CHAPTER 1

GENERAL INTRODUCTION

In the context of increasing interest in penaeid prawn culture in coastal waters, establishment of commercial hatcheries for large scale production of seed is being planned by all the maritime states of India. In the hatchery operations, adopting different systems, mortalities (ranging from 1% to 100%) of larvae and postlarvae have been reported frequently. Such mortalities are brought forth by several biotic and abiotic factors, among which diseases contribute significantly to the cause of large scale mortality.

Most of the scientific studies on the diseases of marine animals have come forth only during the past four decades. Sindermann (1970) has given an excellent review and a bibliography on the diseases of commercially important marine fish and shellfish. A perusal of this literature reveals that the significant contributions published prior to 1970 on the diseases of crustaceans relate to the works by Reinhard (1956) on parasitic castration and to the accounts by Gordon (1966), Sindermann and Rosenfield (1967) and Johnson (1968). Anderson and Conroy (1968) discussed the role of diseases in the aquaculture of crustaceans.

Several Institutes and workers are actively involved in the investigations on penaeid prawn diseases and this paved the way for

Among the different groups of crustaceans, much emphasis of disease investigations has been on prawns, obviously due to their economic value and demand. Knowledge of the disease of penaeid prawns has been reviewed a number of times within the past 15 years (Overstreet, 1973, 1983; Sindermann, 1974; Johnson, 1978; Lightner, 1977, 1983, 1985; Couch, 1978, 1983). Besides these, the valuable studies by Villela et al. (1970), Barkate (1972), Feigenbaum (1973), Barkate et al. (1974), Johnson (1974a), Lightner et al. (1975), Delves-Broughton and Poupard (1976), Gacutan et al. (1977), Liao et al. (1977), Nurdjana et al. (1977) and Perez Alvidrez (1977) have greatly contributed to the fund of data on the diseases of prawns. While the knowledge on the diseases of crustaceans in general and of penaeid prawns in particular is fairly developed and progressive in the advanced countries and as revealed from the above cited investigations and reviews, the information on the subject from India is limited. Among the earlier works, the most significant contribution to the knowledge of crustacean parasites was by Chopra (1923). Further studies in this field came forth only since
the last decade.

In the following section, an attempt is made to briefly review the most valuable studies carried out on penaeid prawn diseases abroad and in India.

**An overview of the studies carried out abroad**

Viruses, bacteria, fungi, protozoans, trematodes, cestodes, nematodes and parasitic crustaceans cause diseases in penaeid prawns. Apart from these, dietary deficiencies, environmental stress as well as pollution and toxic algal blooms in the water also bring forth diseases.

**Viral diseases**

Six viral diseases have been reported in cultured penaeid prawns and several additional diseases have been noted to have associated with virus-like or rickettsia-like structures. Three baculoviruses namely *Baculovirus penaei*, baculoviral midgut gland necrosis virus (BMNV) and *Peneaus monodon* type baculovirus (MBV), the picorna-like virus, infectious haematopoietic and hypodermal necrosis virus (IHHNV) and hepatopancreatic parvo-like virus (HPV) and a reo-like virus in the hepatopancreas have been recognised to cause disease in cultured penaeids (Couch, 1974; Sano et al., 1981; Lightner and Redman, 1981, 1985b; Lightner et al., 1983a, 1983; Tsing and Bonami, 1987).
The occurrence of baculoviruses has been reported in several penaeids such as *Penaeus duorarum*, *P. aztecus*, *P. japonicus*, *P. setiferus*, *P. vannamei*, *P. stylophorus* and *P. plebejus* cultured on the Northern Gulf of Mexico, the Pacific coast of Central America and New South Wales, Australia (Lightner, 1983; Lester et al., 1987; Momoyama, 1988). MBV has been encountered in *P. monodon* in Philippines, Taiwan, Tahiti, Hawaii, Mexico, Malaysia and Indonesia (Lightner and Redman, 1981; Lightner et al., 1983a; Anderson et al., 1987; Nash et al., 1988) and the BMNV has been reported in *P. japonicus* cultured in southern Japan (Sano et al., 1981). These baculoviruses infect epithelial cells of the hepatopancreas of protozoa through adult life stages and the midgut epithelium of larvae and postlarvae, often resulting in high mortalities. *B. penaei* and BMNV have often caused serious epizootics in the larval and early postlarval stages in the hatcheries (Couch, 1981; Sano et al., 1981). The viral attack on the epithelial cells causes nuclear hypertrophy, proliferation of nuclear membrane, chromatin diminution and nuclear degeneration. In nature, the transmission of *B. penaei* probably takes place by feeding of the infected prawn by the non-infected ones (Couch, 1978) or by waterborne exposure (Sano et al., 1981). Recently Momoyama and Sano (1988) successfully transmitted BMN virus to the mysis larvae of *P. japonicus* exposed to the medium inoculated with the virus. Couch (1976) however was not able to enhance *Baculovirus* prevalence in *P. duorarum* by exposing them to low levels of Aroclor 1254, Mirex or Cadmium.
Infectious hypodermal and haematopoietic necrosis virus (IHHN V) has been reported in P. stylirostris (Lightner et al., 1983b; Bell and Lightner, 1987). Positive IHHN infections have been achieved in juvenile P. aztecus, P. duorarum, P. setiferus and P. japonicus following experimental exposure to IHHNV (Lightner et al., 1985). This viral disease is diagnosed by the presence of eosinophilic inclusion bodies within the nuclei of cuticular hypodermis, haematopoietic or connective tissue cells which are completely destroyed in acute cases.

