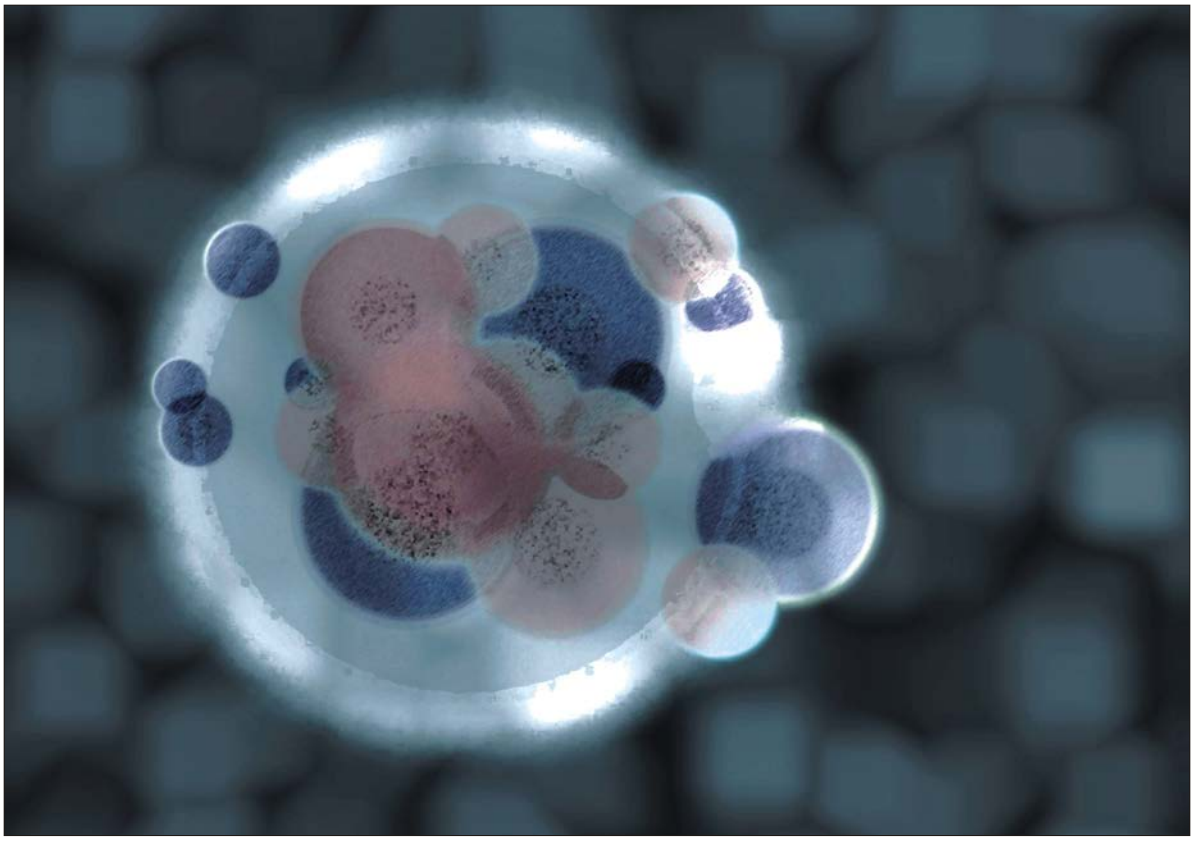

Journal of Biological Sciences

ISSN 1727-3048

science alert

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Journal of Biological Sciences 12 (3): 117-127, 2012
ISSN 1727-3048 / DOI: 10.3923/jbs.2012.117.127

Shell Diseases of Brachyuran Crabs

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Abstract: Shell fishes like crustaceans and molluscs are often prone to shell diseases. Among crustaceans, crabs, lobsters and shrimps are largely infected by infectious pathogens and various environmental stresses. Viruses are commonly seen in shells of shrimps, whereas dinoflagellate is predominant in lobsters. Crabs are infected in higher extend than any other crustacean. Shell disease syndrome in brachyuran crabs is characterized by damaged external manifestation of colored lesions in the exoskeletons. Bacteria (*Vibrio*, *Pseudomonas* and *Aeromonas*), viruses (WSSV), fungi (yeast) and several other pathogens influence the higher percent of shell disease in crabs. Crabs with shell diseases are prone to internal damages causing variation in haemocyte counts and histopathological alteration in internal tissue and organs. Infections in crab may lead to great economic loss. This review discusses about the causes, types, histopathology, genetic variability, hematology, humoral defense mechanism and host susceptibility hypothesis of brachyuran crab shell diseases.

