



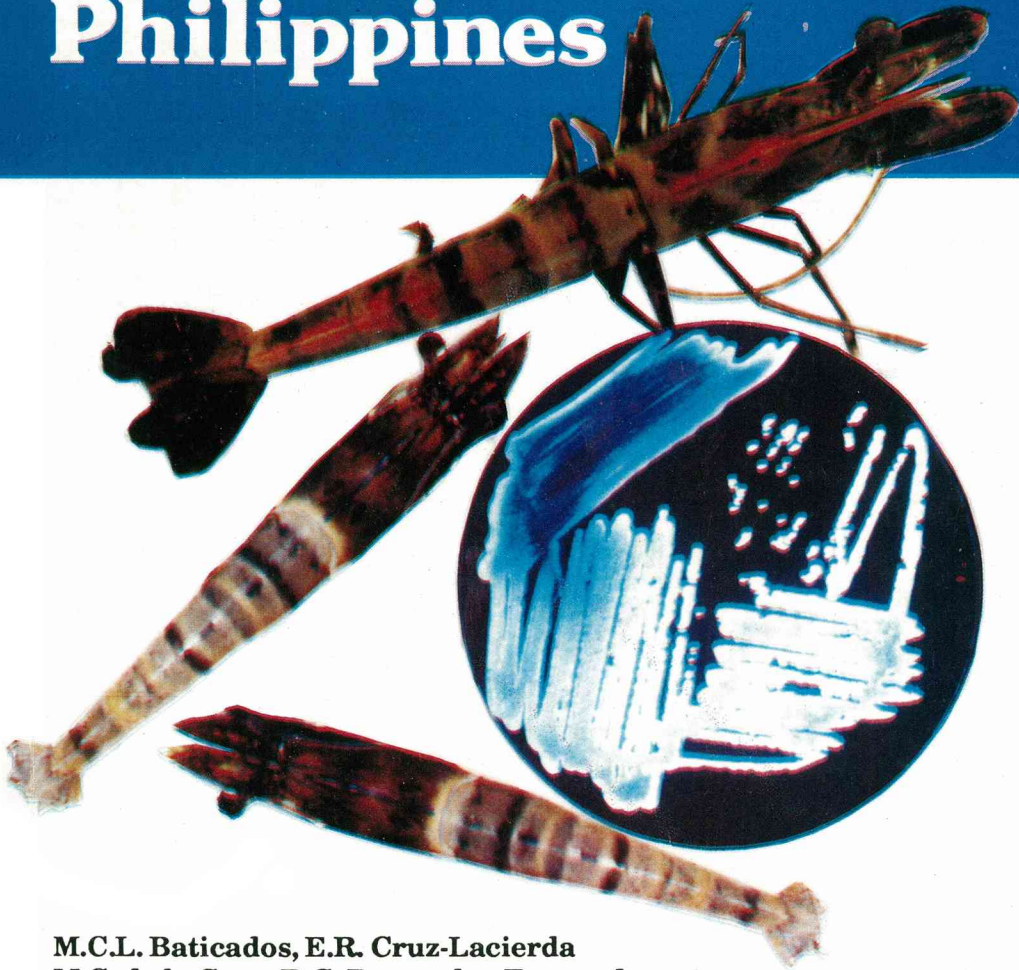
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Diseases of Penaeid Shrimps in the Philippines



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AQUACULTURE DEPARTMENT
SOUTHEAST ASIAN FISHERIES DEVELOPMENT CENTER

AQUACULTURE EXTENSION MANUAL NO. 16
MAY 1990
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DISEASES OF PENAEID SHRIMPS IN THE PHILIPPINES

by

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IN THE PHILIPPINES**
Aquaculture Extension Manual No. 16

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Cover: A shrimp with red disease (top), two
shrimps afflicted with tail rot, and luminescent
bacteria, *Vibrio harveyi*, on agar medium.

Design by I. T. Tendencia

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INTRODUCTION

The high market demand for shrimps has triggered the intensification of culture systems in the Philippines. As a consequence, shrimps are now a major export commodity of the country. The culture of shrimps at very high stocking densities, however, has brought about a proliferation of diseases resulting in growth retardation, physical deformation, reduced fecundity, physiological malfunction, and mortality of stock. Moreover, diseased shrimps command a lower price in the market. Diseases of penaeid shrimps may be caused by living agents like viruses, bacteria, fungi, and parasites, as well as by non-living factors such as nutritional deficiencies, toxic substances and environmental problems.

To increase the production of good quality shrimps, there is a need to counteract one of the primary causes of production losses - disease. The shrimp farmer must be familiar with potential shrimp disease problems that will most likely occur. This manual aims to meet this need by providing information on the diseases that affect 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. 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. Also, many of the drugs available in the market are used for treatment of human diseases and would thus pose a danger to public health.

ACKNOWLEDGMENT

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VIRAL DISEASES

- *Monodon* Baculovirus (MBV) Disease
- Infectious Hypodermal and Hematopoietic Necrosis Virus (IHHNV) Disease
- Hepatopancreatic Parvo-like Virus (HPV) Disease

4 Diseases of Penaeid Shrimps

Common name : **MONODON BACULOVIRUS (MBV) DISEASE** **Fig. 1**

Causative agent : *P. monodon*-type Baculovirus

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

Stages affected : 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.
- Yellowish-white hepatopancreas.

Effects on host:

- Presence of the virus causes destruction of the hepatopancreas and lining of the digestive tract. Damage in these organs could weaken the shrimp and lead to gradual mortalities.

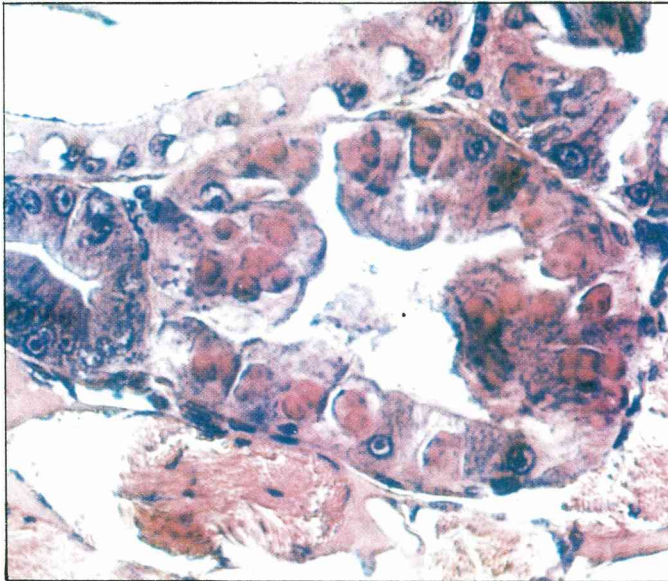


Fig. 1. Monodon baculovirus (MBV) occlusion bodies in hypertrophied nuclei of *P. monodon* postlarva.

