



## Effects of Quinalphos 25 EC and Dimethoate 30 EC on Activities of AchE, Catalase, GOT and GPT in the Freshwater Prawn *Macrobrachium rosenbergii*

## KEYWORDS

Prawn, quinalphos, dimethoate, AChE, catalase, GOT, GPT

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## ABSTRACT

The commercially important freshwater prawn, *Macrobrachium rosenbergii* post larvae (PL),  $1.5 \pm 0.2$  cm and  $0.1 \pm 0.03$  g were subjected to static renewal type acute toxicity bioassays against two organophosphate insecticides, quinalphos (Ekalux EC 25) and dimethoate 30% EC (TAFGOR). The 96 hr  $LC_{50}$  values were determined to be  $0.774 \mu\text{g l}^{-1}$  for quinalphos and  $0.856 \text{ mg l}^{-1}$  for dimethoate. The PL were exposed to lethal (the 96 hr  $LC_{50}$ ) and sub-lethal ( $1/2^{\text{nd}}$  and  $1/4^{\text{th}}$  of the 96 hr  $LC_{50}$ ) concentrations of these insecticides (quinalphos:  $0.774$ ,  $0.384$  and  $0.193 \mu\text{g l}^{-1}$ ; dimethoate:  $0.856$ ,  $0.428$ ,  $0.214 \text{ mg l}^{-1}$ ) for a duration of 4, 8 and 12 days to study their acute and chronic impacts on whole body activities of enzymes, the neurotransmitter, acetyl cholinesterase (AChE), the antioxidant, catalase and metabolic enzymes, glutamate oxaloacetate transaminase (GOT) and glutamate pyruvate transaminase (GPT). The activity levels of AChE and catalase were found to be significantly ( $P < 0.05$ ) decreased in test prawns when compared with control, whereas, there were significant elevations in GOT and GPT levels ( $P < 0.05$ ). Among GOT and GPT, the impact was more on GOT than GPT. The dosage and time dependent manner of inhibition or elevation in activities of these enzymes were recorded. Among these two insecticides, quinalphos showed more impacts than dimethoate on this non-target organism.

## Introduction

Pesticides have tremendous benefits to man by increasing crop protection and thereby increasing food production, and controlling the vectors of man and animal diseases. They are transported over long distances by global circulation, and through run-off, find their way into aquatic systems. At the same time the pollution of freshwater ecosystem by chemical pesticides has become one of the most critical environmental problems (Northoff and William, 2004). This causes extensive damage to the activities of the living resources of food-web due to their toxicity, persistency with half-lives of decades and tendency to accumulate in the organisms (Joseph and Raj, 2010; Joseph et al., 2010; Joseph and Raj, 2011).

Quinalphos ( $\text{C}_{12}\text{H}_{15}\text{N}_2\text{O}_3\text{PS}$ ), O,O-diethyl O-quinoxalin-2-yl phosphorothioate, an ester of OP is used as insecticide and acaricide having a quick knock down effect through contact and stomach poisoning (David and Kumaraswami, 1988; Hassal, 1990). It is frequently used in many countries and represents a source of toxicity to humans and vertebrate animals (Kegley et al., 2010). Dimethoate ( $\text{C}_5\text{H}_{12}\text{NO}_3\text{PS}_2$ ), O,O-dimethyl S-[2-(methylamino)-2-oxoethyl] dithiophosphate is a broad-spectrum OP insecticide and acaricide exhibit both contact and systemic activity (David and Kumaraswami, 1988; Hassal, 1990). Its degradation by esterases and amidases are very low in insects as compared with those of mammals (Rose and Hodgson, 2004).

The effects of pesticides, such as endosulfan, carbaryl, dichlorvos, lindane, chlorpyrifos, monocrotophos, carbofuran and methomyl have been studied on acute toxicity (Bhavan et al., 1997a, b, 2008; Key and Fulton, 2006; Satapornvanit et al., 2009) and biochemistry (Bhavan and Geraldine, 1997, 2000a, b; 2001, 2002, 2004, 2007, 2009; Geraldine et al., 1999; Bhavan et al., 2011) of freshwater prawns. However, no data is available pertaining to quinalphos and dimethoate toxic-

ity induced changes on the activities of the neurotransmitter, acetyl cholinesterase (AChE), the enzymatic antioxidant, catalase and metabolic enzymes, glutamate oxaloacetate transaminase (GOT) and glutamate-pyruvate transaminase (GPT) in *Macrobrachium*. Therefore, it was necessitated to generate information on these pesticides in *Macrobrachium rosenbergii* to establish the potential of predictive biomarkers for use in water pollution monitoring.