Key words: Shell disease, histopathology, haemocytes, humoral defense mechanism

INTRODUCTION

Brachyurans (Linnaeus, 1758), the true crabs contain more than 6500 species and is the largest clade of the decapods crustacean. The brachyuran crabs have a high scientific and economic value. They also possess anti-microbial, anti-leukemic, anti-coagulant and cardio active properties. They are also used as tools in drug action mechanisms.

Shell disease syndrome is the progressive degradation of the cuticle and is characterized by the appearance of the black and white spot lesions and pale coloration in the exoskeleton surface. The black coloration of the lesions is a result of melanisation, defense response triggered by cuticular damage (Nyhlen and Unestam, 1980). Exoskeleton erosion is largely attributed to the chitinolytic activities of microorganisms. Lesion initiation requires removal of the outermost, non-chitin-containing layer of the cuticle and may occur by lipolytic microbial activities (Cipriani *et al*., 1980), predatory or cannibalistic attacks (Dyrynda, 1998), chemical attack or the abrasive action of sediment or articulated body parts (Young, 1991).

Pathogenic microbes have been identified from different parts of various crustaceans and factors causing damage to crustaceans were also analyzed. Occurrence of pathogenic bacteria in gills, skin and buccal cavity of *Lutjanus agennes, Pseudotolithus elongatus* and *Sphyraena barracuda* were proven (Akinyemi and Buoro, 2011). Presence of bacteria in mud crab *Scylla serrata* from Malaysia was also reported (Najiah *et al*., 2010). Antibacterial activity of brachyuran crabs like *Scylla serrata, S. tranquebarica, Nanosesarma minutum, Neoepisesarma tetragonum, Metapograpsus maculates* and *Macrophthalmus depressus* (Veeruraj *et al*., 2008) and in fishes (Ravichandran *et al*., 2010c) were determined. Krishnika and Ramasamy (2012) have demonstrated that *Artemia* sp. cysts carry pathogenic bacteria like *Vibrio* spp. in their shell and have initiated ways to eliminate them. Nutritional quality (Sudhakar *et al*., 2011) and heavy metal accumulation has also been studied in brachyuran crabs (Kamaruzzaman *et al*., 2011). Very few studies were known to have conducted detailed research on shell diseases caused by chitinolytic bacteria and other pathogenic microbes in these crustaceans especially in brachyuran crabs.

The shell disease may be categorized in to two stages, Firstly, lesion initiation, which involves removal of the externally situated non-chitinous epicuticle, may be the result of abrasive damage (Vogan *et al*., 1999), fighting injuries (Dyrynda, 1998), chemical attack or a bacterial degradation (Cipriani *et al*., 1980). Secondly, lesion development and dissolution of the underlying procuticle is thought to proceed largely via the liberation of extracellular chitinase from epibiotic microorganisms (Stewart, 1993).

The disease is not believed to be fatal in its initial stages but may turn lethal causing adhesion of successive moult shells at lesion sites leading to

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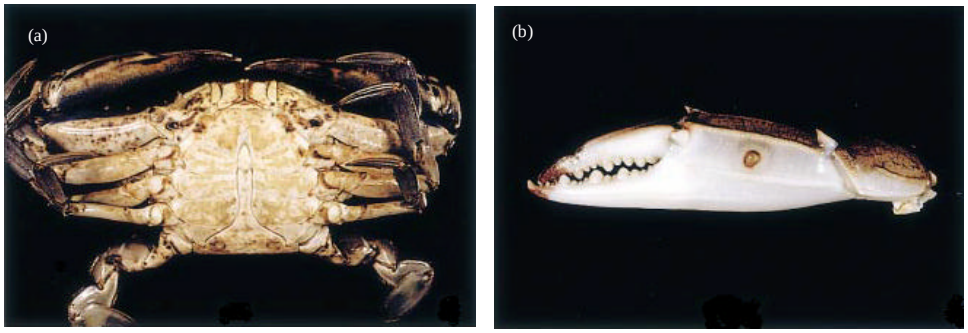

Fig. 1(a-b): (a) Brown melanization in ventral carapace, (b) Ulceration on a chela (Adapted from Noga *et al*., 2000)

incomplete withdrawal from the exuviate at moult. It is suggested by Baross *et al*. (1978) that haemocoelic infections by pathogenic bacteria originating by entry through the lesion site may also lead to death. Though there are numerous studies carried out by many authors related to the shell diseases of brachyuran crabs, there is no cumulative paper on histopathology, genetic variability, hematology and humoral defense mechanism.

CAUSES OF SHELL DISEASES

The incidence of shell disease in natural populations of crustaceans is very low. When cultured together, a high incidence occurs may be due to mechanical damage. The crab shell diseases are associated with infectious agents like bacteria, virus, fungi, dinoflagellates, protistans, ciliates, metazoans and foulers. Majority of them are linked with bacteria, while fungi are occasionally involved in shell diseases (Sindermann, 1989; Prince *et al*., 1993; Hameed, 1994). Parasitic infestations in fin fishes were reported in South-East Coast of India while shell fishes were merely considered for infection. Plenty of works support visible infestation by parasites in fin fishes in India (Ravichandran *et al*., 2010a, 2011; Trilles *et al*., 2011).