- Accumulated mortality of 70% was observed among *P. monodon* juveniles cultured in raceways and tanks.
- Spherical, eosinophilic occlusion bodies of the virus fill up enlarged nuclei of hepatopancreatic cells or may be found in the lumen after cells have been destroyed.

Preventive methods:

- Use MBV-free stocks.
- Reduce stress by use of good husbandry practices and proper nutrition.
- Destroy infected shrimps by burning or burying in pits lined with lime.
- Disinfect rearing facilities (Appendix I).

Treatment:

- None reported.

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

Causative agent : Unclassified virus probably belonging to Picornavirus

Species affected : *P. 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.
- The motion of the pleopods (Appendix III) and pereopods ceases and the animal sinks to the bottom.
- Shrimps would eventually right themselves up, 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.

Effects on host:

- Larval stages (zoea and mysis) of *P. monodon* are presumed to be latently infected.
- Presence of the virus can cause death of the cells of the cuticle, blood-forming tissues and connective tissues of the shrimp.
- Death of the cells in these tissues can cause abnormal metabolism which eventually leads to mortalities.

6 Diseases of Penaeid Shrimps

- Mortality rates of above 90% were observed among penaeid juveniles in intensive culture systems.

Preventive methods:

- Avoid introduction of IHNV-infected postlarvae, juvenile or adult shrimp into culture areas.
- If the disease agent is suspected among cultured shrimp stocks, destroy exposed shrimps and disinfect contaminated premises.
- Strictly adhere to quarantine practices for all live and newly acquired shrimps.

Treatment:

- None reported.

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

Causative agent : Parvovirus

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

Stages affected : 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 abdominal muscles.

Effects on hosts:

- The virus causes cell death and shrinkage of the hepatopancreas. Damage in this organ can cause abnormal metabolism and eventual death of the organism.
- 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.

BACTERIAL DISEASES

- Luminous Bacterial Disease
- Shell Disease, Brown/Black Spot, Black Rot/Erosion, Blisters, Necrosis of Appendages, Tail Rot
- Filamentous Bacterial Disease

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Common name : **LUMINOUS BACTERIAL DISEASE** Fig. 2a, b

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

Species affected : *P. 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.

Effects on host:

- Systemic infections result in mortalities in larvae and postlarvae, reaching up to nearly 100% of affected population.

Preventive methods:

- Prevent the entry of luminous bacteria into the hatchery system by using ultraviolet-irradiated water or by employing a series of filtration equipment (sandfilters, filter bags, cartridge filters, 0.45 micron pore-sized microfilter, etc.) and chlorination procedures (Appendix II).
- Adhere to strict sanitation procedures prior to and during the larval stages of growth.
- Use only previously chlorinated water during spawning and rearing to ensure a clean environment for newly hatched and developing larvae.
- 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 (Appendix IV) followed by a complete clean-up and disinfection of hatchery paraphernalia after every larval rearing period.

Treatment:

- Water change must be 80 to 90% replacement daily.

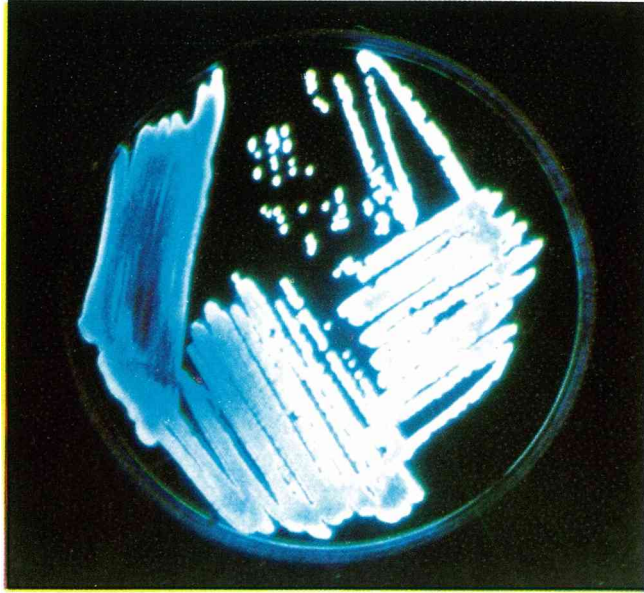


Fig. 2a. Seawater agar culture of the luminous bacterium *Vibrio harveyi*. Photo taken in total darkness.

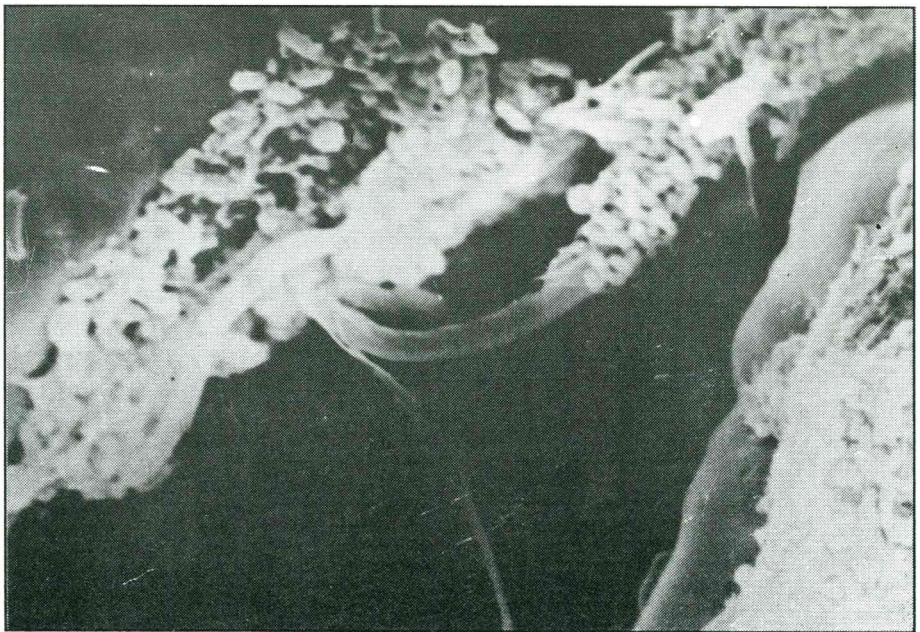


Fig. 2b. Bacterial plaques on the mouth parts of *P. monodon* (mysis I) exposed to *V. harveyi* for 48 h.

