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## Chapter 16

# Ecotoxicological Risks of Potential Toxicants for Brown Tree Snake Control on Guam

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## INTRODUCTION

The brown tree snake (*Boiga irregularis*) is a nocturnal, arboreal, rear-fanged, mildly venomous, colubrid snake which can reach lengths of up to 2.3 m and weigh as much as 2 kg(1). Originally, the species' range included the northern and eastern coasts of Australia, Papua New Guinea and nearby islands (2). It is believed that sometime in the 1950's, that snakes were inadvertently transported from New Guinea to Guam, where they proliferated (3). By the mid-1960's, marked decreases in Guam's bird life were observed. By the mid-1980's, snake densities were estimated at 50 to 100/hectare (13,000 to 26,000/sq mile), higher densities than those recorded for any other snake (3,4).

Brown tree snakes are dietary generalists, being observed to eat chicken bones, cooked spare ribs, lizards, birds, rodents, domestic fowl hatchlings, puppies, piglets, rabbits (in hutches), and pet birds (in cages inside homes) (1,5). Human infants have also been attacked, resulting in very serious bites (6,7). Snake predation has resulted in the extirpation or severe reduction in the populations of virtually all Guam's avifauna and has essentially resulted in the extinction of four endemic species/subspecies: 1) Bridled white-eye (*Zosterops conspicillatus conspicillatus*), 2) Guam flycatcher (*Myiagra freycineti*), 3) Micronesian kingfisher (*Halcyon cinnamomina cinnamomina*), and 4) Rufous fantail (*Rhipidura rufifrons uraniae*) (8,9). The Mariana crow (*Corvus kubaryi*) has also been severely reduced, with 8 birds remaining on Guam, and an additional 300 to 600 remaining on the nearby island of Rota (10). The crow is listed as an endangered species and, as a scavenger that might consume lethally-dosed snake carcasses resulting from chemical toxicant control operations, plays a significant role in secondary hazard assessments of the use of such toxicants.

In addition to the ecological and agricultural damage, snakes crawl along power lines in search of prey. This activity frequently results in short circuits leading to extensive damage to power transmission equipment, subsequent power blackouts to human population centers, and millions of dollars in economic losses (7).

The large military presence on Guam and shipment of associated cargo coupled with the high snake densities increase the likelihood of dispersal of the snake to other locations where the whole damage scenario might be repeated. Individual snakes have been observed on other islands in the Marianas (Saipan, Tinian) and other islands such as Kwajalein, Wake, Diego Garcia, and Hawaii (11,7). One individual was found in a cargo container in Corpus Christi, TX which had been shipped from Guam some six months earlier (12). The United States Department of Agriculture Wildlife Services personnel on Guam utilize a variety of measures to prevent snake accidental snake relocations, but the only long term solution is the reduction or eradication of the brown tree snake population on Guam. As part of a multiagency snake control program funded by the U.S. Department of Defense's Legacy Program scientists from the NWRC were asked to evaluate traps, lures and chemical toxicants. Several candidate compounds with demonstrated toxicity to poikilothermous vertebrates were screened for effectiveness. Among the most effective were: 1) pyrethrum, an extract of *Chrysanthemum* flowers containing a family of six pyrethrins that is registered with the U.S. EPA for insecticidal use, 2) rotenone, a natural product extracted primarily from roots of the tropical plant genus *Derris*, registered as both a piscicide and insecticide, and 3) propoxur, a carbamate insecticide.

## EXPERIMENTAL

### Toxicity Testing

On Guam, the acute toxicity of toxicants to brown tree snakes was evaluated by oral gavage, oral dosing in bait, and dermal application. For oral gavage, the toxicants were dissolved in propylene glycol or ethanol and introduced directly into the entrance of the snake's esophagus by means of a ball tipped feeding needle (13). As future wide scale snake population reduction might utilize a baiting program, snake preferences for various potential bait matrices were evaluated (14). Of 21 bait matrices tested, geckos and mice, processed meat (SPAM) and juvenile quail were well accepted. These latter matrices were subsequently combined with each toxicant at varying concentrations and offered to snakes. Lastly, toxicants dissolved in ethanol were applied to the dorsal surface of restrained snakes using a syringe fitted with a ball tipped needle. In all procedures, five snakes were used to test each toxicant concentration. During experimentation, snakes were housed in plastic cages in racks kept outdoors under shade cloth. Snakes which received non-lethal toxicant doses

were euthanized using halothane (14). All snakes were wrapped in aluminum foil and frozen for subsequent residue determination.