## Materials and Methods

The post larvae of freshwater prawn, *M. rosenbergii* were purchased from Happy Bay Aqua Nova Hatchery, Mugaiyur, Marakanam Taluk, and Kancheepuram District, Tamilnadu, India. They were safely brought to the laboratory in polythene bags filled with hatchery water and well-oxygenated. They were stocked in large cement tank ( $6' \times 4' \times 3'$ ) and acclimatized for 2 weeks in ground water. During which they were fed with boiled egg albumin, *Artemia* nauplii and commercially available scampi crumble feed alternatively thrice a day. The excreta, unfed feed and exuvia if any were removed daily, three fourth of the water was renewed daily and adequately aerated.

Quinalphos (Ekalux EC 25) and dimethoate 30% EC (TAFGOR) were purchased from local agro service centre. Ten concentrations of each quinalphos ( $0.250$ - $1.375 \mu\text{g l}^{-1}$ ) and dimethoate ( $0.060$ - $1.350 \text{ mg l}^{-1}$ ) were prepared by mixing in distilled water afresh on every day. Post larvae (PL) of *M. rosenbergii* ( $1.5 \pm 0.2$  cm and  $0.1 \pm 0.03$ g) were transferred to plastic aquaria of 10 l capacity (each with 10 PL) with ground water for 2 days. Out of eleven groups one PL group was served as control and others were exposed to ten different known concentrations (quinalphos:  $0.250$ - $1.375 \mu\text{g l}^{-1}$ ; dimethoate:  $0.060$ - $1.350 \text{ mg l}^{-1}$ ) of each insecticide for 96 hours to assess their  $LC_{50}$  value as per the guidelines prescribed by ASTM (1980). The experiment was conducted

in triplicates. The toxic water medium was renewed daily by siphoning method, causing minimum disturbance to the prawns and freshly prepared concentrations of quinalphos and dimethoate were added separately to maintain the toxic level in a steady state. During the experiment the prawns were neither fed nor aerated. The concentrations and their respective mortality percentage were subjected to computation for calculation of the median lethal concentration. The 96 h LC<sub>50</sub> value with 95% confidence limits was assessed using computerized program of Finney (1971) method of probit analysis.

Based on 96 hr LC<sub>50</sub> values of quinalphos and dimethoate, three concentrations each for quinalphos (0.774 µg l<sup>-1</sup>, 0.384 µg l<sup>-1</sup> and 0.193 µg l<sup>-1</sup>) and dimethoate (0.856 mg l<sup>-1</sup>, 0.428 mg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup>) were selected for treatment during 12 days. A common control was also maintained. Each group comprised 5 aquaria (15 l capacity) and each aquarium housed 20 PL. Sampling was done on day 4, 8 and 12. The entire quantity of medium in each aquarium was gently siphoned out daily and replaced by medium containing freshly prepared concentrations of quinalphos and dimethoate with minimal disturbance to the prawns. During the period of the experiment, the toxic medium was not aerated and the animals were fed with commercial scampi feed. The dead post larvae prawns were removed during the experiment. The activities of enzymes in post larvae prawns, such as AChE (Ellman et al., 1961), catalase (Sinha et al., 1972), and GOT and GPT (Reitman and Frankel, 1957) were assayed on 4<sup>th</sup>, 8<sup>th</sup> and 12<sup>th</sup> day of exposure by sacrificing the test PL in each group. Control prawns were similarly assayed at the same time as the test prawns. The differences between control and pesticide exposed groups were analyzed by adopting student-'t' test using SPSS software (version 16.0). All measurements were performed in triplicates and the results are expressed as mean ± SD of three individual observations. P<0.05 was fixed to assess the statistical significance.

### Results and Discussion

The 96 hr LC<sub>50</sub> of quinalphos and dimethoate for *M. rosenbergii* PL was assessed to be 0.774 µg l<sup>-1</sup> and 0.856 mg l<sup>-1</sup> respectively (Tables 1 and 2). During bio-assay tests, the mortality of PL was found to be increased in response to higher concentrations of quinalphos and dimethoate (Tables 1 and 2). A comparison of the 96 hr LC<sub>50</sub> values assessed in the present study revealed that quinalphos was >1000 fold more toxic than that of dimethoate to *M. rosenbergii* PL (Tables 1 and 2). Therefore, it is clear that *M. rosenbergii* was more sensitive to quinalphos toxicity than that of dimethoate. The toxicity of quinalphos and dimethoate caused severe metabolic distress, which was evident from the escaping tendency of test PL from the aquaria and such behavior was based on dosage of these pesticides, which eventually leads to death of test PL.