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Common name : SHELL DISEASE, BROWN/ BLACK SPOT, BLACK ROT/EROSION, BLISTERS, NECROSIS OF APPENDAGES **Fig. 3a-c**

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

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

Stages affected : Larvae, postlarvae, juveniles, adults

Gross signs:

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

Effects on host:

- Infection is usually initiated at sites of punctures or injuries made from either telson or rostrum, cracks on the abdominal segment from sudden flexure of the shrimp body, or from other damage caused by cannibalism.
- Progressive erosion of these exoskeletal lesions follows upon entry and multiplication of bacterial pathogens. The infection may lead to loss of the affected appendage(s) or of the exoskeleton and may reach the underlying musculature. When these occur, normal locomotion or molting is hampered and may result in shrimp losses.
- The affected shrimp becomes susceptible to cannibalism or dies from stress or energy exhaustion.

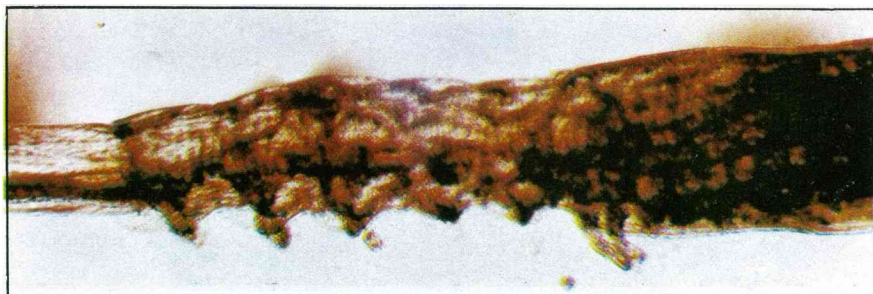


Fig. 3a. Loss of appendages of larval *P. monodon* due to progressive erosion of the pleopod cuticle.



Fig. 3b. Erosion of appendage as in Fig. 3a.

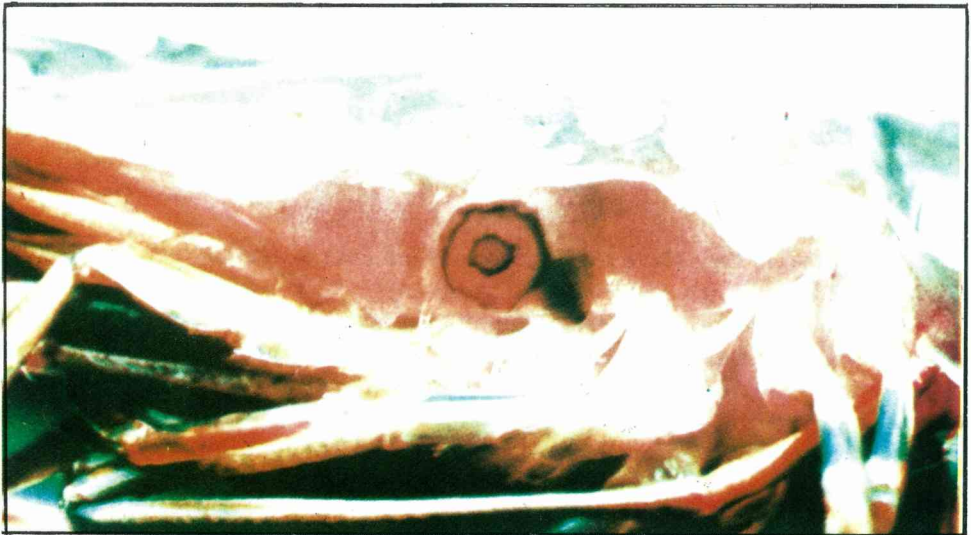


Fig. 3c. Shell erosion and black spot on the carapace of adult *P. monodon*.

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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.

Fig. 4

Common name : **FILAMENTOUS BACTERIAL DISEASE**

Causative agent : *Leucothrix* sp.

Species affected : *P. 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.

Effects on host:

- Infected eggs show a thick mat of filaments on the surface which may interfere with respiration or hatching.
- In larvae and postlarvae, filamentous growth on appendages and body surface may interfere with normal locomotory process 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 levels and low organic matter levels.



Fig. 4. Strands of the filamentous bacterium *Leucothrix* sp. on heavily infested gills of juvenile *P. monodon*. At upper left is the protozoan *Zoothamnium*.

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Treatment:

- Cutrine Plus at 0.15 ppm copper in 24-h flowthrough treatments or at 0.5 ppm copper in 4- to 6-h static treatments for postlarva 2 or older. For treatment guidelines, see Appendix V.

FUNGAL DISEASE

Common name : LARVAL MYCOSIS Fig. 5

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

Species affected : *P. monodon*

Stages affected : Eggs, larvae, early postlarvae

Gross signs:

- Infected eggs, larvae, and postlarvae appear whitish, become weak, and may eventually die.
- Signs are readily apparent when the 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 tanks with 100 ppm detergent (Tide*) for 24 h.

*Mention of brand names in this manual does not mean endorsement of the product.

16 Diseases of Penaeid Shrimps

- Observe rigid water management and sanitation.
- Disinfect eggs with detergent at 20 ppm for 2 h long before hatching. For spawners, use Treflan at 5 ppm for 1 h (Appendix VI).
- 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:

- Treflan or trifluralin at 0.2 ppm for 24 h (Appendix VII).

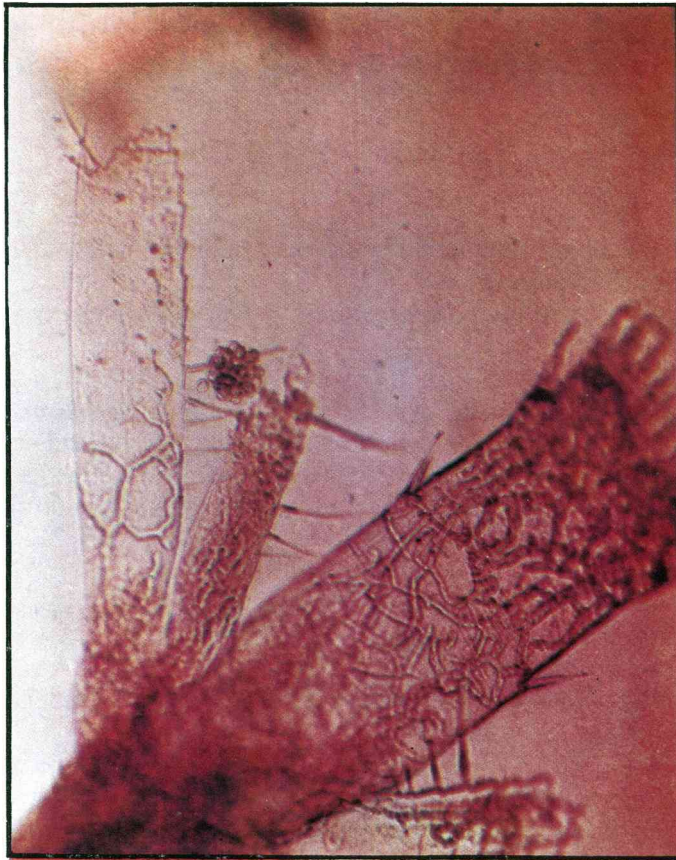


Fig. 5. Hyphae of *Lagenidium* in the tail of *P. monodon*. Zoospores in a vesicle (left) are about to be released by the fungus.

