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Study on the effects of exposure of sub lethal dose of cadmium and zinc on the enzymatic activity in the tissues of green mussel – *Perna viridis* (L) from Ratnagiri coast, Maharashtra

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Abstract: *This research paper reports on the effects of exposure of green mussel to sub lethal concentrations of cadmium and zinc on GOT, GPT, ACP and ALP activities in the tissues of mantle, gill, hepatopancreas, adductor muscle, siphon, foot and gonad of Perna viridis. Exposure of sub lethal concentration of cadmium for 15 days resulted in the increase of GOT activity in all the tissues and was found to be the maximum in mantle and gonad but declined in hepatopancreas, siphon and foot. 30 days exposure of cadmium caused over all increase in GOT activity but the maximum was found to be in gonad although hepatopancreas and mantle showed higher levels of this activity. The GOT activity increased in all the tissues except in adductor muscle and the maximum in hepatopancreas and gonad as a result of exposure to lower concentration of zinc. Higher concentration of zinc for 30 days resulted in the increase in the activity in all the tissues but being higher in hepatopancreas, gill and gonad. GPT activity due to exposure to lower concentration of cadmium declined in mantle, adductor muscle, siphon, gill and gonad but it showed gentle rise in case of hepatopancreas and foot after 30 days of exposure. The exposure of lower concentration of zinc caused decline in GPT activity in mantle, adductor muscle, siphon, foot and gonad while in gill it increased. The ACP activity increased in all the tissues except in adductor and gonad during 15 days exposure to cadmium while exposure to zinc brought about elevation in all the tissues except gill. The exposure for 30 days resulted in decline in ACP activity in hepatopancreas, mantle and foot. 15 days exposure to cadmium enhanced the ALP activity in all the tissues but not in foot where as 30 days exposure resulted in decline in ALP activity in mantle, gill and adductor muscle. Exposure of zinc caused gradual increase in ALP activity in all the tissues studied. Thus, there is a possibility that Perna viridis can act as an indicator of pollutant contamination due to cadmium and zinc.*

Key words: Green mussel, Cadmium, Zinc, GOT, GPT, ACP, ALP

Introduction

Bonsignore *et al* (1965) have suggested that enzyme activities in the body fluids and tissues are reliable indicators of heavy metal contamination. Hernberg and Nikkanen (1970) have demonstrated that enzyme activities are of diagnostic significance and act as an indicator of lead poisoning. Chandravarti and Reddy (1994), Tendulkar (1996) and Kerkar (2000) have also stressed on the importance of monitoring the enzyme activities in crustaceans, molluscs and fish in response to heavy metals and pesticides exposure. Halliwell and Gutteridge (1989) and Winston (1991) reported that exposure of trace metals plays an effective role in the formation of reactive oxygen species which are the cause of oxidative stress. Oxidative stress is likely to result in protein degradation, inactivation of enzyme, damage of DNA, carcinogenesis etc. Mohite (2002) has mentioned about the possibility of the enzymes which are more specific in nature, can act as an indicator of pollutant

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contamination. The role of zinc finger domains of dimerised pair of steroid hormone receptors have been explained. The zinc ions are included within the protein and maintain the stability of its structure. Subhayu (1994) reviewed the bioaccumulation of cadmium in marine organisms. Rivonkar and Parulekar (1998) have reported the seasonal variations in the major and trace metals in sea water. Thus there is every chance of these metals to be present in the environment. It has been observed that zinc and cadmium are among those metals which are likely to be effective components of industrial effluent released in the environment. In the recent times, many indices of stress, measuring effects of pollutants at various levels such as biochemical, cytochemical, physiological and specific enzyme activities have been identified. *Perna* vir*i*dis is widely distributed along the east and west coast of India, is one of the intertidal benthos. This green mussel is also consumed as food by common folks along the coastal belt. Therefore, it was thought imperative to study the effects of exposure sub lethal concentration of zinc and cadmium on some of the enzymes activities in the tissues of green mussel.