### Analytical Chemistry

Chemical analyses were required to generate the data required for risk assessments. Residues of the toxicants in snakes following dosing were determined. To determine the potential secondary hazards associated with the use of these potential snake toxicants, residue methods were developed to quantify rotenone, pyrethrins and propoxur in whole body brown tree snakes. For all three methods, frozen (-20 °C) snakes were cut into 2 inch pieces and placed into a cylindrical stainless steel container containing liquid nitrogen. The frozen snake was then shattered into a homogeneous powder with a steel bar (15).

To quantify propoxur residues, a silica gel matrix solid phase dispersion method was developed to clean up and concentrate the residues in 2 g portions of homogenized tissue. Extracts were analyzed by reversed phase high performance liquid chromatography (HPLC) with fluorescence detection (excitation = 225 nm, emission = 305 nm). The mean recovery and standard deviation (std dev) were 86.7 and 7.8 percent, respectively. The method limit of detection (MLOD) was 9 parts per billion (ppb) (16).

To quantify pyrethrins, a liquid extraction followed by C8 solid phase extraction clean up was developed to clean up and concentrate pyrethrins in 6 g portions of homogenized tissue. Extracts were analyzed by gas chromatography (GC)/electron capture detection (ECD). Mean recovery was 70.8% with a std dev of 5.7%. MLOD was 6.5 ng/g (17)

To quantify rotenone residues, a silica/florisil solid phase extraction method was developed to clean up and concentrate rotenone residues in 2 g portions of homogenized tissue. Rotenone residues in the extracts were separated by HPLC and quantified by ultraviolet detection at 295 nm. Mean recovery was 84.7% with a std dev of 7.4%. MLOD was 0.012 µg/g (18).

## RESULTS

### Toxicity testing

#### Pyrethrins

Snakes were gavaged with pyrethrum solutions in ethanol and propylene glycol (Table I). Doses ranged from 5 to 40 mg/kg. Oral gavage with pyrethrins yielded 100% mortality only at the highest tested dose of 40 mg/kg. This dose is equivalent to

a dose of approximately 0.25 to 8 mg active ingredients per snake for average snakes ranging in weight from 50 to 200 g. No mortality was found in controls given ethanol or propylene glycol only.

**Table I. Mortality Following Gavage with Pyrethrum**

Carrier	Dose (mg/kg)			
	5	10	20	40
Ethanol	ND*	1/5**	4/5	5/5
Propylene glycol	0/5	4/5	3/5	5/5

\*Not determined

\*\* #Dead/ #tested

Source: Reference 13.

Incorporation of pyrethrum into SPAM and quail chick bait matrices greatly reduced toxicity (Table II). For example, when given in a treated bait, only 50% mortality was achieved at the highest dose of 40 mg/bait (40 mg/snake). This is about 10 to 20 times greater dose than the highest dose administered by oral gavage (which produced 100% mortality). Obviously, combination with a bait severely attenuated the effectiveness of the pyrethrum.

**Table II. Acute toxicity of pyrethrum fortified baits**

Dose (mg/bait)	Number of Snakes Consuming Bait	Percent Mortality
20	5	20
40	4	50

Source: Reference 14.

The whole body pyrethrin residues in snakes given 40 mg baits ranged from 4.1 to 501 µg/g (Table III). The higher residues were found in fatally dosed snakes. This suggests that snakes surviving the initial pyrethrin dose will rapidly metabolize/excrete the pyrethrins. Residue concentrations in surviving snakes were less than in fatally dosed snakes. Quantification of these residue levels was necessary to estimate the potential secondary hazards to predators and/or scavengers potentially feeding on pyrethrin-containing brown tree snake carcasses. For secondary hazard estimates, the highest residue concentrations for each toxicant was used. This conservative approach generally results in "worst case" risk assessment calculations. For pyrethrins, the concentration of 501 µg/g was used.

**Table III. Pyrethrin dose and residues**

<i>Bodyweight (grams)</i>	<i>Dose (mg/kg)</i>	<i>Tissue Residue (µg/g)</i>	<i>Fate</i>
121	331	113	Died
83	482	501	Died
206	194	29	Survived
45	889	4.1	Survived

Source: References 19,20.

