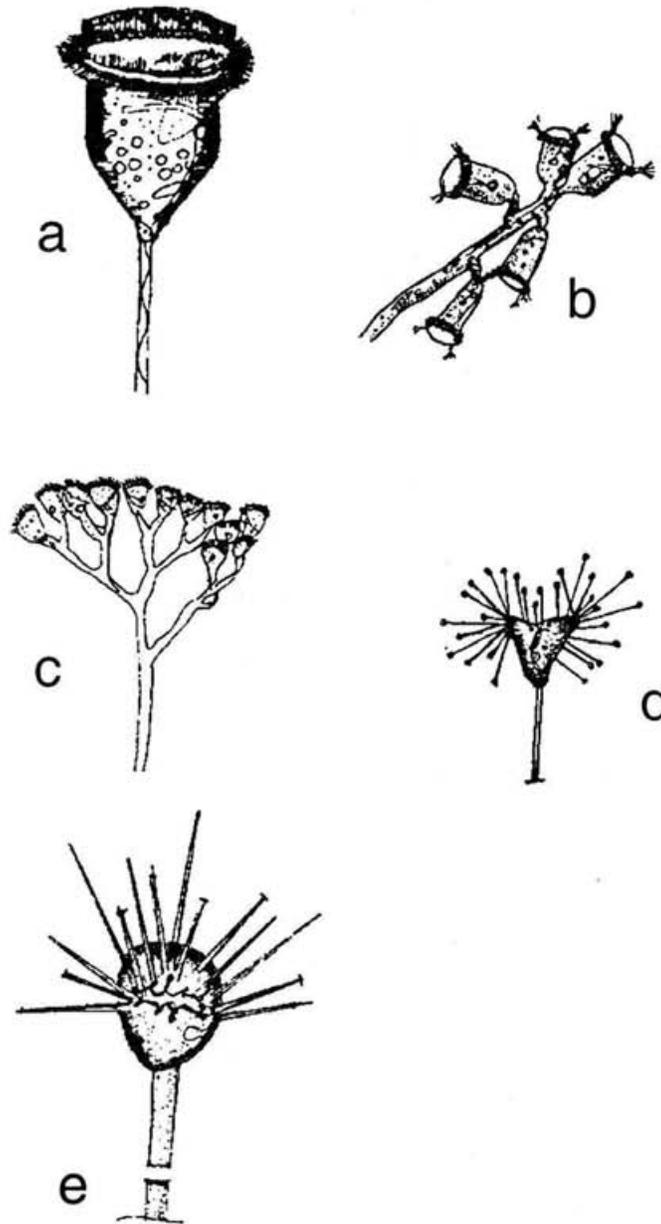


Figure 10a. The protozoans a. *Vorticella*, b. *Epistylis*, c. *Zoothamnium* d. *Aniceta* and e. *Ephelota*.

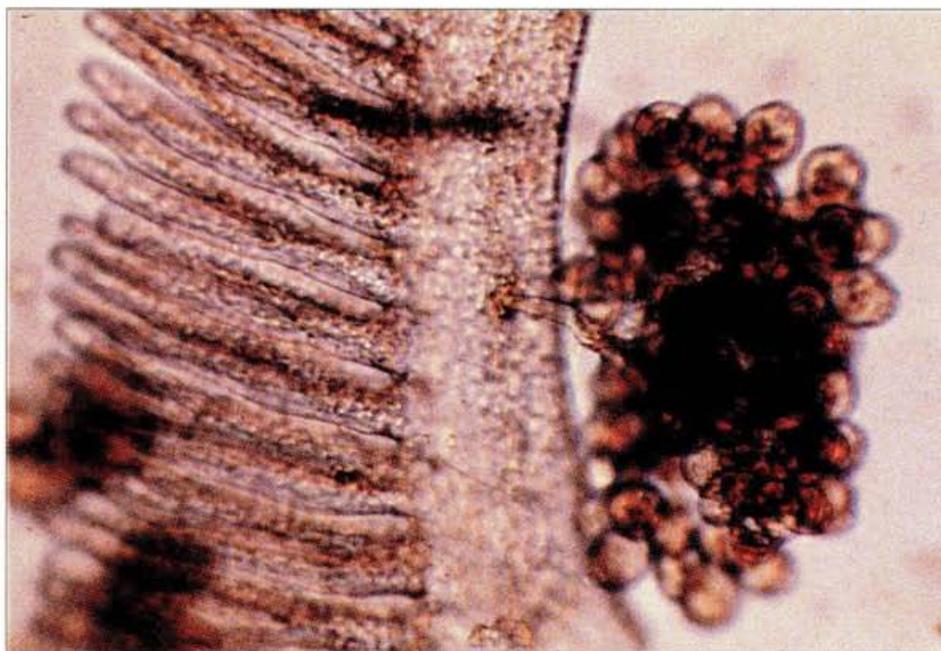


Figure 10b. The ciliate *Zoothamnium* on gills of *Penaeus monodon* adult. (Wet mount, 200X).

Common name: **MICROSPORIDIOSIS, WHITE OVARIES, MICROSPORIDIAN INFECTION**

Causative agent: Microsporidia. These are endoparasitic protozoans that may be diagnosed only through microscopic examination of the infected tissues.

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Juveniles, adults

Gross signs:

- Affected tissues/organs turn opaque white (Figure 11)

Effects on host:

- Spores and other stages of the parasite replace the affected tissues
- Infection may result in sterility of spawners with white ovaries
- Infection rate is relatively low (usually <10%) but the parasite is highly pathogenic

Preventive methods:

- Disinfect culture facilities with chlorine or iodine-containing compounds
- Isolate and destroy infected shrimps by burning or boiling

Treatment:

- None reported



Figure 11. Microsporidian infection of the abdominal muscles of *Penaeus indicus* (top) and ovary of *P. monodon* (bottom).

Common name: **GREGARINE INFESTATION**

Causative agent: Gregarines. These are protozoan parasites commonly found in the digestive tract of penaeid shrimps. They utilize a mollusc species as intermediate host.

Species affected: *Penaeus monodon*, other penaeids may also be affected

Stages affected: Larvae, postlarvae, juveniles, adults

Gross sign:

- Gregarines may be detected in the digestive tract microscopically (Figures 12 a - c)

Effects on host:

- Large numbers of this protozoans could interfere with particle filtration through the gut or the hepatopancreatic duct
- Infection rate in pond-grown prawns was reported to reach 94%

Preventive method:

- In the hatchery, filter or chlorinate seawater used for rearing
- In grow-out ponds, eliminate the molluscan intermediate host

Treatment:

- None reported

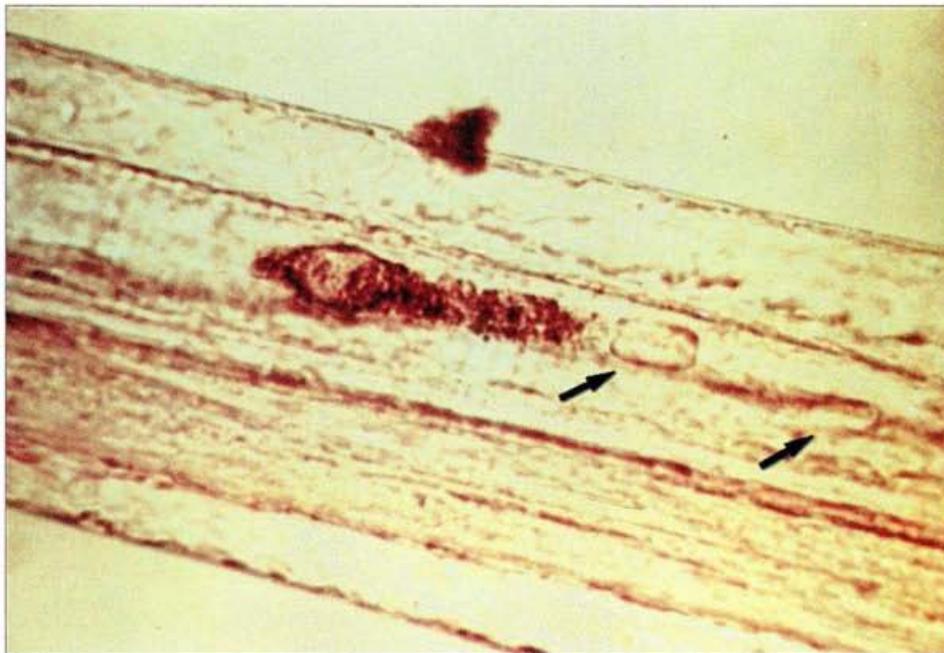


Figure 12a. Gregarines (arrows) in the gut of *Penaeus monodon* postlarva. (Wet mount, 200X).

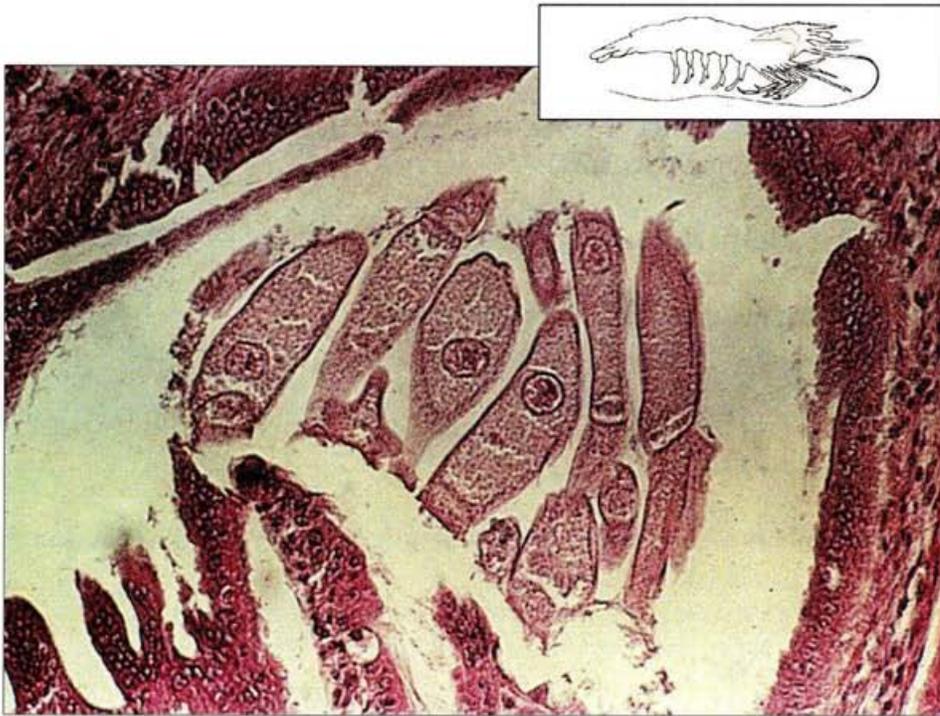


Figure 12b. Histological section showing gregarines in the anterior midgut cecum of pond-reared shrimp juvenile. (H & E, 200X).

