Chapter Six

DISEASES

Ma. Cecilia L. Baticados*

The great losses suffered by the industry due to diseases attest to the need to focus more attention on this aspect of aquaculture. Prawn diseases have been the subject of many reports from various culture facilities in the Philippines (Villaluz 1975, Gacutan 1979, Vicente et al 1979), Taiwan (Liao et al 1977), Thailand (Ruangpan 1982), and Mexico (Lightner et al 1984). Reviews of diseases causing significant losses in penaeid shrimp and prawn culture have been made by Lightner (1983, 1985) with special emphasis on more recent trends and developments. The present review deals specifically with diseases of the giant tiger prawn, *Penaeus monodon* Fabricius, including the diagnosis and pathology of disease or disease agents involved and their prevention and control.

VIRAL DISEASES

Perhaps because of the specialized methods of diagnosis involved in the study of viral diseases, reports on these are relatively recent. A group of scientists led by Dr. Donald V. Lightner of the University of Arizona, U.S.A. first identified and reported the occurrence of viruses in *P. monodon.* At present, three types of viruses have been found in *P. monodon*: the *monodon* baculovirus or MBV, the infectious hypodermal and hematopoietic necrosis virus or IHHNV, and the hepatopancreatic parvo-like virus or HPV.

P. monodon Baculovirus (MBV) Disease

In 1980, Lightner et al reported a disease which wiped out a whole population of adult *P. monodon* in Mexico. The mortalities were suspected to be due to the destruction of hepatopancreatic tubule epithelium or to secondary bacterial septicemias (Lightner et al 1984). The disease agent was found to be a singly enveloped, rod-shaped, presumed DNA virus of

*Research Associate of SEAFDEC Aquaculture Department

the genus *Baculovirus* subgroup A (Brock et al 1983, Lightner et al 1983b). *P. monodon* baculovirus (MBV) occurs free or within proteinaceous polyhedral occlusion bodies in the nucleus, with nucleocapcids measuring 42 ± 3 nm x 246 ± 15 nm and enveloped virions measuring 75 + 4 nm x 324 ± 33 nm (Lightner et al 1983b).

MBV has been found in *P. monodon* coming from Taiwan, the Philippines, Malaysia, French Polynesia, Hawaii (Brock et al 1983), and Kenya (Colorni et al 1987), Mexico, Singapore (Lightner 1985), and Indonesia (Baticados, unpubl.). It affects the hepatopancreatic tubule and duct epithelium of postlarvae, juveniles, and adults as well as the anterior midgut epithelium of the very young postlarvae (Lightner 1983, Lightner et al 1983b). The pathogenesis of MBV disease in the prawn hepatopancreas starts with the presence of slightly hypertrophied hepatopancreatocyte nuclei and few completed virions and eventually ends up with cell necrosis and cytolysis, releasing the virus and occlusion bodies into the gut lumen (Lightner et al 1983b). Shrimp populations affected by MBV reach up to 70% cumulative mortality among older stages (juvenile to adults) in tanks and raceways (Brock et al 1983). Younger stages (PL_5 and PL_{10}) examined had low incidence and severity, making it difficult to diagnose the disease until the shrimp were 20-30 days old (Lightner et al 1983b). A case of multiple infection with MBV, a reo-like virus, rickettsia-like organisms, and bacteria was recently reported in extensively cultured *P. monodon* juveniles in Malaysia (Anderson et al 1987). About 61% of all shrimp examined showed the typical intranuclear MBV occlusion bodies. Anderson and his co-workers (1987) believe that the MBV and the reo-like virus may have facilitated the initial rickettsial infection or may have become patent after shrimp had been weakened from rickettsia and secondary bacterial infections.

The reservoir of infection for MBV is believed to be feral *P. monodon* as indicated by its enzootic nature in Taiwan and Philippine hatcheries using wild-caught broodstock (Brock et al 1983). The same report suggests that patently infected prawns may be the source of infection in culture settings, as MBV virions contained in occlusion bodies shed in feces may remain intact for years and could be a potential source of infection to susceptible shrimp (Brock et al 1983).

The presence of MBV in prawn may be detected through direct microscopic examination of wet mounts of infected tissue stained with 0.1% aqueous solution of malachite green to demonstrate one or more bright green spherical occlusion bodies in hypertrophied nuclei of hepatopancreatic or midgut epithelial cells (Lightner 1985). A more sensitive method is the histological preparation of tissues from prawns with enhanced infections, enhancement being accomplished by crowding in small tanks to increase the prevalence and severity of infection especially among late postlarvae (Lightner et al 1983b, in press a in Lightner 1985). Although not specific for MBV, Brown and Brenn's gram stain intensely dyes the occlusion bodies, making them visible in light microscopy; otherwise, histological sections are processed and stained with uranyl acetate and lead citrate for transmission electron microscopy (Lightner and Redman 1981, Lightner et al in press b in Lightner 1985). MBV has never been grown in cell culture so diagnosis using serologic or immunologic procedures are not available for distinguishing MBV viral antigens (Brock et al 1983).

Observing the gross appearance of the prawn may sometimes aid in the diagnosis of suspected MBV infections. Moderately to severely infected postlarvae are usually much smaller and darker in color (pale bluish-grey to dark blue-black) than are less affected or unaffected postlarvae (tan or buff base) (Lightner et al 1983b). General signs of severe infections are lethargy, loss of appetite, decreased preening activity, and therefore greater susceptibility to surface and gill fouling by benthic microorganisms (Lightner et al 1983b).

MBV infections may be prevented only through avoidance, i.e., by using MBV-free stages of prawn (Brock et al 1983), quite a difficult feat particularly when dealing with younger stages (e.g., PL_{25}) where the incidence and severity of infection are low and thus not easily diagnosed (Lightner et al 1983b). Crowding stress, cannibalism of dead prawns, or ingestion of free virus or occlusion bodies with virus from tank sediments are believed to increase the prevalence and severity of MBV infections (Lightner et al 1983b). Good and sanitary husbandry practices as well as proper nutrition, therefore, must always prevail in the culture facility to control such infections (Lightner et al 1984, Brock et al 1983). Antibiotic therapy, i.e., by using medicated feeds could also control secondary bacterial infections (Lightner et al 1984). Otherwise, eradication would be the only alternative left, particularly in highly prevalent and

severe infections in cultured prawn populations (Brock et al 1983).

Infectious Hypodermal and Hematopoietic Necrosis Virus (IHHNV) Disease

The infectious hypodermal and hematopoietic necrosis virus is an unclassified, probable picornavirus (Lightner 1985). The virions are icosahedral and small, 17-28 nm, and the inclusions or polyhedra are eosinophilic, intranuclear, single, and basophilic (Brock et al 1983, Lightner 1983, Lightner et al 1983a).

IHHNV has been found in the United States, South America, Southeast Asia, and Israel (Brock et al 1983). Its extensive range may have resulted from contaminated shipments of live *P. stylirostris* and *P. vannamei* from South or Central American countries (Bell and Lightner 1983 in Brock et al 1983) which could be the natural geographic range of IHHNV (Brock et al 1983). IHHNV was detected in histological samples coming from *P. monodon* reared in Ecuador, Guam, Tahiti, the Philippines, and Hawaii (Bell and Lightner 1983), Singapore, and possibly Taiwan (Lightner 1985).

Lightner (1983) reported 80-90% cumulative mortalities within two weeks from onset of IHHNV in 0.05-l g *P. monodon.* This virus could cause epizootics in cultured prawn populations (Bell and Lightner 1983), particularly among postlarvae and juveniles (Lightner 1983). The target organs or tissues are the cuticular hypodermis, hemocytes, hematopoietic organs, and connective tissues (Lightner 1983, Lightner et al 1983a). It may be present in larval stages as a latent or inapparent infection (Brock et al 1983).

The incubation period for IHHNV is 5-14 days after exposure to the virus based on experimental infection (Bell and Lightner 1983a in Brock et al 1983). The virus may be transmitted via parenteral infection, direct contact, indirect contact with contaminated water, or by *per os* exposure (Bell and Lightner 1983b in Brock et al 1983). In culture, *P. monodon* was shown to develop clinical IHHNV disease and harbor IHHNV as a latent carrier (Brock et al 1983).

IHHNV may be detected through histological preparations of tissues having the characteristics Cowdry type A intranuclear inclusion bodies (Lightner et al in press a) and direct samplings for microscopic examination of wet mounts (Lightner et al in press b in Lightner 1985). Enhancement of infection (through crowded and stressful conditions) or bioassay of a suspect shrimp population with a sensitive indicator species (i.e., juveniles or 0.05-4 g *P. stylirostris*) may also be done, followed by sampling and histopathology (Lightner et al in press b in Lightner 1985). Direct sampling and histopathology have limited sensitivity because they demonstrate only prawns with acute/subacute infections in populations with higher prevalence of the disease. Enhancement of infection, on the other hand, is more sensitive but is not suitable for demonstration of IHHNV in asymptomatic carriers (Lightner et al in press b in Lightner 1985).

Prevention of IHHNV disease consists only of avoidance, i.e., by preventing the introduction of IHHNV-infected prawn into the culture area while control would mean eradication of the virus through depopulation of infected or exposed prawns and disinfection of contaminated areas (Brock et al 1983). There should also be absolute quarantine of all live prawns (Brock et al 1983).