Materials and Methods

The specimens of green mussel were randomly collected from Bhatye Creek, Ratnagiri. On arrival at laboratory the encrusting epifauna and mud were removed. The clean specimens were kept in running sea-water for about six hours. The specimens with length (4.5 to 7.5 cm) and weight (75 to 125 g) were selected for the current study. $CdCl_2$ and $ZnSO_4.7H_2O$ were used as a source of cadmium and zinc exposure. Their solutions were made in double distilled water and added to sea water to prepare the respective exposure media for the experimental animals. Sub lethal concentrations were selected after performing acute toxicity test. Thereafter for LC_0 0.10 ppm and for LC_{50} 0.15 ppm sub lethal concentrations were selected incase of cadmium. Similarly sub lethal concentrations for LC_0 0.20 ppm and for LC_{50} 0.50 ppm were selected for zinc exposure. Ten specimens of acclimatized individuals were exposed to each concentration as mentioned. The experimental and control aquaria of appropriate size containing 5 liters of sea water each were used, over crowding and contamination was prevented. The media was continuously aerated and the water was changed at an interval of 12 hours. Five specimens of *Perna viridis* were dissected open and tissues such as mantle, gill, hepato-pancreas, adductor muscle, siphon, foot and gonad were extracted in cold conditions. 100 mg of the respective tissue were homogenized with 2.0 ml of buffer solution. The homogenates were centrifuged at 3000 rpm for 15 minutes and the clear respective supernatants were used as a source of respective enzyme activities.

For the study of glutamic oxaloacetic transaminases activity (GOT) method of (Bergmeyer and Bernt, 1965a) was adopted with slight modification (Mohite, 2002). The reaction mixture for the study of glutamic oxaloacetic transaminases consisted of 1.0 ml of substrate buffer solution (0.1N phosphate buffer pH 7.4 and 0.1N l-aspartate oxoglutarate) and 0.2 ml of tissue homogenate. This reaction mixture was incubated for 1 hour at 30 ^oC and 1.0 ml of ketone reagent (10 N 2-4Dinitrophenyl hydrozine) was added and the reaction mixture was kept at room temperature for 20 minutes. 10 .0 ml of 0.4 N NaOH were added and the O D was read after 5 minutes at 540 nm. The specific activity of GOT is expressed as unit/g protein. The glutamic pyruvate transaminases activity (GPT) was assessed by the method of Bergmeyer and Bernt (1965b) with slight modifications (Mohite, 2002). 1.0 ml of substrate buffer (pH 7.4, 0.2M DL alanine and $2X10^3$ M oxoglutarate) was added to 0.2 ml of the respective tissues homogenates, the mixtures were incubated for 30 minutes at 37 ^oC. There

after 1.0 ml of ketone reagent (10 N 2-4 Dinitrophenyl hydrozine) was added to it. This resultant mixture was kept for 20 minutes at room temperature followed by addition of 10 ml of 0.4 N NaOH. The O D was read after five minutes at 540 nm. The specific activity of GPT is expressed as unit/g protein. The acid phosphatase activity (ACP) and alkaline phosphatase activity was estimated by following the method of (Andersch and Szcypinski 1947) with slight modifications (Mohite, 2002). To assess acid phosphatase activity 1.0 ml of citrate buffer and 0.2 ml of p-nitro phenol phosphate were mixed. To this reaction mixture 0.20 ml of respective homogenates were added. Thus the final reaction mixtures were incubated at 37°C for 30 minutes. After the completion of this step the reaction was stopped by adding 4.0 ml of 0.2 N NaOH. The resultant mixtures were shaken thoroughly and the O D was read at 410 nm. The specific activity of acid phosphatase (ACP) was the measured as n-mole of p-nitro phenol liberated/30 minutes/mg protein. To assess the alkaline phosphatase activity 1.0 ml of glycine-NaOH buffer and 0.2 ml of p-nitro phenol phosphate were added to 0.2 ml of tissue homogenates. The resultant reaction mixtures were incubated at 37°C for 30 minutes. After the incubation was over 4.0 ml of 0.2N NaOH were added to prevent further reaction. The final resultant reaction mixtures were thoroughly shaken and respective O D was read at 410 nm. One phosphate unit is the amount of enzyme which librates an n-mole of p-nitro phenol under assay conditions and specific activity is presented as n-mole of p-nitro phenol liberated/30 minute/mg protein. The protein estimation was carried out according to the method of (Lowery *et al.,* 1951).