Available literature revealed that the 96 hr LC<sub>50</sub> values reported for quinalphos in another species of freshwater prawn, *Macrobrachium lamarrai* (0.461 mg l<sup>-1</sup>) was many more times higher toxic (Omkar and Shukla, 1985) when compared with the result observed in the present study, 0.774 µg l<sup>-1</sup> (Table 1). In the case of dimethoate, the reported value of 96 hr LC<sub>50</sub> (540 µg l<sup>-1</sup> (0.540 mg l<sup>-1</sup>)) in a marine crustacean species, the opossum shrimp, *Neomysis integer* (Roast et al., 1999) was closer to the result recorded in the present study, 0.856 mg l<sup>-1</sup> (Table 2). However, in a report on the freshwater shrimp, *Paratya australiensis*, the reported 96 hr LC<sub>50</sub> value of dimethoate (8.00 µg l<sup>-1</sup> (0.008 mg l<sup>-1</sup>)) by Kumar et al., (2010) was 100 times lower than that of the value observed in the present study (Table 2). It is important to mention here that toxicity of a xenobiotic is governed by many factors, like water temperature, purity of the toxin, life stage of an organism, size of the individual etc.

The activity of AChE was found to be significantly (P<0.05) lower in test PL on all sampling days irrespective of concen-

trations of quinalphos and dimethoate when compared with control (Table 3). However, maximum inhibition was seen on day-12 in 0.774 µg l<sup>-1</sup> concentration of quinalphos (58.03%) and 0.856 mg l<sup>-1</sup> concentration of dimethoate (54.40%) followed by 0.384 µg l<sup>-1</sup> and 0.193 µg l<sup>-1</sup> concentrations of quinalphos (54.14 & 49.74%) and 0.428 mg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup> concentration of dimethoate (51.81 & 48.96%). Among the two pesticides, quinalphos showed maximum inhibition in AChE activity than dimethoate (quinalphos: 40.48-58.03% inhibition; dimethoate: 36.68-54.40% inhibition).

Similar inhibition in AChE activity has been reported in the grass shrimp, *Palaemonetes pugio* embryos exposed to OP pesticides, chlorpyrifos and malathion (Lund et al., 2000), in *Macrobrachium malcolmsonii* exposed to dichlorvos, endosulfan, and carbaryl (Geraldine et al., 1999; Bhavan and Geraldine, 2001, 2002), in the freshwater shrimp *Paratya traliensis* exposed to dimethoate (Kumar et al., 2010), in the clam, *Ruditapes decussatus* exposed to malathion (Nadji et al., 2010), in the Riceland prawn, *Macrobrachium lanchesteri* on exposure to chlorpyrifos (Tongbai and Damrongphol, 2011) and in freshwater fairy shrimp, *Streptocephalus dichotomus* exposed to malathion and glyphosate (Kumar and Ali, 2013). The inhibition of AChE recorded in *M. rosenbergii* PL indicates impairment in hydrolysis of ACh, which suggests disruption of synaptic transmission in the cholinergic system.

The activity of catalase was found to be significantly (P<0.05) lower in test PL on all sampling days irrespective of concentrations of pesticides when compared with control (Table 3). However, maximum inhibition was seen on day-12 in 0.774 µg l<sup>-1</sup> concentration of quinalphos (19.15%) and 0.856 mg l<sup>-1</sup> concentration of dimethoate (17.70%) followed by 0.428 mg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup> concentration of dimethoate (16.78 & 15.07%) and 0.384 µg l<sup>-1</sup> and 0.193 µg l<sup>-1</sup> concentrations of quinalphos (15.26 & 12.36%). Among the two pesticides, quinalphos showed maximum inhibition (12.08-19.15%) of catalase activity than that of dimethoate (8.62-17.7%) in *M. rosenbergii* PL when exposed to 0.774 µg l<sup>-1</sup> of quinalphos and 0.856 mg l<sup>-1</sup> of dimethoate (lethal concentrations of each pesticide). In the cases of sub lethal concentrations (0.384 µg l<sup>-1</sup> and 0.193 µg l<sup>-1</sup> of quinalphos, and 0.428 mg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup> of dimethoate) just the reverse was seen, the maximum inhibition of catalase was recorded in dimethoate (6.58-16.78%), than that of quinalphos (5.00-15.26%).