Exoskeleton or cuticle of crustaceans is made up of thin proteolipoidal material. Cipriani *et al*. (1980) suggests that lipoidal nature of the epicuticle may induce micro-organisms producing extracellular lipase to initiate lesions. Meyers *et al*. (1987) reported bacteria and unidentified ciliate parasites in the haemolymph and phagocytes of the hepatopancreas of *Hematodinium*-infected crabs. Arcier *et al*. (1999) investigated the occurrence of secondary bacterial infections in invertebrates, particularly for virus-infected crustaceans under aquaculture conditions.

Shell diseases are characterized by various types of erosive lesions on carapace (Johnson, 1983; Sindermann and Lightner, 1988). Black spot and white spot lesions are some of the classic and common shell diseases in brachyuran crabs (Fig. 1) which are characterized by various-sized foci of hyper pigmentation (Rosen, 1970; Noga *et al*., 2000; Vogan and Rowley, 2001; Vogan *et al*., 2002).

TYPES OF SHELL DISEASES

Among crabs of Indian waters *Scylla* spp. are reported to have more infections by bacteria and virus. The most common bacteria causing shell diseases in crabs are *Vibrio* and *Pseudomonas* (Getchell, 1989). A new type of shell disease, rust spot syndrome (Fig. 2) has been reported in *Scylla serrata* (Forskal) in Port Curtis, Queensland (Andersen *et al*., 2000). WSSV (White Spot Syndrome Virus) infects the shells of marine and fresh water crabs equally (Hameed *et al*., 2003; Islam *et al*., 2007). Other pathogens like fungi, dinoflagellate, protistans and foulers are also reported to infect brachyuran crabs (Table 1).

Bacteria: Heavy bacterial growth has been observed both in normal as well as in shell diseased carapace of crabs like *Cancer pagurus* (Linnaeus), *Callinectes sapidus* (Rathbun) (Table 2) and *S. serrata*. Among *Vibrio, Pseudomonas* and *Aeromonas*, Vibrios are predominant in high salinity sites whereas *Pseudomonas* and *Aeromonas* are common in low salinity sites. Natural feed such as trash fish, molluscs, farm waste etc facilitate the entry of microbial pathogens in ponds. Molecular identification of shell disease causing bacteria was done in India in the ecologically important mangrove crab, *Neoepisesarma mederi* and presence of chitinolytic bacteria like *Aeromonas* and *Pseudomonas* were confirmed to be the cause for the shell infection. The disease also caused secondary infection which resulted

Table 1: Characteristic features of shell micro-flora and key references

Shell diseased crab	Disease	Pathogen	Reference(s)
Bacteria			
Scylla sp. (Mud crab)	Bacterial necrosis	*Vibrio* spp. *Pseudomonas* spp. *Aeromonas* spp. *Spirillium* spp.	Lavilla-Pitogo *et al.* (2001)
Cancer pagurus (Shore crab)	Black spot shell necrosis Burn spot disease	Chitinolytic bacteria (*Vibrio*, *Pseudomonas*) Heamocoelic bacteria	Ayre and Edwards (1982), Vogan *et al.* (1999) and Vogan *et al.* (2002)
Cancer irroratus (Say) (Rock crab)	Shell blackening, Carapace erosion	Bacteria	Sawyer (1991) and Young and Pearce (1975)
Callinectes sapidus (Blue crab)	Burnt spot disease, Rust disease	*Vibrio* *Beneckea*	David and Lofton (1973)
Virus			
Scylla serrata and *S. tranquebarica* (Fabricius)	White spot syndrome	White Spot Syndrome Virus (WSSV)	Lo *et al.* (1996), Rajendran *et al.* (1999), Chen *et al.* (2000), Hameed *et al.* (2003), Lavilla-Pitogo and de la Pena (2004), Escobedo-Bonilla *et al.* (2008) and Gopalakrishnan *et al.* (2011)
Podophthalmus vigil (Fabricius)	White spot syndrome	WSSV	Hameed *et al.* (2003)
Portunus sanguinolentus (Herbst)	White spot syndrome	WSSV	Hameed *et al.* (2003)
Philyra syndactyla (Ortmann)	White spot syndrome	WSSV	Hameed *et al.* (2003)
Charybdis annulata (Fabricius)	White spot syndrome	WSSV	Hameed *et al.* (2003)
Charybdis lucifera (Fabricius) and *Charybdis natator* (Herbst)			
Paratelphusa hydrodomous and *P. pulvinata*	White spot syndrome	WSSV	Hameed *et al.* (2001)
Fungi			
Cancer pagurus and *Necora puber*	Secondary infection	Yeast like organism	Stentiford *et al.* (2003)
Dinoflagellate			
Chionoecetes opilio and *Chionoecetes bairdi*	Bitter Crab Disease (BCD)	*Hematodinium* sp.	Gottfried *et al.* (2003), Meyers *et al.* (1990) and Eaton *et al.* (1991)
Callinectes sapidus	BCD	*Hematodinium perezi*	Messick (1994) and Shields and Squyars (2000)
Cancer pagurus	Pink Crab Disease (PCD)	*Hematodinium* like parasite	Stentiford *et al.* (2002)