Results and Discussion

The data of this study is presented in tabulated form (Table 1- 8). This study indicates that the exposure of sub lethal concentration of cadmium and zinc to green mussel bring about the variations in the glutamic oxaloacetate transaminase (GOT), glutamic pyruvate transaminases (GPT), acid phosphatase (ACP) and alkaline phosphatase (ALP) activities. Mussels are one of the many intertidal marine invertebrates which face varied types of natural fluctuations and also those caused due to the activities by man. Marine bivalves and mussels are known to exhibit greater metabolic diversity in anaerobic energy demands during the environmental fluctuations. Verma *et al.,* (1981), Santhamma *et al.,* (1999) and Kerkar (2000) have reported on the correlation between aminotransferase activity and stress in response to pollutants.

The GOT activity was observed to be higher in tissues like hepatopancreas, mantle, gonad and gill while in adductor muscle, siphon and foot, it was found to be lower in the control set. When the green mussel was exposed to cadmium (0.10 ppm for LC_0) for 15 days the GOT activity increased in all the tissues, being the maximum in mantle but declined in hepatopancreas. The 15 days exposure of cadmium (0.15 ppm for LC_{50}) caused further elevation in the GOT activity in all the tissues but not in siphon and foot. Maximum GOT activity was observed in gonad followed by hepatopancreas. The 30 days exposure of cadmium (0.10 ppm for LC_0) resulted in the maximum GOT activity in mantle and remaining tissues it elevated while in adductor muscle and gonad it slightly declined. The 30 days exposure (0.15 ppm for LC_{50}) caused over all increase in GOT activity but it was observed to be the maximum in gonad although hepatopancreas and mantle also showed comparatively higher activity (Table 1). When *Perna viridis* was exposed to sub lethal concentration of zinc (0.20 ppm for LC_0) for 15 days the GOT activity increased in all the tissues except in adductor muscle and the maximum were found to be in hepatopancreas and gonad. The exposure of zinc (0.50 ppm for LC_{50}) for 15 days further raised the activity of GOT enzyme in all the

tissues but declined in hepatopancreas and siphon. When the green mussels were exposed to 0.20 ppm (LC_0) of zinc for 30 days the GOT activity elevated in all the tissues studied. The exposure of 0.50 ppm (LC_{50}) of zinc for 30 days also caused an increase in the GOT activity in all the organs studied being very high in hepatopancreas, gill and gonad (Table 2).