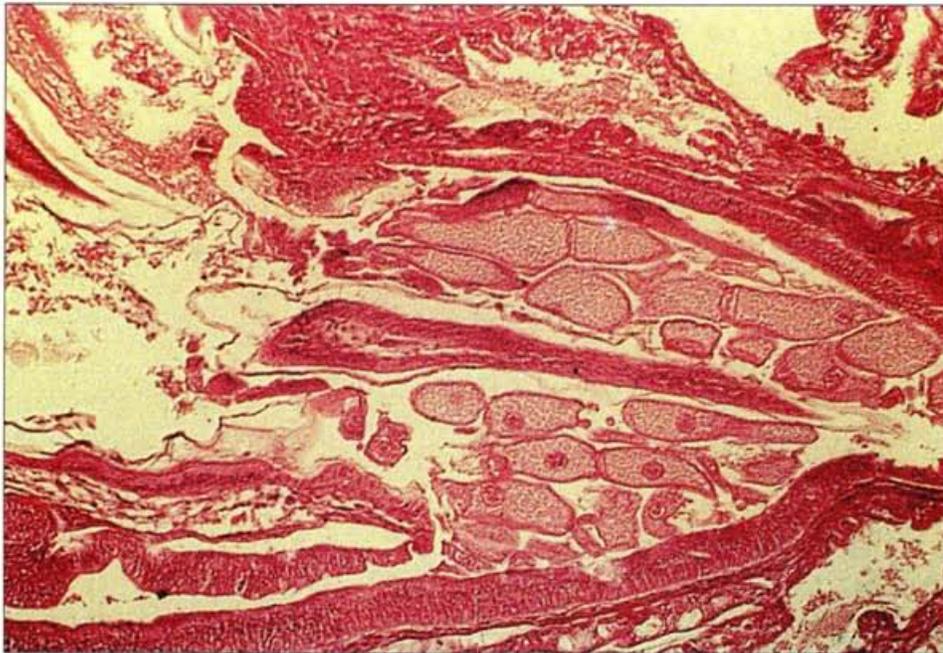


Figure 12c. Histological section showing gregarines in the stomach of pond-reared shrimp juvenile. (H & E, 100X).

Nutritional, Toxic and Environmental Diseases

Incomplete Molting

**Swollen Hindgut
Syndrome**

**Chronic Soft-shell
Syndrome, Soft-
shelling**

Black Gill Disease

**Blue Disease, Sky
Blue Shrimp
Disease, Blue
Shell Syndrome**

**Red Disease, Red
Discoloration**

Underfeeding

Muscle Necrosis

**Cramped Tails, Bent
Tails, Body Cramp**

**Acid Sulfate Disease
Syndrome**

Asphyxiation, Hypoxia

**Bamboo Back
Syndrome**

Common name: **INCOMPLETE MOLTING**

Causative agent: Unknown, although this occurrence is closely associated with low rearing water temperature

Species affected: Hatchery-reared *Penaeus monodon*; may also affect other species of penaeids

Stages affected: Zoea and mysis stages, occasionally in postlarvae

Gross signs:

- Presence of old skeleton attached to newly molted larvae, especially in the area of the appendages (Figures 13 a - b)

Effects on host:

- Abnormal swimming movement which could lead to easy predation
- Mortality

Preventive methods:

- Maintain optimum temperature in the rearing water

Treatment:

- Use of heaters to raise and maintain temperature at optimum levels

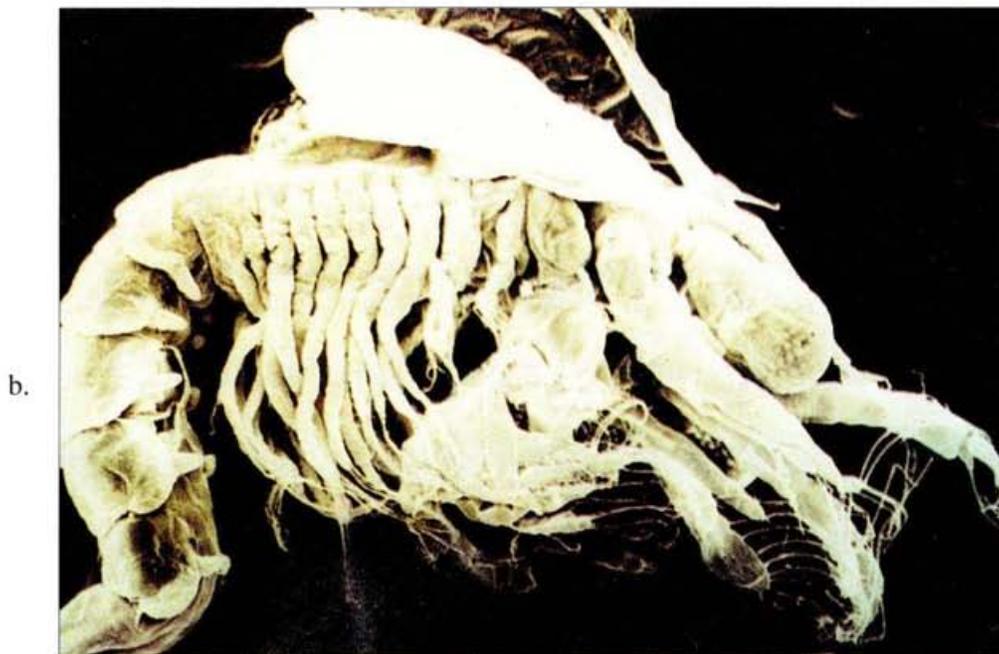
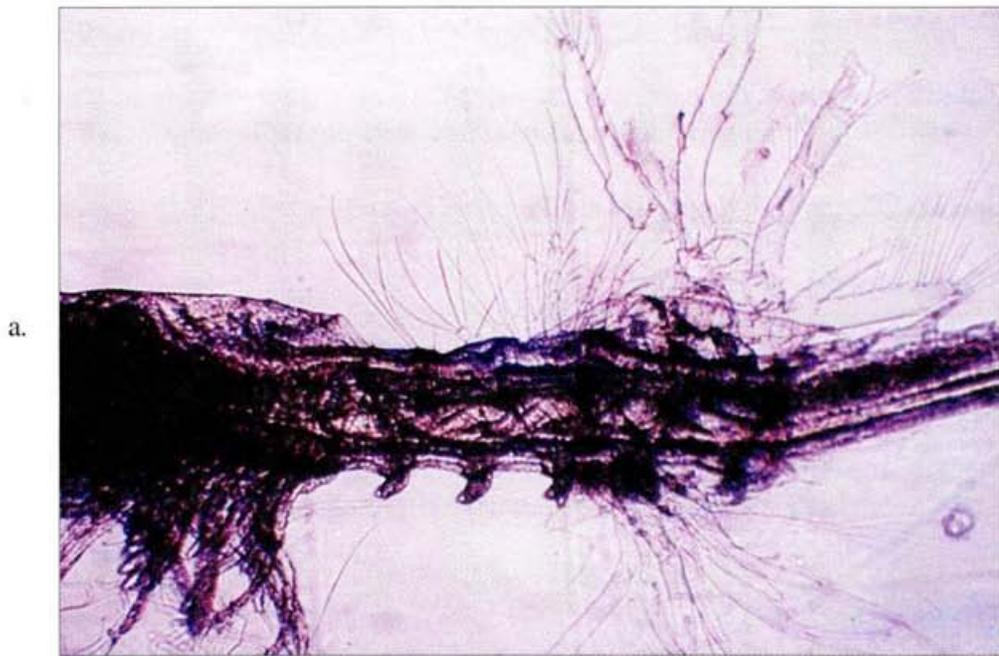


Figure 13. Incomplete molting in mysis stage larva. The old exoskeleton remains adhered to the abdominal segments (a), and the anterior appendages and carapace of the larvae (b). Photo in Figure 13a is a wet mount while Figure 13b is a scanning electron microphotograph.

Common name: **SWOLLEN HINDGUT (SHG) SYNDROME**

Causative agent: Unknown, but condition may be triggered by poor feed quality such as senescent and contaminated phytoplankton

Species affected: Hatchery-reared *Penaeus monodon*

Stages affected: Postlarvae

Gross signs:

- The normally triangular hindgut (Figure 14a) becomes swollen and bulbous (Figure 14b)
- In some cases, swelling occurs in the midgut-hindgut junction (Figure 14c)
- Melanization may sometimes appear giving the hindgut a brown color (Figure 14d)

Effects on host:

- Affected postlarvae fail to expel their fecal pellets due to the cessation of rhythmic movements of the rectal pads
- Delayed development due to molting failure
- Cumulative mortality of up to 65% in postlarvae exhibiting severely swollen hindgut

Preventive methods:

- Rigid sanitary procedures in water management and handling of natural food
- Proper storage of artificial feeds

Treatment:

- Complete reversal of swollen hindgut syndrome has been observed under experimental conditions after 10 days of rearing affected postlarvae in UV-sterilized seawater with rinsed *Artemia* nauplii as its sole food source