Hepatopancreatic Parvo-like Virus (HPV) Disease

Another virus reported by Lightner and Redman (1985b) infected the hepatopancreas of *P. monodon* in Malaysia, appearing as aggregations of 22-24 nm diameter, basophilic, PAS-negative, Feulgen-positive, intranuclear inclusion bodies in necrotic and atrophied hepatopancreatocytes. The same workers examined samples coming from a private company in the Philippines and referred to the virus as HPV for hepatopancreatic parvo-like virus.

Prawns with HPV exhibited poor growth rates, anorexia, reduced preening activity, increased surface fouling, and occasional opacity of tail musculature. The incidence of HPV (1 juvenile) was relatively lower than those of IHHNV infections (6 juveniles and adults) and MBV infections (69 juveniles) within the same batch of 114 prawns examined. The non-specific signs of the disease were accompanied by mortalities during the juvenile stages after apparently normal development

through the larval and post-larval stages (Lightner and Redman 1985b).

P. monodon adults imported by Israel from Kenya were also found to be infected with HPV during quarantine so the entire stock was destroyed (Colorni et al 1987).

Available data on the viral diseases of *P. monodon* are summarized in Table 1.

BACTERIAL DISEASES

Like many other pathogens, bacteria may always be present in the water and are "opportunistic" organisms. They affect prawns both as primary and secondary invaders, and

Table 1. Viral diseases of *Penaeus monodon*

Disease Agent	Stages Affected	Target Organ/ Tissues	Geographic Distribution	Diagnostic Method
MBV	PL_5 to adults	Hepatopancreatic tubule and duct epithelium; anterior midgut epithelium	Taiwan, Indonesia, Philippines, Malaysia, Singapore, French Polynesia, Mexico, and Hawaii	Microscopic examination of wet mounts of infected tissue Histological examination of infected tissues under light and electron microscopy
IHHNV	Post-larva (0.05-1g) to juveniles	Cuticular hypodermis, hemocytes, hematopoeitic organs and connective tissues	Singapore, Philippines, Guam, Tahiti, Hawaii, USA, Israel, Ecuador, and other South/Central American countries, and possibly in Taiwan	Microscopic examination of wet mounts Histological examination Enhancement of infection or bioassay of sensitive indicator species followed by sampling and histopathology
HPV	Juveniles	Hepatopancreas	Malaysia, Philippines and Kenya	Microscopic examination

infections develop usually as a result of adverse changes in the environment which easily stress the prawn and thus lower its resistance. A number of these bacterial diseases have been reported to occur in different stages of *P. monodon.*

Necrosis of Appendages

Larvae and young postlarvae (PL_{10}) of *P. monodon* may be affected by a bacterial disease causing necrosis of the appendages, with postlarvae relatively more resistant than the younger stages (Aquacop 1977, 1979; Vicente et al 1979). In protozoeae, the disease starts with the liquefaction of gut contents. Necrosis begins as a "browning" of the exoskeleton or tip of the appendage, e.g., uropods in protozoea III or pleopods in mysis, spreads towards the base and finally appears as an

Morphology	Pathology/ Signs	Prevention/ Control	References
Singly enveloped; free or with polyhedral occlusion bodies; intranuclear; nucleocapsids 42±3 nm X 246 ± 15 nm; enveloped virions 75±4 nm X 324 ± 33 nm	Necrosis and cytolysis of hepatopancreatic epithelial cells and anterior midgut epithelium, up to 70% mortality, postlarvae small and dark, lethargy, loss of appetite, reduced preening activity	Prevention through avoidance, sanitation and proper nutrition Eradication through depopulation and disinfection	Lightner 1983, 1985 Lightner and Redman 1981 Lightner et al 1983b, 1984 Brock et al 1983, Colorni et al 1987, Anderson et al 1987
Probable picornavirus; virions icosahedral, small 17-28 nm; inclusions, eosinophilic intranuclear, single	80-90% cumulative mortalities, latent infection in larvae possible	Prevention through avoidance Eradication through depopulation and disinfection and quarantine of all live prawns	Bell and Lightner 1983 Brock et al 1983 Lightner 1983, 1985 Lightner et al 1983a
Aggregations of 22-24 nm dia., intranuclear, basophilic inclusion bodies	Necrosis and atrophy of hepatopancreatocytes but very low incidence, poor growth rates, anorexia, reduced preening activity, occasionally opaque tail, mortalities		Lightner and Redman 1985a Colorni et al 1987

erosion of affected areas (Aquacop 1977, Gacutan 1979, Vicente et al 1979). Affected mysis and postlarvae have twisted antennae, broken setae, deformed abdomen, opaque and eroded appendages, and exhibit incomplete molting (Aquacop 1977, Vicente et al 1979). Gacutan (1979) suggested that mortalities were due to secondary bacterial infection after cuticular injury or breakdown, or to the inability of larvae to molt. This infection played a minor role in SEAFDEC AQD hatchery mortalities (Gacutan 1979) but wiped out the whole larval population at the MSU-IFRD hatchery in 24 hours (Vicente et al 1979). The disease has also been observed in Indonesia, Malaysia, Singapore, Taiwan and Thailand (Baticados, unpubl.).

The use of antibiotics to control bacterial infections has been instituted against necrosis of larvae and heavy mortalities (Aquacop 1983). Aquacop (1977) has tested and used prophylactic/therapeutic drugs, namely, erythromycin phosphate at 1 ppm active product (from the second day of protozoea II), streptomycin-bipenicillin at 2 ppm AP (2 UI/ml), tetracycline chlorhydrate at 1 ppm AP, sulfamethazin at 3 ppm AP, and Furanace at 0.1 ppm AP. The drug is applied every other day until the post-larval stage is reached. Treated larvae resume feeding, recover quickly, and lose any trace of necrosis after regenerating new appendages at the next molt (Aquacop 1977). More recently, chloramphenicol has been used in hatcheries at 1 ppm every 3 days for prophylactic treatment (Sunaryanto 1986) and found to be most effective at prophylactic levels of 2-6 ppm every 2 days or therapeutic levels of 2-10 ppm with variation adjusted according to the larval stage (Aquacop 1983).

Vibrio Disease

The bacterial species *Vibrio* has also been observed to affect the protozoeal stages and to cause heavy mortalities of up to 80% in hatcheries (Ruangpan 1982, PCARRD 1985). The disease is asymptomatic but occurred primarily during the period December to February in Thailand's Phuket Fishery Station (Ruangpan 1982). Ruangpan (1982) believed that perhaps *Vibrio* preferred to grow in colder temperature while Delves-Broughton and Poupard (1976 in Aquacop 1977) suggested that the *Vibrio* disease observed in *P. monodon* may have been due to the prevailing environmental conditions.

Luminous Bacterial Disease

Luminous bacteria were recently isolated from larval and post-larval sediments and water samples from hatcheries in Panay Island, Philippines. The bacteria were recovered in dominant proportions from weak and dead prawns and rearing water samples and were isolated from sediments and sea-water samples indicating its occurrence in the natural environment (Pitogo, pers. comm.). The luminous bacteria were classified as predominantly *Vibrio harveyi* and a few isolates as *V. splendidus* (Pitogo, pers. comm.). Heavily infected larvae were moribund and luminescent in the dark due to the large numbers of motile bacteria in the tissues. The bacteria were also isolated from apparently active larvae in low numbers. Mass mortalities were often encountered during heavy infection. The disease is also is also a serious problem in Indonesia, Malaysia, and Thailand (Baticados, unpubl.).

Several luminous bacterial isolates were tested and found to be resistant to many of the more commonly used antibiotics such as erythromycin, penicillin, streptomycin, and sulfadiazine (Baticados, unpubl. data). Screening of other chemicals and drugs that could control luminous bacteria is being conducted and tolerance of the larvae and postlarvae to effective levels of the chemicals is being investigated at SEAFDEC AQD. It appears that resistance of the bacteria to certain drugs depended on the kind of antibiotic commonly used in a particular area (Baticados, unpubl. data).

Rigid sanitary practices which include chlorination of the rearing water, removal of the wastes and sediments from the tank bottom, and more frequent water change (flow-through twice daily) were observed to reduce mortalities due to luminous bacteria.

Filamentous Bacterial Disease

The filamentous bacterial species *Leucothrix mucor* has been found to occur as an ectocommensal of *P. monodon* larvae and postlarvae particularly during the months of October to December (Gacutan 1979). The bacteria attach themselves to the gills, setae, appendages, and body surface of the prawn and flourish particularly in waters rich in organic and inorganic substances such as phosphates and nitrates (Gacutan 1979).

When present in large numbers, the bacteria can cause mortalities due to hypoxia and impairment of the molting process (Gacutan 1979, Lightner et al 1984). Losses in the prawn population may also occur in conjunction with stressful conditions resulting from crowding, molting, or low oxygen levels (Lightner et al 1984). The disease occurs in the Philippines, Indonesia, and Malaysia (Baticados, unpubl.).

The disease may be controlled by Cutrine-Plus, a copper compound, given upon onset of the disease at 0.1 mg Cu/l for 24 h 0.25-0.5 mg Cu/l for 4-8 h (Lightner et al 1984).

Shell Disease

Juveniles and adult prawns from grow-out earthen ponds in the Philippines, Indonesia, Malaysia, Taiwan, and Singapore are affected by a shell disease or erosion of the exoskeleton (Villaluz 1975, Chong and Chao 1986, Baticados, unpubl.). Affected juveniles exhibit eroded and brownish dorsal shell surface which may be eliminated when the prawn molts except when the underlying membranes or tissues are already damaged by the bacteria (Villaluz 1975). Chitinolytic forms of *Vibrio* and *Aeromonas* species have been isolated from shell lesions of pond-grown prawns, and the disease was observed during experimental infection when accompanied by mechanical injury of the shell (Baticados et al 1986). Most of the isolates from the haemolymph of adult *P. monodon* with eroded uropods and carapace lesions were of the species *Vibrio alginolyticus* (Chong and Chao 1986). *Vibrio* and *Aeromonas* are very common in sea water and may also be part of the natural flora of the prawn so that the disease could be a result of secondary infection after mechanical injury or trauma of the shell and the underlying membranes.

