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OBSERVATIONS ON DISEASES OF CRUSTACEAN CULTURES IN POLYNESIA

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ABSTRACT

First important problems in pathology at the Centre Océanologique
du Pacifique (COP) have been met during larval rearings of penaeid
shrimps and Macrobrachium rosenbergii. In penaeids, heavy mortalities
during the zoea and mysis stages have led to the diagnosis of two main
types of diseases: a bacterial necrosis of the appendages and a fungus
disease. Two preventive treatments with antibiotics during the zoea
stages and a continuous treatment with a selective herbicide (Treflan)
allow a consistent production of 100 post-larvae/liter (80% survival
from the nauplius stage). With Macrobrachium rosenbergii, during the
larval rearing, mortalities of bacterial origin have been recorded in
some experiments. The observation of appendage necrosis allows early
diagnosis and treatment with antibiotics. Chlorination of the water
helps to control the disease.

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During the growth of penaeids, different diseases have been encountered and are briefly described. "White pleura disease" due to a Vibrio sp. is the only one that has produced important mortalities in P. aztecus, P. merguiensis and P. japonicus but does not seem harmful to P. monodon.

These observations are discussed and conclusions about the importance of diseases in crustacean cultures in tropical area are stated.

INTRODUCTION

The goal of the aquaculture team of the Centre Océanologique du Pacifique (Aquacop), built by the Centre National pour l'Exploitation des Océans (CNEXO, France) on the island of Tahiti in 1972, is to develop intensive aquaculture techniques in tropical areas. This implies the control of each step of the rearing cycle to ensure the consistency of results essential to industrial expansion. During the first experiments, mainly concerned with crustaceans, Macrobrachium rosenbergii and different species of penaeids, sudden deaths could not be explained by an incorrect methodology or technical errors. It quickly appeared that the health of the animals was involved and that the methodology had to be improved.

This paper describes pathological problems met during the rearing of Macrobrachium and penaeids, all recently introduced in Polynesia and bred in captivity through one generation or more and the therapeutics that have been used to limit or prevent deaths. Diseases that have involved heavy mortality are the only ones considered in detail; others are mentioned briefly and conclusions are stated about the importance of diseases in crustacean tropical culture.

MATERIALS AND METHODS

Macrobrachium rosenbergii (de Man), Penaeus merguiensis de Man, Penaeus aztecus Yves, Penaeus japonicus Bate, Metapenaeus ensis de Hann and Penaeus monodon Fabricius are reared at the COP and reproduce in captivity (Aquacop 1976, Aquacop 1977a, Aquacop 1977c); Penaeus vannamei Boone and Penaeus stylirostris have been introduced more recently.

Sea-water is pumped from the lagoon of Vairao and has stable physicochemical characteristics all year round: temperature 25 to 29 C, pH 8.2, salinity 35 ppt. For the hatcheries, after adjusting of salinity, water is chlorinated (approximately 1.5 ppm) and dechlorinated by intensive bubbling and natural illumination.

Larvae are reared at high density (100 larvae/liter) in thermostated tanks equipped with a conical bottom. For penaeids, the nauplii are siphoned out of the incubation tank and stocked in treated water tanks. This water is totally changed at the mysis stage and then half of it is renewed each day. Different foods consist of Cylindrotheca (10,000/cc) and Tetraselmis (30,000/cc) algae for zoea and then rotifers (Brachionus plicatilis, 10/cc), Artemia nauplii, Artemia and pelleted food for the next stages. During Macrobrachium larval rearing, water is changed daily and animals are fed with Artemia nauplii and fresh skipjack flesh. There are three different types of grow-out systems: 12 m^2 sandy bottom tank with 100% daily water renewal, 600 m^2 sandy bottom tank with 50% daily water renewal through the sand and 700 m^2 or more, earthen ponds with 15% daily water renewal. Animals are fed exclusively with pellets adapted to their size and species from P_{10}.

