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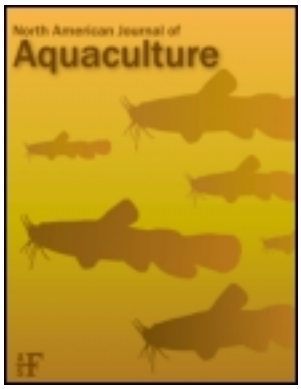

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ARTICLE

# Toxicity of Rotenone to Giant River Freshwater Prawn *Macrobrachium rosenbergii*

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***Abstract***

**Aquaculturists have often suffered predation losses in the production of freshwater giant river prawn *Macrobrachium rosenbergii* due to the presence of wild fish species in culture ponds. The piscicide rotenone is widely used to remove undesirable fish species from ponds. Although evidence in the technical literature suggests that crustaceans generally have a higher tolerance to rotenone than fish, there are currently no data on the acute or chronic toxicity of rotenone to juvenile freshwater prawns. In this study, two static acute-toxicity bioassays (96 h) were conducted using Prentox Prenfish (5% active ingredient) rotenone to determine the median lethal concentration (LC50) for juvenile freshwater prawns (average weight = 0.55 g, SD = 0.25; length = 41.43 mm, SD = 6.45). In bioassay 1, prawns were exposed to rotenone concentrations of 1.0, 3.0, 5.0, and 10.0 mg/L. In bioassay 2, prawns were exposed to rotenone concentrations of 2.2, 3.6, 6.0, 10.0, and 16.7 mg/L. All rotenone concentrations used in the study were based on the total product of the commercial rotenone formulation. The LC50 calculated in bioassay 1 was 6.2 mg/L, and the LC50 calculated in bioassay 2 was 7.5 mg/L. Freshwater prawns were able to tolerate 3.0 to 3.6 mg/L of rotenone with no mortality or apparent adverse effects during the study. Prawns held at the end of each bioassay for 5 d showed no signs of delayed effects from rotenone exposure. Data from this study indicate that juvenile prawns should be able to tolerate the concentrations of rotenone required to eradicate certain problematic wild fish species.**

The giant river prawn *Macrobrachium rosenbergii* is a popular aquaculture species. Since 2002 prawn culture has increased dramatically (FAO 2007), and in 2008 the global prawn industry produced 208,000 metric tons, worth US$1 billion (New 2010). China is currently the largest producer of *M. rosenbergii* at 124,520 metric tons (t), followed by Thailand (27,650 t), India (27,262 t), and Bangladesh (23,240 t; FAO 2009). In tropical regions prawns can be raised all year; however, in temperate zones the grow-out period is limited to 3 to 5 months due to seasonal low temperatures (D'Abramo et al. 2006). During the

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months when ponds are not in production, wild fish can be inadvertently introduced in various ways, such as filling the ponds from source waters and flooding.

Wild fish should be removed from ponds prior to stocking prawns. However, unwanted fish species can be introduced into culture ponds even after prawns have been stocked. Wild fish are problematic because they can compete with prawn for natural foods (D'Abramo et al. 2006) and prey on small juvenile prawns (Valenti and New 2000), negatively impacting prawn production and reducing financial returns for farmers.

Chlorine is frequently used in Asia to eliminate undesirable finfish species from culture ponds. Unfortunately, it is difficult to find the correct concentration of free chlorine that will kill wild fish because it oxidizes organic matter in the pond and only the remaining residual chlorine kills the undesirable species (Husnah and Lin 2002). Saponin in tea seed cake has also been used as a piscicide in prawn culture ponds. Chen et al. (1996) reported that the 96-h LC50 value of kuruma prawn *Penaeus japonicus* to saponin was 18.1 mg/L. Also, chlorine and tea seed cake are not approved in some countries (such as the USA) for piscicidal use (Finlayson et al. 2000). Antimycin A can be used as a piscicide in culture ponds (USEPA 2007a), but it is expensive to use for total finfish removal (Smith 1972).