Table 2: Bacteria isolated from carapace of normal and shell diseased blue crab, *Callinectes sapidus* (Modified from Noga *et al.*, 2000)

Bacteria from shell lesion	Bacteria from normal shell
Achromobacter xylosoxidans	*Achromobacter xylosoxidans*
Acinetobacter calcoaceticus	*Acinetobacter calcoaceticus*
Aeromonas punctata	*A. calcoaceticus*
Aeromonas sobria	*A. calcoaceticus*
Plesiomonas shigelloides	*Aeromonas hydrophila*
Pseudomonas acidovorans	*Aeromonas punctata*
Pseudomonas alcaligenes	*Escherichia coli*
Pseudomonas pntrefacieus	*Plesiomonas shigelloides*
Pseudomonas sp.	*Pseudomonas alcaligenes*
Pseudomonas testosteroni	*Pseudomonas cepacia*
Serratia sp.	*Pseudomonas putrefacieus*
Vibrio alginolyticus	*P. vesicularis*
V. mimicus	*Vibrio mimicus*
V. parahaemolyticus	
V. vulnificus	

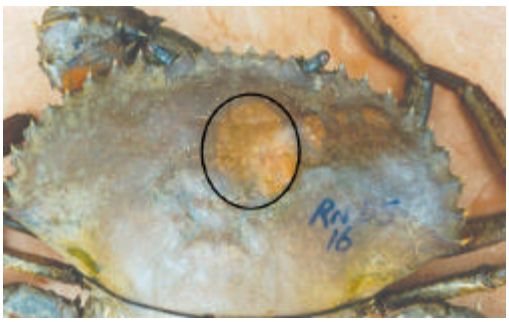

Fig. 2: Carapace of an adult female crab with rust spot lesion (circled) (Adapted from Andersen *et al.*, 2000)

in abnormality in haemocytes count, proximate composition and histopathology of the animal (Sharmila, 2011).

Virus: WSSV (White Spot Syndrome Virus), TSV (Taura Syndrome Virus), YHV (Yellow Head Virus) are the most commonly described viruses affecting the shells of crustaceans. Brachyurans with only White Spot Syndrome Virus (WSSV) are identified. Secondary bacterial infections are also seen in virus infected crustaceans.

Fungi: Dinoflagellate (*Hematodinium* sp.) infection causing Pink Crab Disease (PCD) in *Cancer pagurus* and *Necora puber* (Linnaeus) suffered with

a systemic co-infection of yeast like organism (Stentiford *et al*., 2003).

Dinoflagellate: Infection of *Hematodinium*, a Dinoflagellate parasite is seasonal and also amplified by abiotic factors. Infection peaks result in sporulation (Stentiford and Shields, 2005; Hamilton *et al*., 2009). Bitter Crab Disease (BCD) caused by the dinoflagellate, *Hematodinium* sp. is predominant in snow crab, *Chionoecetes opilio* (Fabricius) (Gottfried *et al*., 2003) and tanner crabs *Chionoecetes bairdi* (Rathbun) in the subarctic waters of Newfoundland and the Bering Sea (Meyers *et al*., 1990; Eaton *et al*., 1991). The disease is also seen in some populations of blue crab *Callinectes sapidus* by *H. perezi* and occurs well after main moulting period (Messick, 1994; Shields, 1994; Shields and Squyars, 2000; Messick and Shields, 2000). Pink Crab Disease (PCD) caused by the *Hematodinium* like parasitic dinoflagellate in *Cancer pagurus* from Ireland and from the English Channel and is characterized by hyper-pigmentation of carapace and appendages (Stentiford *et al*., 2002). *Hematodinium* infection was also detected in *Cancer pagurus*, *Carcinus maenas* (Linnaeus), *Liocarcinus depurator* (Linnaeus) and *Necora puber* from the Clyde Sea in Scotland (Hamilton *et al*., 2009).