Catalase is a sensitive antioxidant biomarker against oxygen free radicals, the reactive oxygen species generated due to oxidative stress (Regoli et al., 2004; Atli and Canli, 2007). In the present study, due to toxicity of quinalphos and dimethoate excessive hydrogen peroxide or superoxide radical may have produced, which in turn inactivated the catalase activity (Table 3). Therefore, the protective mechanism was hampered in test PL even at lower sub lethal level of quinalphos and dimethoate (0.193 µg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup> respectively). OP inhibited catalase activity due to oxidative damage has also been reported in the brackish water prawn *Penaeus monodon* exposed to fenvalerate (Vijayavel and Balasubramanian, 2009), in the clam, *Ruditapes decussates* (Nadji et al., 2010) and in freshwater fish, *Labeo rohita* (Thenmozhi et al., 2011) exposed to malathion.

The activities of GOT (aspartate aminotransferase, ASAT) and GPT (alanine aminotransferase, ALAT) were found to be significantly (P<0.05) higher in test PL on all sampling days irrespective of concentrations of pesticides when compared with control (Table 3). However, maximum elevation in these enzymes activities was seen on day-12 in 0.856 mg l<sup>-1</sup> concentration of dimethoate and 0.774 µg l<sup>-1</sup> concentration of quinalphos followed by 0.428 mg l<sup>-1</sup> and 0.214 mg l<sup>-1</sup> concentration of dimethoate and 0.384 µg l<sup>-1</sup> and 0.193 µg l<sup>-1</sup> concentrations of quinalphos. Among the two pesticides, dimethoate showed maximum elevations in GOT and GPT activities than that of quinalphos (dimethoate-GOT: 6.06-25.10%; quinalphos-GOT: 3.25-17.95%; dimethoate-GPT: 24.39-29.98%;

quinalphos-GPT: 1.29-23.29%). Similar elevation in activities of GOT and GPT has also been reported in the marine shrimp, *Litopenaeus vannamei* exposed to DDT, lorsban, diazinon, folidal, guzation and lindane (Galindo-Reyes et al., 2000) and in the green mussel, *Perna viridis* exposed to copper, lead and zinc (Anand et al., 2010).

Increases or decreases in GOT and GPT activity levels are suggested as reflection of tissue damage or organ disfunction (Oluah, 1999; Rao, 2006). In the present study, the increase recorded in GOT activity suggests that an important reaction of the molecular rearrangement involving amino acids linked to the citric acid cycle at two points (oxaloacetic and ketoglutaric acids) that GOT catalyzes was affected. Similarly, the increase in GPT indicates the fact that the test PL terribly required intensive glycogenesis to coop-up the severe energy crisis occurred due to quinalphos and dimethoate toxic stress.

In conclusion, quinalphos and dimethoate exhibited dose and time dependent responses to coop-up with toxic stress, and thus, adversely modulate the activities of AChE, catalase, GOT and GPT in *M. rosenbergii*. Therefore, these enzymes can be taken as biomarkers for monitoring water pollution by these pesticides in natural environment.

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**Table 1: 96 h LC<sub>50</sub> evaluation of quinalphos on *M. rosenbergii* PL**

Quinalphos (µg l <sup>-1</sup> )	Observed Mortality			Mortality (mean)	Mortality (%)	LC <sub>50</sub> (µg l <sup>-1</sup> )	95% confidence limit	
	T1	T2	T3				Upper (µg l <sup>-1</sup> )	Lower (µg l <sup>-1</sup> )
Control	0	0	0	0.00	0.0	0.774	0.826	0.721
0.250	0	0	0	0.00	0.0			
0.375	1	1	1	1.00	10.0			
0.500	2	3	3	2.66	26.6			
0.625	3	4	3	3.33	33.3			
0.750	5	5	5	5.00	50.0			
0.875	6	6	6	6.00	60.0			
1.000	7	7	7	7.00	70.0			
1.125	8	8	9	8.33	83.3			
1.250	10	9	10	9.66	96.6			
1.375	10	10	10	10.00	100.0	--	--	--

T1, T2, and T3 represent triplicates of exposure each with ten numbers of *M. rosenbergii* PL

**Table 2: 96 h LC<sub>50</sub> evaluation of dimethoate on *M. rosenbergii* PL**

Dimethoate (mg l <sup>-1</sup> )	Observed Mortality			Mortality (mean)	Mortality (%)	LC <sub>50</sub> (mg l <sup>-1</sup> )	95% confidence limit	
	T1	T2	T3				Upper (mg l <sup>-1</sup> )	Lower (mg l <sup>-1</sup> )
Control	0	0	0	0.00	0.0	0.856	0.891	0.822
0.060	0	0	0	0.00	0.0			
0.150	1	0	0	0.33	3.3			
0.300	1	1	0	0.66	6.6			
0.450	2	3	0	1.66	16.6			
0.600	3	4	1	2.66	26.6			
0.750	4	4	3	3.66	36.6			
0.900	5	5	5	5.00	50.0			
1.050	6	7	7	6.66	66.6			
1.200	7	8	9	8.00	80.0			
1.350	9	9	10	9.30	93.0	--	--	--