Glutamate pyruvate transaminase activity in the tissues of *Perna viridis* exhibited fluctuations as result of exposure to sub lethal concentration of cadmium. The exposure of cadmium (0.10 ppm LC_0 and 0, 15 ppm LC_{50}) the tissues such as mantle, adductor muscle, siphon, gill and gonad exhibited decline in GPT activity while hepatopancreas and foot showed gentle rise at LC_{50} after 30 days of exposure. In case of hepatopancreas, the rise was very significant (Table 3). When *Perna viridis* was exposed to zinc (0.20 ppm for LC_0 and 0.50 ppm for LC_{50}) the GPT activity declined through out the duration of observation in mantle, adductor muscle, siphon, foot and gonad while in gill it increased after30 days of exposure. In hepatopancreas, the GPT activity gradually elevated reaching the maximum after 30 days of exposure (Table 4). The current findings on GOT and GPT support the views expressed by (Kulkarni and Kulkarni, 1987; Ray 1994; Reddy Bhagylakshmi*.,* 1994; Tendulkar 1996 and Kerkar 2000). Halliwell and Gutteridge (1989) have reported that trace metals enhance the formation of reactive oxygen species which are the cause of oxidative stress and lead to many toxic effects like degradation of proteins, inactivation of enzymes, damage to DNA and carcinogenesis. Mohite (2002) has reported that damage to tissue due to the exposure of cadmium and zinc can be caused. Agwuocha *et al.,* (2009) observed cellular damage to gills as result of xylene exposure which might lead to anoxia and oxidative stress. Mohite (2002) has suggested that aminotransferases are the probable strategic link between carbohydrate and protein metabolism because of the reason that they inter convert metabolites like ketogluteric acid, pyruvic acid and oxaloacetic acid on one side and aspartic acid and alanine on other side. Thus the study of GOT and GPT enzyme activities might reflect of the effects of role of cadmium and zinc in an intertidal marine animals and may be considered as bio indicator models to study environmental disturbances.

Due to the exposure of sub lethal concentration of cadmium (0.10 ppm and 0.15 ppm LC_0) for 15 days caused an increase in acid phosphatase activity (ACP) in all the tissues studied except in adductor muscle but after exposure for 15 days (LC_{50}) the ACP activity increased in all the tissues except in gonad. The exposure for 30 days (LC_0) to cadmium elevated ACP activity in all the tissues except mantle and gill while for LC50 there was rise in this activity in all the tissues studied (Table 5). The exposure to zinc (0.20 ppm for LC_0) for 15 days resulted in elevation in ACP activity in all the tissues except gill and siphon but when exposed to higher concentration of zinc (0.50ppm for LC_{50}) 15 days the activity increased in all the tissues. The exposure for 30 days for both concentrations (0.20 ppm and 0.50 ppm for LC_0 and LC_{50}) the ACP activity declined in hepatopancreas while in mantle and foot it declined with 0.20 ppm concentration but with higher concentration the ACP activity increased in all the tissues (Table 6).

When *Perna viridis* were exposed to cadmium sub lethal concentration (0.10 ppm LC_0 and 0.15 ppm LC_{50}) for 15 days the alkaline phosphatase activity (ALP) was found to be higher in all the tissues except the foot (LC_{50}, 0.15 ppm); but when the exposure was for 30 days (0.10 ppm, LC_0) the ALP activity declined in mantle, gill and adductor muscle and increased in the remaining tissues. The ALP activity was observed to be increased after the exposure to 0.15 ppm LC_{50} for 30 days in all the tissues studied (Table 7). It was quite surprising that the

Table 1. Effects of exposure of sub lethal concentration of cadmium on GOT activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure		30 days exposure	
			LC_0 (µg/g) (0.10ppm)	LC_{50} (µg/g) (0.15ppm)	LC_0 (µg/g) 0.10 ppm	LC_{50} (µg/g) (0.15 ppm)
1.	Mantle	4.245 ± 0.145	6.325 ±0.312	6.773 ±0.607	9.645 ±0.947	12.354 ±1.245
2.	Gill	3.654 ±0.321	3.987 ±0.212	4.354 ±0.111	5.541 ±0.213	6.325 ±1.215
3.	Hepato pancreas	6.255 ±0.746	4.354 ±0.547	7.514 ±1.245	7.658 ±0.541	15.254 ±0.249
4.	Adductor muscle	2.541 ±0.012	3.110 ±0.125	4.215 ±0.311	3.546 ±0.210	6.457 ±0.321
5.	Siphon	3.215 ±0.215	5.483 ±0.842	2.987 ±0.214	5.781 ±0.654	8.125 ±0.245
6.	Foot	1.954 ±0.321	2.745 ±0.211	2.125 ±0.524	3.578 ±0.452	4.533 ±0.549
7.	Gonad	4.124 ±0.945	5.845 ±0.428	7.852 ±0.289	6.531 ±0.479	18.645 ±1.655

Values are mean of 5 readings± SD

Table 2. Effect of exposure of sub lethal concentration of zinc on GOT activity in the tissues of *Perna viridis*.