Shell disease was also reported to affect wild adult prawns caught with cast or drag nets off the Cochin backwaters of India (Gopalan et al 1980). Prevalence, though, was quite low with only 6 out of 155 specimens of 7.9-8.8 cm length (Gopalan et al 1980).

General control procedures for bacterial diseases. Bacterial infections may be reduced by frequently changing the water during the protozoeal stages to keep water quality at an optimum level (Vicente et al 1979). Sanitary measures like

drying, cleaning, and disinfection of culture tanks could also considerably minimize the occurrence of bacterial diseases in larvae and postlarvae (Lightner 1983).

Chemotherapy of bacterial infections may be done through direct addition of antibiotics to culture tank water during hatching, larval, or post-larval rearing while for older stages, incorporation of antibiotics in the diet could prove a better method (Lightner 1983). Liao et al (1981 in Liao 1984) suggested the use of furazolidone, glutaraldehyde, and oxolinic acid to prevent the spread of cholera from imported spawners.

Table 2 summarizes information on bacterial diseases of *P. monodon.*

FUNGAL DISEASES

Among the fungi attacking *P. monodon, Lagenidium* has been considered to be the most prevalent and pathogenic in the larval and early post-larval stages. As a result, it was also the most frequently reported and most extensively studied. It has occurred under the same conditions as *Haliphthoros* and *Sirolpidium* which belong to the same group of aquatic fungi, the Phycomycetes (Gacutan 1979, PCARRD 1985).

Larval Mycosis

Lagenidium. In 1977, a fungus which caused heavy mortalities among larvae and postlarvae at the SEAFDEC AQD hatchery was isolated, identified as *Lagenidium callinectes,* and grown in culture media (Baticados et al 1977b, Gacutan and Baticados 1979). *In vivo* and *in vitro* observations of the sporulation process left no doubt as to its reproductive capacity. The hyphal system extends from inside (intramatrical) the body of the prawn to the outside (extramatrical), developing short discharge tubes. A vesicle forms at the end of the discharge tube with sporogenic cytoplasm coming from the hyphal system in 5-10 min. Spore formation proceeds for 10-20 min and spores start to move slowly inside the vesicle, after which numerous motile spores become active and are released 10-15 min later to infect other prawns (Baticados et al 1977b). The fungus has a wide range of tolerance for temperature, pH, and salinity and appears to have the same growth and sporulation conditions as

Table 2. Bacterial diseases of *Penaeus monodon*

Diseases	Agent	Stages Affected	Pathology/Signs
Necrosis of appendages	Bacteria	Zoea, mysis, post-larva	Necrosis of appendages, twisted antennae or broken setae, "browning" of exoskeleton or tip of appendages, liquefaction of gut contents in zoea.
Vibrio disease	*Vibrio*	Zoea	Heavy mortalities up to 80%
Luminous bacterial disease	*Vibrio harveyi* *V. splendidus*	Nauplii, zoea, mysis, post-larva	Prawn luminiscent in the dark, heavy mortalities
Filamentous bacterial disease	*Leucothrix mucor*	Larvae, post-larvae	Bacteria on gills, setae, appendages and body surface; mortalities due to hypoxia; and impaired molting
Shell disease	*V. alginolyticus* *V. parahaemolyticus* *Aeromonas*	Juveniles, adults	Erosion of exoskeleton

for larval rearing (Gacutan 1979). *Lagenidium* spp. have also been observed (Vicente et al 1979) and isolated from *P. monodon* eggs and protozoea (Bautista 1983) at the MSU-IFRD Hatchery in Mindanao, Philippines.

Lagenidium attacks eggs, larvae, and postlarvae of *P. monodon* and has been the cause of mass mortalities in many hatcheries. Infected eggs appear opaque white and do not hatch while larvae become weak, lose equilibrium, respire irregularly, twitch their appendages, and appear either whitish (Bautista 1983) or reddish (Vicente et al 1979). In all cases, the fungal hyphae fill the eggs, replace the tissues of the larvae/postlarvae, and may cause 100% mortality (Villaluz 1975, Aquacop 1977, Baticados et al 1977b, Gacutan 1979, Gacutan and Baticados 1979, Ruangpan 1982). The signs of the disease, however, are apparent only when it is already widespread (Gacutan 1979).

Prevention/Control	References
Erythromycin phosphate 1 ppm, Streptomycin-bipenicillin 2 ppm, Tetracyclin chlorhydrate 1 ppm, Sulfamethazine 3 ppm, Furanace 0.1 ppm, every other day; Chloramphenicol, 1 ppm every 3 days or 2-6 ppm every 2 days (prophylactic) or 2-10 ppm (therapeutic)	Aquacop 1977, 1979, 1983 Gacutan 1979 Vicente et al 1979 Sunaryanto 1986
	Aquacop 1977 Ruangpan 1982 PCARRD 1985
Chlorination of rearing water, removal of bottom sediments, and more frequent water change	
Cutrine Plus, 0.1 mg Cu/l, 24 h or 0.25 to 0.5 mg Cu/l, 4-8 h	Gacutan 1979 Lightner et al 1984
None reported	Villaluz 1975 Gopalan et al 1980 Baticados et al 1986 Chong and Chao 1986

The disease develops very fast, within 1-2 days, especially in younger stages (Aquacop 1977, Ruangpan 1982). In a case in Thailand, the infection occurred in 20% of the larvae (late protozoea to early mysis) but disappeared at the post-larval stage (Ruangpan 1982). *Lagenidium* infection is likewise a problem in Indonesian hatcheries (Baticados, unpubl.).

At SEAFDEC AQD, *Lagenidium* attacked larvae in 35 out of 51 hatchery runs in 1976 (Gacutan 1979) and 12 out of 31 in 1977 (SEAFDEC 1977). It was observed that when the initial infection rates were higher than 10%, the fungus multiplied rapidly and the larvae did not recover (SEAFDEC 1977). Of the 35 runs affected in 1976, 22 were serious and had to be discarded. The prevalence rate was considerably reduced to 9 out of 43 hatchery runs in 1978 because of safeguards instituted against the fungal infection (Gacutan 1979).

In cases where the incidence rates were quite low (1-5%), siphoning of sediments and dead larvae as well as water management (e.g., 30-min flow-through and replenishment of water) every 4 h proved to be effective in controlling *Lagenidium* infections (Vicente et al 1979, Vicente 1981 in PCARRD 1985). Other precautionary measures include the reduction of stocking density and faster water circulation (Ruangpan 1982).

Several chemicals have been screened to combat *Lagenidium* spp. isolated from *P. monodon.* Lio-Po et al (1982) determined the sensitivity of *Lagenidium* isolates to 34 antifungal compounds and found the mycostatic levels of malachite green at 0.1-0.5 ppm, Treflan at 0.1-5 ppm, trifluralin (the active ingredient of Treflan) at 0.005-10 ppm, formalin at 10 ppm, and detergent (Tide) at 5-50 ppm. The mycocidal levels of Tide (50-100 ppm), formalin (50 ppm), and potassium permanganate (100 ppm) could be used for disinfection (Lio-Po et al 1982). The mycostatic levels of the fungicides were then tested on the prawn eggs and larvae and based on the experiments conducted, only Treflan and trifluralin could be safely used for the eggs, larvae, and postlarvae at therapeutic levels up to 0.2 ppm for 24 h, while a 20 ppm Tide bath for 2-4 h followed by complete water change before hatching could be used for disinfection of eggs (Lio-Po and Sanvictores 1986). Spawners may be disinfected with a high concentration of Treflan R (5 ppm) for 1 h, followed by an equal duration of thorough rinsing (Gacutan 1979). Aquacop (1983) preferred to use Treflan R at 5 ppm as continuous preventive treatment against attacks of *Lagenidium* as trifluralin is very volatile and drifts away with tank aeration (Aquacop 1977). Because of these characteristics, it is believed that a combination of the single dose treatment and continuous dosing regimes might be more effective than only either of the two methods, i.e., a single dose producing an effective 0.01-0.1 ppm trifluralin level followed by a continuous application at a rate that will compensate for a 20-30% loss per hour (Williams et al 1986). Considered economical and easy to use, trifluralin gave consistent results without secondary effects (Vicente et al 1979). Malachite green is fungitoxic at 0.006 ppm (Ruangpan 1982) but is safe only for the mysis stage (Lio-Po et al 1978). This chemical is also reported to have potential carcinogenic and teratogenic properties (Bailey 1983). Another chemical, 2,4-D (2.4-dichloro-phenoxy-acetic acid), is well tolerated, has a 96-h

LD50 of 0.6 ppm for M_I, and is also effective in arresting *Lagenidium* infections (Gacutan 1979).

Sirolpidium. Another fungus attacking prawn larvae is *Sirolpidium* (Gacutan 1979, Aquacop 1983, Bautista 1983). At the MSU-IFRD hatchery, this fungus was found to be as pathogenic as *Lagenidium* sp. (Bautista 1983). This fungus does not produce vesicles, has very short discharge tubes, and small numerous zoospores which may enter through the anus, mouth, or wounds (Bautista 1983). *Sirolpidium* infections exhibit the same signs as in *Lagenidium* infections.