Parvo-like virus (HPV) was first recognised in P. merguiensis cultured in Singapore and in Malaysia (Lightner and Redman, 1985b). In addition to P. merguiensis, HPV has caused high mortalities in cultured populations of juvenile P. orientalis from Quing dao, Peoples Republic of China, in P. semisulcatus from Kuwait and in P. monodon from Philippines (Lightner et al., 1985). This disease has been diagnosed by necrosis and atrophy of the hepatopancreas, accompanied by the presence of large prominent basophilic, PAS-negative, Feulgen-positive intranuclear bodies in affected hepatopancreatic tubular epithelial cells.

Tsing and Bonami (1987) isolated and characterised a reo-like virus associated with high mortalities in tank-reared P. japonicus in South France. It was found in the cytoplasm of F. cells and R. cells of the hepatopancreatic tubular epithelium, where it formed large cytoplasmic viral inclusion. The disease experimentally transferred by inoculation of new
hosts with purified virus, or by feeding pieces of hepatopancreas from infected shrimp to new hosts.

Bacterial diseases

A number of diseases caused by bacteria have been reported from penaeid prawns. The majority of bacterial diseases are of a secondary etiology (Lightner, 1977). In most of the cases of bacterial infections in penaeid prawns, motile, Gram-negative, oxidase-positive and fermentative rods have been isolated (Barkate, 1972; Lewis, 1973a,b; Lightner and Lewis, 1975; Lightner 1977; AQUACOP, 1977; Zeng, 1986a,b). Most isolates have been *Vibrio* species, usually *V. alginolyticus*, *V. parahaemolyticus* or *V. anguillarum*. Certain other Gram-negative rods including *Pseudomonas* spp. and *Aeromonas* spp. may occasionally be involved in bacterial syndromes in penaeid prawns.

The bacteria affect all the life stages of penaeid prawns (Lightner, 1977). Bacterial infections in prawns are of two types, localized pits in the cuticle (Anderson and Conroy, 1968; Cook and Lofton, 1973; Cipriani et al., 1980) or localized infections in the body and generalized septicaemia (Lightner, 1983). Vibriosis has been implicated as a frequent mortality factor in juvenile and larval penaeid prawns in culture (Sindermann, 1971b; Lightner and Lewis, 1975). The signs of bacterial infected prawns were gradual change from the usual colourlessness to increasing opaqueness of abdominal muscles, prolongation of clotting time of haemolymph and reduction of haemocyte number (Lightner, 1977; Lightner and Lewis, 1975).
**V. parahaemolyticus** is the causative agent of gastroenteritis associated with the consumption of raw sea food during warm summer months (Vanderzant et al., 1970; Thatcher and Clarke, 1968). *V. parahaemolyticus* has caused death of the blue crab (*Callinectes sapidus*) (Krantz et al., 1969) and of the Gulf of Mexico shrimp (*P. azteca*) (Vanderzant et al., 1970). Vanderzant et al. (1970) have reported that addition of 3% inoculum of *V. parahaemolyticus* (24 hr culture in BHI broth) to an aquarium caused the death of the brown shrimp (*P. azteca*) in a few hours.