PROTOZOAN DISEASES

- Protozoan Infestation
- Microsporidiosis
- Gregarine Disease

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Common name : PROTOZOAN INFESTATION Fig. 6, 7

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.

Species affected : *P. 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.

Effects on host:

- Microscopically, protozoans may be observed attached to any external part of the shrimp.
- The protozoans cause locomotory and respiratory difficulties when present in large numbers on the appendages and gills, respectively, particularly at low dissolved oxygen levels.
- Loss of appetite.

Preventive methods:

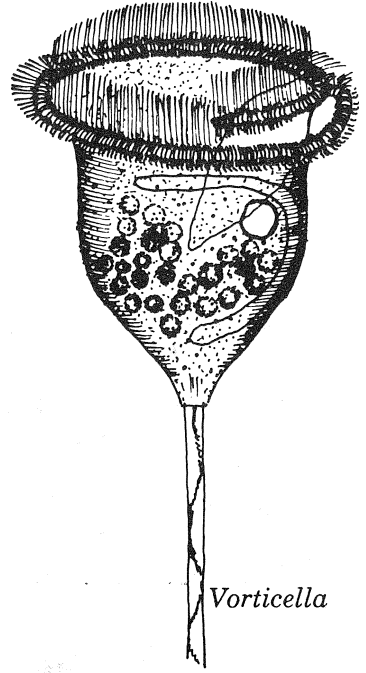
- Maintain good water quality.
- Avoid high organic load, heavy siltation, turbidity, and low oxygen levels.

Treatment:

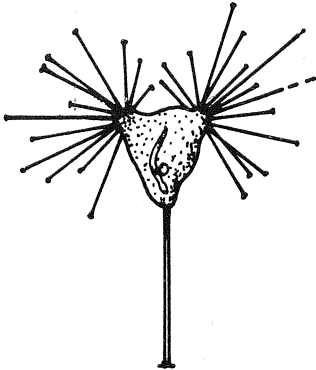
- Among juveniles in nursery tanks, application of chloroquin diphosphate at 1.1 ppm for 2 days was reported to be effective against the ciliates after three treatments.
- *Zoothamnium* infestation in adults was reported to be effectively treated with 50-100 ppm formalin for 30 min.
- *Epistylis* infestation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