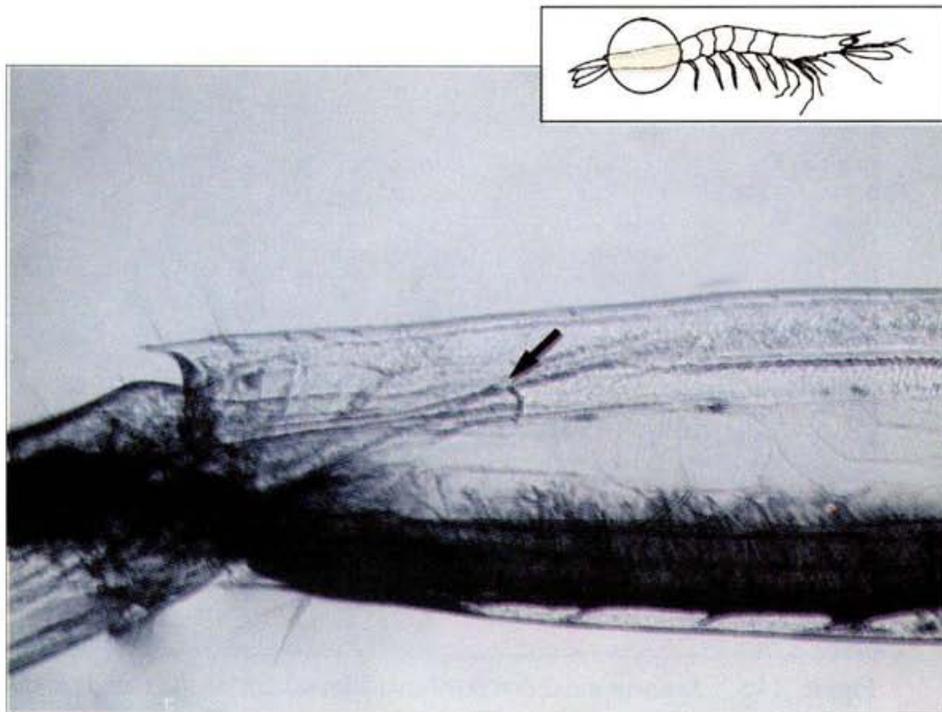


Figure 14a. The sixth abdominal segment of a *Penaeus monodon* postlarva showing normal hindgut (arrow). (Wet mount, 200X).

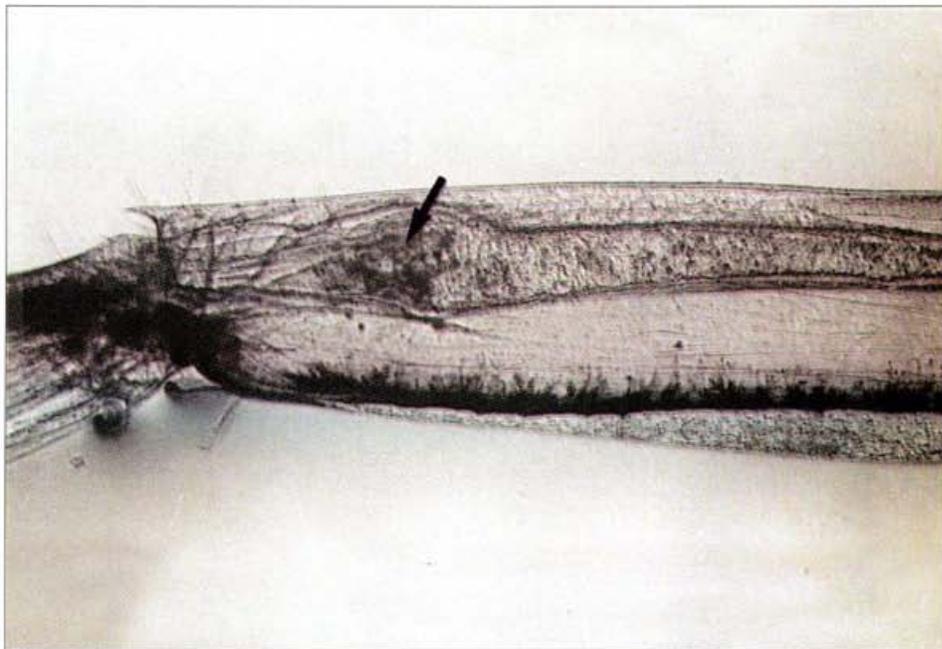


Figure 14b. *Penaeus monodon* postlarva with severe swollen hindgut syndrome showing necrotic and distended hindgut pad tissues (arrow). (Wet mount, 200X).

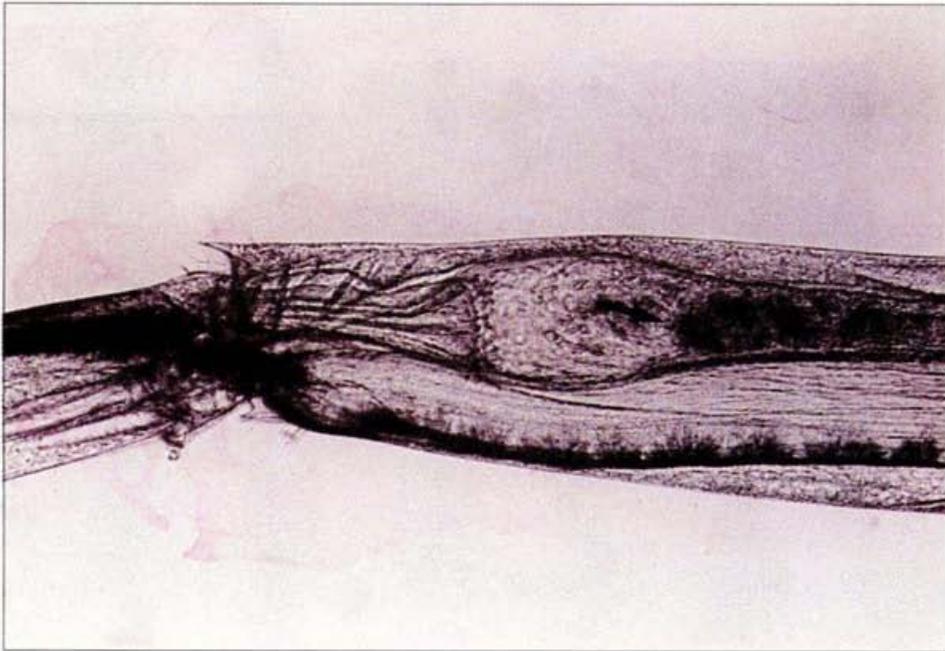


Figure 14c. *Penaeus monodon* postlarva with severe swollen hindgut syndrome affecting the midgut-hindgut junction. The hindgut pads appear slightly affected, but there is a dense accumulation of fecal material (arrow) resulting from the cessation of rhythmic movements to expel them. (Wet mount, 200X).

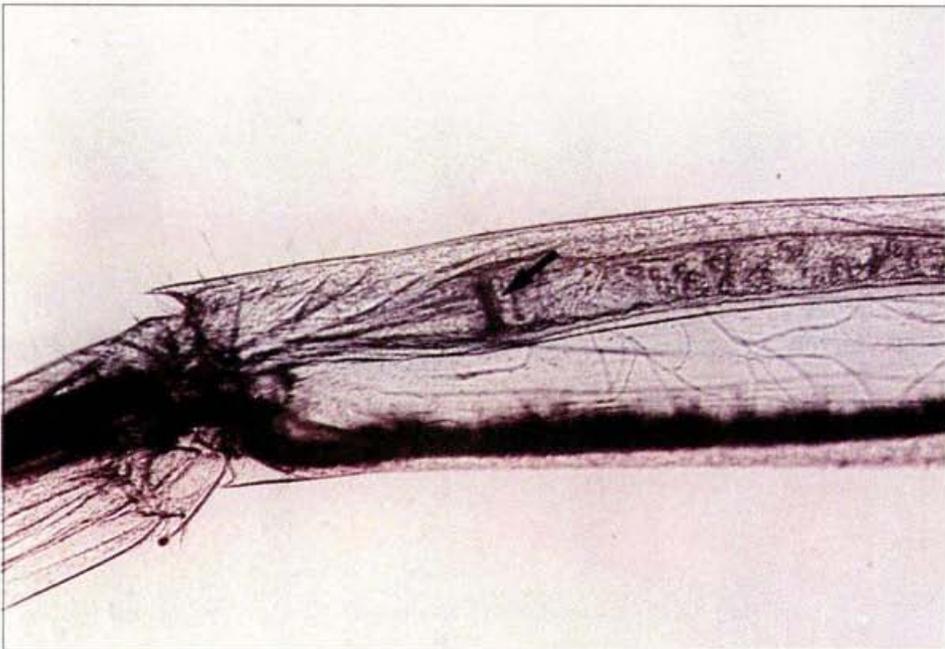


Figure 14d. *Penaeus monodon* postlarva with slightly swollen hindgut with melanization (arrow) in the anterior portion of the hindgut pads. (Wet mount, 200X).

Common name: **CHRONIC SOFT-SHELL SYNDROME, SOFT-SHELLING**

Causative agents:

- Nutritional deficiency; pesticide contamination; and poor pond water and soil conditions
- Exposure of normal hard-shelled shrimps to pesticides and piscicides. Aquatin or Gusathion A at 75-150 ppb, rotenone at 10 - 50 ppm, and saponin at 100 ppm for 4 days induced soft-shelling in initially hard-shelled stocks.
- Pond surveys also indicated that the occurrence of soft-shelling could be predicted with 98% accuracy under conditions of high soil pH, low water phosphate, and low organic matter content in the soil. Of the ponds that were surveyed and had soft-shelling of shrimps, 70% had high soil pH (>6), low water phosphate (<1ppm), and low organic matter content (<7%).
- Inadequate feeding practices like improper storage of feeds, use of rancid or low-quality feeds, and lack of supplementary feeding in ponds with relatively higher stocking densities were also highly correlated with significantly high incidence of soft-shelling
- Insufficient or infrequent water exchange was highly correlated with soft-shelling

Species affected: *Penaeus monodon*

Stages affected: Juveniles, adults

Gross signs:

- Shell is thin and persistently soft for several weeks, shell surface is often dark, rough and wrinkled, and affected shrimps are weak (Figure 15)
- The disease must not be confused with the condition of newly molted shrimps, which have clean, smooth, and soft shells that harden within 1-2 days

Effects on host:

- Affected shrimps are soft-shelled, grow slowly, and eventually die
- Shrimps become more susceptible to wounding, cannibalism, and surface fouling by *Zoothamnium* and other epicommsals
- Histopathology of shrimps exposed to Gusathion A shows slight hyperplasia of the gill epithelium, delamination of the cells lining the tubules of the hepatopancreas, and general necrosis and degeneration of these tissues

Preventive methods:

- During pond preparation, flush ponds thoroughly particularly when pesticide contamination is suspected
- Maintain pond water and soil of good quality
- Feed shrimps adequately and use only good-quality feeds

Treatment:

- Provide rigid water management, e.g., water change of 20-50% daily in ponds
- Provide supplementary feed, e.g., mussel meat at 8-14% of the body weight daily for 2-4 weeks, or a diet containing 1:1 ratio of calcium-to-phosphorus
- Water must be changed immediately, particularly when pesticide contamination is suspected



Figure 15. *Penaeus monodon* with soft shell syndrome showing dark, rough and soft shell.