Other vibrios such as *V. anguillarum*, *V. alginolyticus*, and *V. algosus* have been found to be pathogenic to shrimps (Lightner and Lewis, 1975; Leong and Fontaine, 1979). Most of the strains of *V. alginolyticus* have caused death of all the shrimps tested so far within 24 hr of inoculation (Lightner and Lewis, 1975). Leong and Fontaine (1979) have assessed the virulence of four species of *Vibrio* in penaeid prawn (*P. setiferus*) and reported *V. parahaemolyticus* to be the most virulent species to white shrimp, followed by *V. anguillarum*, *V. algosus* and *V. alginolyticus* in that order. Larval mortalities due to *V. harveyi* and *V. splendidus* have been reported in *P. monodon* hatcheries in many parts of Panay Island, Philippines (Pitago, 1988).

*Vibrio* spp. as well as members of the genera *Beneckea* and *Pseudomonas* with chitinolytic capacities are also responsible for another significant shell disease in the cultured penaeids (Cook and Lofton, 1973).
P. aztecus, P. japonicus and P. merguiensis succumb often due to white pleura disease, whereas P. monodon does not get affected by this disease even if it is reared in the pond containing the bacteria carrying the disease or fed with the infected prawns (AQUACOP, 1977). Recently Takahashi et al. (1984, 1985) have isolated *Vibrio* from the diseased postlarvae of Kuruma prawn *P. japonicus*, and have reported it to be pathogenic as revealed from the inoculation experiments. The efficiency of antibiotic therapy seems to indicate a bacterial origin for an abnormal swimming behaviour seen in *P. merguiensis* and *P. aztecus*, where the prawns whirl with confused movements and then die lying on their backs (AQUACOP, 1977).

Besides the above mentioned bacteria, *Leucothrix mucor* and *Leucothrix* - like filamentous ectocommensal bacteria occur on many species of marine and estuarine crabs, shrimps, prawns, their eggs, and on cultured *Artemia salina* (Johnson et al., 1971; Shelton et al., 1975). *L. mucor* is a saprophyte and does not penetrate the cuticle (Shelton et al., 1975; Couch, 1978). The thick mat formed by filamentous bacteria on eggs and on gills interferes with respiration and other metabolic exchanges. The larvae and postlarvae get entangled with the filaments, which in turn interferes with their normal behaviour and moulting (Nilson et al., 1975; Lightner and Supplee, 1976). This filamentous bacteria appear in culture systems particularly when the stocking density is high, the water is rich with organic substrate and high temperature prevails (Ishikawa, 1966, 1967; Barkate et al., 1974; Johnson, 1974a; Lightner, 1975, 1977, 1978a, 1983;
Fungal diseases

Fungal diseases are very common in penaeid prawns, particularly in larval and postlarval stages. Several species belonging to phycomycetes fungi and a single genus of the imperfect fungi are involved in causing fungal disease in all the life stages of penaeid prawns. Two general types of fungal diseases, systemic mycosis and localized mycosis, occur in cultured penaeid prawns. The systemic mycosis of larval and postlarval penaeids causes severe mortalities in penaeid hatcheries throughout the world (Lightner, 1977; AQUACOP, 1977; Lightner and Fontaine, 1973; Barkate et al., 1974; Bland, 1975). Chytriodinium parasiticum is found to be parasitic on the eggs believed to belong to penaeid shrimps in the Mediterranean region (Cachon, 1968). Lagenidium callinectes and related species including Sirolpidium like fungus belonging to the phycomycetes fungi have been responsible for epizootics in eggs and larvae of cultured penaeid prawns (Cook, 1971; Lightner and Fontaine, 1973; Barkate et al., 1974; Bland, 1974; 1975; Lightner, 1975, 1981, 1983, 1985; Baticados et al., 1977; Gacutan and Baticados, 1979). Other phycomycetes fungi such as Atkinsiela dubia in P. azteca (Lightner, 1983), Haliphoros milfordensis in P. duorarum and P. setiferus (Lightner, 1977; Tharp and Bland, 1977), H. philippinensis in P. monodon (Hatai et al., 1980) and an unidentified phycomycete in P. azteca (Overstreet, 1973) have also been reported.

L. callinectes is apparently a very active pathogen in larvae of the
brown shrimp _P. aztecut_ (Lightner, 1975). It replaces the larval tissues and produces extramtrical germtubes and mortalities may reach 100% within two days (Gacutan and Baticados, 1979; Lightner, 1977). The pathogenesis of the disease has been described in detail by Lightner and Fontaine (1973) and Lightner (1981). _P. aztecut_ is the most sensitive to fungal disease followed by _P. monodon, P. merguiensis_ and _P. japonicus_ in the decreasing order (AQUACOP, 1977). The infection by _Lagenidium_ and _Sirolpidium_ to the larval shrimp occurs through the parent brood stock or through the carrier hosts in the sea water supply, when the fungal zoospore attaches to and encysts in the egg or the larva (Lightner, 1983).

