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ACUTE TOXICITY OF PESTICIDE SEVIN (CARBAMATE) AND ITS EFFECTS ON THE OXYGEN CONSUMPTION OF JUVENILES OF THE TIGER PRAWN *PENAEUS MONODON* FABRICIUS

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ABSTRACT. Acute (96 hr) static bioassay experiments were designed to determine the median lethal concentration (LC_{50}) of a carbamate pesticide (Sevin) employing juveniles of the tiger prawn, *Penaeus monodon* Fabricius. The 96 hr LC_{50} value was 43 ppb (with 95% confidence limits of 30.71-60.20 ppb). On the basis of LC_{50} values, the juveniles were exposed to three different sublethal concentrations of the pesticide. The oxygen uptake by the juveniles of prawn was found to be depressed in all the three sublethal concentrations.

INTRODUCTION

Sevin (1-napthyl N-methyl carbamate), a synthetic carbamate insecticide widely used both in household and agricultural activities enters the aquatic environment passes through different trophic levels. Reports are available on the acute toxicity of Sevin on a variety of marine and estuarine crabs and shrimps.[3,18,20]. Previous investigations on the toxicity of Sevin and other pesticides to aquatic organisms indicate that penaeid prawns are more sensitive than fishes and molluscs[7]. In general carbamate pesticides have not been tested much in regard to penaeid prawns, but it is known that Sevin is lethal to other shrimps and crustaceans when applied to field sites in the marine environment[9].

Short term toxicity test results in a physiological stress of the organism and measurement of oxygen consumption had been frequently employed to evaluate changes in the metabolic rate. This provides an index of stress to organisms due to the presence of toxicants[2]. Reports on the pesticide toxicity to prawns in ralation to oxygen consumption and other respiratory physiology are limited. [3,15,16] In view of the general paucity of information on the toxicity of Sevin to tiger prawn *P. monodon* the present study was made to determine the acute (96 hr) toxicity of commercial grade Sevin and its effects in sublethal concentrations on the oxygen consumption of jeveniles of *P. monodon*.

MATERIALS AND METHODS

The juveniles of tiger prawn *Penaeus monodon* (50-60 mm from tip of the rostrum to tip of the telson) were chosen for the present study. Collections of juveniles were made in shallow inlets of Vellar estuary using a nylon hand seine. Acclimation and testing procedures were compatible with standard procedures[1]. Acute static bioassay experiments were conducted to determine the 96 hr median lethal concentration (96 hr LC_{50}) of the pesticide employing the method described by Sprague[17] and APHA[1]. During the period of experiments the juveniles were kept in fibreglass tanks containing filtered estuarine water with a salinity of 27±0.5% dissolved oxygen content of 3.9±0.2 ml/l and a temperature of 29±1°C. Commercial grade Sevin 50 WP (50% active ingradient and wetable powder, supplied by the Union Carbide India Limited, Calcutta) was dissolved in distilled water and desired concentrations were chosen for initial screening and further bisections of the concentrations used. Mortality data were collected after 96 hr exposure in each concentration and the median lethal concentration was calculated employing the method outlined by Litchfield and Wilcoxon[13].

Based on the LC_{50} values obtained, the juveniles were exposed to three different sublethal concentrations, viz., 0.001, 0.01 and 0.1 toxic units (1 toxic unit = 96 hr LC_{50}). The respiratory apparatus designed by Lingaraja *et al.*[12] was employed in the present study to determine the oxygen consumption (O_2/g wet weight of tissue/hr) of the animals. The modified Winkler's method[19] was used to determine the dissolved oxygen content.