Observations are made directly in the tanks or by means of binocular and microscope. The daily check of the larvae includes digestive tract, setae and appendages, to look for external parasites (fungus, bacteria, etc.). In some cases, observations have been complemented by laboratory tests like bacteriological studies of animals and water and ammonia and nitrite analysis as specified in Aquacop (1977c). Total bacterial counts are made on 12 ppt saline agar and bacterial identification from the hemolymph or the muscles of shrimps are made after Lewis (1973).

In each case of mortality, different types of treatments have been successively tested including sanitary precautions to keep high water quality (frequent renewal, filtration, chlorination, etc.), limitation of different kinds of stress (temperature, salinity, manipulations) and use of drugs. Antibiotic and antifungal therapeutics are tried as curative or preventive measures: streptomycin, bipenicillin, erythromycin-phosphate, chloramphenicol, sulfamethazin, sulfamethoxypyridazin, furanace, spiramycin, malachite green. The trifuralin (Treflan), an herbicide reported effective on fungi by Armstrong (1975), was tested. For the larvae, drugs are dissolved in the water and for the adults, they are incorporated in the pellets. Active levels are tested in 50-liter tanks. For the adults, the dosages are those used in piscicultural or veterinary practice. One therapeutic experiment on P. aztecus with white pleura disease was done in six 2 m^2 tanks (control standing water, control flowing water, terramycin, chloramphenicol, sulfamethazin treatments). The 6th tank held P. monodon exclusively fed with P. aztecus which had died with typical symptoms of the disease.

RESULTS

During the larval rearing, three types of diseases leading to mass mortalities were seen in penaeids and one in Macrobrachium.

During the growth, six different types of diseases have been encountered on penaeids only.

Only frequent observations permit detection of the first signs of illness in the larvae. Once they are affected, the situation degrades quickly. Healthy larvae are active, well pigmented with a digestive tract full of food but the check must be focused on larvae with abnormal behavior: weak swimming, abnormal pigmentation (whitish and reddish for the penaeids or bluish for the Macrobrachium), empty digestive tract or broken or deformed setae. The appearance of the first dead larvae means that a large proportion of the animals are already ill.

Deaths Due to Deformed Nauplii (Figure 1)

Disease is often seen among P. stylirostris, P. monodon and P. merguiensis and is seldom seen among P. aztecus and P. japonicus. Some spawners can produce normal eggs at one time and abnormal ones at another. In any spawning, some unfertilized eggs appear showing an anarchic cellular development but no fecundation membrane; if their percentage is low, the hatching is good and the nauplii have long and well-shaped setae; if their percentage reaches 30% or more, the hatching of the fertilized eggs is poor; some of the nauplii are unable to get out of the eggs, and some are weak with twisted setae and swim on the bottom of the tank whereas normal nauplii swim up to the surface of the water. Abnormal nauplii reach the first zoea but subsequently die without taking any food. The forementioned treatments, sanitary precautions and drug therapeutics, were ineffective.

Necrosis of Appendages (Figures 3, 4, 5, 6)

This syndrome occurs in larval and post-larval stages of all the species that have been reared. With the naked eye, necrosis can only be seen on the mysis and the post-larvae which present twisted antennae, deformed abdomen and incomplete molting. Under the microscope, the first trouble can be seen in zoea and it often starts with a liquefaction of the gut contents. Algae are incompletely digested and a green liquid fills the gut instead of a shaped feces as in healthy larvae. Necrosis often starts on an antenna or a newly formed appendage like the uropods in zoea III or the pleopods in mysis. The body of the animal is seldom affected. Necrosis starts at the tip of the appendage which turns to a brownish color and progresses toward the base. Weakened larvae have broken setae, their appendages are opaque, more or less eroded. Dying larvae are often ready to molt or have failed to molt, the old carapace being held back by the necrosed appendages. The attack is more serious if it starts early in larval

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development. Beginning at zoea I, the whole tank is destroyed by zoea II or zoea III in 24 hours. If necrosis starts at zoea III, part of them reach mysis and mortality lasts for a few days. Post-larvae are rather resistant to the disease which at this stage seems to be related to water and food quality.