Only one member of imperfect fungus _Fusarium solani_ has been responsible for mortalities in captive populations of several penaeid prawns (Johnson, 1983a). This fungus has been reported from _P. japonicus_ (Egusa and Ueda, 1972; Fukuyo, 1974; Fukuyo and Egusa, 1974; Guary et al., 1974; Hatai et al., 1978; Momoyama, 1987), _P. aztecut_ (Johnson, 1974b), _P. setiferus, P. occidentalis_ (Lightner, 1977), _P. californiensis, P. stylirostris_ and _P. vannamei_ (Lightner, 1975; Laramore et al., 1977; Lightner et al., 1979b). This is an opportunistic pathogen (Lightner, 1981) and has been responsible for mortalities in several species of captive penaeids in North and Central America and Tahiti (Lightner et al., 1975; Lightner, 1977).

Egusa and Ueda (1972) have described a serious disease known as "Black gill disease" in _P. japonicus_ caused by _F. solani_. Lesions in the gills, at the bases of the appendages and on the cuticle are the internal
symptoms of this disease (Lightner, 1981; Egusa and Ueda, 1972; Shigueno, 1975).

The pathogenesis of *F. solani* has been studied in artificially infected penaeid prawn (Lightner et al., 1981). The histopathology of "Black gill disease" caused by *F. solani* in *P. japonicus* has been worked out by Bian and Egusa (1981) while Solangi and Lightner (1976) have studied the cellular inflammatory response of *P. aztecus* and *P. setiferus* to injected suspension of conidia of *F. solani*.

Protozoan diseases

Prawns serve as hosts of symbiotic, commensal, parasitic, and pathogenic protozoans. Sprague and Couch (1971) published an annotated list of protozoan parasites, hyperparasites and commensals of decapod crustacea. A disease, observed by Couch (1978) in protozoal and mysis stage of brown shrimp (*Penaeus aztecus*), is caused by an amoeboflagellate placed in the genus *Leptomonas*. This organism eventually fills the blood spaces and replaces certain soft tissues of the shrimp. It invades the appendages, including eye stalks and eyes (Couch, 1983).

Gregarines are common inhabitants of the guts of wild and pond-reared *P. aztecus, P. duorarum, P. setiferus, P. vannamei* and *P. brasiliensis* (Hutton et al., 1959; Kruse, 1959; Sprague and Couch 1971; Overstreet, 1973, 1978; Feigenbaum, 1975; Johnson, 1978; Couch, 1978). Gregarines were not causing any disease in penaeids even when present in large
numbers in the gut (Johnson, 1978). Two genera, Nematopsis and Cephalobulus have been known from penaeids (Kruse, 1959; Overstreet, 1973; Johnson, 1978; Feighenbaum, 1975).

Microsporidians have caused a characteristic disease called as "Cotton" or "Milk shrimp disease" both in the wild as well as pond cultured prawns incurring considerable loss to the production and value (Kruse, 1959; Overstreet, 1973; Lightner, 1977; Johnson, 1978). Microsporidian infected prawns have distinctly opaque body muscle with dark blue or blackish discolouration due to expansion of the cuticular chromatophores (Lightner, 1983). Incidences of cotton shrimp has been reported in penaeid prawns in different parts of the world (Hutton et al., 1959; Iversen and Manning, 1959; Iversen and Vanmeter, 1964; Baxter et al., 1970; Overstreet, 1973). Four species of pathogenic microsporidian are known to occur in the penaeid prawns: Perezia (=Nosema) nelsoni has been found in the muscle of P. azteceus, P. duorarum and P. setiferus (Sprague, 1950; Hutton et al., 1959; Overstreet, 1973; Couch, 1978; Lightner, 1985); Agmasoma (=Theolohania) penaei, has been found infecting the blood vessels, foregut, hindgut, gonads and occasionally the muscle of P. setiferus (Sprague, 1950; Hutton et al., 1959; Overstreet, 1973; Rigdon et al., 1975); a similar but unnamed species infecting ovaries of P. merguiensis has been described by Baticados (1980); a third microsporidian, Theolohania duorara has been reported to infect muscle, gonads and other organ tissues of P. azteceus, P. duorarum and P. brasiliensis (Iversen and Manning, 1959; Kruse, 1959; Iversen and Vanmeter, 1964; Overstreet, 1973, Iverse et al., 1987) and the
fourth microsporidian, *Pleistophora* sp. and *P. penaei* have been found infecting the different tissues of *P. aztecus*, *P. setiferus* and *P. duorarum* (Baxter *et al.*, 1970; Constrasitch, 1970; Sparague, 1970; Overstreet, 1973). An unrecorded haplosporean has been found in the hepatopancreas of *P. vannamei* (Dykova *et al.*, 1988).