Preventive measures include disinfection of equipment and tanks, filtration of water, chemical treatment of spawners, environmental sanitation, proper disposal of infected larvae and contaminated tank water, and occasional siphoning of debris and dead larvae (Bautista 1983). Treflan at 5 ppm may also be used continuously against *Sirolpidium* infection (Aquacop 1982b).

Haliphthoros. A new species, *Haliphthoros philippinensis* was found and isolated from mysis of *P. monodon* at the SEAFDEC AQD hatchery (Hatai et al 1980). It grows in culture media at wide ranges of temperature (13.5-36.3°C), sodium chloride concentration (0.3-7%), and pH (5-11) (Hatai et al 1980). The fungus also replaces the tissues of the prawn larvae.

Lio-Po et al (1985) tested a number of fungicides against *H. philippinensis* and found that for therapeutic purposes the following could be used because they inhibit the growth of and kill the fungus within a 24 h exposure: Furanace (10% active product) at 0.2-1 ppm, malachite green at 0.01-1.3 ppm, formalin at 6-30 ppm, and potassium permanganate at 10 ppm. For disinfection purposes, the detergent Tide (100 ppm),/calcium hypochlorite (200 ppm), and Resiguard (200 ppm) may be used (Lio-Po et al 1985). Furanace was found to have a 24-hour LD50 of 1.6 ppm for protozoea II and 2.0 ppm for mysis (Gacutan and Llobrera 1977). Protozoea II may be safely exposed to a furanace bath of 1 ppm for 6 h; overdosage could result in morphological changes and necrosis (Gacutan et al 1979b), reduced swimming activity, loss of appetite, and weak gill movements (Gacutan and Llobrera 1977). The same level of 1 ppm, however, significantly reduced the algal food *Chaetoceros* populations after a 6-h exposure (Baticados and Gacutan 1977) but did not have considerable effect on the rotifer *Brachionus*

populations (Baticados et al 1977a). Likewise, the use of malachite green, potassium permanganate, and formalin may not be wise, since the fungitoxic levels of the first two chemicals are also possibly toxic to the larvae while formalin is tolerated by larvae and postlarvae only at 5 ppm for 24 h (Lio-Po and Lavilla in Lio-Po et al 1985).

Fusarium Infection

Fusarium, a fungus which produces oval-shaped microconidia and boat-shaped microconidia, was reportedly isolated from nauplius and protozoea of *P. monodon* at the MSU-IFRD hatchery (Bautista 1983). The fungus has scanty white cottony strands of hyphae which attach to the larvae and hinder their movement (Bautista 1983) In adult prawns, *Fusarium* caused the black gill disease in a private prawn farm in Thailand (Ruangpan 1982). About 50% of the prawn population had black gills, reduced feeding activity, and gill tissues destroyed by the fungal mycelium, resulting in large losses of the stock (Ruangpan 1982).

The black gill disease was controlled by harvesting all the prawns and treating the pond with chlorine (Ruangpan 1982).

Other Fungal Diseases

Another fungal species, *Hyphomyces* sp., was isolated from cultured prawn nauplii and protozoea at the MSU-IFRD hatchery. Affected larvae moved sluggishly (Bautista 1983).

In India, adult prawns were found to be infected with two species of fungi - *Saprolegnia parasitica* and *Leptolegnia marina* (Gopalan et al 1980). The hyphae of *L. marina* were found to be older than those of *S. parasitica* in the prawn, indicating that the prawn may have been infected with the former (a marine form) in the sea and later on with *S. parasitica* (a predominantly freshwater form) in the estuary. *L. marina* has mostly intramatrical, long, delicate, densely interwoven, microscopic hyphae with terminal sporangia containing a row of smaller spores while *S. parasitica* has straight, simple, cottony, long filaments with terminal clavate sporangia and motile zoospores. The fungi attack primarily the calcified layer of the

shell, with *L. marina* producing wider and more necrotic lesions. Infection with *L. marina* starts as tiny brownish dots which later fuse to form deeply pitted irregular patches. Affected prawns were generally sluggish, very weak, exhibited gradual mortalities in 32 days, molted irregularly, and had dark brown necrotic lesions all over the body (Gopalan et al 1980).

These reports on fungal diseases of prawn are summarized in Table 3.

PROTOZOAN DISEASES

Protozoans which can cause diseases in prawns may be classified as either ectocommensal or parasitic. The first group consists of opportunistic organisms which cause disease only when present in large numbers. The second group comprises gregarines and microsporidians, endoparasites of the prawn, which may do little harm but could also be fatal to the prawn.

Ciliate Infestation

Several species of protozoans attach to the eyes, gills, appendages, and shell or body surface of prawns, causing respiratory and locomotory difficulties when present in large numbers. These include the peritrichs *Epistylis, Vorticella,* and *Zoothamnium,* and suctorians *Ephelota gemmipara* and *Acineta* which are often seen on prawn larvae and postlarvae (Gacutan et al 1977, Gacutan 1979). Among these, *Epistylis* is commonly observed in the Philippines (Gacutan 1979, Vicente and Valdez 1979, Vicente et al 1979) and Taiwan (Liao et al 1977, Chen 1978, Liao and Chao 1983, Liao 1984). *Epistylis* grows well when there is stratification of dissolved gases, particularly with minimal aeration (Vicente et al 1979). *Zoothamnium* is as prevalent among prawns in Thailand (Ruangpan 1982), Indonesia, Malaysia, and Taiwan (Baticados, unpubl.), and among juveniles and adults particularly soft-shelled ones, in the Philippines (Baticados et al 1986). Less often observed was *Ephelota gemmipara* which nevertheless caused heavy mortalities in several runs at the SEAFDEC AQD hatchery in 1976 (Gacutan 1979). *E. gemmipara* primarily attached to broad and relatively immobile parts of the larvae, e.g., body segments, carapace, and uropods (Gacutan et al 1979a). Larvae infected with this suctorian kicked violently, possibly to shake off the

Table 3. Fungal diseases of *P. monodon*

Disease Agent	Stages Affected	Pathology/Signs
Lagenidium (L. *callinectes*, *Lagenidium* sp.)	Eggs, larvae, early post-larvae	Fungus replaces internal tissues of prawn, eggs do not hatch, larvae weak, up to 100% within 1-2 days
Sirolpidium	Larvae	Same signs as above
Haliphthoros philippinensis	Zoea, mysis	Same signs as above
Fusarium	Nauplius, zoea, juvenile to adult	Scanty white cottony strands attach to larvae; may cause blackening of gills of 50% of pond stock; destroyed gill tissues; mortalities
Hyphomyces	Nauplius, zoea	Larvae move sluggishly
Saprolegnia parasitica and *Leptolegnia marina*	Adults	Attack shell of prawn; affected prawn sluggish, weak, gradual mortalities for 32 days, irregular molting, necrotic dark lesions on body

Prevention/Control	References
Water management; reduction of stocking density Treflan or trifluralin at 0.2 ppm, 24 h treatment Disinfection of spawners with Treflan R, 5 ppm, 1 h Malachite green, 0.006 ppm, only for mysis Disinfection of eggs with Tide, 20 ppm, 2-4 h	Villaluz 1975 Aquacop 1977, 1983 Baticados et al 1977b SEAFDEC 1977 Lio-Po et al 1978, 1982 Gacutan 1979 Gacutan and Baticados 1979 Vicente et al 1979 Ruangpan 1982 Bailey 1983 Bautista 1983 PCARRD 1985 Lio-Po and Sanvictores 1986 Williams et al 1986
Disinfection of equipment and tanks; filtration of water; chemical treatment of spawners with Treflan, 5 ppm, 1 h; sanitation; disposal of infected larvae	Gacutan 1979 Aquacop 1983 Bautista 1983
Furanace, 1 ppm, 6 h	Baticados and Gacutan 1977 Baticados et al 1977a Gacutan and Llobrera 1977 Gacutan and et al 1979b Hatai et al 1980 Lio-Po et al 1985
Harvest affected pond-reared prawns and treat pond with chlorine	Ruangpan 1982 Bautista 1983
None reported	Bautista 1983
None reported	Gopalan et al 1980

pathogen which might have resulted in larval weakening and loss of appetite (Gacutan et al 1979a).

Heavy infestations of these ciliates, particularly *Zoothamniun,* may result in the formation of a mat-like material on the shell and gills of juvenile and adult prawns (Baticados 1988, Villaluz 1975). Increase in their populations may come as a result of the deterioration of the culture water, i.e., high nutrient load, heavy siltation and turbidity, and low oxygen tension. These ciliates do not cause any mechanical damage but do harm to the prawns through immobility, starvation, and hypoxia (Villaluz 1975, Vicente and Valdez 1979, Ruangpan 1982).

Peritrichous ciliates attacking juveniles in nursery tanks were controlled by applying chloroquin diphosphate at a rate of 1.1 ppm for 2 days, the ciliates dying after 3 treatments (Yang 1979). *Zoothamnium* infestation was treated with a 50-100 ppm formalin bath for 30 min (Ruangpan 1982). Pond-reared juveniles with *Epistylis* infestation were exposed to 30 ppm formalin which effectively controlled the disease (Chen 1978). The same concentration was applied in natural food culture tanks before feeding the food organisms to the larvae (Vicente et al 1979). Formalin at 30 ppm did not have any effect on the alga Chlorella and the rotifer *Brachionus* but was found to be the LC_{50} for larvae at 12 hours and to kill mysis and postlarvae after an 8-hour exposure (Vicente and Valdez 1979). A higher dose, 40 ppm, resulted in coarsening of the shell and weakening of pond-reared juveniles (Chen 1978).

