CHAPTER I

TOXICITY EVALUATION
INTRODUCTION

Environmental pollution has become a major global problem because of an undesirable change in the physical, chemical or biological characteristics of air, land and water, that may or will be harmful to human life or that of desirable species. Whatever may be the mode of contamination, polluted environment is less suitable for existing life forms (Menzel, 1977).

Since the industrial revolution, the efforts of removing man made pollutants from the natural environment have been unable to keep pace with the increasing amount of waste materials and a growing population that further aggravates the situation. This has often resulted in transformation of lakes, river and coastal waters into sewage deposit, where the natural biological balance is severely upset in some cases totally disrupted.

Aquatic pollution from agricultural chemicals can result from wide spread application of highly toxic chemical in agricultural farm, forest, garden etc. In addition to this the promiscuous dumping of industrial, agricultural and domestic pollutants had its impact on water quality, in our major water ways, lakes, ponds and ground water supplies (Kraybill, 1964).
In India pollution problem has noticeably increased after growth of industrialisation and increase in population with diversification of human activities. Marine pollution is observed in limited areas along the coast but increasing inland water pollution is pronounced because the water is used for the discharge of sewage and industrial effluents which contain vast array of inorganic and organic compounds. Many substances in sewage, industrial wastewater and waste from the agricultural run off are toxic to aquatic organisms.

The intrusion of metal contaminants into aquatic system has various sources, like smelting process and fuel combustion via atmospheric fall out effluents, and dumping activities, run off of terrestrial systems and land applications of sewage material and leading of garbage. The amount of heavy metals discharged as industrial and agricultural wastes and sewage effluents have promoted concern of their effects on freshwater aquatic life. The increase of every day discharge of heavy metals in urban environment disturbs the equilibrium state of the aquatic communities.

Heavy metals are most hazardous pollutants because of their nondegradable nature and property to affect all kinds of ecological systems. The salts of metals which find their way into
commercial, industrial application possess certain biocidal properties. The first property of metal is that they are immutable. They can neither be created nor destroyed, nor can one metal be transformed into another. This means that once the metal is metabolised in the environment, its total amount there remains the same regardless of form, until it is immobilized again. Its form may be altered by biochemical processes, so that the particular salt in which it originally enters the environment no longer exists, but total amount of the metal present as other compounds or ions remains unchanged. In the long run, heavy metals may give worst problem, although some of them at low concentrations may be valuable micronutrients and essential for life (Ferrous, zinc, copper, manganese) some metals act as toxins (mercury, silver, cadmium, copper) contrary to general belief, all of the heavy metals have the potential to cause adverse effects on human beings at certain level of exposure and absorption.

Some metals such as copper, zinc can act either as stimulatory or inhibitory depending on their level of availability. Investigations on interaction between trace metals and aquatic organisms have been identified recently because of increased anthropogenic impacts of these metals into aquatic system from highly sophisticated man made industries. Trace metals can exist
in variety of different chemical forms in natural waters including free ions, inorganic complexes, organic complexes and metal absorbed or incorporated into particulate matter. They are added to water system both from man made and natural sources. They may be contributed by soluble materials chemically weathered from soil and rocks due to geochemical alterations. Heavy metals which are selectively concentrated by vegetation may also find their way to surface mainly due to the existence of individual gardens, lawns and their cuttings.

The chemical forms of a metal in aqueous media will depend on both the chemical properties of the individual metal and the chemical composition of natural waters. Recent investigations with copper (Sunda and Guillard, 1975; Andrew et al., 1977; Anderson and Morel, 1978) have demonstrated that the toxicity and bioavailability of trace metals is highly dependent on their chemical form. These investigations have shown that biological response (i.e. toxicity and accumulation) to dissolved trace metal is the function of the free metal ion concentration which is determined not only by the total dissolved concentration, but also by the extent of metal complexion to both organic and inorganic legends.