Rotenone is the most widely used fish poison used for assessment, management, and eradication of fish, including undesirable species (Bettoli and Macenia 1996; McClay 2000; Ling 2003). Prentox Prenfish rotenone is a commercial piscicide that is commonly used to remove undesirable fish species from lakes and ponds (Turner et al. 2007). Rotenone causes mortality by inhibiting cellular respiration within the mitochondria, thereby preventing the reduction–oxidation reaction of NADH within complex 1 of the electron transport system (Singer and Ramsay 1994; USEPA 2007b). In the later stages of toxicosis, fish experience respiratory paralysis (Perry and Conway 1977; Hyatt 2004) then death due to tissue hypoxia of the heart and nervous system (Ling 2003).

Information on the toxicity of rotenone to crustaceans is limited, and no published information was found specific to *M. rosenbergii*. Toxicity studies preformed on crustaceans and fish suggest that under similar environmental conditions, rotenone is usually more lethal to fish than to crustaceans. This could potentially allow producers to selectively remove unwanted fish species without harming the prawn crop. The objective of this study was to evaluate the 96-h static acute toxicity of a commercial grade formulation of rotenone to juvenile giant river prawns and to provide toxicity data to prawn producers.

## METHODS

The study was conducted at the Aquaculture Research Center, Kentucky State University, Frankfort. Prentox Prenfish rotenone (Prentiss, Sandersville, Georgia) with 5% active ingredient (a.i.) was used. The emulsified formulation of Prentox Prenfish was utilized because it is known to provide more consistent results than the powder formulations (Wujtewicz and Petrosky 1997). Concentrations of Prentox Prenfish rotenone were based on, and presented in terms of, the total formulation and not on the active ingredient. Prawn juveniles weighing an average of 0.55 g (SD, 0.25) and 41.43 mm (SD, 6.45) in length (tail to rostrum) were obtained from a commercial supplier (Aquaculture of Texas, Weatherford, Texas). All animals were acclimated to dilution water (tap water dechlorinated by carbon filtration; USEPA 2002) and held for 5–10 d prior to rotenone exposure. During this quarantine period, animals were kept under conditions of fluorescent lighting (15 h light : 9 h dark) and fed 2% of their body weight using Otohime larval feed 55% protein (Aquatic Eco Systems, Apopka, Florida) daily. However, the animals were not fed 24 h before each bioassay or during each 96-h toxicity test.

For bioassay 1, 20 glass bowls (10 L) were partially submerged in two fiberglass tanks (236 × 102 × 15 cm) filled with tap water (28°C) to provide a consistent temperature. Each glass bowl was filled with 7 L of dilution water (tap water dechlorinated by carbon filtration; USEPA 2002). Sufficient quantities of Prentox Prenfish were pipetted directly into the glass bowls to achieve the following final concentrations in milligrams per liter (mg/L) of 0.0 (control), 1.0, 3.0, 5.0, and 10.0 mg/L. The Prentox Prenfish was homogeneously mixed throughout the water column by gentle aeration. There were four replicates of each concentration of Prentox Prenfish, and all glass bowls were aerated throughout the bioassay. Juvenile prawns were stocked six animals per glass bowl, and each prawn was placed inside a cylindrical ventilated container (10.2 × 3.8 cm; Aquatic Eco Systems) to prevent cannibalism during the bioassay. Juvenile prawns were placed into each glass bowl within 30 min after the rotenone was added (ASTM 2002). A total of 120, 66-d nursed juvenile prawns were used.

For bioassay 2, the toxicity test was carried out with the same basic methods and conditions as bioassay 1 except that a total of 108 71-d nursed juvenile prawns were used and there were three replications per treatment. Prentox Prenfish was pipetted directly into the glass bowls in the following concentrations: 0.0 (control), 2.2, 3.6, 6.0, 10.0, and 16.7 mg/L. Juvenile prawns were exposed to different concentrations of Prentox Prenfish 5% a.i. rotenone during bioassay 1 and bioassay 2 at a range of concentrations estimated to result in a range of mortalities from 0% to 100%. During each bioassay, the animals were evaluated at the following times posttreatment: 0, 3, 6, 12, 24, 48, 72, and 96 h. Death was defined as "no movement after gentle stimulation of the legs with a probe" (Jarboe and Romaire 1991), and dead prawns were removed when observed.