Gregarine Disease

Gregarines are among the common microorganisms associated with *P. monodon* larvae (Gacutan 1979). Microscopic examination of the larvae would show the motile stage, the trophozoite, gliding about or attached to the digestive tract (Baticados 1984). Large numbers of this protozoan in the filter apparatus could interfere with particle filtration to the hepatopancreatic ducts or through the gut (Baticados 1984) and could cause heavy mortalities in prawn hatcheries. Gregarines were observed to occur together with luminous bacteria in protozoea I, resulting in mass mortalities within 2 days (Cruz and Pitogo, pers. comm.). In Thailand, two species of gregarines

were found in the gut of 94% of the prawns from a private farm (Ruangpan 1982).

Gregarines utilize a molluscan bivalve as intermediate host so that one way of controlling them is by eliminating this host.

Microsporidiosis

Parasitic infection of the female reproductive organ may result in sterility, weakness, and greater susceptibility to other environmental stresses (Villaluz 1975, Baticados 1984, PCARRD 1985). The disease microsporidiosis has been observed in *P. monodon* spawners and is manifested by the whitening of the ovaries due to the presence of spores and other stages of the parasite which have replaced the ovarian tissues. Microsporidiosis may be diagnosed through gross examination of the ovaries coupled with microscopic examination of fresh squashes or Giemsa-stained smears from infected tissue or histological examination of suspected tissue samples (Baticados 1984).

Microsporidiosis may be prevented or controlled by isolating or destroying infected individuals, avoiding contact of infected spawners with non-infected individuals, and disinfecting culture systems with commercial bleach or disinfectants containing iodine (Baticados 1984).

Table 4 summarizes the information on protozoan diseases of *P. monodon.*

DISEASES CAUSED BY OTHER ORGANISMS

Other organisms may also be present in prawn as ectocommensals or parasites. The damage done by ectocommensals usually depend on their number while damage due to parasites may depend on their pathogenicity/virulence as well as their quantity. There are no reported control measures for these organisms. In any case, however, rigid water management and sanitation would help considerably.

Table 4. Protozoan diseases of *Penaeus monodon*

Disease	Agent	Stages Affected	Pathology/Signs	Prevent/Control	References
Ciliate infestation	*Epistylis* *Vorticella* *Zoothamnium* *Ephelota* *Acineta*	All stages	Respiratory and locomotory difficulties, loss of appetite, sometimes form fuzzy mat on shell	Chloroquin diphosphate, 1.1 pmm, 2 days For *Zoothamnium:* formalin, 50-100 ppm, 30 min. For *Epistylis*: formalin, 30 ppm, for juveniles but not for larvae and post-larvae	Villaluz 1975 Liao et al 1977 Gacutan et al 1977 Chen 1978 Gacutan 1979 Vicente and Valdez 1979 Vicente et al 1979 Yang 1979 Ruangpan 1982 Liao and Chao 1983 Liao 1984 Baticados et al 1986
Gregarine disease	Gregarines	Larvae	Interferes with particle filtration to hepatopancreatic duct or through gut, rate of infection up to 94%	Eliminate molluscan intermediate host	Gacutan 1979 Ruangpan 1982 Baticados 1984
Microsporidiosis	Microsporidia	Female spawners	Whitening of ovaries, microsporidian spores, and other stages of parasite replaced ovarian tissues	Isolation and destruction of infected individuals, disinfection of culture system with commercial bleach or disinfectants with iodine	Villaluz 1975 Baticados 1984 PCARRD 1985

Dinoflagellates

A major cause of loss in prawn hatchery production in Singapore is the red vein disease of postlarvae caused by a peridinian dinoflagellate related to *Haematodinium* sp., a crab pathogen in temperate waters (Chong and Chao 1986). Moribund prawns are luminescent at night and microscopic examination revealed the presence of numerous red-pigmented microorganisms filling the entire ventral sinus and hemolymph spaces as well as spreading to various appendages (Chong and Chao 1986).

In another case in Taiwan, ponds with prawn juveniles infested with *Epistylis* were characterized by brownish water resulting from the presence of the dinoflagellate *Pyrocystis* sp. and the diatom *Pleurosigma* (Chen 1978).

Algae

Pond-grown prawn may sometimes exhibit ectozoic algal growth resulting in sluggishness, inactivity, or during heavy infestation, death (Liao et al 1977, Liao 1984). Also found on mysis and postlarvae in hatcheries is the algal species *Schizothrix calcicola* (Gacutan et al 1977) which attaches to the prawn and does harm when present in large numbers.

Helminths

An unidentified nematode has been implicated in many mass mortalities among postlarvae. In one case, the nematode was found in low numbers in PL_5 and PL_8 and mass mortality occurred after two days in indoor tanks (Gacutan et al 1977). In another case, nematode infestations caused heavy mortalities among M_{II} and M_{III} within two days following use of non-chlorinated water (Baticados, unpubl.). Nematode insfestations have also been observed in Indonesia, and Malaysia (Baticados, unpubl.).

A nematode has also been found in the gut of adult *P. monodon* caught in Andaman Sea (Ruangpan 1982) but no apparent effect on the prawn was reported.

Copepods

A crustacean ectoparasite, *Caligus epidemicus*, was observed to parasitize the pleopods and tail of 72% of adult *P. monodon* in a Thai farm (Ruangpan 1982). The rate of parasite burden, however, was quite low (Ruangpan 1982).

NUTRITIONAL, TOXIC AND ENVIRONMENTAL DISEASES

Prawns may be adversely affected by deficiencies in their diet, toxins or pollutants in the water, or other environmental factors like temperature extremes and oxygen depletion. Their responses to these factors may vary from slight changes in morphological or histological make-up to mortalities within a few hours.

Chronic Soft-shell Syndrome

One of the disease problems which is largely responsible for low prawn production in ponds is the chronic soft-shell syndrome. The chronic soft-shell syndrome is a condition in which the prawn exoskeleton remains persistently soft for several weeks, thus making the prawn weak, more susceptible to cannibalism, and low-priced upon harvest (Baticados et al 1986). The syndrome affects juveniles and adults of *P. monodon.* The cuticular layers of the hard shell, particularly the exocuticle and endocuticle, are considerably thinner, often have a rough or wrinkled surface and are usually disrupted and separated from the epidermis, while those of the hard shell are thicker, generally intact, and attached to the epidermis (Baticados et al 1987). The condition was found to be caused by nutritional deficiency, pesticide contamination in the water, and poor pond water and soil conditions. Because of the significant differences in tissue calcium and phosphorus levels of soft-shelled prawns from those of hard-shelled ones, Baticados et al (1986, 1987) also believed that mobilization of calcium and phosphorus from the storage organ, the hepatopancreas, to the exoskeleton where they are needed for shell formation and hardening, could have been impaired. The disease also occurs among tank-reared prawns and in ponds in Indonesia, Malaysia, and Thailand (Baticados, unpubl.).

The disease may be prevented or controlled through environmental and dietary manipulation, i.e., by providing favorable pond water and soil conditions for shell formation and hardening and by adequate nutrition. Reversal of soft-shelling, general improvement of shell quality, and best growth and survival rates were obtained after feeding prawn juveniles with frozen mussel meat at 14% of the body weight, compared with those fed 2% and 8%. Some management practices like good water management through regular water change and proper storage or use of good quality feeds could also prevent the occurrence of the chronic soft-shell syndrome (Baticados et al 1986).

Red Disease

Red disease was first reported by Liao et al (1977) among prawn juveniles and adults in Taiwan. Affected prawns exhibited reddish discoloration of the cuticle, gill fouling, internal tissues with foul odor, and pale hepatopancreas and heart which eventually led to heavy mortalities (Liao et al 1977, Liao and Chao 1983, Liao 1984). Lightner and Redman (1985a) much later observed red disease among 3-15 g pond-reared juveniles and 40-60 g wild-caught adults. The same disease was also observed among 55-104 g *P. monodon* in broodstock tanks (Baticados et al, unpubl.) and among 10-15 g prawns in ponds in the Philippines (Gacutan and Billiones, unpubl.), Malaysia, and Thailand (Baticados, unpubl.). Aside from the reddish bodies, the prawns also had reddish gills and pleopods (Lightner and Redman 1985a, Baticados et al, unpubl., Gacutan and Billiones, unpubl.) In ponds, juveniles stocked at PL_{40} to PL_{50} exhibited reddening about a week after stocking, became very sensitive to handling stress, and showed gradual mortalities in a period of three months, eventually reaching 98% (Gacutan and Billiones, unpubl.). Histopathological examination of the samples revealed atrophy and necrosis of hepatopancreas, intense cellular inflammatory response, and less commonly, melanized cellular inflammatory lesions in antennal gland, mandibular organ, midgut, and gills (Lightner and Redman 1985a). The degree of reddening of the prawn broodstock appeared to be directly related to the degree of atrophy and necrosis of the hepatopancreas (Baticados et al, unpubl.). Destruction of the hepatopancreas resulted in the release, distribution and deposition of the stored carotenoids into the tissues which then become reddish (Lightner 1985). It is believed that the disease

might be due to microbial toxins (e.g., mycotoxins) in rancid or spoiled diets or in detritus of ponds rich in organic matter (Liao et al 1977, Lightner and Redman 1985a). Gacutan and Billiones (unpubl.) also observed unusually high inputs of lime during pond preparation (4 tons/ha) and very high levels of carbon dioxide (30-60 ppm) in affected ponds.