Sr. No.	Tissues	Control (µg/g)	15 days exposure LC_0 (µg/g) (0.20 ppm)	15 days exposure LC_{50} (µg/g) (0.50 ppm)	30 days exposure LC_0 (µg/g) (0.20 ppm)	30 days exposure LC_{50} (µg/g) (0.50 ppm)
1.	Mantle	4.245 ±0.145	4.524 ±0.426	4.978 ±0.257	6.542 ±1.214	5.541 ±1.452
2.	Gill	3.654 ±0.321	5.687 ±1.546	8.458 ±0.824	5.122 ±1.241	11.321 ±0.546
3.	Hepato pancreas	6.255 ±0.746	6.872 ±1.124	4.895 ±0.452	8.548 ±1.245	19.326 ±1.255
4.	Adductor muscle	2.541 ±0.012	1.542 ±0.101	3.110 ±0.120	3.425 ±0.345	5.841 ±0.451
5.	Siphon	3.215 ±0.215	6.547 ±0.311	5.354 ±0.432	5.897 ±0.716	8.254 ±1.642
6.	Foot	1.954 ±0.321	2.432 ±1.221	2.578 ±0.614	2.955 ±0.451	5.025 ±0.400
7.	Gonad	4.124 ±0.945	6.852 ±1.004	7.584 ±0.541	5.958 ±1.825	8.978 ±0.541

Values are mean of 5 readings± SD

Table 3. Effects of exposure of sub lethal concentration of cadmium on GPT activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure LC_0 (µg/g) (0.10 ppm)	15 days exposure LC_{50} (µg/g) (0.15 ppm)	30 days exposure LC_0 (µg/g) (0.10ppm)	30 days exposure LC_{50}(µg/g) (0.15 ppm)
1.	Mantle	8.541 ±1.421	6.654 ±0.587	6.452±2.341	6.745±0.255	5.466±1.249
2.	Gill	15.645±1.462	14.987±3.451	13.549±0.124	6.549±1.649	8.965±1.985
3.	Hepato pancreas	5.542 ±0.245	6.542±1.210	6.841±0.465	9.358±2.450	17.976±0.254
4.	Adductor muscle	13.654±0.125	10.357±1.871	12.452±0.587	6.875±1.871	3.652±0.578
5.	Siphon	6.654 ±0.325	6.412±1.210	5.642±0.885	3.845±0.211	1.847±0.045
6.	Foot	5.645 ±1.642	3.458±1.245	3.875±0.145	0.947±0.012	1.578±0.141
7.	Gonad	15.124 ±2.541	10.542±0.460	9.548±0.148	6.977±0.485	3.574±1.810

Values are mean of 5 readings± SD

Table 4. Effect of exposure of sub lethal concentration of zinc on GPT activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure (µg/g)LC_0 (0.20 ppm)	15 days exposure (µg/g)LC_{50} (0.50ppm)	30 days exposure (µg/g)LC_0 (0.20ppm)	30 days exposure (µg/g)LC_{50} (0.50 ppm)
1.	Mantle	8.541 ±1.421	3.147 ±0.875	2.848 ±0.471	1.717 ±0.297	0.946 ±0.714
2.	Gill	15.645 ±1.462	10.942 ±0.453	6.498 ±0.278	4.514 ±0.469	5.757 ±0.839
3.	Hepato pancreas	5.542 ±0.245	7.451 ±1.745	8.844 ±0.656	10.578 ±2.546	11.318 ±0.876
4.	Adductor muscle	13.654 ±0.125	12.242 ±0.988	12.033 ±0.784	8.781 ±0.782	6.978 ±1.401
5.	Siphon	6.654 ±0.325	7.401 ±0.175	6.462 ±0.478	4.826 ±0.279	3.174 ±0.141
6.	Foot	5.645 ±1.642	4.718 ±0.654	4.879 ±0.246	3.905 ±0.345	3.458 ±0.782
7.	Gonad	15.124 ±2.541	8.345 ±1.241	6.471 ±0.164	5.245 ±0.876	2.545 ±1.125