Foulers: Carapace fouling organisms are identified in the exoskeleton of *Cancer pagurus* and are described by Holt (1890). Several species of spirorbid polychaete (*Janua pagenstecheri, Spirorbis rupestris* and *S. tridentatus*) have also been described as inhabiting the external carapace of *C. pagurus* (Knight-Jones and Knight-Jones, 1977). Several cirripede crustacean species (barnacles) like *Balanus balanus* (Linnaeus), *B. crenatus* (Bruguiere), *Chelonibia patula* (Ranzani), *Chirona hameri* (Ascanius), *Elminius modestus* (Darwin) and *Verruca stroemia* (Muller) inhabit the carapace of intermoult *C. pagurus* (Richard, 1899; Heath, 1976). Molluscs are identified on the carapace of *C. pagurus* which include the saddle oyster *Anomia sp.* and the common whelk *Buccinum undatum* (Renouf, 1932; Ingle, 1996). Renouf (1932) and Ingle (1996) has explained about bryozoan infestations by *Scruparia chelata* in *C. pagurus*.

HISTOPATHOLOGY

A positive correlation is found between shell disease and degree of infection (Fig. 3). Bacteria are seen prominently within the body cavities of healthy crustaceans (Colwell *et al*., 1975; Sizemore *et al*., 1975; Ueda *et al*., 1993). The existence of bacteria in the body cavity has apparent detrimental effects such as higher percentage of limb loss on the affected part of the animal. Haemolymph infection frequently co-occurs with limb loss and this suggests a potential route for microbial entry. Bacteria can enter in to the haemocoel through breach of the cuticle either at the gill lamella or through other external lesions. Severe internal damage is associated with crabs with higher external damage in regions surrounding the branchial chambers (Vogan *et al*., 1999; Vogan and Rowley, 2001). Number of free haemocytes vary and decrease dramatically during an infection (Persson *et al*., 1987; Smith and Soderhall, 1983; Smith *et al*., 1984; Lorenzon *et al*., 1999). It is believed that the haemocytes will be continuously produced but at varying rates. Haematopoietic tissue producing new haemocytes has been identified in several crustacean species (Ghiretti-Magaldi *et al*., 1977; Hose *et al*., 1992; Martin *et al*., 1993; Chaga *et al*., 1995). Shell disease (Rust spot shell disease) of non-infectious nature has been

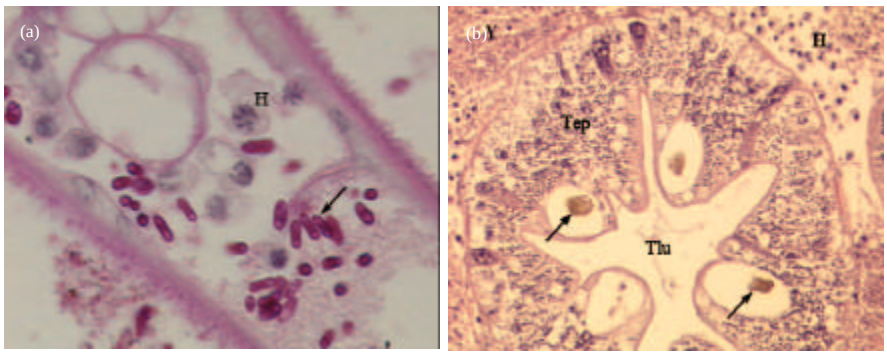

Fig. 3(a-b): (a) Gill lamellae of *Necora puber* with *Hematodinium* species and a yeast like organism, (b) Hepatopancreas of *Cancer pagurus* infected with *Hematodinium* species and a yeast like organism (Adapted from Stentiford *et al*., 2003)

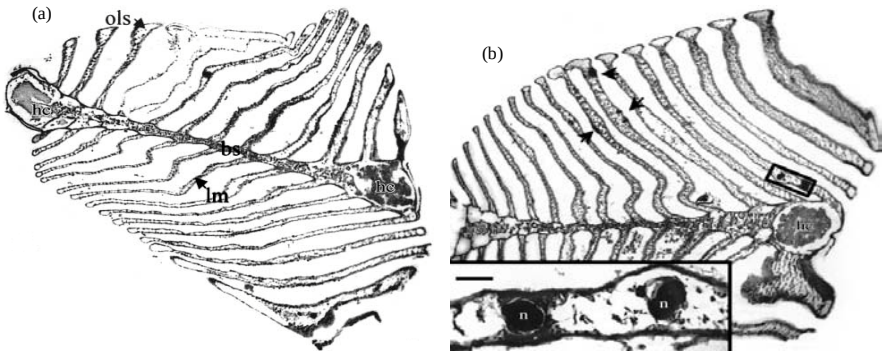

Fig. 4(a-b): Low power micrographs of the gills of *Cancer pagurus*, (a) Gill from an uninfected crab showing haemal channels, (b) Gill from shell diseased crab showing haemocytic (Adapted from Vogan and Rowley, 2001)

reported in *Scylla serrata* from Australia is characterized by irregular lesions and unique histopathological alterations (Andersen *et al*., 2000).