Common name: **BLACK GILL DISEASE**

Causative agents:

- Chemical contaminants like cadmium, copper, oil, zinc, potassium permanganate, ozone, ammonia, and nitrate in rearing water
- Ascorbic acid deficiency
- Heavy siltation
- High organic load due to residual feed, debris, and fecal matter on pond bottom (i.e., the black soil)

Species affected: *Penaeus monodon*

Stages affected: Larvae, postlarvae, juveniles, adults

Gross signs:

- The gills show reddish or brownish to black discoloration, and atrophy at the tip of the filaments (Figure 16)
- In advanced cases, most of the filaments are affected and the gills become totally black
- Dorsal side of the body may be covered with fog-like substance
- Loss of appetite
- Mortalities

Effects on host:

- Histological observation shows that the blackening of the gills may be due to the heavy deposition of black pigment at sites of heavy hemocyte activity (inflammation)
- Extensive accumulation of blood cells in the gill filaments may result in respiratory disturbances
- Absorption of silt on the gills may also result in respiratory difficulties
- Secondary infections by bacteria, fungi, and protozoans via the dying cells of the gills

Preventive methods:

- Avoid overfeeding
- Change water frequently
- Remove black soil by scraping after harvest and draining from the bottom or “vacuuming” during culture period
- Flush out ponds several times during pond preparation
- Avoid heavy metal discharge of nearby factories from getting into the rearing facilities

Treatment:

- If the disease is due to heavy siltation or chemical contamination, change water immediately and daily by draining from the bottom
- If the disease is due to ascorbic acid deficiency, supplement diet with adequate amounts of ascorbic acid (>2000 mg/kg of feed) or fresh algae

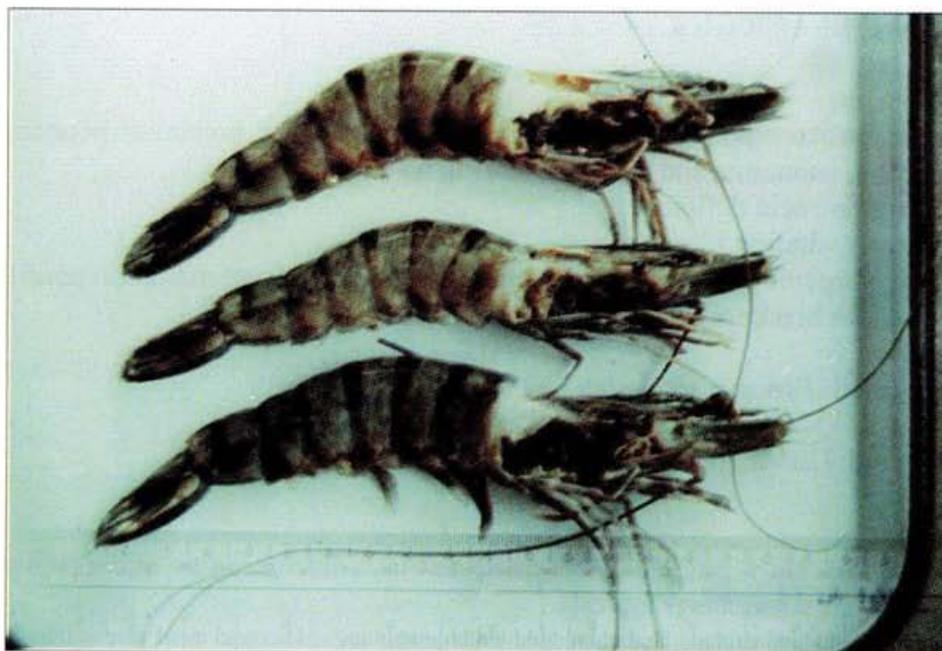


Figure 16. *Penaeus monodon* juveniles showing severely discolored and blackish gills.

Common name: **BLUE DISEASE, SKY BLUE SHRIMP DISEASE, BLUE SHELL SYNDROME**

Causative agents:

- Soil-water quality problems, e.g. acid-sulfate soil, high organic wastes, and low dissolved oxygen levels. The disease is commonly observed in intensive culture systems toward the end of the grow-out culture period.
- Low levels of carotenoid astaxanthin in the diet

Species affected: *Penaeus monodon*

Stages affected: Juveniles, adults

Gross signs:

- Sky-blue color instead of the normal brown-black (Figure 17)
- Lethargic shrimps show shells that are soft and thin with rough surface
- The shell becomes prone to shell disease
- Absence of intense red color after cooking

Effects on host:

- Histopathological changes in the hepatopancreas, e.g., disruption of the tubules

Preventive methods:

- Incorporate Vitamin A or carotenoid sources, like yellow corn, in the diet 45 days after stocking
- Change 10 to 15% of the water volume daily to diminish the hydrogen sulfide-rich bottom layers of water
- Reduce stocking density
- Provide high quality food

Treatment:

- Supplement diet with carotenoid sources



Figure 17. *Penaeus monodon* adults with blue shell syndrome (2nd and 4th from left).

Common name: **RED DISEASE, RED DISCOLORATION**

Causative agents:

- Rancid feeds due to prolonged storage at high temperatures
- Presence of aflatoxin B₁ (produced by the fungi *Aspergillus* spp.) in feeds or feed ingredients
- High water pH due to large inputs of lime (2-6 tons/ha)
- Prolonged exposure to low salinity (6-15 ppt)
- Secondary infection with *Vibrio parahaemolyticus* and *V. harveyi*

Species affected: *Penaeus monodon*, may also affect other cultured penaeids

Stages affected: Late postlarvae, juveniles, adult

Gross signs:

- The first sign of the disease is a sudden drop in feed consumption
- The animals then become lethargic and show general body weakness; death ensues within minutes after being lifted out of the water
- Many of the affected animals are confined to shallow waters at the pond periphery
- Body and appendages of affected shrimp become yellowish, then yellowish pink, then pink and eventually red (Figure 18a)
- Red, short streaks on gills
- Brown to brownish red fecal matter
- Increased fluid in the cephalothorax emitting foul odor

Effects on host:

- Yellow to red discoloration in affected shrimps
- Slow growth and reduced resistance to stress
- Increased susceptibility to systemic bacterial infection and shell disease
- Aflatoxin B₁ levels of 75 parts per billion (ppb) or more in feed causes atrophy and necrosis of the hepatopancreas after 60 days of continuous feeding
- Histopathology of the hepatopancreas shows hemocytic infiltration in the spaces in between the tubules; more advance lesions are in the form of fibrotic and melanized encapsulation of necrotic tissues, either in the tubule itself or the sinuses around it (Figures 18 b - c)
- Experimental infection through injection of healthy shrimp with *V. parahaemolyticus* (10⁷, 10⁶, 10⁵ colony-forming-units (cfu) per shrimp) and *V. harveyi* (10⁷ cfu per shrimp) reproduced the characteristic red discoloration in juvenile shrimp suggesting bacterial involvement in some cases

Preventive methods:

- Use only recently manufactured feeds
- Store feeds properly in well-ventilated and cool rooms for not more than 2 months
- Submit suspected batches of feeds for aflatoxin B₁ and rancidity analysis

- Withdraw and stop giving contaminated feeds as rations
- Prepare pond bottom properly
- Reduce lime input during pond preparation
- Maintain a low organic load in the environment to prevent the proliferation of potentially pathogenic bacteria

Treatment: None reported

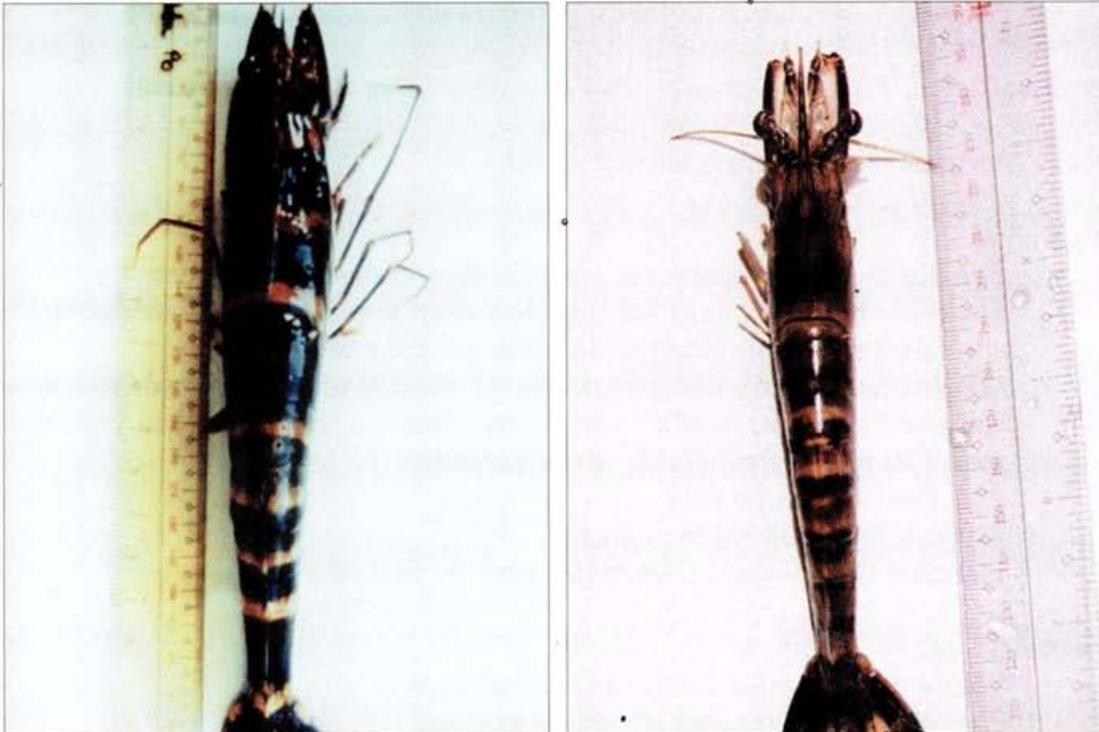


Figure 18a. *Penaeus monodon* adults with red disease. Left: early signs, natural incidence; right: red discoloration after 3 weeks of exposure to aflatoxin B₁.

