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Notochord in *Tilapia nilotica* Exposed to Sublethal Dose of Malathion, S[1,2-Di(Ethoxycarbonyl)Ethyl]Dimethyl Phosphorothiolothionate

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ABSTRACT

Exposure of Tilapia nilotica *embryos to sublethal dose of 1.0ppm commercial grade malathion, S[1,2-di(ethoxycarbonyl)ethyl] dimethyl phosphorothiolothionate from day-10 post fertilization resulted in notochordal aberrations. Pesticide-treated fishes exhibited constriction of the notochordal sheath, folding at the posterior sections and larger notochord than those of the control group.*

INTRODUCTION

Organophosphate pesticides, although not as deadly or as persistent as the organochlorines, are potent anticholinesterases (Anthony, 1986). Their anticholinesterase activities resulted in peripheral nerve lesions (Shell et al., 1988) ataxia and paraplegia (Metcalf et al., 1983) and reduced swimming stamina (Lockhart et al., 1985). These chemicals produce histopathologic aberrations in the liver (Areechon and Plumb, 1990), brain (Amparado, 1989), gills (Richmond and Dutta, 1989), digestive tract (Triebskorn et al., 1990), gonads (Wester and Canton, 1986), kidney (Gills and Pant, 1988), enzyme dysfunction (Facage-Elawar and Francis, 1988), and impairment of sex steroid metabolism (Bagchi et al., 1990; Singh and Singh, 1987).

Organophosphates produce teratogenic effects. Vertebral malformations include parrot beak syndrome, short and twisted neck, shortened digits and vertebral fusion in fowls (Greenberg and Graham, 1969; Van Leeuwen

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et al., 1986) and curved vertebral column in fish (Areechon and Plumb, 1990). That organophosphates have mutagenic properties were shown by the development of resistant population in *Anopheles stephensi* (Roland and Hemingway, 1987), *Anopheles culicifacies* (Herath et al., 1987) and predacious mite *Ambylsius polentillae* (Anber and Overmeer, 1988) and identification of altered acetyl-cholinesterase gene in *Culex pipiens* (Villani and Hemingway, 1986).

This study is aimed at determining the teratogenicity of commercial grade malathion, S[1,2-di(ethoxycarbonyl)ethyl] dimethyl phosphorothiolothionate] on *Tilapia nilotica*. Results of this study will be valuable in the assessment of pesticide use in agriculture.

MATERIALS AND METHODS

Tilapia nilotica eggs obtained from the Natural Science Research Institute of the University of the Philippines were cultured in finger bowls until day-10 post fertilization. The hatchlings were then divided into control and experimental groups, each consisting of 300 larvae and reared in 1.0 x 1.0 x 1.5m concrete ponds until day 45. Malathion, commercial grade, EC 57, diluted to 0.1 and 1.0 ppm served as medium for the experimental groups.

Specimens were harvested at day-36, post fertilization, fixed in Bouin's solution, dehydrated in ethanol series and embedded in paraffin. Fishes were sectioned at 7 m using the American Optical rotatory microtome and were stained with hematoxylin-eosin for light microscopy.

RESULTS AND DISCUSSION

CONTROL FISH

Figure 1 shows the notochordal histology in the control fish. The structure exhibited as vacuolated interior which was surrounded by a thin primary sheath and thick secondary sheath. It was located dorsal to the kidney, ventral to the spinal cord and between the myotome.

TREATED FISH

Animals that were exposed to 1.0ppm malathion exhibited histopathologic effects. These fishes showed abnormally enlarged notochord (Figure 2), dorso-lateral folding, constriction in the chordal sheath and absence of the secondary sheath (Figures 3 and 4). The notochord diameter ranged from 152–160 m which was 1.3 times larger than that in the control group.

The aberrations in the notochord observed in this study were similar to those exhibited by white leghorn chicken injected with malathion (Garrison and Wyttenbach, 1985). These workers attributed the teratogenic effects to undersulfation of chondroitin-4 and chondroitin-6 sulfate, the major glycosaminoglycans of the sheath. Van Leeuwen et al. (1986), in their studies on the effects of dithiocarbamates and related compounds on the rainbow trout, *Salmo gairdneri*, found too, the catalytic function disruption of the chordal sheath, a structure that directs and coordinates notochordal growth. The pathology may be due to the chelating action of the pesticide on copper. Apparently dithiocarbamates reduce the cellular level of copper thus reducing the amount of active lysyl oxidase, which is responsible for catalyzing the oxidative deamination of peptidyl lysine and hydroxylysine residues in procollagen. Chronic exposure to malathion, an anti-cholinesterase like the carbamates, has interfered with the chordal sheath formation thus disturbing the growth of the notochord. Such abnormality has an important consequence on the arrangement and disposition of other organs (Van Leeuwen et al., 1986b) and ultimately on the metabolic processes of the organism.