Dilution water (tap water dechlorinated by carbon filtration; USEPA 2002) used in this study was measured for various water quality parameters. Dissolved oxygen (DO), pH, and temperature were recorded for each tank three times per day. Alkalinity, hardness, nitrite, total ammonia nitrogen (TAN), and un-ionized ammonia were measured at the beginning and at the

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conclusion of each bioassay (ASTM 2002). Dissolved oxygen was measured using a YSI 550A dissolved oxygen meter (YSI Instruments, Yellow Springs, Ohio), pH was analyzed by Waterproof pH Tester 20 (Oakton Instruments, Vernon Hills, Illinois), and the temperature was monitored using an ASTM brand thermometer (Fisher Scientific, Pittsburgh, Pennsylvania). The nitrite, TAN, and un-ionized ammonia were analyzed using an Odyssey DR/2500 spectrophotometer (HACH, Loveland, Colorado). A digital titrator was used to measure the alkalinity and hardness (HACH).

At the end of each bioassay, randomly selected groups of prawns (10 in bioassay 1, 8 in bioassay 2) that survived the exposure to rotenone were placed into clean cylindrical ventilated containers (10.2 × 3.8 cm; Aquatic Eco Systems). These animals were held in freshwater, fed, and observed for 5 d to determine whether delayed effects from rotenone exposure occurred. No statistical analysis was performed to detect differences between treatments.

The mortality data generated from each bioassay were analyzed with Probit analysis using PoloPlus version 2.0 (LeOra Software, Petaluma, California) to determine the 96-h median lethal concentrations (LC50) and the 95% confidence limits for Prentox Prenfish rotenone exposure during each toxicity test. A linear regression was used to characterize the relationship between Prentox Prenfish rotenone concentrations and prawn survival in bioassay 1 using Statistix Analytical Software, version 9.0 (Tallahassee, Florida). Another linear regression was used in bioassay 2 to characterize the relationship between Prentox Prenfish rotenone concentrations and prawn survival using Statistix Analytical Software, version 9.0. All data were tested based on $\alpha$ equal to 0.05.

## RESULTS

During quarantine, 4% mortality occurred. The water quality parameters measured during Bioassay 1 (Table 1) were within the ideal range for *M. rosenbergii* (Boyd and Zimmermann 2000). The 96-h LC50 value calculated in bioassay 1 was 6.2 mg/L (the lower boundary was 5.1 mg/L and the upper boundary was 7.8 mg/L, based on the 95% confidence interval; Figure 1). Figure 2 shows the percentage of prawns alive in each concentration after the 96-h exposure to rotenone. At concentrations of 0, 1.0, and 3.0 mg/L of Prentox Prenfish rotenone, there was 100% survival. The linear regression was highly statistically significant ($P$-value $< 0.01$) indicating a negative relationship between prawn survival and rotenone concentration, and that relationship was well described by a straight line. The $R^2$-value of 0.80 indicates that 80% of the variation in prawn survival in bioassay 1 was accounted for by Prentox Prenfish rotenone concentration.

Table 1 lists the water quality parameters measured during bioassay 2. The 96-h LC50 value calculated in bioassay 2 was 7.5 mg/L (the lower boundary was 6.5 mg/L and the upper boundary was 8.7 mg/L, based on the 95% confidence interval;

TABLE 1. Water quality parameters for bioassays in which the 96-h static acute toxicity of Prentox was determined for giant river prawns.