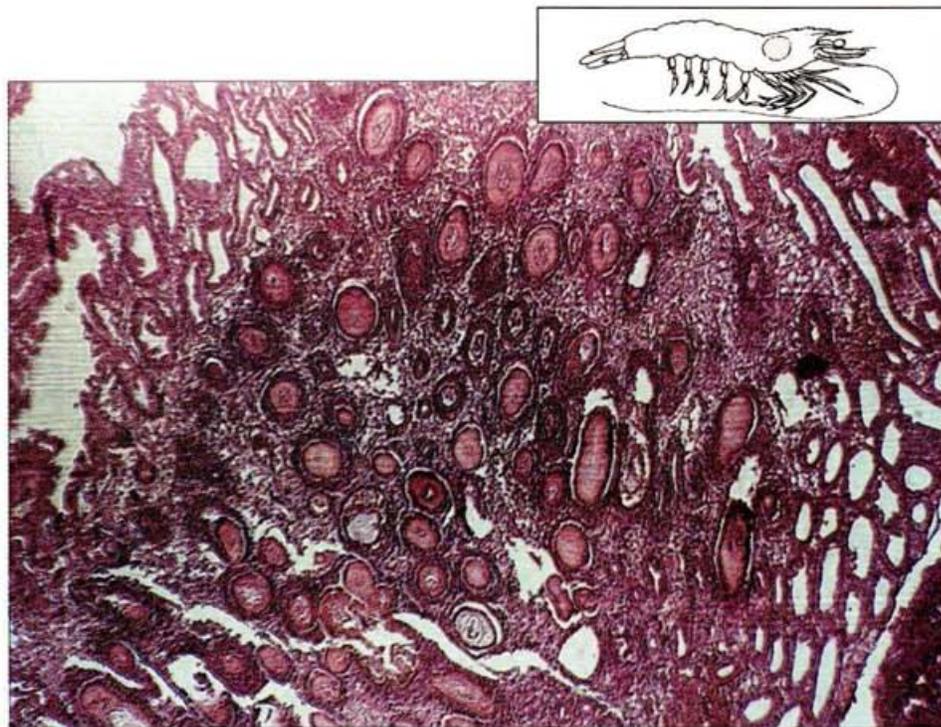


Figure 18b. Histological section of the hepatopancreas of a pond-reared juvenile with advanced red disease. Note the melanized tubules, and fibrosis and hemocyte infiltration in the intertubular spaces. Affected tubules are non-functional at this state. (H & E, 40X).

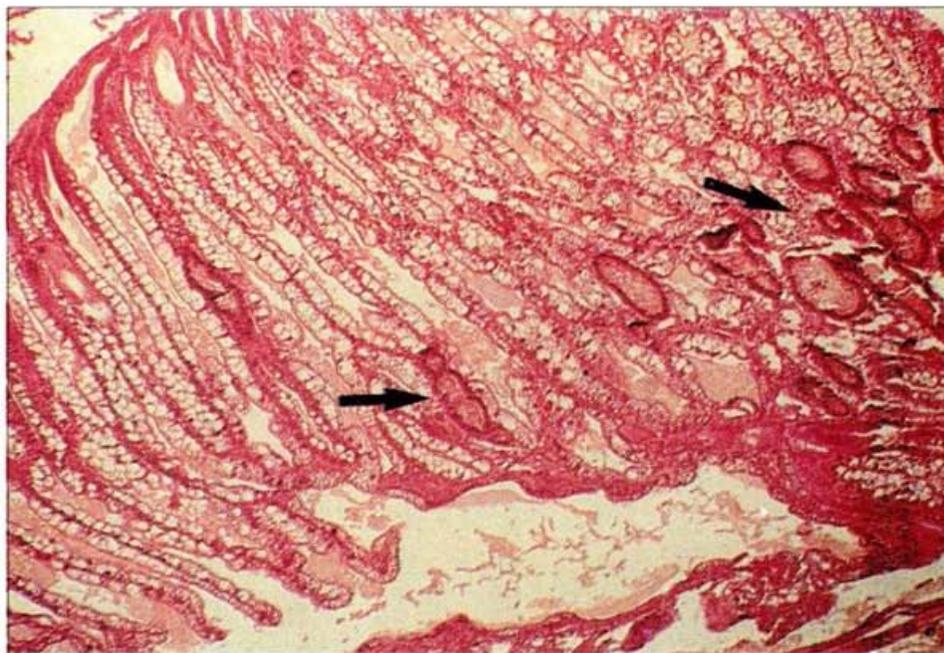


Figure 18c. Hepatopancreas of shrimp experimentally exposed to 200 ppb aflatoxin B₁ for 60 days via feed. Note the presence of melanized and necrotic tubules similar to red disease in the proximal tubules of the hepatopancreas (arrows). (H & E, 40X).

Common name: **UNDERFEEDING**

Causative agent: Lack of food

Species affected: *Penaeus monodon*, may also affect other species of crustaceans

Stages affected: Juveniles to adults

Gross signs:

- Loose shells, reduced muscular mass in the abdominal segments
- Histological sections show the marked absence of storage vacuoles, which are abundant in the hepatopancreas of normal shrimp (Figure 19a), in the hepatopancreas of affected shrimp resulting in atrophy of cells (Figure 19b)

Effects on host: Slow growth or weight loss in severely starved shrimps

Preventive methods:

- Provide supplemental feeds
- Exclude or eliminate competitors from the ponds
- For extensive ponds, apply fertilizer to induce a healthy bloom of algae and trigger a good growth of zooplankton and natural food

Treatment: Same as above

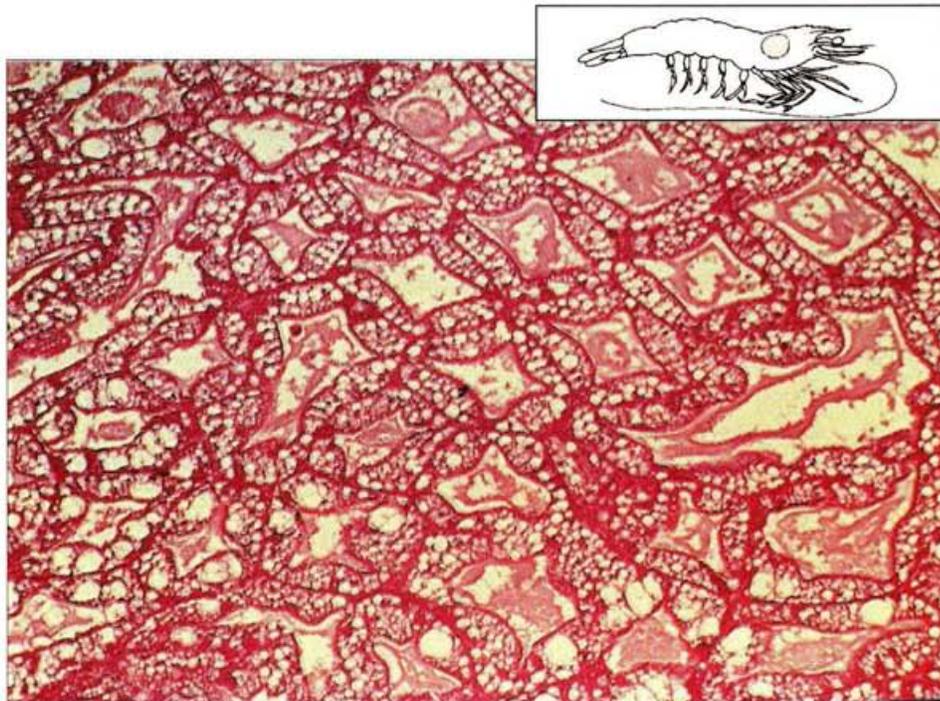


Figure 19a. Normal structure of the hepatopancreas of juvenile shrimp showing well vacuolated cells lining the tubules. (H & E, 100X).

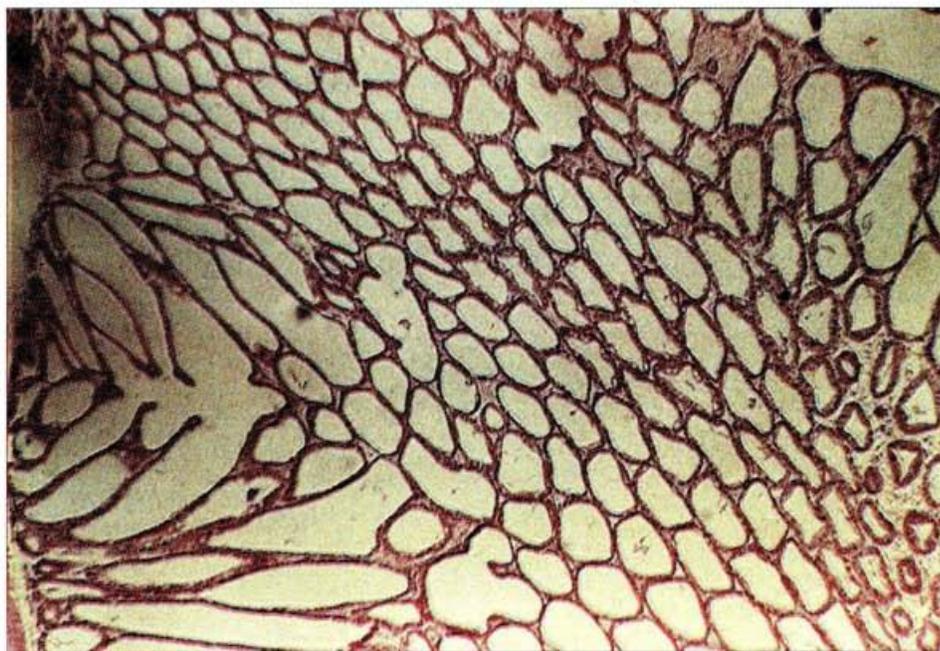


Figure 19b. Hepatopancreas of underfed shrimp juvenile showing severely atrophied tubular cells due to the absence of storage vacuoles. (H & E, 40X).