Fatty Infiltration of the Hepatopancreas

In Texas, Mexico, and Hawaii, prawn juveniles and adults, particularly those which were extensively cultured, exhibited excessive deposits of lipid in the hepatopancreatic tubule epithelium (Lightner 1983). It was believed that this disease might have developed from improper dietary lipid levels, improper caloric-lipid balance, or dietary toxins (Lightner 1983).

Blue Disease

The "blue disease" or the soft blue shell syndrome (Aquacop 1984b) was first observed in 1978-79 when prawn broodstock in Tahiti were affected, resulting in large mortalities (Aquacop 1984a). Affected prawns are lethargic and had a rough cuticle surface and a pale blue discoloration of the often thin and soft cuticle. Both juvenile and adult stages from the ponds are affected (Lightner 1983, Aquacop 1984b, Baticados 1988). The disease was thought to be mainly due to a nutritional deficiency but a probable viral etiology was not discounted (Lightner 1983, Aquacop 1984b). Blue prawns have been found to have very low levels of the carotenoid astaxanthin, the major pigment responsible for the color in prawns (Chiu, pers. comm.). Histological observations on the hepatopancreas of blue prawns also revealed a disruption of the hepatopancreatic tubules (Fernandez and de la Cruz, pers. comm.). Bluish discoloration has likewise been observed in Indonesia, Malaysia, Taiwan and Thailand (Baticados, unpubl.).

To control "blue disease" among breeders, Aquacop (1984a) instituted new procedures for broodstock production from postlarvae based on the principles of low density, high quality food, and frequent change of pond water.

Cramped Tails

Cramped tails or "body cramp" has been reported in Taiwan (Liao et al 1977, Liao 1984) but also occurs in the Philippines (Baticados 1988), and Indonesia (Baticados, unpubl.). Juveniles and adults exhibit flexed, rigid abdomen which may be due to a sudden increase in temperature, e.g., during handling of prawns in air relatively warmer than the rearing water (Liao et al 1977).

Prawns which have just had "body cramp" could still recover by putting them back into well-aerated culture water and straightening the bodies back to their normal shape, which is practical only for the more expensive stages, like spawners or experimental animals (Baticados 1988). Handling of prawns during hot weather conditions should be avoided to prevent the disease.

Hemocytic Enteritis

Blooms of blue-green algae belonging to the family Oscillatoriaceae caused hemocytic enteritis in primarily young juveniles as well as subadult prawns in the Philippines (Lightner 1985). The algal endotoxin released in the gut resulted in necrosis and considerable hemocytic inflammation of mucosal epithelium of the midgut and its caeca as well as necrosis and degeneration of hepatopancreas. Deaths were due to osmotic imbalance, poor absorption of nutrients, or secondary bacterial infections (e.g., *Vibrio alginolyticus*) in septic hemolymph (Lightner 1985).

Growth of the filamentous blue-green algae may be prevented by "shading out" these benthic mats with typical plankton blooms maintained at sufficient density and water depth (Lightner 1988a).

Heavy Metal Poisoning

The presence of heavy metals in the water was proven to be harmful to 2.3-cm long *P. monodon* in bioassay experiments (Chen 1979). Among the metals tested (mercury, copper, cadmium, and zinc), mercury was the most toxic followed by copper, cadmium, and zinc. Cadmium toxicity was found to be the most rapid (Chen 1979).

In a separate case, cadmium and copper poisoning were suspected to be the cause of mortalities in hatchery farms in Taiwan in 1980-81 (Kuo et al 1984). The heavy metals came from the waste water discharged by nearby industries. Signs of poisoning in protozoea and mysis included the presence of a fog-like substance on the dorsal surface of the prawn body, enlarged and laterally curled carapace, irregular chelae and palps, loss of appetite and swimming ability, presence of bubble-like processes on uropod edges which became orange swellings when prawns got worse, and sometimes, red spots on the body (Kuo et al 1984). Mortalities occurred within 6 h to 2 days after signs were observed (Kuo et al 1984). Heavy metals generally damage gill tissues and inhibit the enzyme system (Chen 1979). The 48-h LC50's of copper sulfate on $protozoea_I$, $mysis_I$, and postlarva were shown to be 0.225, 0.350, and 0.125 ppm, respectively (Canto 1977).

Mortality rates in larvae poisoned by heavy metals (particularly cadmium and copper) may be reduced by substituting clean sea water for culture (Kuo et al 1984).

Black Gill Disease

The black gill disease in prawns has been reported to accompany many other syndromes such as viral/bacterial/fungal/protozoan infections, black death or shrimp scurvy, and contamination of toxic pollutants like cadmium, copper, potassium permanganate, ozone, ammonia, and nitrate (Lightner 1985). Black gill disease in *P. monodon* takes the form of reddish, brownish to black gills. Adult *P. monodon* reared in ponds in a Thai farm were found to have black gills during fungal (*Fusarium*) infections (Ruangpan 1982). Red discoloration of gills was also manifested in prawns with red disease (Lightner and Redman 1985b). Examination of affected prawns from some farms in the Philippines also revealed that the black gill disease might also be due to *Zoothamnium* infestation as well as to accumulated soil particles in the gills during heavy siltation (Baticados 1988). The disease may cause respiratory difficulties and mortalities. The disease has been observed also in Indonesia, Malaysia and Taiwan (unpubl.).

Avoidance of the primary causes of the disease is the best way to prevent it. Pond conditions must always be kept at the optimum and the exact cause of the disease must be

identified so that proper treatment could be implemented (Baticados 1988).

Muscle Necrosis

All penaeid species may be affected by a condition called "muscle necrosis" which is characterized by whitish opaque areas in the muscles, particularly in the posterior portion of the abdomen (Lightner 1985). The disease has been associated with stressful environmental conditions like low oxygen levels, temperature or salinity shock, overcrowding, and severe gill fouling. The chronic and infected form of the disease when affecting the distal portion of the abdomen is the more commonly observed "tail rot" (Lightner 1985). Prawns with tail rot have tails that are necrotic, eroded, and reddish to black. Tail rot is highly prevalent in intensive culture farms where environmental conditions are often very poor because of very high stocking densities (Baticados 1988). The disease may cause mortalities if large areas of the abdomen are affected (Lightner 1985). Tail rot is a common problem in Indonesia, Malaysia, Taiwan, Thailand (Baticados, unpubl.) and the Philippines (Baticados 1988).

Control of the disease consists of avoiding possible causes and, when already present, improving the environmental conditions (Baticados 1988).

Gas-bubble Disease

Gas-bubble disease may affect all penaeids (Lightner 1988b). The disease is due to supersaturation of seawater with atmospheric gases and oxygen (Lightner 1983). Gas bubbles appear in the gills, under the cuticle (Lightner 1988b) or in the gut and prawns that die from it float (Baticados 1988).

Supersaturation of gases must be avoided and once the disease is present, mechanical aeration should immediately be done to reduce dissolved oxygen levels (Lightner 1988b).

Available reports on these diseases are summarized in Table 5.

Table 5. Nutritional, toxic and environmental diseases of *Penaeus monodon*

Disease	Agent	Stages Affected
Chronic softshell syndrome	Nutritional deficiency; pesticide contamination; poor pond water and soil conditions (high soil pH, low water phosphate, and low organic matter content)	Juveniles, adults
Red disease	Possibly microbial toxins (mycotoxins) in rancid or spoiled diets or in detritus of ponds rich in organic matter	Juveniles, adults
Fatty infiltration of hepatopancreas	Possibly nutritional; improper dietary lipids or caloric lipid balance or dietary toxins	Juveniles, adults
"Blue disease" or soft blue shell syndrome	Possibly nutritional deficiency or viral etiology	Broodstock Juveniles, adults
Cramped tails or "body cramp"	Temperature shock (e.g., handling of prawns in air relatively warmer than culture water)	Juveniles, adults
Hemocytic enteritis	Endotoxin blooms of blue green algae	Young juveniles and subadults
Heavy metal poisoning	Cadmium and copper contamination	Larvae, post-larvae to young juveniles
Black gill disease	Viral/bacterial/fungal protozoan infections; black death; Cd, Cu, $KMnO_4$, ozone, ammonia and nitrate contamination; heavy siltation	Juveniles to adults
Muscle necrosis	Associated with poor environmental conditions like low oxygen levels, temperature or salinity shock, severe gill fouling, overcrowding	Juveniles to adults
Gas-bubble disease	Supersaturation of seawater with atmospheric gases	All stages