The first attempted control method was frequent renewal of the water during zoeal stages to keep water quality at its optimum level. Attacks were only slightly decreased. Chlorination or UV treatment of the water were not enough to prevent necrosis and it has been necessary to add antibiotics. Streptomycin-bipenicillin (2 ppm active product, 2 UI/ml), erythromycin phosphate (1 ppm AP), tetracyclin chlorhydrate (1 ppm AP), sulfamethazin (3 ppm AP), furanace (0.1 ppm AP) are all efficient as preventive or curative drugs. The treated larvae recover quickly and at the next molt new appendages regenerate without any trace of necrosis. Soon after the treatment, larvae swim actively and start to eat again. Erythromycin phosphate at 1 ppm AP is new used as a preventive measure from the second day of the zoea I stage since antibiotics seem to be toxic to the nauplii. Treatment is repeated every other day until the post-larval stage is reached.

Fungus Mortality (Figure 2)

All the species reared at the COP are sensitive to this disease but to a different degree. P. aztecus is the most sensitive; even eggs and nauplii are affected, whereas in other species, attacks start on the zoea III or the mysis. P. monodon mysis are also very sensitive. In this case the fungus kill the larvae without developing far and therefore are difficult to see in dead bodies. P. japonicus, on the contrary seems rather resistant. In simultaneous rearing of P. aztecus, P. merguiensis and P. japonicus, only P. japonicus get through; all the others are destroyed. In short, P. aztecus is the most sensitive and then in decreasing order, P. monodon, P. merguiensis and P. japonicus. Infected larvae sometimes become reddish. Infection can start anywhere. Thalli soon replace all the tissues of the larvae which form spore reservoirs. Evolution of the disease is fast (24 hours to 48 hours) especially in young stages. From zoea III and mysis, the disease destroys 10 to 20% of the larvae each day. The disease often starts after a bacterial necrosis attack.

Two genera of fungi are involved in this disease: Lagenidium sp. and Sirolpidium sp. (Bland, pers. comm.). In Lagenidium, the reproductive form is an external tube which gives birth to a sporangium, but in Sirolpidium spores are formed in the mycelium, inside the larvae. Spores are attracted by dead eggs or dead bodies where they settle, germinate and reproduce rapidly. These 2 genera show the same disease signs and react to the same therapeutics. Trifuralin (Treflan) was successfully tested and proved to be efficient on the spores and non-toxic to the larvae. First it was

used at 0.01 ppm, 3 to 6 times a day though dosages 100 times higher
are non-toxic, 0.01 ppm is enough to kill the spores but has no
effect on the thalli which continue to grow and reproduce. As it is
very volatile and drifts away with tank aeration, now 0.005 ml of
Trifuralin (AP) in one liter of water are dispensed drop by drop
during 6 hours and are renewed each morning and evening. Malachite
green is also being tested.

The routine techniques of penaeid larval rearing now include the
use of chlorinated water, preventive antibiotic and antifungal
treatment; with these methods, 80% post-larvae P4 from the nauplii
stage can be produced at 28 C.

Bacterial Necrosis in Macrobrachium Larvae
(Figures 7, 8, 9, 10, 11, 12)