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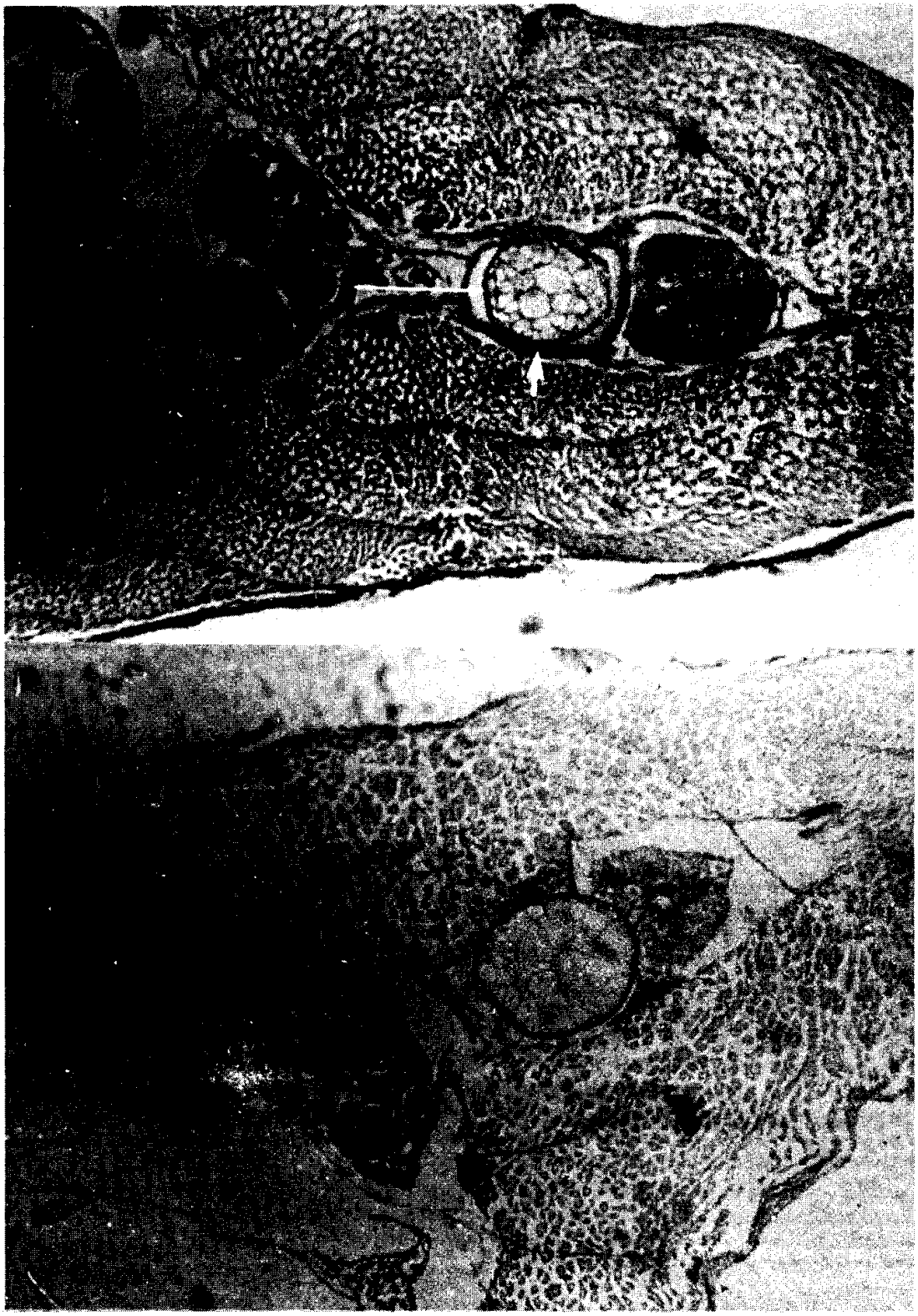