Values are mean of 5 readings± SD

Table 5. Effect of exposure of sub lethal concentration of cadmium on ACP activity in the Tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure LC_0 (µg/g) (0.10 ppm)	15 days exposure LC_{50} (µg/g) (0.15ppm)	30 days exposure LC_0 (µg/g) (0.10 ppm)	30 days exposure LC_{50} (µg/g) (0.15ppm)
1.	Mantle	0.354 ±0.012	0.368 ±0.023	0.702 ±0.059	0.417 ±0.089	0.908 ±0.314
2.	Gill	0.248 ±0.147	0.315 ±0.214	0.895 ±0.313	0.578 ±0.216	1.325 ±0.321
3.	Hepato pancreas	0.421 ±0.059	0.648 ±0.214	0.871 ±0.398	1.655 ±0.321	1.654 ±0.198
4.	Adductor muscle	0.318 ±0.110	0.245 ±0.103	0.541 ±0.023	0.688 ±0.046	0.975 ±0.054
5.	Siphon	0.108 ±0.011	0.120 ±0.010	0.215 ±0.015	0.548 ±0.099	0.502 ±0.015
6.	Foot	0.249 ±0.024	0.964 ±0.105	0.355 ±0.060	0.458 ±0.049	0.926 ±0.140
7.	Gonad	0.297 ±0.031	0.654 ±0.021	0.517 ±0.055	0.875 ±0.080	1.584 ±0.106

Values are mean of 5 readings± SD

Table 6. Effect of exposure of sub lethal concentration of zinc on ACP activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure LC_0 (µg/g)	15 days exposure LC_{50} (µg/g)	30 days exposure LC_0 (µg/g)	30 days exposure LC_{50} (µg/g)
1.	Mantle	0.354 ±0.012	0.538 ±0.182	0.819 ±0.078	0.714 ±0.201	1.049 ±0.271
2.	Gill	0.248 ±0.147	0.110 ±0.003	0.295 ±0.142	0.739 ±0.116	0.925 ±0.011
3.	Hepato pancreas	0.422 ±0.059	0.860 ±0.278	1.012 ±0.018	0.855 ±0.055	2.584 ±0.181
4.	Adductor muscle	0.318 ±0.110	0.415 ±0.025	0.521 ±0.048	0.845 ±0.041	1.745 ±0.105
5.	Siphon	0.109 ±0.011	0.101 ±0.015	0.301 ±0.0.51	0.425 ±0.011	0.821 ±0.009
6.	Foot	0.249 ±o.024	0.421 ±0.036	0.585 ±0.038	0.568 ±0.107	0.720 ±0.102
7.	Gonad	0.297 ±0.031	0.345 ±0.082	0.572 ±0.018	0.794 ±0.092	2.432 ±0.125

Values are mean of 5 readings± SD

Table 7. Effect of exposure of sub lethal concentration of cadmium on ALP activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15 days exposure LC_0 (µg/g)	15 days exposure LC_{50} (µg/g)	30 days exposure LC_0 (µg/g)	30 days exposure LC_{50} (µg/g)
1.	Mantle	0.099 ±0.045	0.142 ±0.011	0.678 ±0.055	0.422 ±0.061	0.851 ±0.037
2.	Gill	0.039 ±0.015	0.064 ±0.011	0.910 ±0.005	0.103 ±0.011	0.305 ±0.024
3.	Hepato pancreas	0.079 ±0.013	0.104 ±0.034	0.161 ±0.094	0.381 ±0.112	0.805 ±0.054
4.	Adductor muscle	0.066 ±0.030	0.084 ±0.012	0.215 ±0.022	0.097 ±0.001	0.412 ±0.046
5.	Siphon	0.059 ±0.023	0.088 ±0.003	0.145 ±0.079	0.278 ±0.102	0.346 ±0.012
6.	Foot	0.047 ±0.012	0.065 ±0.005	0.024 ±0.014	0.135 ±0.008	0.678 ±0.141
7.	Gonad	0.019 ±0.009	0.028 ±0.001	0.065 ±0.014	0.098 ±0.005	0.310 ±0.112

