Bull. Environm. Contam. Toxicol. 24, 37-39 (1980)

Toxic Effects of the Phytoalkaloid Colchicine on Oviposition and Neurosecretion of the Vector Snail (*Indoplanorbis exustus*)[1]

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Chemical control of the population of freshwater gastropods that transmit trematode parasites, is still the promising method of curbing snail-borne diseases (CHENG 1974). However, the use of chemoagents in sterilizing or inhibiting snail reproduction is sparse though it is known that the molluscicides have potential to suppress egg-laying, e.g. organotin (TBTO) in the case of Biomphalaria glabrata (RITCHIE et al. 1974). Effect of the phytoalkaloid which affects in other organisms, both fecundity and nervous system, on snails has not been screened. Colchicine (COLC) blocks fertility and ovarian development in many insects (see INDIRA et al. 1969) and upsets neurosecretory axonal transport in vertebrates (HINDELANG-GERTNER et al. 1976) and an invertebrate (FITZHARRIS 1973).

Oviposition in the intermediate host of several trematodes, Indoplanorbis exustus appears to be neurohormonally regulated (NAGABHUSHANAM & HANUMANTE 1979). Thus any intervention in its breeding biology is likely to be mediated and/or reflected in the oviposition modulating neurosecretory cells. The lack of information about the impact of the antimitotic phytoalkaloid COLC on reproduction of I. exustus prompted us to initiate this work. Here we report sublethal, chronic effects of COLC on oviposition and neurosecretion.

TABLE 1

Snail category	No. of egg capsules laid/ snail/day	B cell	
		NSMI	Nuclear diameter (microns)
Control	12.0 ± 1.9	5	8.0 ± 0.31
Colchicinized (10 ppm) for 7 days	1.7 ± 0.1*	1.7	3.5 ± 0.1*

* P < 0.001

[1]Supported in part through PL-480 Project (A7-ADP-39)

0007-4861/80/0024-0037 $01.00
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MATERIAL AND METHODS

Laboratory adapted (25 ± 3°C, 14L : 10D), healthy, breeding adults of I. exustus were exposed to 10 ppm (a sublethal concentration) of COLC for 7 days with ad libitum food and aeration. Dechlorinated tap-water maintained snails served as controls. The number of egg-capsules laid by both of them was monitored every day. After 7 days, CNS of treated and control animals were excised, fixed, histologically processed, stained and their neurosecretory activity was measured as reported by HANUMANTE et al. (1979).

RESULTS AND DISCUSSION

The effects of 10 ppm COLC on oviposition and cerebral B cell AF stainable neurosecretory material intensity (NSMI) and nuclear diameter are profiled in Table 1.

COLC in sublethal (10 ppm) concentration have critical effects both at behavioural (oviposition) and cellular (neurosecretory) levels of I. exustus. The significant inhibition of the egg-capsule laying ability indicates a disruption in the mechanism(s) implicated in oviposition. Since in some of the deposited eggs, membranes were malformed and the number of eggs per capsule was less (50 in control and 42 in experimental), it seems COLC interferes with oogenetic and vitellogenic dynamics also, probably by direct toxic action on female gonocytes. However, the overall result of seven days phytoalkaloid exposure can be categorised as a particular kind of sterility. Since viability of the eggs was lowered, COLC can be regarded to have molluscicidal effects eventhough in the experimental concentration, survival of the adult snails was unaffected.

Amongst the two types of probable neurosecretory (NS) cells (A and B) that AF stain distinguishes in I. exustus CNS (HANUMANTE et al. 1979), COLC provoked cognizable changes only in cerebral B cells which are probably implied in reproduction (CHINTWAR 1974). As such it is likely that the changes, at least partly, are an indirect sequelae to the inhibition of oviposition and the remaining part of B cell alterations may be resulting owing to the well established direct pharmacological action of COLC on axonal transport of NSM (HINDELANG-GERTNER et al. 1976). A cells are either not responding or the vicissitudes in their morphology following COLC treatment are not detectable with present staining technique.

Diminished B cell NSMI after COLC exposure may be owing to either reduced synthesis, and/or faster transport and/or release of NSM. The latter probability is untenable

in view of (i) absence of NSM in neurohaemol areas and (ii) proven blocking action of COLC on the mobility of axonal transport of secretory material without appreciably modifying its synthesis (HINDELANG- GERTNER et al. 1976). The B cell NS changes are in disagreement with earlier observations that colchicinization enhances AF NSM in NS cells of rats (HINDELANG- GERTNER et al. 1976) and in the invertebrate, Sabella melanostigma (FITZHARRIS 1973) which is due to disruption of microtubules. Hence it is certain that at least part of the cerebral B cell vicissitudes are because of disturbances in its secretory kinetics provoked by COLC in some unknown manner which may emanate owing to COLC action at extramicrotubular site(s).

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