Most of the toxic metals such as mercury, cadmium, silver, copper and others tend to accumulate in bottom sediments from
which they may be released by various processes of remobilization where they affect aquatic life. The main problem with these heavy metals is that they are very persistent and hence once released into the environment, they remain in the biota far a prolonged period.(Bhattacharya and Sarkar, 1996; Bianchini and Gills, 1996; Perez 1999.)

Various heavy metals mercury, Copper, Cadmium, Lead and Silver are perhaps the most problematic (Mettinen, 1975). All these heavy metals are a part of the earth's crust and hence not unknown to organism. Many of these occur in natural, unpolluted waters, either in trace quantities or in significant concentrations. Human activity however, is largely responsible for altering the entire cycle of these elements in the environment. The geochemical cycles are largely upset as more and more metals are removed from the earth's crust, purified and used in a variety of industrial operations. For example it is said that technological sources inject lead into environment at a rate 100 times the natural rate (Patterson, 1977).

The heavy metal content of aquatic animal, originates from two routes of intake. Free ions which are simple compounds dissolved in water are taken up directly through the epithelium of the skin, gills and alimentary canal, while others being accumulated in food organisms are incorporated by nutrition (Salonki et al., 1982).
Mercury is considered a non-essential but highly toxic element for living organisms. Even at low concentration mercury and its compounds create potential hazards due to enrichment in food chain. The discovery of consumption of high concentration of mercury compounds accumulated in fish and shell fish have evoked disastrous effects in nutritional food chain (Goldwater, 1971).

The toxicity of mercury to marine animals has been extensively studied by some workers (Calabrese et al., 1973). Metal pollution is now not limited to marine habitat but it has spread into inland waters. Recently with the deterioration of water quality of Husain Sagar lake, Hyderabad (A.P., India) contamination of the lake water with heavy metal has been reported. (Seenayya et al., 1985 and Pralhad, 1987).

The mercurial compounds are highly toxic to aquatic animals in comparison to other heavy metals (Bryan, 1971). Acute toxicity of mercuric chloride to marine and fresh water was studied in some species of fishes by a few workers (Akiyma, 1970; Wobeser, 1975; and Dhanekar et al., 1985). V. Balogh and Katalin (1988) made a comparative study on the ability of freshwater mussels and copepod species to accumulate heavy metals like Mercury, Cadmium, Chromium, Lead, Zinc, and Ferrous and observed that Mercury accumulation in musels was higher than that in crustaceans.
Inorganic mercurial fungicides are the most toxic of the fungicides. Mercury ions are toxic to all forms of life when they get accumulated in them. Mercury is presently regarded as a trace element of concern from the stand point of marine environment. Due to its high toxicity and increase in anthropogenic inputs to the environment (National Academy of Sciences, 1978; Young et al., 1979) it raised serious problems. Mass scale human poisoning and subsequent tragic events of Minamata in Japan (Kurland et al., 1960) have drawn wide attention to mercurial compounds and their histological effects. This human poisoning was associated with mercury pollution of the sea water and resultant accumulation in food animals stimulated world wide research in this field. Mercury has been reported to occur at elevated level in certain coastal environments and marine fauna inhabiting mercury contaminated areas have been shown to possess relatively high tissue burden of the metal (De wolf, 1975; Davies and Pirie, 1978).

Among various environmental pollutants, cadmium is one of the heavy metals, merits a special reference as a potentially toxic elements, outbreak of cadmium pollution affecting a large population in Japan (Ministry of Health and Welfare, Government of Japan, 1972) and other parts of the world aroused much concern about the toxicity of this heavy metal.
The use of cadmium in fertilization and pesticides are as well as cadmium from sludge contribute to environmental pollution. Most of it gets deposited in the soil or water. The cadmium deposited in water may increase its concentration in marine food products or be converted to more toxic organic form. In the event of flooding or irrigation, cadmium in water might also increase its concentration in soil which in turn may lead to increase of cadmium concentration in agricultural production. Thus it is evident that mankind is exposed to cadmium via food, water and air.

The survey of literature on cadmium toxicity clearly shows that this heavy metal is implicated as a causative agent of several pathological disorders both in laboratory animals and human beings.