RESULTS AND DISCUSSION

The LC_{50} value of Sevin for 96 hr exposure was 43 ppb with 95% confidence limits of 30.71-60.20 ppb. Earlier investigations on the acute toxicity of Sevin to crustaceans showed 2.5 ppb for brown shrimp *Penaeus aztecus*; 7 ppb for Korean shrimp *Palaemon macrodactylus*: 38 ppb for grass shrimp *Palaemon pugio*; 130 ppb for ghost shrimp *Callianassa californiensis*; 400 ppb for mud shrimp *Upogebia pugettensis*.[8,18,20] This differential action of pesticide might be due to species variation, size of the animal[11,14], and physicochemical factors.[5]

The oxygen consumption of juveniles of *P. monodon* exposed to three sublethal concentrations of Sevin showed a steady downward shift in relation to concentration and duration of exposure (Fig. 1). The

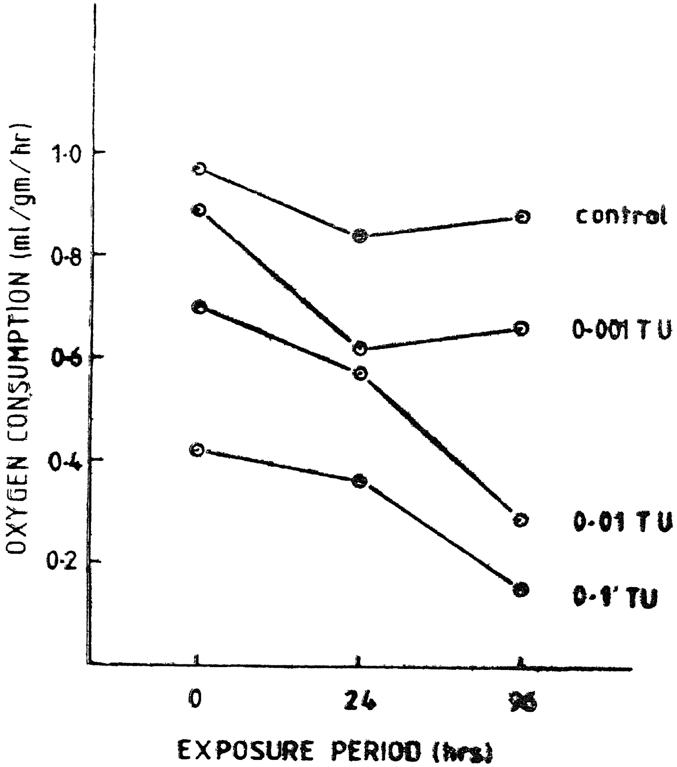

Fig. 1. Effect of Sevin on the rate of oxygen consumption of juveniles of *Penaeus monodon*.

decrease in O_2 consumption ranged from 8.25–56.70% within 24 hr of exposure, 26.19–57 14% after 24 hr and 25.00–82.95% after 96 hr exposures (Table 1). The minimum decrease was noticed in 0 001 TU

Table 1

Percentage of difference in rates of oxygen consumption in *P. monodon* juveniles in different concentrations from those of controls.

Concentration in ppb	Soon after exposure	24 hr after exposure	96 hr after exposure
0.001	8·25	26,19	25 00
0 01	27 84	32.14	78 41
0.1	56 70	57.14	82 95
Control rate of metabolism O_2 ml/g/hr	**0.969**	**0.840**	0.880

and the maximum in 0.1 TU. The mean basal rate of oxygen consumption of juveniles of *P. monodon* in the estuarine water (in control) was 0.896±0.05 ml O_2/g/hr and it decreased upto 0.310±0.115 ml O_2/g/hr in the concentration of 0.1 TU after 96 hr. Exposure of grass shrimp *Palaemonetes pugio* to 5–20 ppm Sodium pentachlorophenate (Carbaryl pesticide) exhibited a decline in oxygen consumption during post ecdysial period.[3,15] Sharp *et al.*[16] reported depressed oxygen consumption (below the control level) in the shrimp *Crangon franciscorum* at all concentrations of Kelthane used viz., 0·0, 0 089, 0.130, 0.208, 0·475, 0.843 mg/l. The decrease in oxygen consumption or mortality may be due to the accumulation of oxaloacetate or due to a disruption of the overall metabolic activity. Fox and Rao[6] reported that Na–PCP inhibits the activity of succinate dehydrogenase in the hepatopancrease of crab *Callinectes sapidus* which would inturn favour the build up of oxaloacetate and reduce the tissue oxygen uptake. Evidences both from *invivo* and *invitro* studies supports the same conclusion[4,10]. However, further studies on the cellular and tissue level changes caused by the toxicant may throw light towards a better understanding of the process.

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