The first visible signs are a bluish color of the larvae, a
slight cannibalistic activity and an incomplete consumption of food.
Swimming is weak and some larvae fall to the bottom of the tank.
Under the microscope, the stomach appears empty. First attacks are
difficult to see being just brown spots on the antennae or on the
newly formed appendages. Next the tissues of the appendage turn
opaque, necrosis develops toward the base of the appendage which may
then be partly or completely destroyed. Different bacterial forms
(filamentous, Leucothrix - like, rods, cocci) can also be seen on
the setae, the appendages and the gills. The occurences are more
serious in the younger larvae. The mortality reaches 100% in 48
hours for stages 4 and 5 but other stages and post-larvae are more
resistant and are able to regenerate necrosed appendages. Detailed
examination of larvae shows necrosis 48 hours before any mortality is
noticed. Deaths are sudden and usually occur at molting. During
simultaneous rearing in several tanks, they were devastated one after
the other, the disease hitting them earlier and earlier (first at
stage 8, then at 6, then at 4) as the disease were propagated from
tank to tank. Table 1 gives the total bacterial counts in different
antibiotic treated and non-treated tanks. Detailed physicochemical
characteristics are given in another paper (Aquacop, 1977b). In one
experiment, a decrease in ammonia concentration in the tank was
noticed a few days before any bacteria could be observed and before
any mortality started. This disease looked so much like the
bacterial necrosis of penaeids that the same antibiotics listed
previously were tested. Tetracyclin chlorhydrate (1 ppm AP),
erythromycin phosphate (0.65 - 1 ppm AP), furanace (0.1 ppm AP), were
effective but a combination of bipenicillin-streptomycin (2 UI/ml,
2 ppm) gave the best results and is now used routinely. Cultures are
treated with this dosage every 3 days as a preventive measure and
with twice the dosage every day as a curative measure if necessary.
Necrotic appendages regenerate at the next molt and larvae get rid of
the epiphytes on their setae. Results of rearing using this
treatment are given separately (Aquacop, 1977a).

Gills Diseases

From time to time prawns with orange or brownish gills can be
seen. The gills themselves are not harmed but they are covered up by
diatoms, filamentous bacteria (Leucothrix-like), filamentous algae
plus fixed and mobile (nematodes) microfauna. This epifauna
disappears at the next molt and seems to be related to an abnormal
increase of the intermolt period. In one case, a proliferation of
protozoan on the gills of P. aztecus and M. ensis causes a chronic
mortality but a change in salinity from 35 to 18 ppt stops it.
Fusarium was seen in the gills of P. japonicus broodstock and produce
chronic mortality. Thalli and spores as described by Egusa et al.
(1974) were observed. Terramycin and furanace treatment did not stop
the mortality and the whole tank died progressively.

Whirling Disease

Whirling prawns were seen in P. merguiensis and P. aztecus tanks.
Some animals would swim, turning on themselves with confused movements
and then die lying on their backs. Distribution of antibiotic food
(spiramycin 1 ppt) stopped the disease. The efficiency of the
antibiotic therapy seems to indicate a bacterial origin.

White Pleura Disease (Figures 13, 14, 15)

In some grow out experiments, P. aztecus, P. japonicus and P.
merguiensis were destroyed by this disease whereas P. monodon reared
in the same pond or fed with dead diseased prawns has never been
affected. For P. japonicus and P. aztecus the first signs of trouble
is abnormal behavior at the surface of the sediment and resting during
the daytime. Gross symptoms are whitish area on the cephalothorax, on
each side of the rostrum and especially on the pleura. The female
thelycum and base of locomotive appendages also turn white. This is
followed by the appearance of a dark area on the cephalothorax-abdomen
junction and sometimes on the base of the gills. Two black nodules
are seen on each side of the digestive tract, behind the heart.

White zones are cellular accumulations just under the shell and
the muscle is not affected. Dissecting the black nodules from the
other tissues is easy but histology shows no particular structure.
Coagulation of the hemolymph is delayed and hemocytes are rare.
Different bacterial forms can be seen in the hemolymph and bacterial
culture shows the constant presence of Vibrio alginolyticus, sensitive
to different antibiotic such as sulfamethazine and chloramphenicol.
Once this disease destroyed a whole pond of P. merguiensis and P.
aztecus in less than a week but chronic mortality has also been
noticed. Table 2 gives the results of an experiment with P. aztecus

showing only slight disease signs. Survival of treated tanks compared to the control shows the efficiency of antibiotics. In a 600 m^2 tank, 25,000 young P. aztecus, P. japonicus and P. monodon were treated with sulfamethazine (3 ppt) pellets as soon as the disease appeared. Mortality was 30 animals per day during the one month treatment and rose to 50 to 100 per day when treatment was stopped. From 10,000 harvested P. aztecus, 16% had gross signs. P. japonicus was also seriously affected, but P. monodon did not suffer any loss.