A number of species of protozoan have been reported to cause fouling and/or gill disease in all life stages of cultured penaeids (Overstreet, 1983, Couch 1983; Lightner, 1983). The most commonly reported protozoans include stalked peritrichs such as *Zoothamnium* spp., *Epistylis* spp. and *Vorticella* spp., the loricate ciliate, *Lagenophrys* sp., an undescribed apostome ciliate and the suctorean *Aclineta* sp. (Couch, 1978; 1983; Overstreet, 1978, 1983; Meng and Yu 1980, 1983; Lightner, 1983). These protozoans have been generally found attached on the gills, appendages and body surface of the larval, postlarval, juvenile and adult penaeids in the culture systems and when abundant on the surface of the gills, could cause hypoxia and death (Overstreet, 1973, 1978; Johnson, 1974a; Lightner, 1975, 1977; Lightner *et al.*, 1975; Couch, 1978). Johnson *et al.* (1973) reported the loss of an estimated 2000 numbers of pond held brown and white shrimp in a single day due to the presence of large numbers of *Zoothamnium* sp. on the gills. An unidentified apostome, which caused black gill disease in penaeid shrimp, has been explained by Couch (1978). A pathogenic suctorean, identified as *Ephelota gemmipara*, has been reported in the larvae of *P. monodon* (Gacutan *et al.*, 1979b).
Metazoan parasites

The metazoan parasites of penaeid prawns comprise of helminth parasites such as worms, and bopyrid isopods. Worms that have been found in the prawns are trematodes, cestodes and nematodes which may be found in various parts of the body. Most of the species reported to date, appear to have little effect on individual shrimp infested and probably little significant effect on populations of penaeids (Couch, 1978). Hutton et al. (1959) reported an undescribed species of microphallid trematode metacercariae from pink shrimp. Overstreet (1973) also reported an unidentified microphallid metacercaria from abdominal muscles of white shrimp. Opecoeloides fimbriatus is a very common parasite of penaeids.

The encyst of this parasite is found in hepatopancreas, other internal organs and beneath the exoskeleton of prawns. Prochristianella hispida (=P. penaei) is found mainly in the hepatopancreas of the host (P. duorarum). Kruse (1959) described two other trypanorhynchans pleocercoid larvae from P. duorarum. Hutton et al. (1959), Kruse (1959), Overstreet (1973), Feigenbaum (1975) and Couch (1978) found a small pyriform cestode larval stage commonly in the intestine of penaeid prawns.

Norris and Overstreet (1976) have found that at least two species of Thynnascaris occurred in penaeid prawns of North America. Overstreet (1973) reported two specimens of Spirocamallanus pereirai in the intestine of P. setiferus. Specimens of Leptolaimus sp. and Croconema sp. have been found by Overstreet (1973) in the brown and white prawns. The
bopyrid isopods have been reported to parasitise the brancial chamber of penaeid prawns in nature (Dawson, 1958; Tuma, 1967; Ahmed, 1978; Cheng and Tseng, 1982; Abu-Hakima, 1984). Although the bopyrid infestations have not generally inhibited the growth of the hosts, they have affected the gonadial development, often causing parasitic castration in the hosts (Tuma, 1967; Abu-Hakima, 1984).

Nutritional disease

In addition to the diseases caused by pathogens and parasites, only one nutritional disease syndrome of cultured penaeids has been identified. This disease occurs due to the ascorbic acid deficiency and is popularly known as 'black death disease'. The disease occurs in penaeid prawns which are reared in closed systems, aquaria or flow-through systems in which most or all of the diet is artificial (Lightner, 1977; Deshimaru and Kuroki, 1976; Lightner et al., 1979a). The disease of black death has not been reported in prawns cultured in ponds, tanks or race ways in which there is at least some algal growth (Lightner, 1977; Lightner et al., 1979a).