| Parameter | Bioassay 1 | Bioassay 2 |
|---|---|---|
| pH | 8.1 ± 0.1 | 8.1 ± 0.1 |
| Temperature (°C) | 25.0 ± 0.3 | 25.6 ± 0.5 |
| Dissolved oxygen (mg/L) | 7.8 ± 0.2 | 8.3 ± 0.2 |
| Alkalinity (mg/L) | 70.0 ± 3.7 | 70.1 ± 11.0 |
| Hardness (mg/L) | 178.5 ± 7.0 | 155.1 ± 34.7 |
| Total ammonia nitrogen (mg/L) | 0.2 ± 0.2 | 0.2 ± 0.1 |
| Un-ionized ammonia (mg/L) | 0.0 ± 0.0 | 0.0 ± 0.0 |

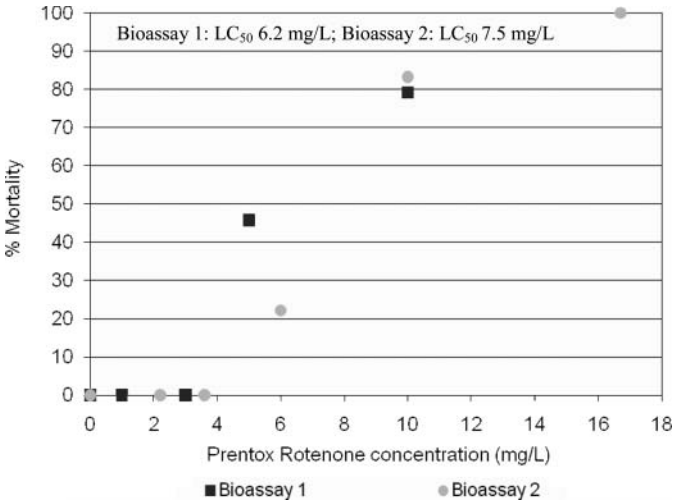


FIGURE 1. Mortality of juvenile giant river prawns in bioassays 1 and 2 after a 96-h exposure to various concentrations of 5% a.i. Prentox Prenfish rotenone. The values along the *x*-axis are milligrams of active ingredient per liter.

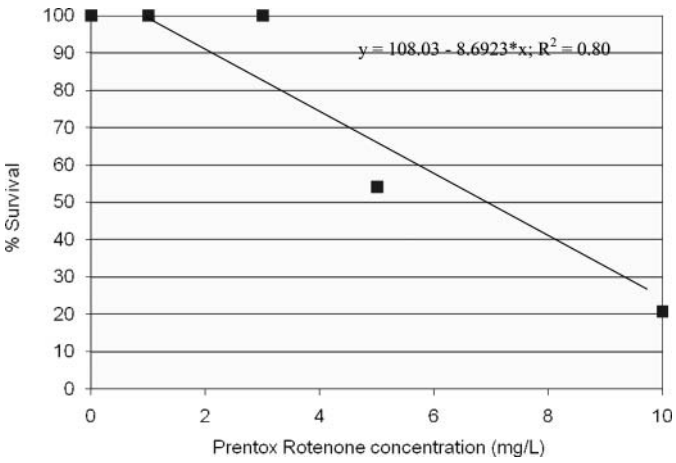


FIGURE 2. Relationship between the survival of juvenile giant river prawns in bioassay 1 and 96-h exposure to various concentrations of 5% a.i. Prentox Prenfish rotenone.

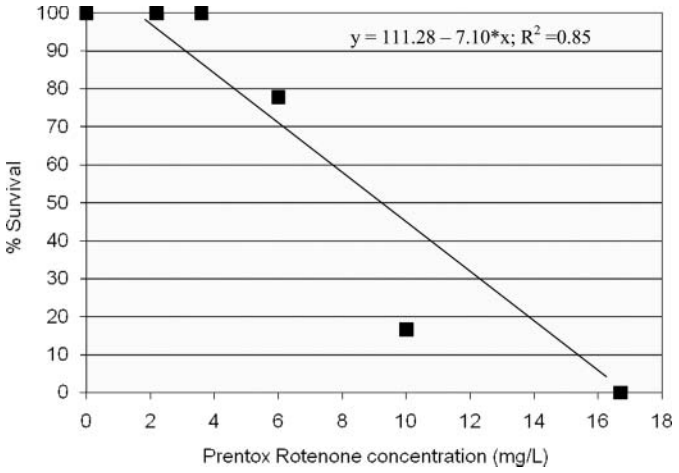


FIGURE 3. Relationship between the survival of juvenile giant river prawns in bioassay 2 and 96-h exposure to various concentrations of 5% a.i. Prentox Prenfish rotenone.