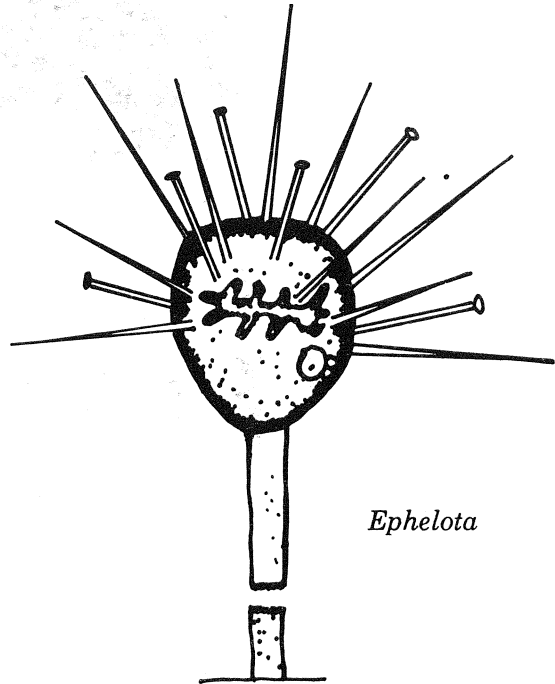
Epistylis



Vorticella



Acineta



Ephelota

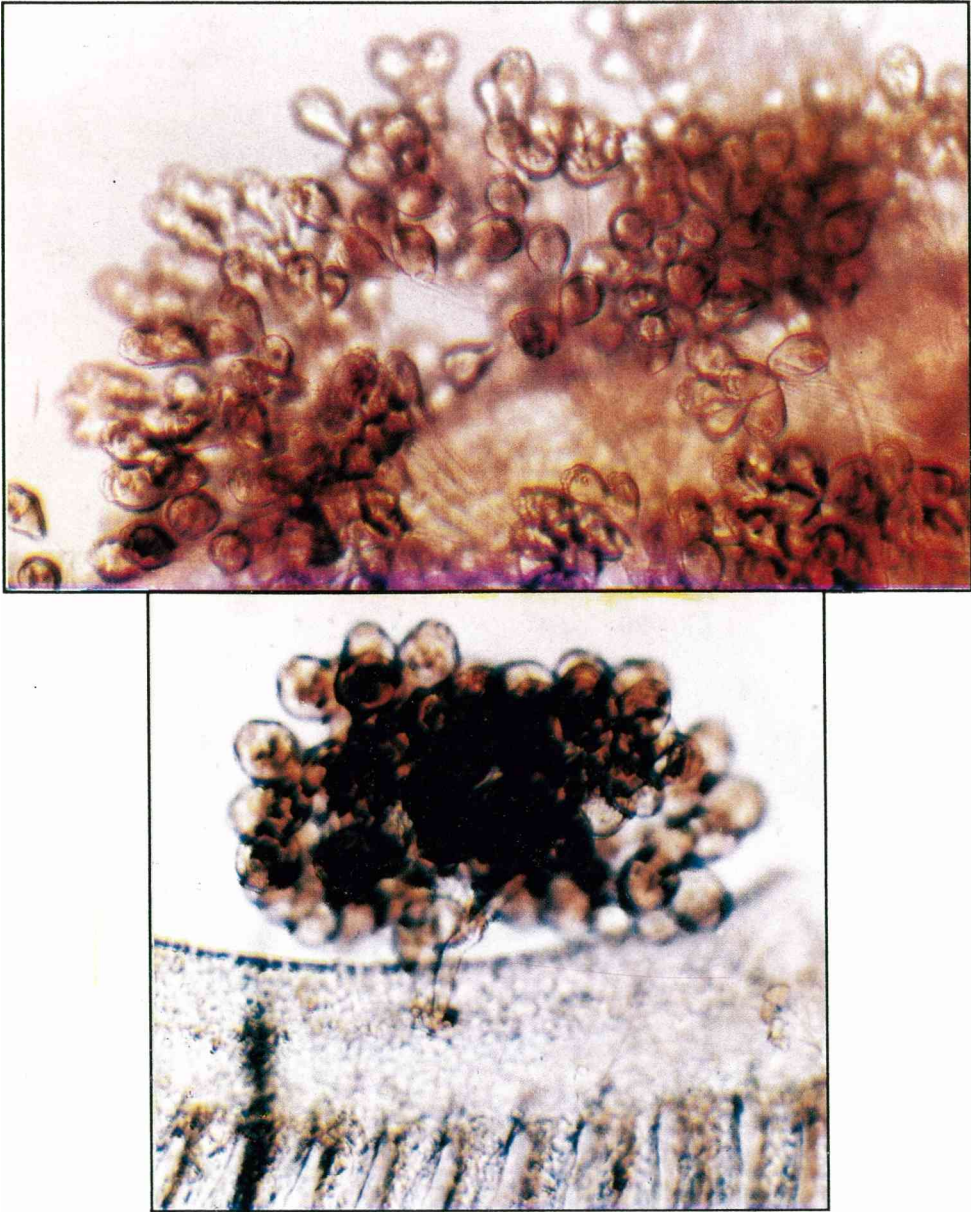


Fig. 7. The ciliate *Zoothamnium* on the shell (above) and gills (below) of *P. monodon* adult.

Fig. 8. Microsporidian infections of the abdominal muscles of *P. indicus* (top) and ovaries (bottom) of *P. monodon* .



Common name : **MICROSPORIDIOSIS, WHITE OVARIES,** Fig. 8
MICROSPORIDIAN INFECTION

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

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

Stages affected : Juveniles, adults

Gross signs:

- Affected tissues/organs turn opaque white.

Effects on host:

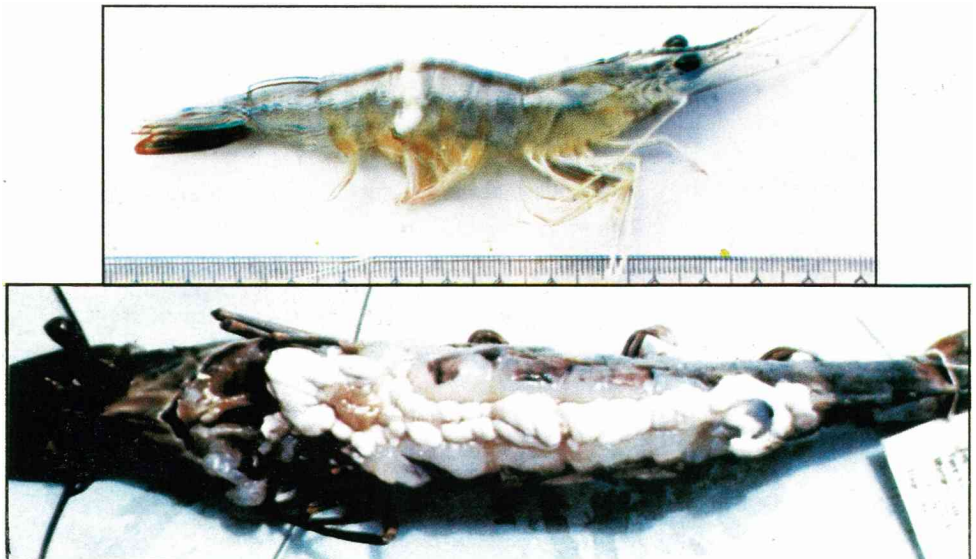
- Spores and other stages of the parasite replace the affected tissues.
- Infection rate is relatively low (usually <10%) but the parasite is highly pathogenic.
- Infection may result in sterility of spawners with white ovaries.

Preventive methods:

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

Treatment:

- None reported.



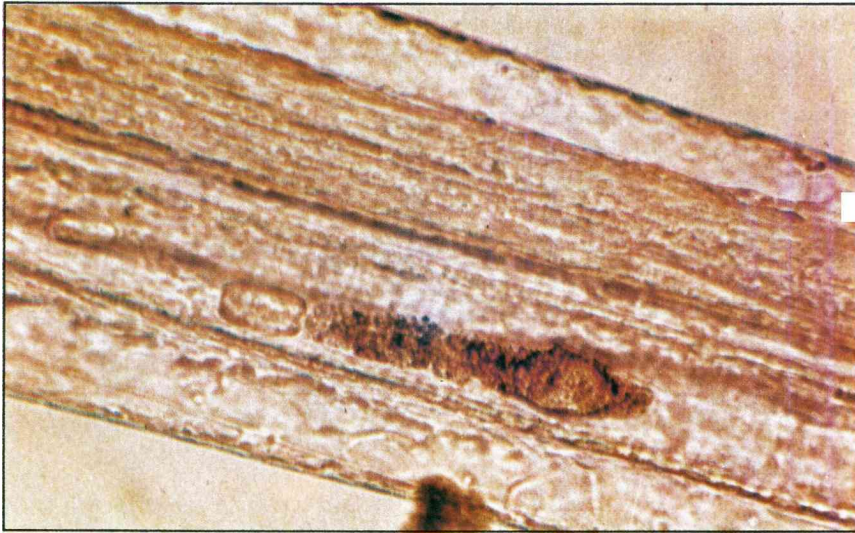


Fig. 9. Gregarines in the gut of *P. monodon* postlarva.

Common name : **GREGARINE DISEASE** **Fig. 9**

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 : *P. monodon*

Stages affected : Larvae, postlarvae, juveniles, adults

Gross signs:

- Gregarines may be detected in the digestive tract microscopically.

Effects on host:

- Large numbers of this protozoan 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:

- Eliminate the molluscan intermediate host.

Treatment:

- None reported.