#### *Rotenone*

Snakes were also orally gavaged with varying doses of rotenone (Table IV). By this route of administration, rotenone appears to be more toxic than pyrethrins as the lowest dose that achieved 100% mortality was 2.5 mg/kg (0.125 mg - 0.50 mg/snake) compared to 40 mg/kg for pyrethrins. All concentrations higher than 2.5 mg/kg also produce 100% mortality. Again, no mortality was noted in control snakes gavaged only with carrier.

**Table IV. Acute Toxicity following gavage with rotenone**

<i>Dose (mg/kg)</i>	0.61	1.25	2.5	5	10	20	40
<i>Mortality (dead/treated)</i>	0/5	1/5	5/5	5/5	5/5	5/5	5/5

Source: Reference 13.

However, when rotenone was incorporated into SPAM and quail chick baits, no acute toxicity was observed at any concentrations tested (Table V). The highest concentration, 10 mg/bait, was 40 - 80 times the 2.5 mg/kg dose which yielded 100% mortality in the orally gavaged snakes. Incorporation of rotenone into baits decreased the toxicity to even a greater extent than was observed for pyrethrins.

**Table V. Acute Toxicity of Rotenone Fortified Baits**

<i>Concentration (mg/bait)</i>	<i>Number of Snakes Consuming Bait</i>	<i>Percent Mortality</i>
2.5	3	0
5	5	0
10	5	0

Source: Reference 14.

Whole body rotenone residues were determined in the snakes fed baits containing 10 mg rotenone (Table VI). As all the snakes survived, the magnitude of residues were similar to those observed for the surviving pyrethrins dosed snakes and less than the fatally pyrethrins dosed snakes. The highest observed level was 61 µg/g.

**Table VI. Rotenone Dose and Residues**

<i>Bodyweight (grams)</i>	<i>Dose (mg/kg)</i>	<i>Tissue Residue (µg/g)</i>	<i>Fate</i>
111	90	61	Survived
131	76	0.67	Survived
185	54	12.4	Survived
98	102	48.4	Survived

Source: Reference 21.

#### *Propoxur*

Brown tree snakes were orally gavaged with solutions containing varying concentrations of propoxur. The lowest concentration to yield 100% mortality was 40 mg/kg which is equivalent to a dose of 2 - 8 mg propoxur per snake.

**Table VII. Acute Toxicity Following Gavage with Propoxur**

<i>Dose (mg/kg)</i>	5	10	20	40
<i>Mortality (dead/treated)</i>	0/5	2/5	3/5	5/5

Source: Reference 13.

Propoxur baits were prepared by fortifying quail chicks and SPAM at 20 mg/bait which delivered a dose ranging from 146 - 220 mg/kg (7 - 43 mg/snake). While this dose is 4 to 5 times the 40 mg/kg oral gavage dose that resulted in 100% mortality, mortality was only 75%. Again, incorporating the toxicant into a biological matrix reduced toxicity (Table VIII).

**Table VIII. Acute Toxicity of Propoxur Fortified Baits**

<i>Concentration (mg/bait)</i>	<i>Number of Snakes Consuming Bait</i>	<i>Percent Mortality</i>
20	4	75

Source: Reference 14.

Propoxur residues in snakes consuming baits containing 20 mg propoxur were similar for both surviving and fatally dosed snakes (Table IX). The highest observed residue was 141  $\mu\text{g/g}$ .

**Table IX. Propoxur Residues in Snakes Fed Propoxur Baits**

Bodyweight (grams)	Dose (mg/kg)	Tissue Residue ( $\mu\text{g/g}$ )	Fate
131	153	106	Died
124	161	134	Died
137	146	116	Survived
91	220	141	Died

Source: Reference 22,23.

Dermal application was evaluated as a potential mean of applying toxicants to snakes. By far the most effective compound tested was rotenone, yielding 100% mortality at 10 mg/kg, or 0.5 to 2 mg/snake (Table X). This level of toxicity was half that observed for administration via oral gavage.

**Table X. Acute toxicity\* Following Dermal Dosing**

Toxicant	Dose (mg/kg)						
	0	2.5	5	10	20	40	80
Pyrethrins	-.**	-	-	-	1/5	2/5	-
Rotenone	-	0/5	2/5	5/5	5/5	5/5	5/5
Propoxur	-	-	-	-	0/5	3/5	2/5

Note: \* # dead/#tested, \*\* Not determined

Source: Reference 13.