Muscular Opacity in P. merguiensis

Under certain stresses, white opacity of the tail of shrimp is a common feature. In P. merguiensis an unusual opacity of the tail muscles has been noticed. The pigmentation of the chromatophores turns to a blue-greenish color and the whole shrimp looks milky-green; the tail is soft and the shell poorly calcified. Animals are very sensitive to handling. Microscopic examination never shows microsporida or other anomalies.

Necrotic Antennula in P. vannamei

Several times, adult P. vannamei were found with one antennula necrotic or even completely eroded. This necrosis is characterized by a nauseous smell. Mortality due to this is limited to a few shrimps and no treatment has been attempted.

DISCUSSION

These disease outbreaks need to be placed in their context: an intensive hatchery system, with semi-intensive grow-out experiments in a tropical area. That means that due to the high temperature the natural bacterial population can develop quickly if conditions permit it and be potentially pathogenic. It also means that rearing continues year round without the interruption of a cold season, thereby encouraging the propagation of disease. Moreover, in water with very low natural productivity, animals fed only an imperfectly formulated experimental diet are subject to the appearance of disease.

Spawnings containing a high percentage of unfertilized eggs and abnormal nauplii seem to be associated with broodstock quality. A pathological origin has not been demonstrated but is not excluded. Lagenidium disease has been described in numerous recent papers (Bland et al., 1976); Barkate et al., 1974; Sindermann, 1975). This fungus can affect many marine animals thus multiplying spore reservoirs. As the spores settle preferentially on dead eggs or dead bodies, it is necessary to remove such decaying matter from the rearing tanks. The high sensitivity of P. aztecus to the disease was

also noticed by Cook (1971). Lightner (1975) observed that P. setiferus seems to be resistant from the mysis stage on. There is still doubt concerning the eventual toxicity of Treflan to the eggs and the nauplii stages. But from the first zoea to the post-larvae, it is an economical therapeutic, easy to use, which gives consistent results and no secondary effects. From Bland et al. (1976), malachite green presents most of these characteristics and is also active on the mycelium.

The bacterial origin of larval necrosis in penaeid and Macrobrachium is almost certain, taking into account the efficiency of the different antibiotic treatments. The use of treated water allows rearing to begin in optimal conditions but bacterial proliferation in an intensive larval system cannot be avoided. Antibiotic do not affect the total bacterial population and Hider and Tubiash (1963) have shown that they can even enhance bacterial growth. These authors explain beneficial action of the drugs by the elimination of the pathogen genera. In our experiments total bacteria counts and the appearance of bacterial cultures do not differ significantly in the waters of treated and untreated tanks. Infectivity experiments need to be conducted to study their virulence. Shigueno (1975) reports the destruction of P. japonicus mysis and post-larvae due to a Vibrio. Lucas and Prieur (1974) show the presence of bacteria pathogenic for mollusc larvae in algal culture. Keel, Lightner and Colwell (1975) and Keel et al. (1975) show the presence of potential pathogens in mollusc and crustacean cultures and report that Vibrio parahaemolyticus has been found by different investigators in the food used in penaeid culture. Even with a total daily water exchange, larvae keep the bacteria inside the gut or fixed on the shell; this flora can be harmful if its level is too high. The development of nitrifying bacteria in Macrobrachium rearing tanks when antibiotics were not used and when the tanks were cleaned daily, leads one to think that the only place where the bacteria could grow are the animals themselves. The action of the antibiotic would be then essentially a partial destruction of this flora. The fact that weakened larvae get back to normal activity and show clean appendages in a few hours without molting favors that hypothesis. The rapid propagation of the infection in zoea of penaeids and young stages of Macrobrachium show that the age of the larvae is certainly an important factor in sensitivity to disease. Cellular response to the necrosis is limited to the accumulation of a few hemocytes and cannot stop its progression. Susceptibility of the larvae is also enhanced as soon as the conditions deteriorate (manipulations, overcrowding, ammonia, organic degradation products of the metabolism, etc.). Monitoring the ammonia level may be a way to check the animal's health. A decrease of ammonia excretion is an indicator of a poor larval metabolism which can provoke bacterial necrosis a few days later. High density rearing certainly enhances the risk of epizootics (Mayr et al., 1968) but rearing techniques are conceived to control the main factors and allow intervention if necessary.