Figure 1). Figure 3 shows the percent of prawns alive in each concentration after the 96-h exposure to rotenone. In treatments of 0, 2.2, and 3.6 mg/L, there was 100% survival. The linear regression was highly statically significant ($P$-value $< 0.01$), indicating a negative relationship between prawn survival and rotenone concentration, and that relationship was again well described by a straight line. The $R^2$-value of 0.85 generated from the linear regression indicated that 85% of the survival in bioassay 2 was accounted for by rotenone concentration. The prawns held at the end of bioassay 1 and bioassay 2 for 5 d survived and showed no signs of delayed effects from rotenone exposure.

## DISCUSSION

The results from this study indicate that juvenile giant river prawns had 96-h LC50 values of 6.2 mg/L (bioassay 1) and 7.5 mg/L (bioassay 2) when exposed to Prentox Prenfish rotenone. These values are higher than those reported for white river crayfish *Procambarus acutus acutus* (4.0 mg/L; Wujtewicz and Petrosky 1997) exposed to the total product of the commercial formulation 5% a.i. rotenone. Neither the juvenile prawns in bioassay 1 nor the white river crayfish (Wujtewicz and Petrosky 1997) showed any signs of adverse toxic effects when exposed to a 3.0 mg/L-concentration of commercial rotenone (concentration based upon the total commercial formulation of 5% a.i. rotenone). Prawn LC50 values were higher than those reported for numerous fish species exposed to the total product of a commercial formulation containing 5% a.i. rotenone. For instance, channel catfish *Ictalurus punctatus* had an LC50 value of 0.2 mg/L (Marking and Bills 1976); and both bluegill *Lepomis macrochirus* and largemouth bass *Micropterus salmoides* had LC50 values of 0.1 mg/L (Howland 1969; Marking and Bills 1976); silver carp *Hypophthalmichthys molitrix*, bighead carp *H. nobilis*, and common carp *Cyprinus carpio* had similar LC50 values, ranging from 0.07 to 0.05 mg/L (Marking and Bills 1976; Rach et al. 2009). The technical literature indicates that unwanted fish species commonly found in prawn ponds have a lower tolerance to rotenone than juvenile prawns in laboratory studies.

Prawn producers wish to kill 100% of the undesirable fish species in a pond. Kinney (1968) reported that a pond treatment of 0.5–2.0 mg/L of 5% a.i. commercial rotenone (treatment based upon the total commercial product) was required to eliminate 100% of finfish species. In this current laboratory study, juvenile giant river prawns showed no signs of adverse effects when exposed to rotenone concentrations higher than the amount required to eliminate 100% of fish species commonly found in prawn ponds. Further testing of Prentox Prenfish rotenone under field conditions is necessary to definitively verify the differential toxicity of rotenone to freshwater prawns and nuisance fish species. Although the prawns showed no signs of adverse effects when exposed to sublethal levels of Prentox Prenfish, there is evidence in the literature that rotenone causes shell softening in tiger shrimp *Penaeus monodon* when exposed to sublethal levels (Cruz-Lacierdia 1993) of rotenone. An experimental field trial should be conducted with Prentox Prenfish to determine its relative toxicity to finfish and juvenile freshwater prawn prior to recommending its use in a prawn production pond.

## CONCLUSIONS

The results of this study indicate that juvenile giant river prawns can tolerate concentrations of rotenone (3.0 to 3.6 mg/L) that are lethal to several wild fish species (that die at concentrations $\leq$2.0 mg/L). Differential toxicity could allow unwanted fish species to be eliminated from active prawn production ponds without affecting prawn survival. Further testing of this compound under field conditions is necessary to validate the differential toxicity of rotenone to freshwater prawns and nuisance fish species.

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