Common name: **MUSCLE NECROSIS**

Causative agent: Stressful environment conditions like low oxygen levels, temperature or salinity shock, overcrowding, and severe gill fouling

Species affected: *Penaeus monodon*

Stages affected: Postlarvae, juveniles, adults

Gross signs:

- Opaque white areas on the abdomen (Figure 20a)
- Blackening on edges of the uropod followed by erosion
- Liquid-filled boils at the tip of uropods in advanced stages
- Weakness and, eventually, death
- In postlarvae appearance in distinct lines in the abdominal muscles giving it a “wood grain” appearance (Figure 20b)

Effects on host:

- The disease causes gradual death of cells of affected parts such as uropods and musculature leading to erosion especially in the tail portion
- Affected areas may serve as portals of entry for a secondary systematic infection by bacteria

Preventive methods:

- Reduce stocking density in ponds
- Give adequate feed but do not overfeed
- Maintain good water quality avoiding extreme fluctuations in salinity, temperature and dissolved oxygen

Treatment: None reported

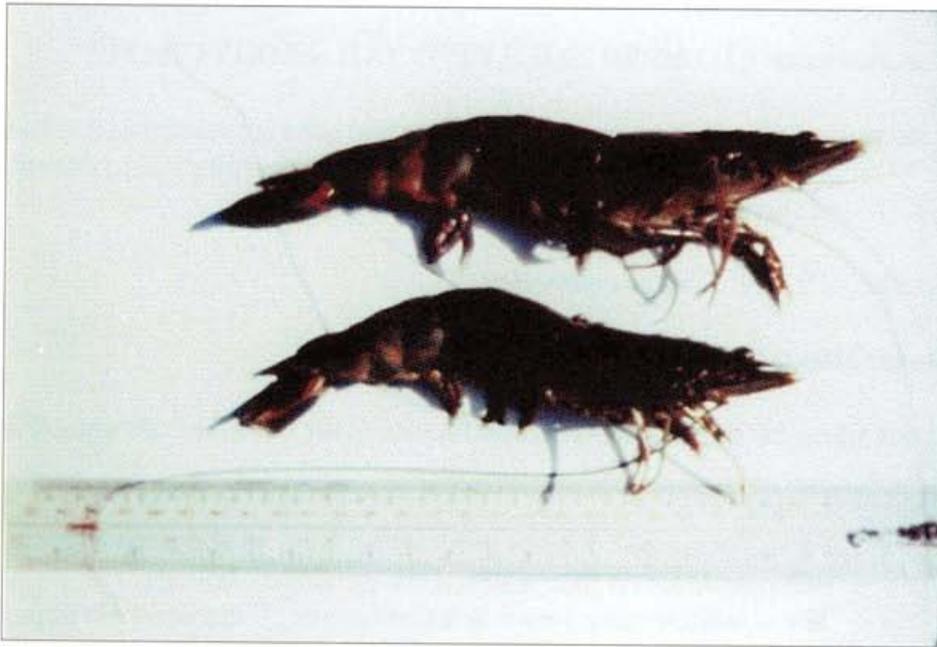


Figure 20a. *Penaeus monodon* juveniles with necrosis in the abdominal muscles.

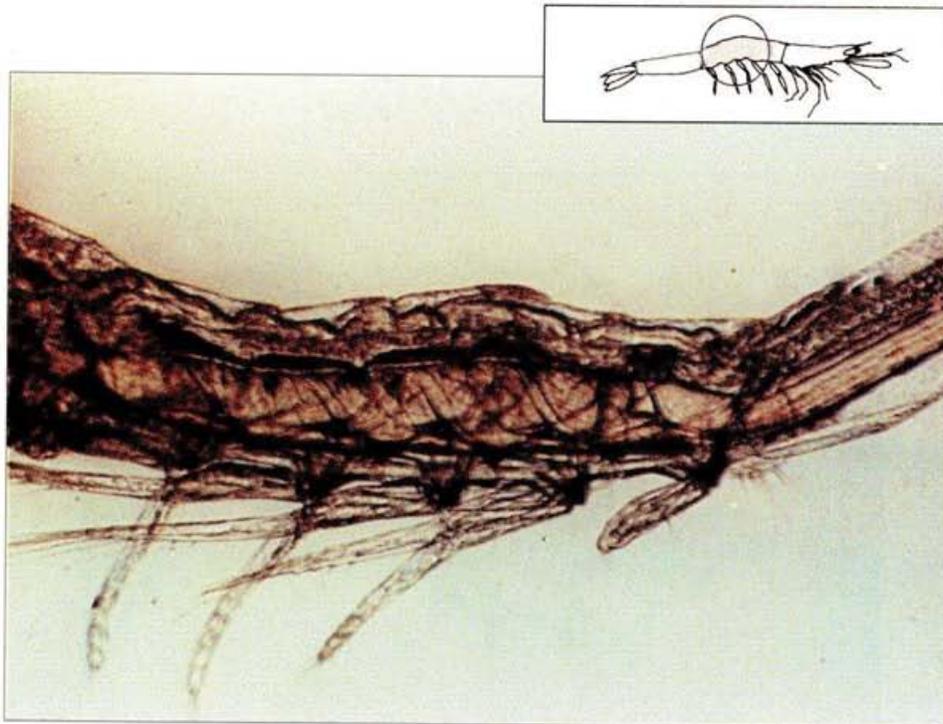


Figure 20b. *Penaeus monodon* postlarva with "grainy" abdominal muscles.

Common name: **CRAMPED TAILS, BENT TAILS, BODY CRAMP**

Causative agents: Unknown, but the disease had been associated with mineral imbalance, and to abrupt change in water and air temperatures, e.g., during handling of shrimps in air warmer than the culture water

Species affected: *Penaeus monodon*

Stages affected: Juveniles, adults

Gross signs: Partial or complete rigid flexure of the tail in live shrimps (Figure 21)

Effects on host:

- Partially cramped shrimps swim with a humped abdomen whereas fully cramped individuals lie on their sides at the pond/tank bottom
- The condition may result in cannibalism of cramped shrimps by unaffected ones, and death
- Injured muscle fibers may turn brown as an outcome of the wound repair process

Preventive methods: Avoid possible causes

Treatment: None reported



Figure 21. *Penaeus monodon* juvenile with body cramp.

Common name: **ACID SULFATE SOIL SYNDROME**

Causative agent: Low water and soil pH

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stage affected: Juveniles

Gross signs:

- Poor growth
- Decreased molting frequency
- Yellow to orange to brown discoloration of the gill and appendage surfaces
- Reddish to orange color of the pond soil (Figure 22)

Effects on host:

- Normal metabolism of the shrimp is hindered resulting in retarded growth and eventual death

Preventive methods:

- Correct low pH soil condition by liming and flushing of pond bottom before stocking.
- Regularly monitor water pH
- Broadcast lime on pond dike surfaces or hang lime bag as the need arises

Treatment: Correct low pH conditions as above



Figure 22. Pond with acidic soil (left).

Common name: **ASPHYXIATION, HYPOXIA**

Causative agent: Very low levels of dissolved oxygen (DO)

Species affected: *Penaeus monodon*, *P. merguensis*, *P. indicus*

Stages affected: Larvae, postlarvae, juveniles, adults

Gross signs:

- Surface swimming
- Sudden mass mortalities

Effects on host:

- Prolonged respiratory distress leads to death
- Below optimum oxygen levels may cause impairment of metabolism resulting in growth retardation

Preventive methods:

- Monitor DO levels late in the afternoon and early in the morning
- Provide aeration facilities and water pump for ready water change
- Decrease stocking density if aeration and water-change facilities are not available
- Control feeding of artificial diets according to consumption rates to prevent accumulation of high organic matter

Common name: **BAMBOO BACK SYNDROME**

Causative agents: Unknown, but may be due to nutritional deficiency

Species affected: *Penaeus monodon*

Stages affected: Juveniles, adults

Gross sign:

- Succeeding exoskeletal plates of the abdominal segments do not overlap properly allowing the muscles to protrude giving the shrimp a bamboo-like appearance
- Shorter rostrum and tail region

Effect on host:

- Protruding muscles become prone to injury and secondary infection

Preventive methods: None reported

Treatment: None reported

Appendices

Glossary

References

Acknowledgement

Appendices

Appendix I. Procedure for disinfection of rearing facilities.

Hatchery

1. Disinfect used and infected rearing water with 220 ppm available chlorine. Soak all used hatchery paraphernalia overnight in this tank.
2. Drain disinfectant. Scrub tank bottom and sidewalls with freshly prepared disinfectant of the same concentration.
3. Rinse thoroughly with clean freshwater several times.
4. Allow to dry under the sun and let stand for several days.
5. Wooden materials used when infection occurred should be burned and replaced for the next operation.

Ponds

1. Used pails and other pond paraphernalia should be soaked overnight in 220 ppm available chlorine. Wash thoroughly with clean water before using. Dry under the sun.
2. Infected ponds may be disinfected immediately after harvest by applying lime while the soil is still wet. The soil pH should rise to 9.
3. Infected ponds should be dried thoroughly (until the soil cracks) before using these for the next operation.
4. Tilling should be done to expose the sub-surface layer.

Appendix II. Procedure for disinfecting rearing water using calcium hypochlorite (70% chlorine activity).

1. Using Table I, dissolve the required amount of powder for a desired volume of water in a small volume of water (500 ml). For example, if the water volume is 0.5 ton or 500 liters and the desired concentration is 15 ppm, the amount of calcium hypochlorite needed is 10.7 g.

Table 1. Guide for determining the amount of calcium hypochlorite (g) to be used for water disinfection.

Volume of water	Chlorine concentration			
	5 ppm	10 ppm	15 ppm	20 ppm
0.25 ton (250 liters)	1.8	3.6	5.4	7.2
0.50 ton (500 liters)	3.6	7.1	10.7	14.3
1.0 ton (1,000 liters)	7.1	14.3	21.4	28.6
2.0 tons (2,000 liters)	14.3	28.6	42.9	57.1
3.0 tons (3,000 liters)	21.4	42.9	64.3	85.7
5.0 tons (5,000 liters)	35.7	71.4	107.1	142.9
10.0 tons (10,000 liters)	71.4	142.9	214.3	285.7

The amount of calcium hypochlorite may be multiplied by different factors to obtain other chlorine concentrations. Ex.: To obtain 400 ppm chlorine solution in 1 ton water, multiply 28.6 g by 20 or 14.3 by 40.