Pathology/Signs	Prevention/Control	References
Shell is thin and persistently soft for several weeks; prawns weak, more susceptible to cannibalism	Environmental and dietary manipulation	Baticados et al 1986, 1987
Reddish cuticle, gills and pleopods; gill fouling; internal tissues with foul odor; pale hepatopancreas and heart; atrophy and necrosis of hepatopancreas: mortalities; intense cellular inflammatory response	None reported	Liao et al 1977 Liao and Chao 1983 Liao 1984 Lightner and Redman 1985b
Excessive deposits of lipid in hepatopancreatic tubule epithelium	None reported	Lightner 1983
Blue discoloration of thin, soft cuticle; rough cuticle surface; lethargy	Maintain low stocking density, high quality food, frequent changes of pond water	Lightner 1983 Aquacop 1984a, 1984b Baticados 1988
Rigid flexure of abdomen	Early stages may be reversed by replacing prawn in water and straightening them back to normal; avoid handling during hot weather	Liao et al 1977 Liao 1984 Baticados 1988
Necrosis and hemocytic inflammation of mucosal epithelium of midgut and caeca; necrosis and degeneration of hepatopancreas; deaths due to osmotic imbalance; poor nutrient absorption; and secondary bacterial infections	"Shade out" benthic blue-green mats with typical plankton blooms	Lightner 1985, 1988a
Morphological deformities, gill damage and inhibition of enzyme system, mortalities	Water change	Canto 1977 Chen 1979 Kuo et al 1984
Gills become reddish, brownish to black; respiratory difficulties, mortalities	Avoid causes, maintain good pond conditions	Lightner 1985 Baticados 1988
Whitish opaque areas on abdominal segment, distal portion of abdomen may become infected and develop into tail rot.	Avoid possible causes, improve environmental conditions	Lightner 1985 Baticados 1988
Gas bubbles in gills, under the cuticle or gut; gills appear whitish and prawn exhibits erratic swimming behavior	Mechanical aeration to reduce dissolved oxygen levels once disease is present	Lightner 1983, 1988b Baticados 1988

OTHER ENVIRONMENTAL STRESS FACTORS

Adverse changes in the environment could cause harm to the prawns either directly or indirectly. Ghosh and Nanda (1985) observed that 8-10.5 cm long *P. monodon* subjected to thermal shock (about 10°C higher or lower) suffered cytomorphological changes in the eye and brain which considerably altered their secretory function.

The build-up of organic wastes and toxic gases due to overcrowding (Villaluz 1975) and excess food (Ruangpan 1982) coupled with improper sanitation deteriorate water quality and contribute to mortalities in hatchery (Villaluz 1975) and broodstock (Primavera et al 1978) tanks.

In ponds, heavy rains could lower the salinity of the surface water and cause oxygen depletion on the lower layer because of water stratification. Mortalities may occur because of the benthic nature of prawns in ponds. Turbid water due to suspended mud particles during periods of high temperature could also be fatal to prawns (Villaluz 1975). Poor quality of the pond bottom has also been associated with rolled up gills and carapace (Aquacop 1984).

PROBLEMS AND PROSPECTS

Even with the available information on the diseases of *P. monodon*, many gaps still exist but could be bridged through a more intensive research program. Viral infections, for instance, are difficult to identify particularly in very young stages (larvae) which appear normal but may have latent viral infection. Diagnostic methods must be improved and simplified to facilitate the identification of viral infections. More effective methods of prevention and control are very much needed to combat not only viral diseases, but bacterial, protozoan, fungal, and non-infectious diseases as well.

It should be noted that not all cases of disease outbreaks are reported--one reason why researches on some disease problems are not conducted. A major hindrance is the prawn farmer's or technician's inability to distinguish or diagnose diseases and to prevent or control them. Training programs in aquaculture should include the study of diseases to alleviate this problem. In addition, disease diagnostic centers in prawn

culture areas could facilitate the identification of prawn diseases and recommend preventive and control measures as needed.

LITERATURE CITED

Anderson IG, Shariff M, Nash G, Nash M. 1987. Mortalities of juvenile shrimp, *Penaeus monodon,* associated with *Penaeus monodon* baculovirus, cytoplasmic reo-like virus, and rickettsial and bacterial infections, from Malaysian brackishwater ponds. Asian Fish. Sci. 1:47-64.

Aquacop. 1977. Observations on diseases of crustacean cultures in Polynesia. Proc. World. Maricult. Soc. 8: 685-703.

Aquacop. 1979. Penaeid reared broodstock: Closing the cycle of *P. monodon, P. stylirostris* and *P. vannamei.* Proc. World. Maricult. Soc. 10:445-452.

Aquacop. 1983. Penaeid larval rearing in the Centre Oceanologique du Pacifique. In: MacVey JP, ed. Crustacean Aquaculture. Boca Raton, Florida:CRC Press, Inc.; p. 123-127. (CRC handbook of mariculture; vol. 1)

Aquacop. 1984a. Constitution of broodstock, maturation, spawning and hatching systems for penaeid shrimps in the Centre Oceanologique du Pacifique. In: Aquaculture en Milieu Tropical. Vairao, Tahiti:Aquacop; p. 304-335.

Aquacop. 1984b. Ten years of experimental rearing of penaeid shrimp in Tahiti and New Caledonia (South Pacific). In: Aquaculture en Milieu Tropical. Vairao, Tahiti: Aquacop; p. 446-477.

Bailey TA. 1983. Screening fungicides for use in fish culture: Evaluation of the agar plug transfer, cellophane transfer, and agar dilution methods. Prog. Fish-Cult. 45:24-27.

Baticados MCL. 1984. Parasites of fishes; NACA Fish Health and Sanitation Course Lecture notes. Tigbauan, Iloilo:UNDP/FAO/SEAFDEC/UPV Training Program for Senior Aquaculturists in Asia. 52 p. (AQU-TRAIN/NACA 84-100).

Baticados MCL. 1988. Typical prawn diseases-causes, prevention and treatment. In:Chiu YN, Santos LM, Juliano RO, eds. Technical Considerations for the Management and Operation of Intensive Prawn Farms. Iloilo City: U.P. Aquaculture Society; p. 134-143.

Baticados MCL and Gacutan RQ. 1977. Reduction in *Chaetoceros* populations by furanace. Q. Res. Rep. SEAFDEC Aquacult. Dep. 1(1):36-37.

Baticados MCL, Gacutan RQ, Gutierrez PJ. 1977a. Effects of furanace on *Brachionus.* Q. Res. Rep. SEAFDEC Aquacult. Dep. 1(1) : 34-35.

Baticados MCL, Po GL, Lavilla CR, Gacutan RQ. 1977b. Isolation and culture in artificial media of *Lagenidium* from *Penaeus monodon* larvae. Q. Res. Rep. SEAFDEC Aquacult. Dep. 1(4):9-10.

Baticados MCL, Coloso RM, Duremdez RC. 1986. Studies on the chronic soft-shell syndrome in the tiger prawn, *Penaeus monodon* Fabricius, from brackishwater ponds. Aquaculture 56:271-285.

Baticados MCL, Coloso RM, Duremdez RC. 1987. Histopathology of the chronic soft-shell syndrome in the tiger prawn, *Penaeus monodon.* Dis. Aquat. Org. 3:13-28.

Bautista QM, Jr. 1983. Parasites and diseases: Mycosis of larvae of prawn and their control. Seminar Workshop on Prawn Culture: Hatchery, Pond Cultivation; 1983; Naawan, Misamis Oriental. Naawan, Misamis Oriental: MSU - Institute of Fisheries Research and Development; 34-41.

Bell TA and Lightner DV. 1983. The penaeid shrimp species affected and known geographic distribution of IHHN virus. Rogers GL, Day R, Lim A, eds. Proceedings of the first international conference on warm water aquaculture-crustacea; 1983 February 9-11; Brigham Young University-Hawaii Campus. Honolulu:Brigham Young University-Hawaii Campus; 280-290.

Brock JA, Lightner DV, Bell TA. 1983. A review of four virus (BP, MBV, BMN and IHHNV) diseases of penaeid shrimp with particular reference to clinical significance, diagnosis and control in shrimp aquaculture. International Council for the Exploration of the Sea. C.M. 1983/Gen. : 10/Mini-Symposium.

Canto J. 1977. Tolerance of *Penaeus monodon* larvae to cupric sulfate added in bath. Q. Res. Rep. SEAFDEC Aquacult. Dep. 1(1):18-23.

Chen HC. 1979. Acute toxicity of heavy metals to some marine prawns. China Fish. Mon. (316): 3-10 (in Chinese with English abstract).

Chen HP. 1978. Diagnosis and control of disease of *Penaeus monodon* culture from overwintered juveniles. China Fish. Mon. (309):3-11.

Chong YN and Chao TM. 1986. Septicaemias of marine crabs and shrimp. Maclean JL, Dizon LB, Hosillos LV, eds. The First Asian Fisheries Forum: proceedings; 1986 May 26-31; Manila, Philippines. Manila: Asian Fisheries Society; 331-332.

Colorni A, Samocha T, Colorni B. 1987. Pathogenic viruses introduced into Israeli mariculture systems by imported penaeid shrimp. Bamidgeh 39(1):21-28.

Gacutan RQ. 1979. Diseases of prawns (pests and diseases of sugpo). Technical Consultation on Available Aquaculture Technology in the Philippines; 1979 February 8-11; Tigbauan, Iloilo. Tigbauan, Iloilo:SEAFDEC Aquaculture Department; 170-179.

Gacutan RQ and Baticados MCL. 1979. Notes on *Lagenidium* from larvae of *Penaeus monodon* Fabricius. 1. Isolation and culture in artificial media. Fish. Res. J. Philipp. 4(l):24-38.

Gacutan RQ and Llobrera AT. 1977. Effects of furanace on the zoea and mysis of *Penaeus monodon.* Kalikasan, Philipp. J. Biol. 6(3):263-268.

Gacutan RQ, Llobrera AT, Santiago CB. 1977. Microorganisms associated with *Penaeus monodon* postlarvae and mysis in hatchery tanks. Kalikasan, Philipp. J. Biol. 6(1):77 (Abstract).