Use of antibiotics was considered unavoidable in recent years in most larval rearing (Loosanoff and Davis, 1963; Shelbourne, 1964). Now it seems that with the development of a better methodology, consistent results can be achieved without their use (Dupuy, pers. com.; Flassch et al., 1975; Mock and Murphy, 1970; Girin and Harache, 1976). This has been realized in temperature areas where environmental conditions change throughout the year. In a continuous culture in tropical areas, potential pathogens are always ready to proliferate, so antibiotic prophylactic treatments seem necessary on a routine basis. That involves a better understanding of the antibiotic actions upon larvae and bacteria to define methods for their use. Fisher et al. (1975) note that the use of antibiotics may enhance the proliferation of fungi; this has also been noticed at the COP but the use of Treflan prevents this secondary effect. Chan and Lawrence (1975) have also observed a negative effect of antibiotics on nauplii and young zoea but they were using higher dosages than those used at the COP. However older larvae and post-larvae seem to tolerate these high dosages without trouble. The health of the larval crustaceans appears as a delicate balance between the host, the potential pathogen and the rearing medium. It can be easily upset in intensive systems where potential stresses are numerous and where all the factors which enhance the host metabolism also favor bacterial development. A low density method, using large volumes of water (Shigueno, 1975; Fujimura and Okamoto, 1970), has a possibility of self regeneration but represents an ecological balance specific to each geographic area. It is also interesting to note that the post-larvae produced from antibiotic treated tanks give growth results as good as untreated larvae.

In juvenile and adult, fungic and parasitic diseases have not been important yet. Fusarium has only attacked one tank of P. japonicus while 11 other tanks in the same conditions containing other species were not harmed. Lightner et al. (1975) succeeded in controlling this disease in P. californiensis. At the COP, the appearance of the disease, its regression after changing the food and then the mortality of the shrimp under conditions that are not fully understood, seem to indicate that it is a secondary infection of weakened animals. For the white pleura disease, the three affected species are two temperate species, P. japonicus and P. aztecus and one tropical P. merguiensis, which needs a high protein diet. Aquacop (1976) reported that each penaeid species needs a different diet, especially for protein and fat contents. If these needs are not perfectly satisfied, as is probably the case in a experimental rearing situation, animals will be more sensitive to any potential pathogen. Potential pathogenic bacteria have been found in crustacea considered in good health (Colwell et al., 1975). Lightner and Lewis (1975) reproduced a septicemic syndrome in different species of penaeids with Vibrio alginolyticus but it was necessary to inject rather high concentrations of bacteria. Delves, Broughton and Poupard (1976) observed Vibrio disease in P. merguiensis

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and $P.$ monodon, and considered environmental conditions responsible
for outbreaks. This also seems to be the case for most of the
diseases seen at the COP (white pleura, fusarium, whirling diseases).
Animals which are not in the ranges of their natural conditions or
which are weakened by an inappropriate diet, support no stress and
are susceptible to disease. They die from a secondary infection and
it seems that the efficiency of antibiotics is limited only to animals
still able to react positively. On the contrary, Macrobrachium
rosenbergii, $P.$ monodon and $P.$ vannamei, living in an environment
closer to their natural conditions and accepting low protein food,
have shown no real pathological troubles yet.