Cadmium, a crystalline silver white, malleable metal is obtained as a byproduct in the refining of zinc and other metals. Although cadmium has been recognized for a relatively short period of time, the environment has been polluted with cadmium for several thousand years. Cadmium and its compounds have been increasing used in industries, causing a sharp increase in environment metal contamination. Some of the compounds are cadmium acetate, cadmium sulfide, cadmium oxide, cadmium carbonate, cadmium chloride and cadmium surface. Of many inorganic
cadmium compounds serveral are quite soluble in water (acetate, chlorides, sulfates), where oxide are insoluble.

Biologically cadmium is neither essential nor beneficial. It can get into environment from smelters from burning of plastics, nickel-cadmium batteries, motor oiled tyres etc. It can also be obtained from food and studies of various investigators in a number of different countries have led to an estimate of 4 to 64 mg/day ingested from food. The concentration of cadmium increase with age. Adults (Aged above 50 years) have from 10 mg to 60 mg, the lowest value occurring in Africa and highest in Japan.

The most serious cases of environmental pollution by cadmium occurred in Japan after world war II. Dr. Noboru Hagino noted a syndrome that occurred in Toyama prefecture, the illness which was named "Itai Itai Kyo" or "Ouch Ouch" disease. This is because of cadmium in the waste of Kamioka mine of them Mitsui mining and smelting company which produces copper lead, zinc. India is also one of the countries which shows marked increase in cadmium levels in industrialized areas especially Ludhiana and Faridabad.

Various sources of water pollution cause untold hazards to several nontarget organisms such as prawns, fishes, frogs, mussels and crabs. Therefore the present investigation was carried
out to study the toxicity evaluation and the effect of heavy metals on the physiology of freshwater crab. *Barytelphusa cunicularis* which is commonly found in the freshwater bodies, especially the Godavari river Paithan, near Aurangabad.
MATERIAL AND METHODS

Freshwater crab *Barytelphusa cunicularis* were collected from Godavari river 50 km. away from Aurangabad city. The animals were collected from downstream of Jaikwadi dam and immediately transported to the laboratory the animals was having habitat in shallow water and getting food as insects from surrounding.

The crabs were brought to the laboratory and kept in the plastic trough with two litre of dechlorinated water. During acclimation animals were fed with earthworms to maintain the healthy conditions. Water from plastic troughs was changed after every 12 hours to get sufficient amount of oxygen for physiological activity.

After acclimatization the animals of equal size and weight were grouped for evaluation of toxicity. Series of static bioassay studies were conducted under laboratory conditions. The crabs were exposed to different concentrations of mercuric chloride and cadmium chloride. Each group was kept in plastic trough containing 2 litre polluted water of definite concentrations. After an interval of 12 hours polluted water in trough were replaced by the same concentration of heavy metal. The resulting mortality was noted
in the trough of 10 to 90% for each concentration for the duration of 24, 46, 72 and 96 hours. Each experiment was repeated thrice to obtain constant results. Similar experiment were performed in summer, monsoon and winter season. Acute toxicity tests were carried out under static condition upto 96 hrs. The data collected was then analysed statistically by means of probit method on transforming toxicity curve (% mortality versus concentrations, Finney 1951) which allows the average lethal concentration or LC<sub>50</sub> value to be calculated for 24, 48 72 and 96 hours. To count mortality, the crabs were confirmed as dead when there was no eyestalk movement and no stimulation. After mechanical disturbances from the number of dead animals.
RESULTS

The acute and chronic studies were carried out in laboratory for 24, 48, 72 and 96 hrs. for two heavy metal mercuric chloride and cadmium chloride. The LC$_{50}$ values were calculated for 24, 48, 72 and 96 hrs. by the method described by Finney (1951) and simplified by Busvine (1971). The results obtained after toxicity evaluation of pollutant to <i>B. cunicularis</i> are summerised in Table 1 and 2 and Figs. 1 to 6.

The percentage mortality of size groups increased progressively up to 96 hrs. in all the concentration of mercuric chloride and cadmium chloride. The LC$_{50}$ values decreased with increasing exposure period showed an inverse relation.