2. Fill the tank with the desired volume of water, then add the dissolved calcium hypochlorite solution.
3. Keep chlorinated water for at least 12 hours, up to 24 hours, then check the residual chlorine level using portable kits available in the market. Neutralize remaining chlorine with equal amount for sodium thiosulfate ($\text{Na}_2\text{S}_2\text{O}_3$) before using the water.

Table 2. Guide for determining the amount of bleach (ml) for water disinfection.

Volume of water	Chlorine concentration			
	5 ppm	10 ppm	15 ppm	20 ppm
0.25 ton	25	50	75	100
0.50 ton	50	100	150	200
1.0 ton	100	200	300	400
2.0 tons	200	400	600	8,000
3.0 tons	300	600	900	1,200
5.0 tons	500	1,000	1,500	2,000
10.0 tons	1,000	2,000	3,000	4,000

Appendix III. Guidelines for discarding infected larval stocks.

Disease-infected larval stock should not be drained back into the sea without prior disinfection. Failure to disinfect would introduce large numbers of disease-causing organisms in the nearshore environment. The following steps are recommended:

1. Calculate the total volume of the contaminated rearing water.
2. Weigh the amount of calcium hypochlorite needed to disinfect the contaminated water volume so that the resulting active chlorine concentration would be equivalent to 200 ppm.
3. Allow the disinfectant to act for at least 1 hour.
4. Drain. Discarded water/dirt/larvae from the hatchery should not be thrown directly into the sea. The dumping area should be located in the sandy portion several meters above the high-tide waterline.

Appendix IV. Guidelines for treatment of shrimp diseases.

1. Clean rearing facilities before treatment. This may be accomplished by siphoning out sediments from the tank bottom and by water change. Organic matter present in dirty tanks could absorb part of the drug being used thus reducing its effectiveness.
2. Apply treatment only during the coolest part of the day (i.e., nighttime). The drug used should provide the least environmental hazard or stress.
3. Monitor dissolved oxygen levels before and during treatment since stressed shrimps need more oxygen. Provide additional aeration if necessary.
4. Always make sure that your computations are correct by having someone else check figures if possible. Unexpected mortalities due to drug overdose may happen.
5. Follow recommended protocol strictly. Regular use of drugs at levels lower than recommended could result in the development of resistant strains of bacteria. Continued use of the drug at the recommended levels but beyond the prescribed period of exposure could result in physical deformities among treated shrimps.
6. Keep records of all treatments, their purpose, and results for future reference.

Appendix V. Chemical prophylaxis against larval mycosis.**Eggs**

1. Prepare 20 ppm laundry detergent.

Total volume of water (liters)	Weight of detergent (gram)
1	0.02
5	0.01
10	0.2
100	2.0
500	10.0
1000	20.0

2. Dissolved the detergent in a small amount of freshwater, add to the egg culture tank, and mix gently.
3. Let stand and aerate for 2 hours.
4. Transfer eggs to an egg washer and rinse eggs thoroughly using flow-through seawater to remove detergent.
5. Chemical prophylaxis should be done long before hatching. Do not let eggs hatch in detergent solution.

Spawners

1. Prepare 5 ppm Treflan.

Total volume of water (liters)	Volume of Treflan (milliliters)
100	0.5
250	1.25
500	2.1
1000	5.0

2. Mix the chemical in a small amount of freshwater, add to the spawning tank, and aerate for 1 h.
3. Cover tank with a black cloth to prevent photodegradation of Treflan.
4. Transfer spawner to another tank with fresh seawater and rinse thoroughly with flow-through seawater to remove the chemical.

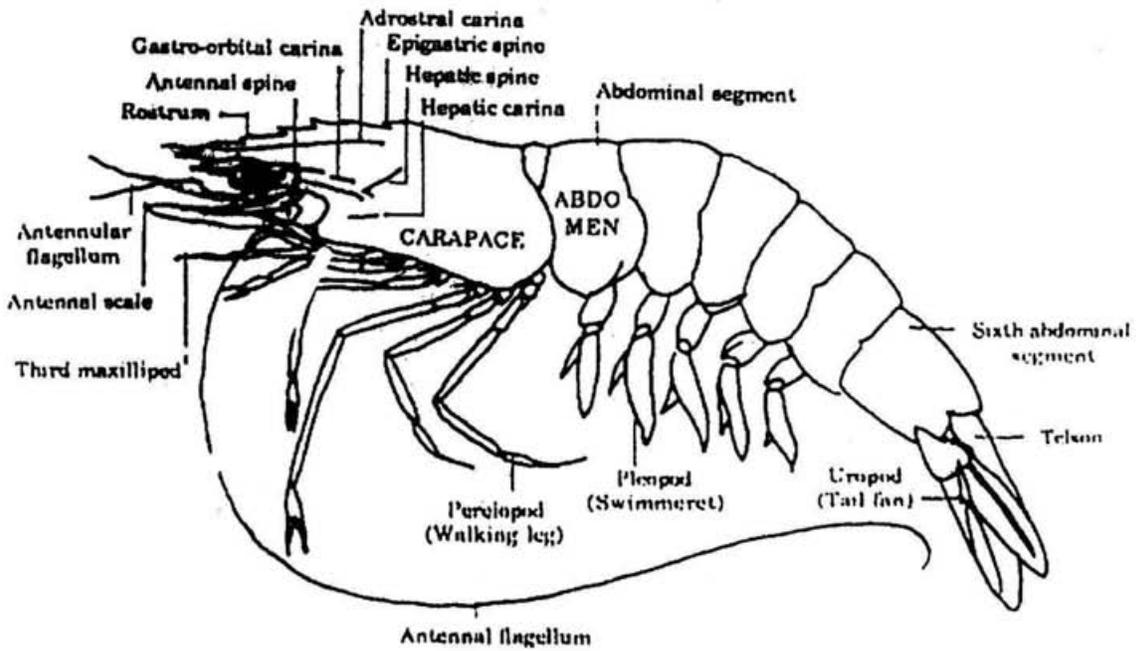
Appendix VI. Treatment of larval mycosis with Treflan using drip method.

1. Prepare 0.2 ppm Treflan. Mix the chemical in a small amount of freshwater (for 1 ton culture water, use 1 liter freshwater) and add to the culture water tank. Mix thoroughly.

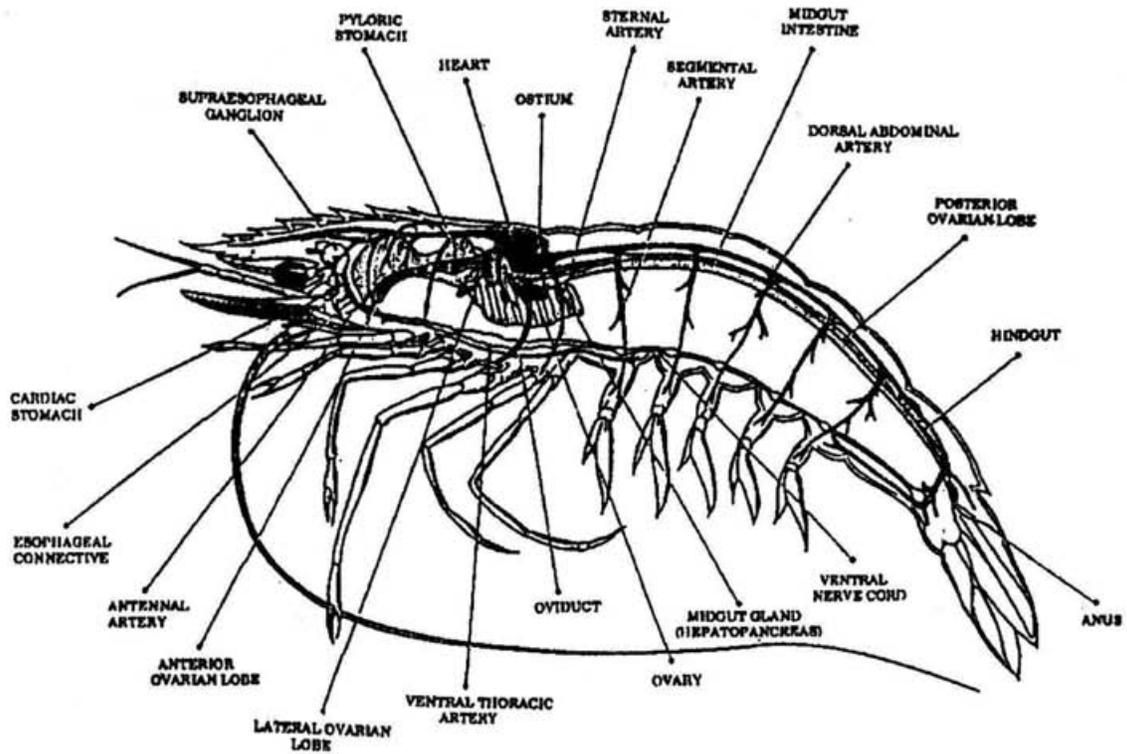
Total volume of water (ton)	Volume of Treflan (ml)
0.5	0.1
1.0	0.2
2.0	0.4
3.0	0.6
4.0	0.8
5.0	1.0

2. After 1 h, prepare a second set of Treflan solution of equal concentration as in #1 and place in dextrose bottles. Hang the bottles inverted so that the solution would drip into the culture water through the plastic hose.
3. Adjust the drip regulator to release 1/4 to 1/3 of the amount of the solution into the water per hour. Administer contents of the bottle within 3-4 h. One ml is equivalent to 15-20 drops, so the flow rate from the bottle would be approximately 60-100 drops per minute.
4. Cover tanks with a black cloth during treatment especially at daytime to prevent photodegradation of Treflan. Treat only during cool times of the day.