Non-diseased animals have found to have 0.05% of their body surface covered with shell disease lesions (named as superficial minor infections). Distinct histological changes in the gill were observed in crabs with shell disease lesions on their body surface. In particular, lesions appeared on the outer surfaces of the lamellae and haemocyte accumulations (nodules) were observed occluding the haemal sinuses at such sites. All gills from shell disease-affected crabs showed distinct changes to the nephrocytes found in the central blood sinus. These cells were enlarged and filled with brown pigment (Fig. 4). In some cases greater numbers of haemocytes, particularly eosinophilic granular cells, were found in association with the nephrocytes (Vogan and Rowley, 2001).

Shell-diseased animals showed varying degrees of necrosis of their hepatopancreatic tubules. It is reported by Vogan and Rowley (2001) that the degree of tubular damage was not uniform within the hepatopancreas. The sections from individual crabs contained tubules apparently in different stages of epithelial cell breakdown and the hepatopancreas of crabs with greater degree of external lesions was found to contain more damage than that of less affected crabs.

HEMATOLOGY

Vogan and Rowley (2001) showed that the crustaceans with shell disease also possessed damages in internal organs. There was a linear relationship between the bacterial load in the haemolymph and the severity of shell disease. This study (Vogan and Rowley, 2001) also revealed breakdown of the hepatopancreas and damage to the gills associated with nodule formation due to haemocyte clumping. A contrary to this, study of Vogan *et al.* (2002) had no clear effects on haemocytes count, PPO (Prophenoloxidase) activity and no changes were found in the immune parameters of the shell diseased crab.

A dinoflagellate, *Hematodinium perezi* is a significant threat to *Callinectes sapidus* (blue crab) fisheries in high-salinity estuaries. Although the parasite infects male and female crabs, it may have a greater impact on mature females as they move to higher salinities to breed. Haemocyte densities declined precipitously and exhibited differential changes in subpopulations of granulocytes and hyalinocytes in infected blue crabs and lasted throughout the course of infection (Shields and Squyars, 2000).

The haemocyte count can vary greatly in response to infection (Fig. 5), environmental stress and endocrine activity during moulting cycle (Smith and Ratcliffe, 1980; Persson *et al*., 1987; Smith and Johnston, 1992).

GENETIC VARIABILITY

The presence of white spot syndrome virus (WSSV) in two species of freshwater crabs, *Paratelphusa hydrodomous* and *P. puloinata* was confirmed by genetic and histological analysis (Hameed *et al*., 2001). PCR confirmation (single-step and nested) of gills, head tissue, heart, muscle and eye stalk for the WSSV injected Indian crabs (Table 3) was reported by Hameed *et al.* (2003). PCR amplification of the first internal transcribed spacer (ITSI) region of ribosomal DNA and flanking 3 end of the small subunit (SSU) was done by Stentiford *et al.* (2002) which amplified a 680 bp PCR product in crabs with the symptoms of PCD (Pink crab disease) while in others there was no amplification (Fig. 6).

Table 3: Cumulative percent mortality of Indian crabs with White spot syndrome at different time intervals after inoculation (intramuscular injection or oral route) with WSSV and results of PCR analysis (Modified from Hameed *et al*., 2003)

			PCR test				
Species	Days to 100% mortality by injection	Mortality by oral route (%)	G	HT	H	M	E
Calappa philargius (Linnaeus)	4.3	70	+	++	+	+	+
Charybdis annulata	3.5	90	+	++	+	++	+
Charybdis lucifera	4.0	80	+	++	++	+	+
Charybdis natator	-	0	++	++	+	+	+
Podophthalmus vigil	4.0	80	++	++	++	+	+
Portunus sanguinolentus	5.0	70	++	++	++	++	++
Scylla serrata	4.0	90	++	++	++	++	++

G: Gill, HT: Head soft-tissue, H: Heart, M: Muscle, E: Eye stalk, ++: WSSV positive by single-step PCR, +: WSSV positive by nested PCR