Higher toxicity to both the heavy metal was observed in summer season where as it was lowest in winter the results are listed in Table 1 and 2 in summer season LC$_{50}$ values for 24, 48, 72 and 96 hrs exposure to mercuric chloride are 1.6873, 1.0961, 0.8001 and 0.7043 ppm respectively. LC$_{50}$ values for cadmium chloride 3.5315, 3.0700, 2.5668 and 2.0010 ppm for 24, 48, 72 and 96 hrs. respectively. In monsoon season LC$_{50}$ values for 24, 48, 72 and 96 hrs. exposure to mercuric chloride are 2.0212, 1.7257, 1.0056 and 0.8022 ppm LC$_{50}$ values for 24, 48, 72 and 96 hrs. exposure to
cadmium chloride are 3.9981, 3.5210, 3.0694 and 2.5698 ppm respectively.

In winter season \( LC_{50} \) values for 24, 48, 72 and 96 hrs. exposure to mercuric chloride are 2.5948, 1.9461, 1.1444 and 0.8736 ppm and \( LC_{50} \) values for 24, 48, 72 and 96 hrs. exposure to cadmium chloride are 4.4811, 3.9981, 3.5201 and 3.1342 ppm respectively.

The fiducial limit for the log \( LC_{50} \) are summarised in Table 1 and 2 under column fiducial limit \( m_1 \) and \( m_2 \). The 95% confidence of \( LC_{50} \) values fiducial limit to heavy metal are \( m_1 \) (Minimum limit) and \( m_2 \) (Maximum limit) in summer season. The minimum and maximum fiducial limit for 24, 48, 72 and 96 hrs. log \( LC_{50} \) values of mercuric chloride are 0.1995 to 0.2814, 0.977 to 1.1026, 0.7646 to 1.0416 and 0.6786 to 1.0168 respectively. In summer season minimum and maximum fiducial limit for 95% confidence to 24, 48, 72 and 96 hrs. log \( LC_{50} \) values of cadmium chloride are 0.3917 to 0.9397, 1.3047 to 1.6695, 1.1081 to 1.7099 and 1.0751 to 1.5273 respectively.

In monsoon season the minimum and maximum fiducial limit for 24, 48, 72 and 96 hrs. log \( LC_{50} \) values mercuric chloride are 1.123 to 1.4882, 0.993 to 1.4808, 0.7874 to 1.2174 and 0.6611 to 1.1475 respectively. In monsoon season minimum and maximum fiducial limit for 24, 48, 72 and 96 hrs. log \( LC_{50} \) values of cadmium
chloride are 0.4627 to 0.7409, 0.3777 to 0.7155, 1.2687 to 1.7053 and 1.1064 to 1.7134 respectively.

In winter seasons the minimum and maximum fiducial limit for 24, 48, 72 and 96 hrs. log \( LC_{50} \) values of mercuric chloride are 1.1459 to 1.6829, 1.0600 to 1.5182, 0.8685 to 1.2485 and 0.8216 to 1.0610 respectively. In winter season minimum and maximum fiducial limit for 24, 48, 72 and 96 hrs. log \( LC_{50} \) value of cadmium chloride are 0.5341 to 0.7685, 0.4627 to 0.7409, 0.3775 to 0.7155, 0.4588 to 0.5334 respectively.
Table 1

Calculated LC$_{50}$ values and regression equation of mercuric chloride toxicity to *Barytelphusa cunicularis* in Summer, Monsoon and Winter.