T1, T2, and T3 represent triplicates of exposure each with ten numbers of *M. rosenbergii* PL

**Table 3: Activities of AChE, catalase, GOT and GPT in *M. rosenbergii* PL exposed to lethal and sub lethal concentrations of quinalphos and dimethoate**

Enzyme	Day	Control	Lethal and sub lethal concentrations of pesticide					
			Quinalphos (µg l <sup>-1</sup> )			Dimethoate (mg l <sup>-1</sup> )		
			Lethal	Sub lethal		Lethal	Sub lethal	
			0.774	0.384	0.193	0.856	0.428	0.214
AChE (µmol/ min <sup>-1</sup> / mg protein <sup>-1</sup> )	4	3.68±0.16	1.97±0.13 (46.46↓)	2.04±0.08 (44.56↓)	2.19±0.07 (40.48↓)	2.17±0.16 (41.03↓)	2.23±0.09 (39.40↓)	2.33±0.04 (36.68↓)
	8	3.77±0.05	1.76±0.27 (53.31↓)	1.97±0.23 (47.74↓)	2.00±0.13 (46.94↓)	2.01±0.04 (46.68↓)	2.08±0.10 (44.82↓)	2.11±0.14 (44.03↓)
	12	3.86 ±0.13	1.62±0.12 (58.03↓)	1.77±0.16 (54.14↓)	1.94±0.19 (49.74↓)	1.76±0.10 (54.40↓)	1.86±0.12 (51.81↓)	1.97±0.10 (48.96↓)
Catalase (H <sub>2</sub> O <sub>2</sub> consumed/ min <sup>-1</sup> / mg protein <sup>-1</sup> )	4	36.74±0.36	32.30±1.13 (12.08↓)	34.39±0.45 (6.39↓)	34.90±0.24 (5.00↓)	33.57±0.69 (8.62↓)	33.77±0.44 (8.08↓)	34.32±0.50 (6.58↓)
	8	37.25±0.25	31.67±0.75 (14.97↓)	33.49±1.165 (10.09↓)	34.53±0.50 (7.30↓)	31.72±1.06 (14.84↓)	32.54±1.72 (12.64↓)	33.50±1.21 (10.06↓)
	12	38.00±0.15	30.72±0.40 (19.15↓)	32.20±0.82 (15.26↓)	33.30±0.57 (12.36↓)	31.27±1.0 (17.7↓)	31.62±1.61 (16.78↓)	32.27±1.25 (15.07↓)

GOT (IU <sup>-1</sup> )	4	26.72±0.57	29.15±1.02 (9.09↑)	28.38±0.77 (6.21↑)	27.59±0.59 <sup>NS</sup> (3.25↑)	31.20±0.53 (16.76↑)	30.22±0.71 (13.09↑)	28.34±0.07 (6.06↑)
	8	27.70±0.60	31.53±0.83 (13.8↑)	30.42±0.71 (9.81↑)	29.37±0.82 (6.02↑)	33.23±0.52 (19.96↑)	31.63±0.83 (14.18↑)	29.44±0.62 (6.28↑)
	12	27.45±0.54	32.38±0.90 (17.95↑)	30.41±0.61 (10.78↑)	29.80±0.39 (8.56↑)	34.34±0.75 (25.10↑)	33.66±1.18 (22.62↑)	30.83±0.87 (12.31↑)
GPT (IU <sup>-1</sup> )	4	23.17±0.11	26.59±0.98 (14.76↑)	24.45±0.20 (5.52↑)	23.47±0.81 <sup>NS</sup> (1.29↑)	28.44±0.15 (22.74↑)	26.40±0.28 (13.94↑)	24.39±0.38 (5.26↑)
	8	23.24±0.21	27.66±1.01 (19.01↑)	25.46±0.68 (9.55↑)	24.54±0.75 <sup>NS</sup> (5.59↑)	29.65±0.13 (27.58↑)	27.23±0.13 (17.16↑)	25.65±0.43 (10.37↑)
	12	23.31±0.17	28.74±1.09 (23.29↑)	26.37±0.27 (13.12↑)	25.48±0.41 (9.30↑)	30.30±0.11 (29.98↑)	28.54±0.27 (22.43↑)	26.62±0.18 (14.19↑)

Each value is mean ± SD of 3 individual observations.  
Values in parentheses are % increase (↑)/ decrease (↓).

Values are significant at P < 0.05.  
<sup>NS</sup>, Not significant statistically.

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