Gacutan RQ, Llobrera AT, Baticados MCL. 1979a. Effects of furanace on the development of larval stages of *Penaeus monodon* Fabricius. Lewis DH and Leong JK, comps. Proceedings of the second biennial crustacean health workshop; 1977 April 20-22; Galveston, Texas. College Station, Texas: Sea Grant College Program, Texas A&M University; 231-244. (TAMU-SG-79-114)

Gacutan RQ, Llobrera AT, Santiago CB, Gutierrez PJ, Po GL. 1979b. A suctorean parasite of *Penaeus monodon* larvae. Lewis DH and Leong JK, comps. Proceedings of the second biennial crustacean health workshop; 1977 April 20-22; Galveston, Texas. College Station, Texas: Sea Grant College Program, Texas A&M University;202-213. (TAMU-SG-79-114).

Ghosh PK and Nanda DK. 1985. Effects of thermal stress on the optic cerebral neurosecretory cells of brackishwater prawn, *Penaeus monodon* (Fabricius). Philipp. J. Sci. 114:113-124.

Gopalan UK, Meenashikunjamma PP, Purushan KS. 1980. Fungal infection in the tiger prawn (*Penaeus monodon*) and in other crustaceans from the Cochin backwaters. Mahasagar 13(4) : 359-365.

Hatai K, Bian BZ, Baticados MCL, Egusa S. 1980. Studies on the fungal diseases in crustaceans. II. *Haliphthoros philippinensis* sp. nov. isolated from cultivated larvae of the jumbo tiger prawn (*Penaeus monodon*). Trans. Mycol. Soc. Japan 21:47-55.

Kuo GH, Lin YS, Chen HC, Lo CF. 1984. Diseases and mortalities of cultured marine fish and shellfish in Taiwan. Liao IC and Hirano R, eds. Proceedings of ROC-Japan symposium on mariculture; 1981 December 14-15; Taipei, Taiwan. Pingtung, Taiwan: Tungkang Marine Laboratory;173-192. (TML Conference proceedings; no. 1)

Liao IC. 1984. Status and problems of grass prawn culture in Taiwan. Liao IC and Hirano R, eds. Proceedings of ROC-Japan Symposium on Mariculture; 1981 December 14-15; Taipei, Taiwan. Pingtung, Taiwan: Tungkang Marine Laboratory; 81-98. (TML Conference proceedings; no. 1)

Liao IC and Chao NH. 1983. Hatchery and grow-out: penaeid prawns. In: McVey JP, ed. Crustacean Aquaculture. Boca Raton, Florida: CRC Press, Inc.; p. 161-167. (CRC handbook of mariculture; vol. 1).

Liao IC, Yang FR, Luo SW. 1977. Preliminary report on some diseases of cultured prawn and their control method. JCRR Fish. Ser. 29:28-33. (Reports on Fish Disease Research, I) (in Chinese with English abstract).

Lightner DV. 1983. Diseases of cultured penaeid shrimp. In: McVey JP ed. Crustacean Aquaculture. Boca Raton, Florida: CRC Press, Inc; p. 289-320. (CRC handbook of mariculture; vol. 1)

Lightner DV. 1985. A review of the diseases of cultured penaeid shrimps and prawns with emphasis on recent discoveries and developments. Taki Y, Primavera JH, Llobrera JA, eds. Proceedings of the first international conference on the culture of penaeid prawns/shrimps; 1984 December 4-7; Iloilo City, Philippines. Iloilo: SEAFDEC Aquaculture Department; 79-103.

Lightner DV. 1988a. Hemocytic enteritis (HE) disease of penaeid shrimp. In: Sindermann CJ and Lightner DV, eds. Disease Diagnosis and Control in North American Marine Aquaculture. Amsterdam: Elsevier; p. 89-91.

Lightner DV. 1988b. Gas-bubble disease of penaeid shrimp. In: Sindermann CJ and Lightner DV, eds. Disease Diagnosis and Control in North American Marine Aquaculture. Amsterdam: Elsevier; p. 108-111.

Lightner DV and Redman RM. 1981. A baculovirus-caused disease of the penaeid shrimp, *Penaeus monodon.* J. Invertebr. Pathol. 38 : 299-302.

Lightner DV and Redman RM. 1985a. Necrosis of the hepatopancreas in *P. monodon* and *P. stylirostris* (Arthropoda, Decapoda) with red disease. J. Fish. Dis. 8:181-188.

Lightner DV and Redman RM. 1985b. A parvo-like virus disease of penaeid shrimp. J. Invertebr. Pathol. 45: 47-53.

Lightner DV, Redman RM, Bell TA. 1983a. Infectious hypodermal and hematopoietic necrosis, a newly recognized virus disease of penaeid shrimp. J. Invertebr. Pathol. 42:62-70.

Lightner DV, Redman RM, Bell TA. 1983b. Observations on the geographic distribution, pathogenesis and morphology of the baculovirus from *Penaeus monodon* Fabricius. Aquaculture 32:209-233.

Lightner DV, Redman RM, Bell TA. 1983c. Histopathology and diagnostic methods for IHHN and MBV diseases in cultured penaeid shrimp. Rogers GL, Day R, Lim A, eds. Proceedings of the first international conference on warm water aquaculture-crustacea; 1983 February 9-11; Brigham Young University-Hawaii Campus. Honolulu:Brigham Young University-Hawaii Campus; 291-296.

Lightner DV, Redman RM, Danald DA, Williams RR, Perez LA. 1984. Major diseases encountered in controlled environment culture of penaeid shrimp at Puerto Penasco, Sonora, Mexico. Sindermann CJ, ed. Proceedings of the ninth and tenth U.S.-Japan Meetings on Aquaculture; 1980 May 26-27; Kyoto, Japan; 1981 October 27, Lewes; Delaware, U.S.A. Seattle, Washington: National Marine Fisheries Service, NOAA; 25-33. (NOAA Technical report NMFS, 16).

Lio-Po GD and Sanvictores EG. 1986. Tolerance of *Penaeus monodon* eggs and larvae to fungicides against *Lagenidium sp.* and *Haliphthoros philippinensis.* Aquaculture 51 : 161-168.

Lio-Po GD, Lavilla CR, Llobrera AT. 1978. Toxicity of malachite green to the larvae of *Penaeus monodon.* Kalikasan, Philipp. J. Biol. 7(3):238-246.

Lio-Po GD, Sanvictores MEG, Baticados MCL, Lavilla CR. 1982. In vitro effect of fungicides on hyphal growth and sporogenesis of *Lagenidium* spp. isolated from *Penaeus monodon* larvae and *Scylla serrata* eggs. J. Fish. Dis. 5:97-112.

Lio-Po GD, Baticados MCL, Lavilla CR, Sanvictores MEG. 1985. In vitro effects of fungicides on *Haliphthoros philippinensis.* J. Fish. Dis. 8:359-365.

PCARRD. 1985. State of the Art and Abstract Bibliography of Shrimp and Prawn Researches. Los Banos, Laguna:Philippine Council for Agriculture and Resources Research Development and National Science and Technology Authority. 102 p.

Primavera JH, Borlongan E, Posadas RA. 1978. Mass production in concrete tanks of sugpo *Penaeus monodon* Fabricius spawners by eyestalk ablation. Fish. Res. J. Phil. 3(1): 1-12.

Ruangpan L. 1982. Diseases and parasites of *Penaeus monodon* Fabricius. Thai. Fish. Gaz. 35:385-387.

SEAFDEC. 1977. Hatchery operation in 1977. Tigbauan, Iloilo:Southeast Asian Fisheries Development Center, Aquaculture Department. 18 p.

Sunaryanto A. 1986. Chemical treatment of larval culture: The use of chloramphenicol, sodium-EDTA and malachite green in larval culture of *Penaeus monodon* Fab. Bull. Brackishwat. Aquacult. Dev. Cent. 8(l):25-30.

Vicente HJ. 1981. Preliminary study on the larval rearing of sugpo with emphasis on water management as an economical measure for maintaining water quality and controlling shrimp mycosis. In:PCARRD, 1985. State of the Art and Abstract Bibliography of Shrimp and Prawn Researches. Philippine Council for Agriculture and Resources Research Development and National Science and Technology Authority, Los Banos, Laguna. (Abstract).

Vicente HJ and Valdez FM. 1979. Preliminary observations on *Epistylis* sp. infestation in food organism and shrimp hatchery tanks in MSU-IFRD. Tech. Rep. Mindanao State Univ. Inst. Fish. Res. Dev. (Philippines) 1(1-8): 82-86.

Vicente HJ, Valdez FM, Dejarme SM, Apongan AB. 1979. Notes on the occurrence of undesirable organisms in MSU-IFRD prawn (*Penaeus indicus* Milne Edwards and *Penaeus monodon* Fabricius) hatchery tanks. Tech. Rep. Mindanao State Univ. Inst. Fish. Res. Dev. (Philippines): 87-100.

Villaluz AC. 1975. Diseases and other causes of mortalities of sugpo in the hatchery and in ponds. In: Proceedings of the Seminar of the Fishpond Cooperator's Program on Prawn Culture in Mindanao; 1974; Naawan, Misamis Oriental. 5p.

Williams RR, Bell TA, Lightner DV. 1986. Degradation of trifluralin in seawater when used to control larval mycosis in penaeid shrimp culture. J. World Aquacult. Soc. 17 (1-4): 8-12.

Yang WT. 1979. Shrimp culture research: Indonesia: a report prepared for the Brackishwater Shrimp and Milkfish Culture Applied Research and Training Project. Rome: Food and Agriculture Organization of the United Nations. 40 p.