<table>
<thead>
<tr>
<th>Season</th>
<th>Time of exposure in hrs</th>
<th>Calculated LC$_{50}$ values (ppm)</th>
<th>Regression equation</th>
<th>Fiducial limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summer</td>
<td>24 hrs</td>
<td>1.6873</td>
<td>Y=2.4156+11.3744X</td>
<td>0.1995-0.2814</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>1.0961</td>
<td>Y=-1.3971+6.1519X</td>
<td>0.977-1.1026</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>0.8001</td>
<td>Y=1.2072+4.1994X</td>
<td>0.7646-1.0416</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>0.7043</td>
<td>Y=2.0662+3.4606X</td>
<td>0.6786-1.0168</td>
</tr>
<tr>
<td>Monsoon</td>
<td>24 hrs</td>
<td>2.0212</td>
<td>Y=0.821+3.2008X</td>
<td>1.123-1.4882</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>1.7257</td>
<td>Y=2.0258+2.4044X</td>
<td>0.993-1.4808</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>1.0056</td>
<td>Y=1.7645+3.2276X</td>
<td>0.7874-1.2174</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>0.8022</td>
<td>Y=2.3201+2.9634X</td>
<td>0.6611-1.1475</td>
</tr>
<tr>
<td>Winter</td>
<td>24 hrs</td>
<td>2.5948</td>
<td>Y=1.5418+2.4458X</td>
<td>1.1459-1.6823</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>1.9461</td>
<td>Y=1.5143+2.7038X</td>
<td>1.0600-1.5182</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>1.1444</td>
<td>Y=1.7933+3.0292X</td>
<td>0.8685-1.2485</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>0.8736</td>
<td>Y=0.9343+4.3190X</td>
<td>0.8216-1.0610</td>
</tr>
</tbody>
</table>
Table 2

Calculated LC$_{50}$ values and regression equation of cadmium chloride toxicity to *Barytelphusa cunicularis* in Summer, Monsoon and Winter.

<table>
<thead>
<tr>
<th>Season</th>
<th>Time of exposure in hrs</th>
<th>Calculated LC$_{50}$ values (ppm)</th>
<th>Regression equation</th>
<th>Fiducial limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Summer</td>
<td>24 hrs</td>
<td>3.5315</td>
<td>$Y=3.1074+3.4622X$</td>
<td>0.3917 - 0.9397</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>3.0700</td>
<td>$Y=1.0109+2.6824X$</td>
<td>1.3047 - 1.6695</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>2.5668</td>
<td>$Y=2.0263+2.1099X$</td>
<td>1.1081 - 1.7099</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>2.0010</td>
<td>$Y=2.4762+1.9395X$</td>
<td>1.0751 - 1.5273</td>
</tr>
<tr>
<td>Monsoon</td>
<td>24 hrs</td>
<td>3.9981</td>
<td>$Y=2.4655+4.2111X$</td>
<td>0.4627 - 0.7409</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>3.5210</td>
<td>$Y=3.1076+3.4617X$</td>
<td>0.3777 - 0.7155</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>3.0694</td>
<td>$Y=1.012+2.6818X$</td>
<td>1.2687 - 1.7053</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>2.5698</td>
<td>$Y=2.0124+2.1190X$</td>
<td>1.1064 - 1.7134</td>
</tr>
<tr>
<td>Winter</td>
<td>24 hrs</td>
<td>4.4811</td>
<td>$Y=1.7642+4.9675X$</td>
<td>0.5341 - 0.7685</td>
</tr>
<tr>
<td></td>
<td>48 hrs</td>
<td>3.9981</td>
<td>$Y=2.4655+4.2111X$</td>
<td>0.4627 - 0.7409</td>
</tr>
<tr>
<td></td>
<td>72 hrs</td>
<td>3.5201</td>
<td>$Y=3.1059+3.4655X$</td>
<td>0.3775 - 0.7155</td>
</tr>
<tr>
<td></td>
<td>96 hrs</td>
<td>3.1342</td>
<td>$Y=3.7457+2.5201X$</td>
<td>0.4588 - 0.5334</td>
</tr>
</tbody>
</table>
Fig : 1. Regression lines of *Barytelphusa cunicularis* exposed to mercuric chloride in summer showing LC$_{50}$ values in log of concentration at different time duration.

- Summer - 24 hrs.
- Summer - 48 hrs.
- Summer - 72 hrs.
- Summer - 96 hrs.
Fig : 2. Regression lines of *Barytelphusa cunicularis* exposed to mercuric chloride in Monsoon showing LC$_{50}$ values in log of concentration at different time duration.