Appendix VII. External and internal anatomy of penaeid shrimps. (after Motoh, 1981)



External



Internal

Appendix VIII. Guidelines for sending specimens for disease diagnosis

1. In the absence of facilities or personnel capable of diagnosing shrimp diseases, samples for diagnosis may be sent to a disease diagnostic laboratory (Fish Health Section of the Southeast Asian Fisheries Development Center, Aquaculture Department, Tigbauan, Iloilo). If the laboratory is located nearby, live samples may be sent by packing these in clean, aerated culture water in plastic bags. Diseased shrimps must always be separated from normal ones and stocking density during transport must be reduced by at least 25%. For larval and post-larval stages, at least 20 diseased individuals and an equivalent number of normal shrimps are needed to make a diagnosis. All types of examination and diagnostic procedures may be done on live samples.
2. If the diagnostic laboratory is quite far from the hatchery/farm and there are no facilities for immediate and fast transport, fixed or iced samples may be sent. Specimens are fixed by injecting Davidson's* fixative directly into the tissues of LIVE specimens. Inject an equivalent of 5-10% of the shrimps's body weight until all signs of life should cease and the shrimps appear cooked (orange color). Fixed specimens should be immersed in fixative at a ratio of 1 part shrimp tissue to 9 parts fixative. *If Davidson's fixative is not available, 10% formalin with 1.0% salt may be used.*
3. The same number of specimens are sent as for live samples. Diseased animals must be separated from normal ones. Only direct microscopic examination (for parasites and fungi) and hispathological examination may be done on fixed samples.
4. Iced samples are sent by packing adult/juvenile shrimp in plastic bags (separate diseases from normal) and placing these in between layers of ice in a styrofoam box. Like fixed samples, very limited diagnostic procedures may be done on iced samples.
5. All pertinent data/information must be sent with the samples.
6. Notify the laboratory of the delivery and arrival of samples

**Davidson's Fixative (1 liter)*

95% Ethyl alcohol	330ml
Formalin (saturated formaldehyde solution)	220ml
Glacial acetic acid	115ml
Distilled water	335ml

7. Aside from the Fish Health Section at SEAFDEC, samples may also be sent to:
 - (a) Fish Health Section
Bureau of Fisheries and Aquatic Resources (BFAR)
Arcadia Bldg., Quezon Ave
Quezon City
 - (b) Regional Offices of BFAR throughout the Philippines
 - (c) Negros Prawn Producers Marketing Cooperative, Inc.
JTL Bldg., B.S. Aquino Drive
Bacolod City

Appendix IX. Water quality suitable for rearing penaeid shrimp (Chen, 1985 *in* Licop, 1988)

Hatchery

Parameter	Range
Temperature	24°-31°C
pH	7.5-8.5
Dissolved oxygen (D.O.)	>5 ppm
Salinity	28-33 ppt
Turbidity	<50 FTU
Hg	<0.01 ppb
Heavy metals	<0.01 ppm
Biological oxygen demand (B.O.D.)	<1.0 ppm (5 days)
Unionized ammonia (NH ₃)	<0.1 ppm
Nitrate (NO ₂ -N)	<0.02 ppm

Grow-out ponds

Parameters	Range
Temperature	28°-33°C
pH	8.0-8.5
D.O. (critical)	3.7 ppm
Salinity	15-25 ppt
Heavy metals	
Hg	0.0025 ppm
Cu	0.1 ppm
Cd	0.15 ppm
Zn	0.25 ppm
Hydrogen sulphide (H ₂ S)	0.33 ppm
NH ₃	0.1 ppm

Glossary

Acute	a condition or disease process with a rapid trend
Aflatoxin B1	a highly toxic substance produced by the fungus <i>Aspergillus flavus</i> which induces tumors in some animals
Antennal gland	the organ found in the base of the antennae of shrimps which has an excretory function
Astaxanthin	a violet crystalline carotenoid pigment found in the shell of crustaceans
Atrophy	reduction in the size of tissue or organ
Bacteria	one-celled microorganisms which lack well-defined nucleus. There are three shapes of bacteria: rods, round, and spiral
Basophilic	cell structures that are easily stained with basic stains like hematoxylin
Benthic	related to the bottom of the pond or other aquatic environment
Bioaugmentation	the addition of benign bacteria into the rearing water to promote ecological balance within the system and to digest accumulated organic waste materials
Broodstock	adult animals maintained as a source of eggs and sperms to establish a new population
Chemotherapeutant	a drug or chemical used in the treatment of disease
Chitin	the complex carbohydrate that makes up the exoskeleton of shrimps
Chitinoclastic	that which is capable of digesting chitin
Ciliate	protozoan possessing hair-like structures for movement known as cilia
Clinical signs	signs based on direct observation
Cuticle	the non-living outer layer of skin

Cyanotic	possessing bluish skin due to presence of large quantities of deoxygenated blood in the minute vessel
Diatoms	any of the one-celled or colonial algae having a cell wall with silicon components
Discharge tube	an exit tube formed in the asexual reproductive body of a fungus that penetrates through the host cell wall to the outside; zoospores may be directly released through this tube or maybe formed in a vesicle at the tip of this tube
Distal	anatomically located far from the point of reference
Ectoderm	the outermost layer of the three primary germ layers of the embryo
Endoparasitic	parasites living inside the body of the host
Eosinophilic	cell structures that are readily stained by eosin
Epicommensal	a microorganism that lives on the external surfaces of another organism and derives benefits from it without causing any harm
Epizootic	a disease affecting many animals of one kind at the same time
Fibrosis	excessive proliferation of fiber cells
Fouling	the presence of organisms or organic material on the surface of an animal
Fungus	a general term for a group eukaryotic protists (e.g., mushrooms, yeasts, molds, etc.) characterized by the absence of chlorophyll and the presence of a rigid cell wall
Green water culture system	an innovative system by which shrimp is cultured with water where algae grow abundantly. This green water comes from a pond where <i>Tilapia</i> is grown.
Hematopoietic organ	the blood-forming structure in crustaceans
Hemocyte	a blood cell of shrimp and other arthropods
Hemolymph	the equivalent of blood in crustaceans

Hepatopancreas	the digestive organ of shrimp which also functions in absorption and storage of food
Histology	the study of tissue structure at a microscopic level
Hypha	a tubular filament which is the unit structure of fungi
Hypertrophied	overly developed cell structure or tissue
Inclusion body	any body of material within a cell
Incubation time	elapsed time between exposure to infection and the appearance of disease symptoms
Inflammation/ inflammatory response	is a non-specific response to injury which involves the accumulation of blood cells and the deposition of the pigment melanin
Intermediate host	host in which the larval stages of the parasite develop
Latently infected	infected animal that does not manifest the disease signs but has the potential of transferring the disease agent to other stages of the same or another species
Lethargy	weakness or sluggishness
Luminous/ luminescent	living organisms which give off cold light
Melanin	a dark brown or black pigment produced in response to injury or infection
Mesoderm	the middle layer of the three primary germ layers of an embryo
Microsporidia	a type of protozoa capable of producing resistant spores
Mollusc	an invertebrate animal with a soft body usually covered with a hard shell
Molting	the process of shedding the exoskeleton or skin
Mycosis	any disease caused by fungi
Mysis	a larval stage of higher crustaceans having all the thoracic appendages developed

Nanomicron	one billionth of a micron
Necrosis	the state wherein the cells and the tissues lower their activity and eventually die
Occlusion body	a specialized form of inclusion which contains virus particles
Pasteurize	partially sterilize by heating below the boiling point
Pathogenic	disease-causing
Peptidoglycan	a substance composed of two sugar derivatives found in the rigid layer of the cell wall of both Gram-negative and Gram-positive bacteria
Phytoplankton	minute free-floating aquatic plants
Piscicide	substance that selectively kill fish
Postlarva	the molt stage after mysis where the immature shrimp begins to assume the appearance of a miniature adult
Prevalence	the proportion of diseased animals in population at a specified point in time
Probiotics	benign bacteria which are added into feeds or encapsulated into natural food to stimulate the population of 'friendly bacteria' and prevent the colonization of pathogenic microorganisms in the gut
Protozoa	a single-celled organism with a nucleus
Quarantine	restrictions placed on animals entering or leaving premises; detention on account of suspected disease agents
Secondary pathogen	a disease-causing organism which causes infection only after host has been weakened by other causes such as entry of another pathogens
Spawner	a mature female shrimp that produces eggs in large numbers
Stress	the sum of the biological reactions to any adverse stimulus that tends to disturb an organism's physiological stability
Suctoria	a class of protozoa having cilia only early in development and in which the mature form is fixed to the substrate

Syndrome	a pattern or group of signs which may be associated with more than one disease organism or process
Systemic	affecting the whole animal
Toxin	a poison
Vacuole	a tiny cavity in the cell of animals
Vesicle	a thin, bubble-like structure in which zoospores are formed
Virus	a minute infectious agent which can only be resolved in high-powered microscopes. It lacks independent metabolism and is able to replicate only within a living cell
Zoea	an early larval form of shrimps
Zooplankton	microscopic animals that freely drift in natural bodies of water
Zoospore	motile spores produced by means of asexual reproduction

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About SEAFDEC

The Southeast Asian Fisheries Development Center (SEAFDEC) is a regional treaty organization established in December 1967 to promote fisheries development in the region. Its Member Countries are Japan, Malaysia, the Philippines, Singapore, Thailand, Brunei Darussalam, the Socialist Republic of Viet Nam, and Myanmar.

Representing the Member Countries is the Council of Directors, the policy-making body of SEAFDEC. The chief administrator of SEAFDEC is the Secretary-General whose office the Secretariat, is based in Bangkok, Thailand.

Created to develop fishery potentials in the region in response to the global food crises, SEAFDEC undertakes research on appropriate fishery technologies, trains fisheries and aquaculture technicians, and disseminates fisheries and aquaculture information. Four departments were established to pursue the objectives of SEAFDEC.

- The Training Department (TD) in Samut Prakan, Thailand, established in 1967 for marine capture fisheries training
- The Marine Fisheries Research Department (MFRD) in Singapore, established in 1967 for fishery post-harvest technology
- The Aquaculture Department (AQD) in Tigbauan, Iloilo, Philippines, established in July 1973 for aquaculture research and development
- The Marine Fishery Resources Development and Management Department (MFRDMD) in Kuala Terengganu, Malaysia, established in 1992 for the development and management of the marine fishery resources in the exclusive economic zones (EEZs) of SEAFDEC Member-Countries.