NUTRITIONAL, TOXIC, AND ENVIRONMENTAL DISEASES

- Chronic Soft-shell Syndrome, Soft-shelling
- Black Gill Disease
- Blue Disease, Sky Blue Shrimp Disease, Blue Shell Syndrome
- Red Disease, Red Discoloration
- Muscle Necrosis
- Cramped Tails, Bent Tails, Body Cramp
- Acid Sulfate Disease Syndrome
- Asphyxiation, Hypoxia

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Common name : **CHRONIC SOFT-SHELL SYNDROME, Fig. 10
SOFT-SHELLING**

Causative agents:

- Nutritional deficiency; pesticide contamination; and poor pond water and soil conditions.
- Exposure of normal hard-shelled shrimps to very low levels of chemical pesticides such as Aquatin or Gusathion A or to higher levels of rotenone (10-50 ppm) and saponin (100 ppm) for 4 days resulted in significant soft-shelling of the stock.
- 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 (<1 ppm), and low organic matter content (<7%).
- Insufficient or infrequent water exchange was highly correlated with soft-shelling.
- 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.

Species affected : *P. 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.
- 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 epicommsensals.

Preventive methods:

- Feed shrimps adequately and use only good-quality feeds.
- Flush ponds thoroughly particularly when using chemical pesticides.
- Maintain pond water and soil of good quality.
- Change water daily, if possible, or at least once a week or every two weeks.

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 a 1:1 ratio of calcium-to-phosphorus.
- Water must be changed immediately and frequently, particularly when pesticide contamination is suspected.



Fig. 10. *P. monodon* with dark, rough and soft shell.

Common name : **BLACK GILL DISEASE**

Fig. 11

Causative agents:

- Chemical contaminants like cadmium, copper, oil, zinc, potassium permanganate, ozone, ammonia, and nitrite 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 : *P. monodon*

Stages affected : Larvae, postlarvae, juveniles, adults



Fig. 11. *P. monodon* adults with reddish (top) and blackish (bottom) gills.

Gross signs:

- The gills show reddish, brownish to black discoloration, and atrophy at the tip of the filaments.
- In advanced cases, most of the filaments are affected and the gills become totally black.
- Dorsal side of the body may be covered with a fog-like substance.
- Physical deformities.
- 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 discharges 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.

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

Fig. 12

Causative agents:

- Low levels of the carotenoid astaxanthin.
- 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.

Species affected: *P. monodon*

Stages affected : Juveniles, adults

Gross signs:

- Sky-blue color instead of the normal brown-black.
- Lethargic shrimps with shells sometimes soft and thin with rough surface.
- Absence of intense red color after cooking.

Effects on host:

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

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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.



Fig. 12. *P. monodon* adults with blue shell syndrome (2nd and 4th from left); others are normal.

Common name : **RED DISEASE, RED DISCOLORATION** Fig. 13, 14

Causative agents:

- Presence of aflatoxin (produced by the fungi *Aspergillus* spp.) in feeds.
- Associated with high inputs of lime (2-6 tons/ha) in pond which therefore has high initial pH, prolonged exposure to low salinity (6-15 ppt), and rancid feeds especially after storage at high temperatures.

Species affected: *P. monodon*

Stages affected : Late postlarvae, juveniles, adults

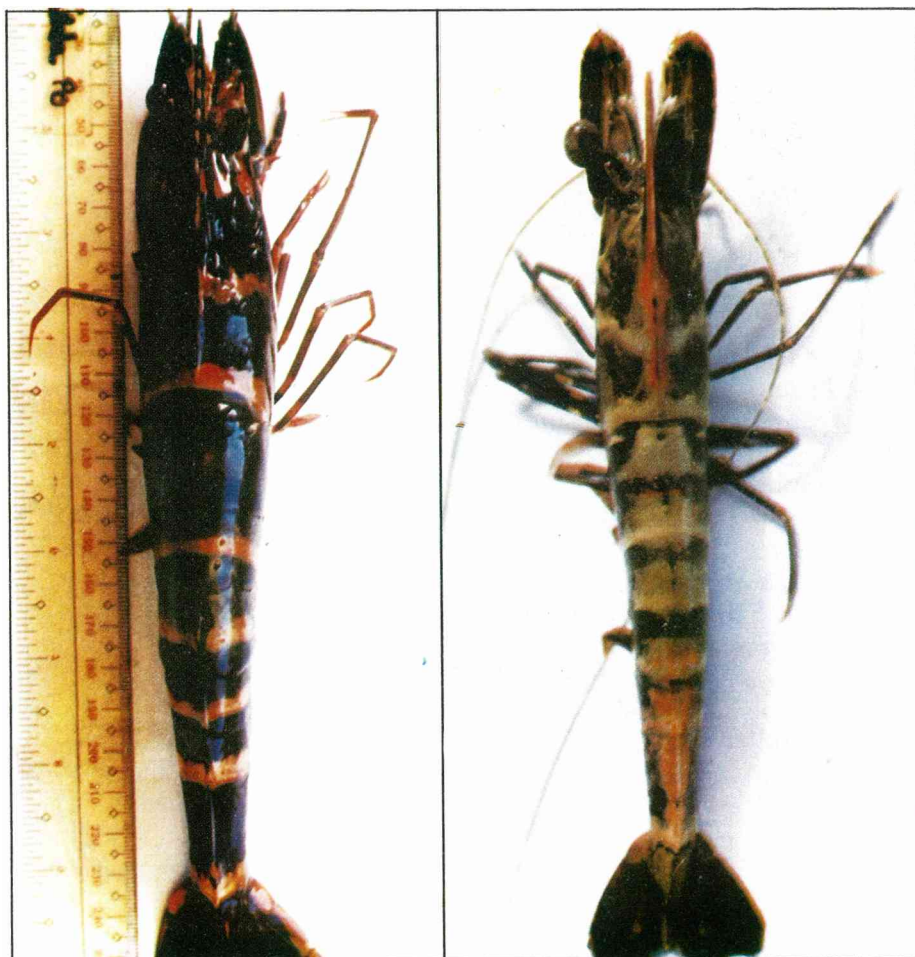


Fig. 13. *P. monodon* adults with red disease. Left: early signs, natural incidence. Right: reddening after 3 weeks of exposure to aflatoxin B₁.