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Table 1. - Random total bacterial counts in Macrobrachium larval culture (800-liter tank)

Antibiotic treated tank (colonies per ml)	Non-treated tank (colonies per ml)
400 000	490 000
660 000	450 000
350 000	260 000
	450 000

Table 2. - Mortality data from therapeutic experiments on white pleura diseased P. aztecus (Treatment begins on second day)

Tank no.	1	2	3	4	5
Original number of diseased prawns	13	12	12	12	12
Number of dead prawns on the first day	3	2	2	1	2
Number of prawns actually tested	10	10	10	11	10
Survival after 23 days	1	3	9	6	9
% survival after 23 days	10	30	90	55	90

Tank no. 1 Control: standing water

Tank no. 2 Control: flowing water

Tank no. 3 Pelleted food + Terramycin (1 ppt)

Tank no. 4 Pelleted food + Chloramphenicol (1 ppt)

Tank no. 5 Pelleted food + Sulfamethazin (4 ppt)

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Figure 1.   Deformed Penaeus stylirostris nauplius with abnormal
              appendages and twisted setae.

Figure 2.   Sirolpidium sp. in a zoea III Penaeus merguiensis
              appendage: empty sporangium containing few spores and
              discharge tube.

Figure 3.   Necrosis of Penaeus merguiensis post-larvae: typical
              twisted antennae.
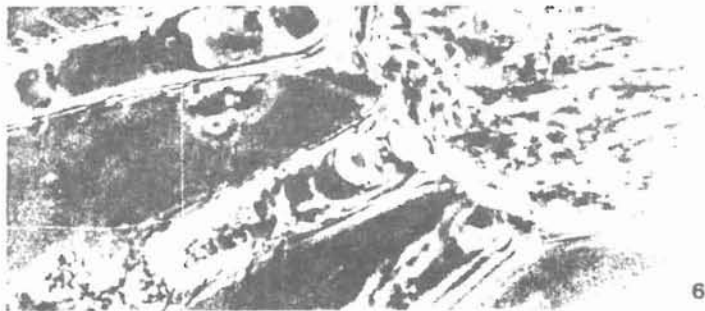
Figure 4.   Necrosis of penaeid larva: the necrosis is limited to
             the superior appendage and the next is ready to molt.

Figure 5.   Necrosis of penaeid larva: starting point at the tip of
             a zoea I antennae.

Figure 6.   Necrosis of penaeid larva: telson with broken setae.

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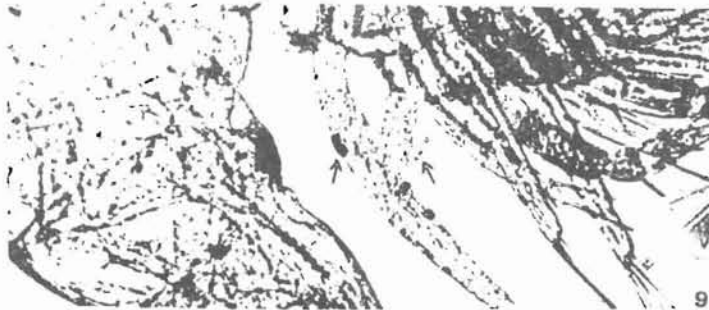
Figure 7.   Necrosis of _Macrobrachium rosenbergii_ larva:
            constriction of the antennal scale.

Figure 8.   Necrosis of _Macrobrachium rosenbergii_ larva:
            starting point in the middle of the antennae with
            filamentous bacteria.

Figure 9.   Necrosis of _Macrobrachium rosenbergii_: starting point
            on thoracic appendages and destroyed appendage.

Figure 10.   Necrosis of _Macrobrachium rosenbergii_ larva: typical
              diseased larva with twisted antenna and necrosed
              appendages.

Figure 11.   Necrosis of _Macrobrachium rosenbergii_ larva: starting
              point on pleopod and completely destroyed pleopod.

Figure 12.   Necrosis of _Macrobrachium rosenbergii_ larva:
              regeneration buds of previously destroyed pleopod after
              antibiotic treatment.

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Figure 13. White pleura disease of penaeid: gross symptoms on juvenile *Penaeus merguiensis*.

Figure 14. White pleura disease of penaeid: detail of diseased *Penaeus aztecus*.

Figure 15. White pleura disease of penaeid: microscopic detail of white pleura tissue.