Values are mean of 5 readings± SD

Table 8. Effect of exposure of sub lethal concentration of zinc on ALP activity in the tissues of *Perna viridis.*

Sr. No.	Tissues	Control (µg/g)	15days exposure LC_0 (µg/g)	15 days exposure LC_{50} (µg/g)	30 days exposure LC_0 (µg/g)	30 days exposure LC_{50} (µg/g)
1.	Mantle	0.099 ±0.045	0.106 ±0.042	0.287 ±0.015	0.282 ±0.013	0.580 ±0.037
2.	Gill	0.039 ±0.015	0.105 ±0.015	0.145 ±0.031	0.321 ±0.002	0.887 ±0.001
3.	Hepato pancreas	0.785 ±0.013	0.098 ±0.006	0.348 ±0.008	0.586 ±0.006	1.460 ±0.005
4.	Adductor Muscle	0.066 ±0.030	0.092 ±0.081	0.135 ±0.022	0.245 ±0.007	0.389 ±0.002
5.	Siphon	0.059 ±0.023	0.028 ±0.002	0.821 ±0.091	0.718 ±0.025	1.348 ±0.001
6.	Foot	0.047 ±0.012	0.080 ±0.064	0.086 ±0.047	0.304 ±0.021	0.708 ±0.401
7.	Gonad	0.029 ±0.009	0.048 ±0.015	0.086 ±0.002	0.287 ±0.051	0.902 ±0.002

Values are mean of 5 readings± SD

exposure to zinc is sub lethal. The concentration of (0.20 ppm, LC_0 and LC_{50}) for 15 days and for 30 days resulted in gradual increase in ALP activity in all the tissues studied (Table 8).

Varley (1969) has mentioned that phosphatase enzyme catalyses the separation of phosphoric acid from some monophosphoric ester; the two types of phosphatase enzymes are acid phosphatase and alkaline phosphatase both having peak activities at pH 5.0 and pH 10.0 respectively. These activities show variations during pathological and environmental conditions. Moor *et al.,* (1989) and Livingstone (1991) have expressed their views regarding the existence of hydrolases (acid phosphatase and alkaline phosphatase), esterases and organo-phosphatase hydrogenases in most of the molluscs. The present observations are in congruency with them. Agwuocha *et al.,* (2011) have reported decline in ACP activity in the tissues of G. *divaricatum* for shorter and longer duration of xylene exposure. They further reported prominent fluctuations in ALP activity in hepatopancreas, gill and adductor muscle as a result of xylene exposure. In the present study the ALP activity was found to be elevated after the exposure of *Perna* viridis to sub lethal concentrations of cadmium and zinc for 15 days and 30 days. This may reflect on the probable different mode of action of these two metals in this intertidal mussel. Mazorra *et al*.,(2002) have suggested that the exposures of heavy metals are likely to cause variations in the ACP and ALP activity in clam – *Scrobicularia plana,* and this may result in stress in the animal. The present observations are likely to be in agreement with their view, and cadmium and zinc in sub lethal concentrations may be the cause of same as reflected through ACP and ALP activities. Morton (1965) has proposed that ACP catalyses hydrolysis of orthomonophosphoric acid and affects the transformation of phosphoric acid and shell deposition; ALP is brush border enzyme and is related to maintaining the orthophosphate pool, transfer of phosphoryl group and hydrolysis and esterification of metabolites moving within the cell and between the extracellular spaces. This view may need further elaboration in case of intertidal mussels.

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