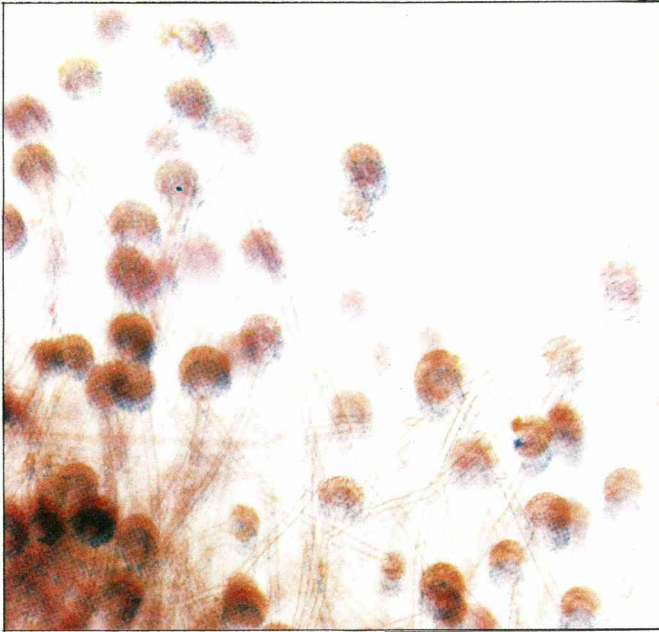


Fig. 14. The mold *Aspergillus* sp., a common contaminant in prawn feeds.

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 as shown by death within minutes after being lifted out of the water.
- Many of the animals are confined to shallow waters at the pond periphery.
- Yellowish and eventually reddish discoloration of the body and appendages.
- Red, short streaks on gills.
- Reddish color of fecal matter.
- Poor growth.
- Increased fluid in the cephalothorax emitting foul odor.

Effects on host:

- 50 μ g aflatoxin/g of feed causes atrophy and necrosis of the hepatopancreas, hemocytic infiltration, and fibrosis after three weeks.
- Gradual mortalities up to 98% in 3 months.
- High doses of aflatoxin speed up the development of the yellow to red discoloration.
- Shrimps are less resistant to stress.
- Histopathology shows necrosis of the hepatopancreas. The degree of reddening appears to be directly related to the degree of atrophy and necrosis of the hepatopancreas.

Preventive methods:

- Use only recently manufactured feeds.
- Store feeds properly in well-ventilated and cool rooms, preferably at 10-20°C or lower.
- Prepare pond bottom properly.
- Reduce organic matter content.
- Reduce lime input during pond preparation.

Treatment:

- None reported.

- Common name* : **MUSCLE NECROSIS** **Fig. 15**
- Causative agents* : Stressful environmental conditions like low oxygen levels, temperature or salinity shock, overcrowding, and severe gill fouling.
- Species affected* : *P. monodon*
- Stages affected* : Postlarvae, juveniles, adults

Gross signs:

- Opaque white areas on the abdomen.
- Blackening on edges of the uropod followed by erosion.
- Liquid-filled boils at the tip of uropods in advanced stages.
- Weakness and, eventually, death.



Fig. 15. *P. monodon* with muscle necrosis.

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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. This condition may serve as portals of entry for a secondary systemic infection by bacteria.

Preventive methods:

- Reduce stocking density in ponds.
- Give adequate feed but do not overfeed.
- Improve water quality by frequent or daily water change (5 - 10%).

Treatment:

- None reported.

Common name : **CRAMPED TAILS, BENT TAILS, BODY CRAMP** **Fig. 16**

Causative agents : Unknown, but the disease is associated with mineral imbalance or increased water and air temperatures, e.g., during handling of shrimps in air warmer than the culture water.

Species affected : *P. monodon*

Stages affected : Juveniles, adults

Gross signs : Partial or complete rigid flexure of the tail (while alive).

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.

Preventive methods:

- Avoid possible causes.

Treatment:

- None reported.



Fig. 16. *P. monodon* juvenile with body cramp.

Common name : **ACID SULFATE DISEASE SYNDROME**

Causative agent : Low water and soil pH

Species affected : *P. 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 color of the pond soil.

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 condition as above.

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Common name : ASPHYXIATION, HYPOXIA

Causative agent : Very low levels of dissolved oxygen (D.O.)

Species affected : *P. 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.
- Sublethal levels may cause impairment of metabolism resulting in growth retardation.

Preventive methods:

- Decrease stocking density if aeration and water-change facilities are not available.
- Monitor D.O. levels late in the afternoon and early in the morning.
- Provide aeration facilities and water pump for ready water change.
- Monitor and control feeding of artificial diets according to consumption rates.

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GLOSSARY

- 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. These cells may occur singly or form simple associations such as chains.
- Cyanotic** : possessing bluish skin due to presence of large quantities of deoxygenated blood in minute vessels.
- 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 may be formed in a vesicle at the tip of this tube.
- Endoparasitic** : parasites living inside the body of the host.
- Epicommensal** : a microorganism that lives on the external surfaces of another organism and derives benefits from it without causing any harm.
- Fibrosis** : excessive proliferation of fibrillar elements.
- Fungus** : a general term for a group of eukaryotic protists (e.g., mushrooms, yeasts, molds, etc.) marked by the absence of chlorophyll and the presence of a rigid cell wall.
- Hemocyte** : a blood cell.
- Hypha** : a tubular filament which is the unit structure of fungi.
- 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.
- Mycosis** : any disease caused by fungi.
- Necrosis** : the state wherein the cells and the tissues are of lower activity and eventually die.
- Pathogenic** : disease-causing.

- 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 the host has been weakened by other causes such as entry of another pathogen.
- Stress** : the sum of the biological reactions to any adverse stimulus that tends to disturb an organism's physiological stability.
- Toxin** : a poison.
- 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.
- Zoospore** : motile spores produced by means of asexual reproduction.

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 should be dried thoroughly (until the soil cracks) before using these for the next operation.

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

1. Using Table 1, 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 of sodium thiosulfate ($\text{Na}_2\text{S}_2\text{O}_3$) before using the water.

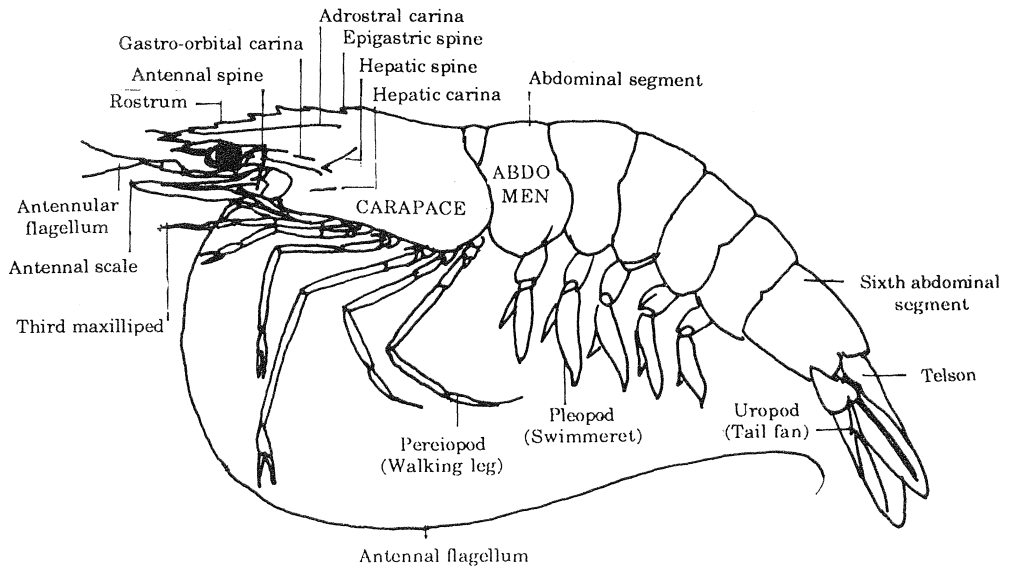
4. If using ordinary household bleach (Purex, Chlorox, etc. with 5% available chlorine), use Table 2 to determine the amount of bleach to be used for a desired volume of water, then follow steps 2 and 3 above.