- Monsoon - 24 hrs.
- Monsoon - 48 hrs.
- Monsoon - 72 hrs.
- Monsoon - 96 hrs.
Fig : 3. Regression lines of *Barytelphusa cunicularis* exposed to mercuric chloride in Winter showing $LC_{30}$ values in log of concentration at different time duration.

Winter - 24 hrs.
Winter - 48 hrs.
Winter - 72 hrs.
Winter - 96 hrs.
Winter 48 hrs
LC50 = 1.9881 ppm

Winter 96 hrs
LC50 = 0.8736 ppm

Winter 24 hrs
LC50 = 2.9348 ppm

Winter 72 hrs
LC50 = 1.344 ppm
Fig: 4. Regression lines of *Barytelphusa cunicularis* exposed to cadmium chloride in summer showing LC$_{50}$ values in log of concentration at different time duration.

- Summer - 24 hrs.
- Summer - 48 hrs.
- Summer - 72 hrs.
- Summer - 96 hrs.
Fig: 5. Regression lines of *Barytelphusa cunicularis* exposed to cadmium chloride in Monsoon showing LC$_{50}$ values in log of concentration at different time duration.

- Monsoon - 24 hrs.
- Monsoon - 48 hrs.
- Monsoon - 72 hrs.
- Monsoon - 96 hrs.
Fig : 6. Regression lines of *Barytelphusa cunicularis* exposed to cadmium chloride in Winter showing $LC_{50}$ values in log of concentration at different time duration.

Winter - 24 hrs.
Winter - 48 hrs.
Winter - 72 hrs.
Winter - 96 hrs.
DISCUSSION

The effect of pollutants are generally characterized by biochemical or physiological changes or on the basis of changes in survival, reproduction or growth occurred by biochemical or physiological alterations of the animals. The physical, chemical and biological components of the environment, play an important role in the manifestation of biological responses to pollutants. The toxicity of a particular pollutants depends on many factors such as animals weights, (Pickering et al. 1968) its developmental stages (Kamaldeep and Toor, 1977) period of exposure, temperature (Macek et al., 1968) pH, hardness of water and dissolved oxygen contents of the medium. Pollutants affects behaviour, life span, and accumulate in test animal. The toxic effects were first seen by changes in the behaviour which were followed in most cases by death.

In the present investigation LC$_{50}$ values were calculated during all seasons such as summer, monsoon and winter, after exposure to both heavy metals mercuric chloride and cadmium chloride. The LC$_{50}$ values were found more during winter season, however, during monsoon and summer, LC$_{50}$ values was least after exposure to heavy metals. The seasonal variation in LC$_{50}$ values
may be due to metabolic activity of the crab. Similar observations have been made by several workers. Joshi et al. (1981) on freshwater prawn, Penaeus mergansis, after acute exposure of mercury, reported that high susceptibility of prawn, P. merguensis to mercury exposure, may be due to impairment in osmoregulatory and oxygen consumption mechanism. There is adverse effect of mercury on normal metabolism in the fiddler crab, Uca pugilator exposed to mercury causes structural damage in gill tissue. Olson et al. (1973) and inhibit Na+, K+, Mg++ dependent ATPase activity (Banting, 1970). Behavioural responses have been used as the sensitive measure of stress in organism (Lolla, 1974, Miller, 1980).