Prawns affected by black death disease typically display blackened lesions in the stomach wall, the hind-gut wall, in the gills and in the subcuticular tissues at various locations especially at the junction of the body and appendages (Lightner, 1983). The disease has been observed in *P. californiensis*, *P. stylirostris*, *P. azteca* and *P. japonicus* (Deshimaru and Kuroki, 1976; Lightner, 1977, 1983; Lightner et al., 1977, 1979a; Magarelli et al., 1979). Deshimaru and Kuroki (1976), Lightner et al. (1979a) and
Magrelli et al. (1979) have reported that a dietary requirement of 2000 to 3000 mg of the ascorbic acid per kg. of feed is necessary to control the disease.

Diseases caused by environmental stress

Environmental stress such as supersaturation of atmospheric gases low dissolved oxygen levels, sudden temperature or salinity changes, over crowding and rough handling lead to unhealthy state in prawns and in severe cases, lead to large scale mortalities. 'Gas bubble' disease has been reported to occur in penaeid prawns as a result of supersaturation of atmospheric gases, particularly when the dissolved oxygen level reaches or exceeds 250 per cent of the normal saturation of medium (Lightner et al., 1974; Supplee and Lightner, 1976; Lightner, 1983, 1985). The first sign of gas-bubble disease in shrimp is a rapid erratic swimming behaviour, followed by a stuporous behaviour (Lightner, 1983). Examination of fresh preparations of gills or whole tissue under the microscope revealed the presence of gas bubbles (Lightner, 1983). Several other diseases such as spontaneous muscle necrosis (Rigdon and Baxter, 1970; Venkataramiah, 1971a,b, Lakshmi et al., 1978; Nash et al., 1987), cramped tail condition (Johnson, 1975, 1978; Lightner, 1977) and broken back syndrome (Couch 1978) occurred due to changes in environmental conditions. Muscle necrosis was characterised by whitish opaque areas in the striated musculature, especially of the distal abdominal segments (Rigdon and Baxter, 1970). The condition follows periods of severe stress such as over crowding, low dissolved oxygen levels, sudden temperature or salinity changes and rough
handling (Lakshmi et al., 1978). The cramped tail condition appears to be related to sudden increase in the temperature of water and air (Lightner, 1983), while the broken back syndrome which displays a characteristic dorsal separation of the pleural plates covering the third and fourth abdominal segments (Couch, 1978), appears due to a combination of severe salinity, cold temperature and handling stresses.

Mortalities due to toxic agents

A number of algae belonging to the family Oscillatoriaceae have been reported to cause mortalities in cultured penaeid prawns. Blooms of the diatom *Chaetoceros gracilis* have been reported to be toxic to the larval stages of *P. stylostris* and *P. vannamei* (Simon, 1978). Filamentous blue green algae such as *Schizothrix calcicola*, *Spirulina subsalina* and *Microcoleus lyngbyaceus* are also toxic to the cultured populations of *P. stylostris*, *P. vannamei* and *P. californiensis* (Lightner 1978b, 1983; Lightner et al., 1978; Simon, 1978; Lightner et al., 1980). The blooms of blue-green algae have been shown to cause haemocytic enteritis (HE), particularly in juveniles, when necrosis and haemocytic inflammation of the mucosal epithelium of those portions of gastrointestinal tract that lack a chitinous lining occur (Lightner, 1978b; 1983; Lightner et al., 1978). This leads not only to osmotic imbalance and poor absorption of nutrients, but also to secondary bacterial infection (Lightner, 1978b, 1983; Lightner et al., 1978, 1980). The occurrence of a toxicity syndrome called "Blue shrimp syndrome unknown" (BSX) in *P. californiensis* and *P. stylostris* farmed in Mexico (Lightner, 1983) has been correlated with the occurrence of red tides. A
dinoflagellate, *Amphora* sp. may infect the prawn and cause melanisation in the gills (Overstreet and Safford, 1980).

Toxic responses of penaeid prawns to pollutants have been reviewed in depth by Couch (1978, 1979). Organochlorines such as DDT, dieldrin, mirex and PCBS; organophosphates such as baytex, dibrom, malathion and parathion and carbamate such as sevin, have adverse effects on penaeids, usually affecting the physiological processes of hepatopancreas and resulting in death of the animal (Butler, 1966; Nimmo *et al.*, 1970; Lowe *et al.*, 1971; Nimmo *et al.*, 1971a,b; Nimmo and Blackman, 1972; Parrish *et al.*, 1973; Coppage and Mathews, 1974; Couch and Nimmo, 1974a,b; Hansen *et al.*, 1974; Conte and Parker, 1975; Couch, 1978; Schoor and Brausch, 1980). Although the information available on the effects of petroleum products to penaeid prawns is limited, they are known to cause necrotic lesions on the body, gills, lining of the gastric mill and eyes (Mills and Culley, 1971; Anderson *et al.*, 1974; Cox *et al.*, 1975; Yarbrough and Minchew, 1975; Minchew *et al.*, 1979; Neff *et al.*, 1976).