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

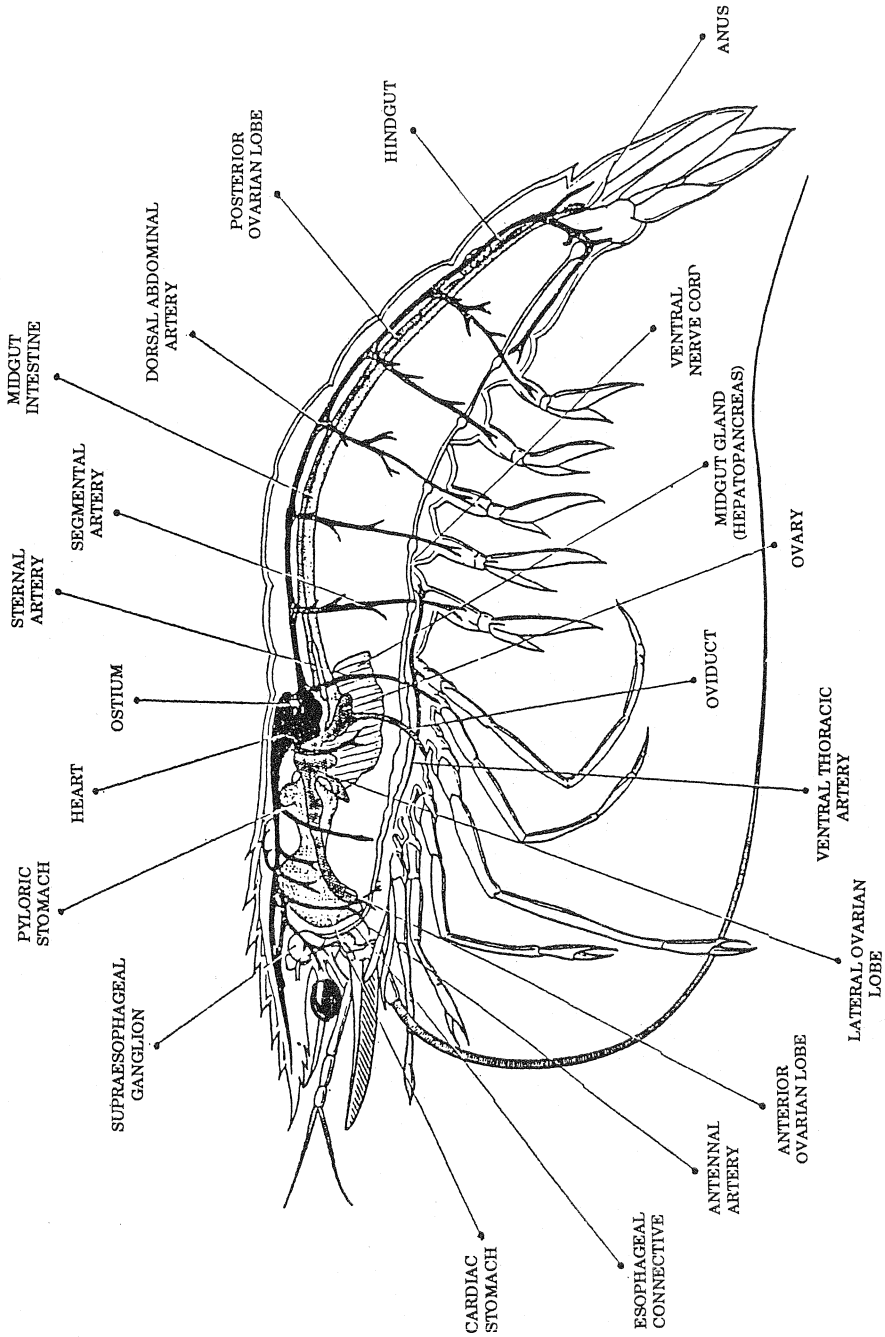
Volume of water	Chlorine concentration			
	5 ppm	10 ppm	15 ppm	20 ppm
0.25 ton	25	50	75	100
0.50 ton	50	100	150	200
1.0 ton	100	200	300	400
2.0 tons	200	400	600	800
3.0 tons	300	600	900	1,200
5.0 tons	500	1,000	1,500	2,000
10.0 tons	1,000	2,000	3,000	4,000

Appendix III. External anatomy of penaeid shrimp

External (after Motosh, 1981)



Internal



Appendix IV. 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 V. 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 VI. 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. Dissolve 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 VII. 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 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 (e.g., Fish Health Section of SEAFDEC/AQD). If the laboratory is located nearby, live samples may be sent by packing these in clean, aerated culture water in plastic bags. Diseased shrimp 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 in 5% (for larvae/postlarvae) or 10% (for adults) buffered formalin* in plastic or glass bottles. The same number of specimens are sent as for live samples and diseased animals must also be separated from normal ones. Only direct microscopic examination (for parasites and fungi) and histopathological examination may be done on fixed samples.

*10% Buffered Formalin (1 liter)

Formalin	100 cc	Sodium phosphate, monobasic	4 g
Seawater	900 cc	Sodium phosphate, dibasic, anhydrous	6 g

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3. Iced samples are sent by packing adult/juvenile shrimp in plastic bags (separate diseased 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.

4. All pertinent data/information must be sent with the samples.

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

Hatchery

<i>Parameter</i>	<i>Range</i>
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
Nitrite (NO ₂ -N)	< 0.02 ppm

Grow-out ponds

<i>Parameter</i>	<i>Range</i>
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

DISEASES OF PENAEID SHRIMPS IN THE PHILIPPINES

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