Immediately after exposure to pollutants animals remains stationary for some time and than various body movements paralysed and finally death was recorded. Sarojini et al.(1989), studied the toxic effect of phosalone to the commercial crab, Scylla serrata. They reported that behavioural toxicity holds a definite promise as sensitive short term indicator of chemical toxicity. Shyamasundari et al. (1996) studied the effect of the pesticides on the survival of the fiddler crab, Uca triangularis. The crabs got paralyzed with increase in the exposure period. This may be due to sudden change in the water medium and animal failed to adjust themselves changed medium.
In the present investigation it was seen that mercuric chloride is more toxic than cadmium chloride. Prosser (1973) has stated that sex and size have a greater influence over the physiology of the animal. Ghate and Mudherkar (1978) observed the toxic effect of copper sulphate to freshwater prawn, Macrobrachium and Caridina, they further reported that cause of death in crustaceans after metal exposure may be due to damage of the respiratory surface or the formation of mucus film over the gills and body surface. Similar observations have been made by Nagabhushanam et al. (1981) on freshwater prawn, Macrobrachium kistnensis exposed to heavy metal pollutants, it may be suggested that as concentration of media increased, there must be an increased osmotic work at the lethal cellular level resulting in an enhanced oxygen consumption, but at the lethal concentration the formation of coagulated mucus film over the body surface and gills must be taking place which interferes with the respiratory function and other vital process of the gills resulting in lowering of respiration in prawn. Similar results were documented by Costa (1965), Chinnayya (1971) in Caridina rajadhari. American workers on antifauling problems have published results supporting the views that copper retains vital processes through inactivation of essential enzyme (Clarke, 1947). Gyananath et al. (1987) reported the toxicity
of potassium ferrocyanide on freshwater prawn, *Macrobrachium lamerrii*. Sambasivarao et al. (1990) studied the toxicity of endocal pesticide on marine edible crab, *Scylla serrata*, they further stated that toxicity of endocal is temperature dependent. O'Hara (1972) have suggested that the high mercury induced mortality is due to the accumulation of mercury in the gills of crab. The increase in toxicity of endocal at higher temperature can also be attributed to high rates of biochemicals metabolic processes, diffusion, active transport across the membrane. Lloyd (1965) hypothesized that higher metabolic activity increase the rate at which poisons reach the gills which are known to be a major site for uptake of certain toxic chemicals. The increased tolerance as is evident by the higher LC$_{50}$ values during winter can also be the results of accumulated biochemical reserves like protein, glycogen and fats during these months (Diwansingh, 1976) which strengthen the tolerance of foreign stress. Decrease in toxicity of endocal might be due to low metabolic rate at sub optimum temperatures, and as a results of this decreased mechanism during summer accumulation of the pesticide might be less.

Toxicity of pesticide on crustaceans have been studied by several workers. Rao (1984) suggested that marine edible crab, *Scylla serrata* were most sensitive to DDT, less sensitive to
Malathion (Organophosphate) and least to sevimol (Carbonate). Mirajkar (1984) stated that organochlorine pesticide endosulfan were most toxic while organophosphate was least toxic. Bodkhe (1983) studied the toxicity of different pesticide to freshwater crab, *Barytelphusa cunicularis* and observed that the order of toxicity was mercuric chloride, sevimol, DDT, CuSO4. It was observed that organophosphate, organochlorine, carbamate and heavy metals caused alterations in the movement of *Macrobrachium kistnensis* when they were subjected to LC<sub>50</sub> concentration of the above mentioned pesticide, the prawn got excited and showed uncoordinated movement. George et al. (1957) have studied disturbed motor coordination in *Uca puginax* and other estuarine organism after their exposure to BHC. The pesticide exposed to *M. kistnensis* hesitated before taking food (Gyananath, 1982). The possibility that pollutants at low concentration could disrupt behaviour of marine animals by blocking test receptors or by mimicking chemical stimuli, was mentioned by Bloumer et al. (1971) the toxicological action of organophosphate results from disturbance of nerve functions due to inhibition of the enzyme acetylcholinesterase (Carbet 1974). Thus it can concluded that organophosphates could interfere with olfactory behaviour of prawns by making the odour, by blocking olfactory receptor or by
disrupting nerve functions. Gyananath (1982) stated that *M. lamertii* when exposed to pesticide in the acute and chronic toxicity studies, were found to consume less food than untreated prawns. Bhagyalakshmi (1981) observed that the field crab, *Oziotelphusa senex senex* when treated with sumithion did not show significant changes in the body weight.

From the above study it can be concluded that nature and seasonal temperature variation could be an important factor, influencing the toxicity of pollutant in water through the possibility of other interacting factors and the physiological state of the animal, can not be ruled out.