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TISSUE LACTIC ACID AND GLYCOGEN LEVEL OF MOLLUSCS EXPOSED TO Cu AND Hg

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OF late, there has been a growing awareness of the toxic effects of trace metals on aquatic animals[1-5]. Most of the studies refer to acute toxicity and uptake kinetics. The present communication is on the effect of two trace metals *viz* Cu and Hg on the lactic acid and glycogen levels in the case of two commercially important bivalve molluscs, *Perna viridis* (Linnaeus) and *Villorita cyprinoides* var *cochinensis* (Hanley).

The animals acclimatized in the laboratory in filtered sea water of habitat salinity and ambient temperature, were exposed to sublethal levels of the metal (tables 1 and 2). The experimental conditions were the same as described earlier[6]. Individual animals were killed at definite intervals of time and soft parts used for determining lactic acid. To estimate glycogen, muscle or liver from 3 to 4 animals were dissected out and pooled for further analysis. Glycogen and lactic acid were estimated by standard methods[7-8]. Results

Table 1 *Change in the tissue lactic acid levels on exposure to Cu/Hg*

Concentration of metal ions (ppm)	Lactic acid μg/g wet wt ± S.D. Exposure time 2 hr	4 hr	24 hr
Perna viridis (Salinity 25‰, temp. 28°C)			
Cu			
0.5	163.56 ± 15.39	190.78 ± 11.07	392.18 ± 33.43
1.0	204.12 ± 21.46	231.95 ± 21.02	445.74 ± 48.82
2.0	297.50 ± 26.08	327.44 ± 18.50	621 44 ± 58.06
Control	48.25 ± 4.13	43.80 ± 3.52	46.61 ± 3.76
Hg			
0.5	127.26 ± 9 67	142.17 ± 9.46	344.17 ± 26.38
1.0	160.52 ± 18.30	176.56 ± 19.28	439.84 ± 37.15
Control	48 25 ± 4.13	46.55 ± 3.82	50.04 ± 4.16
Villorita cyprinoides (Salinity 10‰, temp. 28°C)			
Cu			
0.5	50.22 ± 3.73	74.80 ± 4.84	289.60 ± 33.50
1.0	85.69 ± 5.10	116.78 ± 8.52	423.54 ± 48.05
Control	26.50 ± 1.62	28.82 ± 1.90	37.48 ± 1.77
Hg			
0.5	49.23 ± 3.95	68.12 ± 5.07	254.84 ± 19.78
1.0	73.85 ± 4.68	110.34 ± 7.11	372.65 ± 46.68
Control	25.40 ± 1.44	23.65 ± 1.38	24.73 ± 1.56

Table 2 *Changes in the tissue glycogen content on exposure to Cu/Hg*

Concentration of metal ions (ppm)	Organ	Glycogen content (μg/g wet wt. ± S.D.) after different periods		
P. viridis (Salinity 25‰, temp. 28°C)				
Cu		4 hr	12 hr	24 hr
0.2	Muscle	632.37 ± 20.21	478.50 ± 14.64	380.53 ± 11.63
	Liver	1908.85 ± 42.64	1651.44 ± 31.74	1379.37 ± 25.01
0.5	Muscle	586.90 ± 17.58	372.67 ± 11.31	N.D.
	Liver	1748.59 ± 35.93	1367.36 ± 32.69	811.06 ± 13.48
Control	Muscle	696.69 ± 13.33		
	Liver	2020.29 ± 43.70		
Hg				
0.2	Muscle	657.23 ± 19.62	571.90 ± 14.50	429.70 ± 12.64
	Liver	1943.64 ± 33.98	1638.55 ± 24.49	1405.35 ± 26.87
0.5	Muscle	618.30 ± 11.81	385.63 ± 11.94	N.D.
	Liver	1830.45 ± 41.05	1409.78 ± 25.77	729.68 ± 12.80
Control	Muscle	696.69 ± 13.33	—	—
	Liver	2020.29 ± 43.70		
V. cyprinoides (Salinity 10‰, temp. 28°C).				
Cu		12 hr	24 hr	48 hr
0.5	Muscle	1875.74 ± 22.40	1373.16 ± 44.75	704.54 ± 45.67
1.0	Muscle	1643.50 ± 24.30	900.94 ± 55.01	574.73 ± 29.78
Control	Muscle	2350.53 ± 39.70	2342.60 ± 35.90	2356.24 ± 40.35
Hg				
0.5	Muscle	2016.49 ± 36.21	1487.83 ± 34.70	824.50 ± 30.60
1.0	Muscle	1809.64 ± 33.33	974.10 ± 42.26	486.14 ± 29.56
Control	Muscle	2350.53 ± 39.70	2342.60 ± 35.90	2356.24 ± 40.35

are given in tables 1 and 2. An inverse relationship between lactic acid and glycogen content was observed. The level of lactic acid increased with increasing concentration of the metal ion and time of exposure of the animal. On the other hand, the glycogen content declined, it was reduced to a non-detectable level in the case of *P. viridis* exposed for 24 hr to 0.5 ppm Cu or Hg. Since different animals were used to determine the lactic acid and glycogen in each species, individual variations are not ruled out; nevertheless, the inverse relationship holds good.

These changes could be explained as follows: Metal intoxication might have caused severe anaerobic stress resulting in the breakdown of tissue glycogen. The end-product of the anaerobic degradation of glycogen being lactic acid, its level increases.

It was also observed that the two bivalves produced a lot of mucus, which increased with increasing metal concentration and was maximum for *P. viridis*, on exposure to metal ions. The deposition of mucus on the gills might have interfered with the normal oxygen transfer mechanism, leading to hypoxic stress and consequent tissue glycogen breakdown. The mortality caused by acute heavy metal poisoning could be partly attributed to tissue hypoxia. A similar observation was made by Shaffi[9,10] in the case of freshwater teleosts due to cadmium and copper intoxication.

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