Penaeid prawns are also sensitive to certain heavy metal pollutants. Exposure of prawns to cadmium causes black gill syndrome by impairing the gill cells and consequently leading to the death of the animal (Bahner, 1975; Couch, 1977; Nimmo *et al.*, 1977). Mercuric salts and methylated mercury are extremely toxic with both short term and long term chronic effects to the prawns (Couch, 1978). Mercury is accumulated by prawns and may interfere with their osmoregulatory abilities (Couch, 1978). Nitrogen, which
enters culture systems primarily as organic compounds that are metabolised to ammonia, nitrite, and nitrate by resident culture species and/or bacteria, has also been found to be toxic to cultured crustaceans including penaeid larvae and adult prawns when present in excess (Armstrong, 1979; Chin and Chen, 1987). Nitrite is the most toxic of these three compounds.

Toxic effects of chemotherapeutic chemicals

Certain chemotherapeutic agents, which are used routinely in the treatment of aquatic animals, are found to be toxic to penaeid prawns at certain concentrations (Johnson, 1976a; Hanks, 1976). The optimum exposure time of P. monodon larvae to furanacene has been determined (Gacutan et al., 1979b). Moulting delay and morphological defects have been observed in the larvae of P. monodon resulting from a 24 hour exposure to 1.0 and 2.0 mg/1 furanacene bath (Gacutan et al., 1979a). Schnick et al. (1979) have given a list of chemotherapeutants and anaesthetics with their relative toxicity to crustaceans including penaeid prawns, while Hatai et al. (1974) dealt with the toxicity of a number of fungicides. Lightner (1977) and Lightner and Supplee (1976) have reported that the concentrations of 5 -10 ppm of KMnO4 or 0.5 and 1.0 mg/l of cutrine plus were toxic to P. californiensis respectively.

Miscellaneous diseases

Besides the above mentioned diseases, several other diseases have been reported, these include tumor like growth (Sparks and Lightner, 1973; Lightner et al., 1987b) lymphoma-like neoplasm (Lightner and Brock 1987),
hamartoma (Overstreet and Van Devender, 1978), blisters (Lightner, 1977; Johnson 1978), 'Golden shrimp' (Johnson, 1978; Lightner, 1983), blue or white eye disease (Lightner, 1983), amoebosis of larvae (Laramore and Barkate, 1979), larval encrustation, multifocal opacities (Lightner, 1983), gut and nerve syndrome or GNS (Lightner et al., 1984), deformed nauplii, appendage necrosis in larvae, white pleura disease (AQUACOP, 1977; Lightner, 1983), red disease (Liao et al., 1977; Lightner and Redman, 1985a) nerve disease syndrome (Katzen et al., 1984), aflatoxicosis (Lightner et al., 1982; Wiseman et al., 1982) and fatty acid infiltration of hepatopancreas (Salser et al., 1978; Lightner, 1983).

**Studies carried out in India**

Although information on the capture and culture fisheries of prawns of India and on the biology of economically important penaeid prawns is available from a number of contributions, studies on the diseases of prawns are not many. Chopra (1923) in his excellent monograph entitled "Bopyrid isopod parasitic on India Decapod Macrura", described several bopyrid parasites of *Palaemon* and *Penaeus* along with their geographical distribution and keys for identification. Following this, there have been only occasional and isolated studies on diseases of prawns except one Ph.D. thesis with well documented information on adult prawn diseases by Soni (1986).

Various bacterial diseases such as myxobacteriosis, hemorrhagic septicaemia, vibriosis and enteric bacterial infection have been reported in
penaeid prawns in India (Mahadevan et al., 1978). Among the bacterial diseases, vibriosis caused by 
*V. anguillarum* is the most frequent disease found in *P. indicus* cultivated in the brackish water fields (Mahadevan et al., 1978). Recently, brown spot disease caused by *Vibrio* and *Aeromonas* sp. is also reported in *P. indicus* (Chandramohan et al., 1980; Lakshmanaperumalsamy et al., 1982). The bacterium *Escherichia coli* is found to infect the larvae of *P. indicus* (Mahadevan et al., 1978). The myxobacterial infection caused by *Chondrococcus* sp. is reported in *P. indicus*, *P. monodon*, *M. affinis* and *M. dobsoni* cultured in earthen ponds in the brackishwater areas while *Pseudomonas fluorescence* causing haemorrhagic septicaemia, is encountered mainly in *P. indicus* and *M. monoceros* (Mahadevan et al., 1978). Decay of body surface caused by *Staphylococcus aureus* and *E. coli* in *P. indicus* has been observed by Mahadevan et al. (1978).

