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Aberrations in the Neurosecretory Cells of a Freshwater Pulmonate, *Indoplanorbis exustus*, Chronically Exposed to Sublethal Concentration of Two Molluscicides, $BaCl_2$ and $CuSO_4$

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Majority of the molluscicides, because of their residual effects on adsorption and absorption, act as persistent pollutants. Very little attention has been focused on the toxic effects of molluscicides on the snails and associated fauna. Recently, some workers (MANNA and GHOSH 1972, McINNES and THURBERG 1973, ISHAK and MOHAMED 1975, CHENG and SULLIVAN 1977, SALIBA and VELLA 1977) have reported the changes in the histology of alimentary canal or metabolism or behaviour or osmoregulation of the gastropod snails that were chronically exposed to the different molluscicides. However, histopathological alterations in the neurosecretory cells (NSC) of the snails treated with molluscicides have not been examined so far. Such a study is highly imperative because (i) NSC products of the snails control a variety of behavioural and physiological parameters (NAGABHUSHANAM and HANUMANTE 1978) and (ii) it is likely to give an insight, at least in part, about the cidal action of the molluscicides. In the present probe the effects of sublethal, chronic exposure of I. exustus to two classical molluscicides $BaCl_2$ and $CuSO_4$ on its NSC architecture have been investigated.

MATERIAL, METHODS AND RESULTS

Healthy adults of I. exustus were maintained in the laboratory (21 ± 2.5°C). The snails were fed 'ad libitum' with algal filaments and Hydrilla leaves. For I. exustus, sublethal concentrations of $BaCl_2$ and $CuSO_4$ are 1.0 ppm and 0.1 ppm respectively, for seven days exposure (unpublished data). The solutions were freshly prepared in dechlorinated tap-water every day and the snails (10 snails/molluscicide) were kept in these concentrations for 7 days.

The central nervous systems (CNS) of both molluscicided and control (maintained in dechlorinated tap-water) snails were quickly exicsed and fixed in Bouin's fluid for 24 hr. The tissues were histologically processed, embedded in paraffix wax and the sections were cut at 6 micra. The tissues were stained with AF (EWEN 1962). Measurement of nuclear and cell diameter, and quantification of neurosecretory material intensity (NSMI) was carried as described by HANUMANTE et al. (1978).

The CNS of I. exustus contains two types of NSC, viz. A and B (HANUMANTE et al. 1977). Both the cells from cerebral, pleural, parietal and visceral ganglia revealed histopathological aberrations after 7 days of molluscicidiing (Table I).

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TABLE 1

Molluscicide	Cell Diameter (microns)		Nuclear Diameter (microns)		NSMI	
	A Cell	B Cell	A Cell	B Cell	A Cell	B Cell
Control	13.60 ± 1.12	46.10 ± 2.7	8.00 ± 0.31	28.5 ± 0.0	4	4
$BaCl_2$ (1.0^2ppm)	14.78 ± 1.39	44.1 ± 5.5	10.46 ± 0.24*	28.1 ± 0.1*	1.32	1.32
$CuSO_4$ (0.1^4ppm)	13.00 ± 1.06	43.3 ± 7.3	10.36 ± 0.29*	30.7 ± 0.2*	1.32	1.32

*P = 0.05

A significant (P = 0.05) increase was evident in nuclear diameters of A and B cells without significant change in their cell dimensions. Both the molluscicides provoked striking decline in the NSMI from A and B cell perikarya. Their neurohaemal areas also showed depletion of neurosecretory material. Both the cell perikarya were laden with large number of vacuoles only after $CuSO_4$ treatment.

DISCUSSION

The subtle morphometrical changes induced by $BaCl_2$ and $CuSO_4$ in both NSC from all the ganglia may be a result of generalized chemical stress. This action is consistent with the contention that the insecticide pollutants have unspecific sites of action in insects (NORMANN and SAMARANAYAKA 1977).

The significant increment in nuclear diameter and not in cell diameter of NSC after molluscicide suggests that the rate of synthesis is probably impaired. Additionally, the rate of transport and/or release of neurosecretory material is stimulated thereby causing the observed decline in the NSMI. Since very little neurosecretory material was present in NSC perikarya in comparison with controls, it appears that the rates of transport and/or release of neurosecretory material are greater than that of its synthesis. Since AF, the stain used in identifying NSC in the present investigation, is specific for the amino acids cysteine and/or cystine, it can be postulated that molluscicidal treatment of the snails interferes with the kinetics of these amino acids from NSC.

The appearance of large number of vacuoles in the NSC of $CuSO_4$-treated snails may be because of pronounced toxic effect of copper than barium ions. This is supported by more dramatic

decline in O_2 consumption and survival of $CuSO_4$-exposed I. exustus than $BaCl_2$ in comparatively low doses (unpublished data). Moreover, copper ion is known to be highly cidal for molluscs (CHENG 1974). The observed histopathological changes in the NSC of molluscicided snails may be due to either direct toxic action of molluscicides or indirect effect of a series of aberrations in behavioural (see SALIBA and VELLA 1977) and physiological (see CHENG and SULLIVAN 1974, 1977, ISHAK and MOHAMED 1975) parameters that are regulated by the NSC products (NAGABHUSHANAM and HANUMANTE 1978). In any eventuality, the malfunctioning of neurosecretory centers (which is reflected in the morphological picture of NSC) of molluscicided snails is harmful to them for the efficient survival in their environment.

It is interesting to mention that in the cockroach, Periplaneta americana (NANDA 1974) poisoned with endrin and sumithion (the insecticides) dramatic impairment in both inter- and intracellular structural features of NSC accompanied by heavy vacuolation within perikarya were evident. The changes observed in molluscicided I. exustus NSC cytology are comparable with the above report. In the crab, Uca pugilator (NAGABHUSHANAM et al. 1978) medulla terminalis X-organ neurosecretory material quantity is increased after exposure to the pollutants Aroclor 1242 and PCB. This indicates that there is no uniformity in the responses of the NSC of invertebrates to the pollutants, and the nature of their response is probably linked to the chemical structure of the pollutant.

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