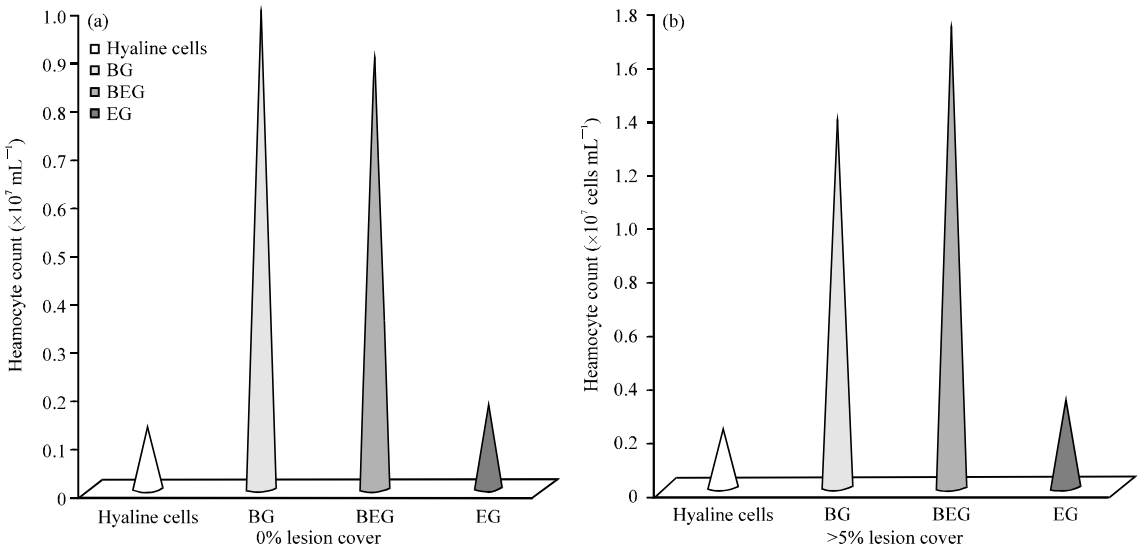

Fig. 5(a-b): (a) Changes in the differential cell count of >5% lesion cover in ventral surface of *Cancer pagurus,* (b) Changes in the differential cell count of 0% lesion cover in ventral surface of *Cancer pagurus* (Modified from Vogan *et al*., 2002)

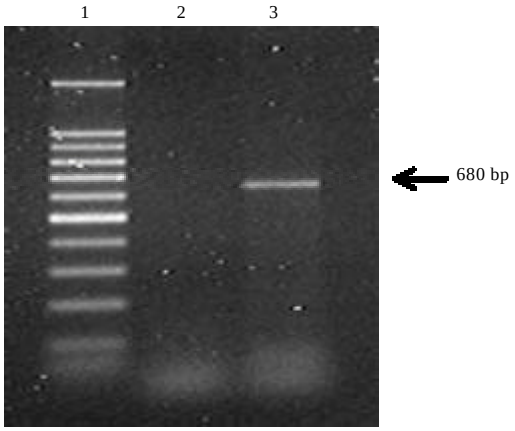

Fig. 6: Agarose gel showing 680 bp amplification product from the hepatopancreas of the crab *Cancer pagurus* exhibiting symptoms of PCD (Pink crab disease) (adopted from Stentiford *et al*., 2002)

The patterns of morphological and genetic variability of many crabs including *Carcinus maenas*, *Scylla serrata* (mud crab) and population studies of species like *Callinectes sapidus* (blue crab) are studied (Brian *et al*., 2006; Fratini and Vannini, 2002). Both genetic and geographical differentiations studies may pave a new way for the identification of the variation in the shell diseased crabs and their genetic impact. The genetic influence of shell disease in brachyuran crabs can be studied for the more confirmation and detailed information of the infection.

PATHOLOGY

Melanization reaction is the defense response triggered by cuticular damage in crustaceans (Lee and Soderhall, 2002). As the disease progresses, degradation extends into the lower layers of the cuticle, the pro-cuticle spreading outwards causing many of the smaller lesions to unite (Smolowitz *et al*., 1992). If the lesions penetrate into soft underlying tissue, secondary infection occurs

through the ruptured cuticle and can result in mortality (Vogan and Rowley, 2001).

Marine crustaceans are reported to have exoskeletons with approximately 70% of the organic fraction being chitin (Brimacombe and Webber, 1964). Hence, microbial chitinases are widely believed to be vital to shell disease lesion progression, at least in the classical form of the disease. Culturable marine microbial chitinoclasts include species within the genera *Vibrio, Photobacterium, Aeromonas, Alteromonas, Pseudoalteromonas, Clostridium, Cytophage* and *Chromobacteria* as well as actinomycetes and fungi (Gooday, 1990; Vogan *et al*., 2002; Costa-Ramos and Rowley, 2004; Bhattacharya *et al*., 2007). Although there are reports of chitinolytic fungal infections of the crustacean cuticle (Noga *et al*., 2000), bacteria are the most commonly cultured microorganisms from the shell disease lesions (Noga *et al*., 2000; Porter *et al*., 2001; Vogan *et al*., 2002; Chistoserdov *et al*., 2005).