Among the diseases caused by fungi, large scale mortality in larvae and juveniles of *P. monodon* raised in the hatchery has been reported due to heavy infection by fungus *Lagenidum* sp. (CMFRI unpublished data). Similarly, the fungi *Saprolegnia parasitica* and *Leptolegnia marina* have been recorded from the juvenile of *P. monodon* caught from the backwaters of Cochin (Gopalan et al., 1980). Five different fungi namely *Saprolegnia* sp., *Achiya* sp., *Aphanomyces* sp., *Pythium* sp. and *Leptomitus* sp. have been reported in the freshwater giant prawn *Macrobrachium rosenbergii* (Shah et al., 1977).
Santhakumari and Gopalan (1980) have reported the protozoan parasites *Zoothamnium rigiduro* and *Stenter coerulens* in *M. monoceros*. Besides these, *Epistylis* sp. together with *Zoothamnium* sp. have been encountered in *P. monodon* causing hypoxia (Issac Rajendran et al., 1982; Venkatesan et al., 1985). Occasionally, these parasites have been found to affect the juvenile prawns in the culture ponds where dissolved oxygen level in pond water decreased to 1.0 ppm due to non-flushing of pond water with tidal water (Issac Rajendran et al., 1982).

The "cotton" or "milk shrimp" disease caused by microsporidian parasites in the natural populations of *P. indicus*, *P. semisulcatus*, *M. monoceros* and *M. brevicornis* caught off Madras, Mandapam, Tuticorin and Cochin has been reported on several occasions (Subrahmanyam, 1974; Thomas 1976; Santhakumari and Gopalan, 1980. Gopalan et al., 1982; Palaniappan et al., 1982; CMFRI unpublished data). Taxonomy, pathogenicity and histopathology of microsporidian parasites have been studied in detail by Soni (1986).

Large number of metacercarian cysts infecting *M. monoceros* inhabiting the Cochin backwater have been reported by Gopalan et al. (1982) and Syed Ismail Koya and Mohandas (1982). Instances of isopod bopyrid parasites infecting the branchial chamber or attaching to the appendages have been reported in *P. indicus*, *P. semisulcatus*, *P. merguiensis*, *P. japonicus*, *Parapenaeopsis stylifera*, *M. monoceros*, *M. dobsoni*, *M.
brevicóñis, M. lysianassa and Palaemon tenuipes from natural population (Chopra, 1923; Menon, 1953; Sawant and Kewalremani, 1964; Thomas, 1977; Soni, 1986).

The ‘soft prawn’ syndrome in P. indicus has been reported (Mahadevan et al., 1978; Rajamani, 1982; Rao, 1983; Soni, 1986, Ramesh, 1988) and being studied at the Central Marine Fisheries Research Institute. This syndrome in cultured prawns is generally encountered during adverse ecological conditions such as low salinities and combinations of higher temperature and salinities. A tumour on the carapace of P. indicus from grow-out ponds of Prawn Hatchery Laboratory at Narakkal has been reported (Soni, 1986).

At the symposium on the diseases of finfishes and shellfishes in India held at the College of Fisheries, University of Agricultural Sciences, Mangalore in 1982, 6 papers relating to the diseases of prawns were presented. Later, the Central Marine Fisheries Research Institute, Cochin, organised a workshop on "Approaches to finfish and shellfish pathology investigation" in January, 1983, where the guidelines for the identification of disease problems and the rational approaches to be undertaken to tackle the same were discussed.

The foregoing review of the literature shows that the studies on the diseases of larvae and postlarvae of penaeid prawns in India are scanty. However this aspect assumes great importance in the context of large scale production of penaeid prawn seed in the hatcheries and their subsequent
culture in nurseries to meet the quality seed requirements for the rapidly expanding prawn culture industry in the country. This thesis, therefore, focuses to identify the diseases encountered in the larvae and postlarvae of penaeid prawns of India, to describe their characteristics and pathological significance and finally attempt on their control measures. The results obtained are presented and discussed.