Fig. 1. Notochord in the normal fish showing the vacuolated structure that is invested by the thin primary sheath (small arrow) and the thick secondary sheath (big arrow) (x 100). Fig. 2. Notochord in the pesticide-treated fish showing the loss of the secondary sheath and an abnormally large size. (x 100)

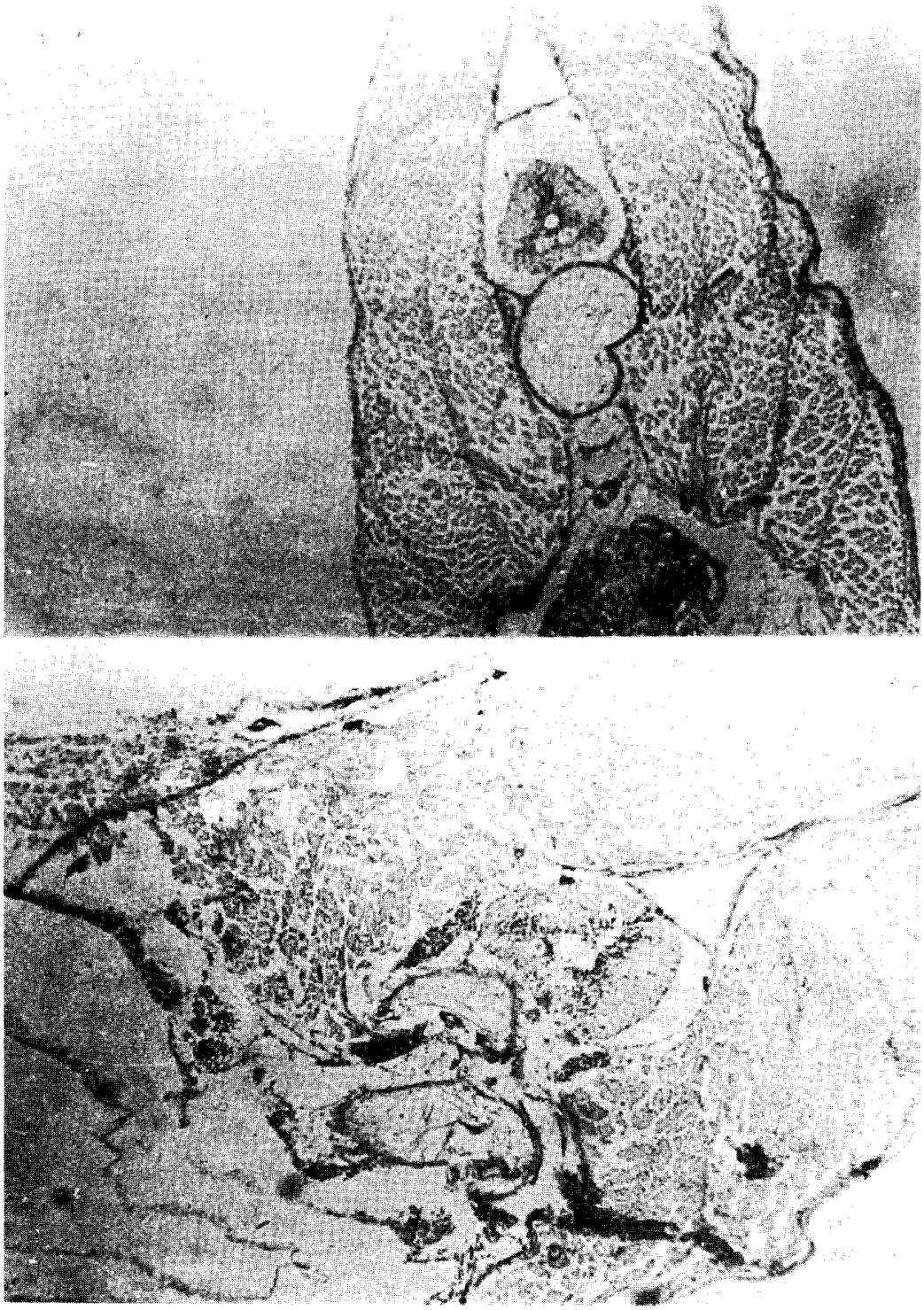

Fig. 3. Notochord in the pesticide-treated fish showing the absence of the secondary sheath and a constriction in the wall of the primary sheath (x 100). Fig. 4. Notochord in the pesticide-treated fish showing the absence of the secondary sheath and the presence of folded posterior (x 100).