EPIDEMIOLOGY

Variation in degree of infection is seen in crabs of natural habitat, impounded population, aquaculture and degraded habitats. Stressors such as inadequate nutrition, temperature extremes are considered to be major cause of shell infection. Another insight of possible importance is that some conditions referred to as shell disease might have a more direct relationship to environmental pollutants and their biochemical effects. Shell diseases are not related to all the effects of chitinolytic microorganisms. Less persistent pesticides like diflubenzuron (Dimlin) could result in contamination of coastal/estuarine waters and damage to shells by interference with chitin synthesis. An example for this is exposure of fiddler crabs, *Uca pugilator* to diflubenzuron showed blackened lesions on regenerating appendages (Weis *et al*., 1987). It is suggested by Young and Pearce (1975) that the lesions reflected a deficiency in chitin formation but they showed a resemblance to shell diseases.

Activity of stressors in the environment especially in high population densities, abnormal temperatures and salinities, presence of toxic chemicals act to disturb metabolic processes and internal defense mechanisms of the species. Lesions most commonly occurred on the dorsal carapace for both sexes. The predominance of lesions to the posterior of the carapace coupled with highest severity in this region suggests that abrasion of the epicuticle may occur as the crab burrows backwards in the sediment. This hypothesis is further supported by the majority of lesions on the walking legs occurring on the ventral surface of the coax. The upper layers of sand sediment are known to support the highest number of chitinoclastic organisms (Hood and Meyers, 1973) and the chitin-rich material is rapidly colonized by microbes (Gooday, 1990).

HUMORAL DEFENSE MECHANISM

The crabs with the shell disease lesions and varying degrees of bacterial septicaemia also displayed differences in the haemogramme, haemolymph phenoloxidase activity, total protein, copper and urea, as well as haemolymph-derived antibacterial activity, compared to uninfected individuals (Vogan *et al*., 2002).

Crustaceans possess one of the more advanced invertebrate immune systems and have both cellular and humoral defense systems. Haemocytes are involved in phagocytosis of external material, confinement of pathogens by clotting, coagulation, hardening (tanning) of the cuticle and encapsulation (Noga *et al*., 2000). Haemocytes are also associated with humoral systems such as the phenoloxidase, prophenoloxidase systems, bactericidins and lectins (Takahashi *et al*., 1995; Kopacek *et al*., 1993). Haemocytes are classified into hyaline, semi-granular and granular cells. Hyalinocytes are found to be associated with initiation of haemolymph coagulation (Aono and Mori, 1996) and phagocytosis in some species (Bell and Smith, 1993). Granulocytes are larger in size and are actively associated with phagocytosis in crustaceans and possess lysosomal enzymes which are important in immune defense system of the host. Drastic reduction in circulating haemocytes maybe indicative of disease, stress or starvation and so the haemocytes can be useful in assessing the overall health of the animal (Noga *et al*., 2000). Antimicrobial lipids from the hemolymph were isolated from brachyuran crabs in India which explains compounds exhibiting immune activity in haemolymph of crabs (Ravichandran *et al*., 2010b).

Humoral defense mechanism of the shell diseased *Cancer pagurus* is reported to be affected. Severity of shell disease in the edible crab, *Cancer pagurus*, correlates with an increase in haemocoelic bacterial infections and may therefore serve as an external marker for the internal health of the animal. A strong correlation was found between the severity of shell disease and a reduction in serum protein, which was further indicative of haemocoelic infection. Levels of copper, urea, phenoloxidase and antibacterial activity in the haemolymph showed no correlations with the proportion of exoskeletal lesion cover. The degree of melanisation of haemolymph samples taken from shell-diseased

individuals was less than that seen in disease-free crabs. Total haemocyte counts were unaffected by the disease, although some minor changes were found in the differential counts. Overall, despite having intra-haemocoelic infections, shell disease-affected individuals few changes in the cellular or humoral defense parameters examined (Vogan *et al*., 2002).

CONCLUSIONS

Crustaceans constitute a major constitute in shellfish culture. Crabs are economically important in several ways. The bio-polymer chitin isolated from carapace of crabs is exported and they support the economic development. They are also useful to humans as most of them are edible. Crabs are the highly infected to cause shell diseases when compared to other crustaceans. Crabs like *Cancer pagurus*, *Scylla serrata* and *Callinectes sapidus* were reported to have more infections when compared to other crabs. This may be due to their mode of living like habitat, feeding and burrowing activities. Bacteria like *Vibrio*, virus like WSSV (white spot syndrome virus) and dinoflagellate like *Hematodinium* are reported to be affected by carapace erosion, pale colouration, hyper pigmentation and spot syndromes. Bacteria are found to cause more infections especially chitinoclastic bacteria which degrade the polymer chitin and result in erosion. This deepens and considerably infects haemolymph and tissues too. Infection in gills seriously affects respiration of the crab. Certain infections are too tedious and may even lead to lethality. The infection is also thought to be caused due to mechanical damage of crabs in densely populated areas, environmental stress like climate and temperature.

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