**Table XI. Residues and Acute Toxicity Following Dermal Application of Rotenone**

	Dose (mg/kg)					
	2.5	5	10	20	40	80
Residue ( $\mu\text{g/g}$ )	0.221 (s)	0.390 (s)	4.07 (d)	6.84 (d)	11.1 (d)	35.2 (d)
	0.183 (s)	0.579 (s)	3.04 (d)	7.76 (d)	14.4 (d)	17.2 (d)
	0.112 (s)	1.70 (d)	4.74 (d)	8.94 (d)	13.5 (d)	23.3 (d)
	-	1.72 (d)	-	-	18.2 (d)	-
	-	-	-	-	16.0 (d)	-
Mean residue	0.172	1.1	3.95	7.85	14.6	25.2
Std. dev.	0.05	0.71	0.86	1.05	2.7	9.2

Note: (s) = survived (d) = died

Source: Reference 22.

As rotenone was the only toxicant that appeared to be promising with respect to dermal application, the residue and toxicity data in Table XI is limited to snakes dermally dosed with rotenone. Using the minimum 100% lethal dosage of 10 mg/kg, the highest tissue concentration found was 4.74  $\mu\text{g/g}$ .

### Secondary hazard assessment

When evaluating the use of chemical toxicants to control snake populations, consideration must be given to those non-target species which could accidentally ingest toxicant by scavenging or preying on dead or dying snakes. On Guam such scavengers include feral cats, wild pigs, feral dogs, monitor lizards (*Varanus indicus*), and the Mariana crow. Obviously, the endangered crow elicits the greatest concern from a secondary hazard standpoint, while the other scavenger species are introduced and may be considered pest species themselves. Ideally, secondary hazard considerations should not be limited to non-target species found on Guam. Toxicants developed for brown tree snakes may be required to control future introduced brown tree snake populations at other locations. These locations will likely contain a wider variety of potential non-target species than are currently found on Guam. Also, the brown tree snake population on Guam may be suitably reduced to permit the reintroduction of other species such as the Micronesian kingfisher, which may have preyed on small snakes (6,24). In this scenario, where regular chemical control may be required to keep snake populations minimized, the reintroduced species represent potential non-target species.

A widely used, straight forward approach for estimating non target hazards is the risk quotient (RQ) method (25). The RQ is the expected dose or dietary concentration divided by the dose or concentration expected to produce lethality in 50% of the population, respectively (dose/LD<sub>50</sub> or concentration/LC<sub>50</sub>). RQs provide a numerical basis for decision making. A RQ greater than 1 indicates that there are appreciable non target risks associated with use of this chemical. A RQ less than 1 indicates that the non target risks from use of this chemical may be acceptable under approved usage guidelines. To provide a "worst case" estimate of non target hazards, we assumed that 100% of the exposed animal's diet would consist of the pesticide formulation (primary hazard) or the tissue, organ, or carcass (secondary hazard) containing the residue highest concentration. EPA further breaks-down RQ values less than 1 into the following categories (26):

For the potential brown tree snake toxicants, RQs were calculated for the crow, dog, pig and cat. Crows, feral dogs and feral cats are potential consumers of brown tree snake carcasses on Guam. For pyrethrin bait-dosed snakes, the highest tissue residue concentration of 501  $\mu\text{g/g}$  was used for all calculations. For a worst case exposure estimate, this concentration was multiplied by the average food consumption for crows, 0.076 g food/g bodyweight/day (27). To estimate the acute toxicity of pyrethrins to crows, we relied on the literature value of 7070  $\mu\text{g/g}$ , the LD<sub>50</sub> for Japanese quail. The resulting RQ for crows consuming brown tree snakes killed by

**Table XII. Risk Quotient Values and Associated Concerns**

<i>RQ Value</i>	<i>Associated Risk</i>
<0.1	Use presents acceptable risk for use under approved guidelines
>0.1	Use restrictions may be imposed to protect endangered species
>0.2	Use may be restricted to certified applicators and/or mitigation techniques may be imposed
>0.5	Mitigation techniques will be imposed to protect all species of the same taxonomic order

**Table XIII. Pyrethrum Risk Quotients**

<i>Animal</i>	<i>Calculation</i>	<i>RQ</i>
Crow	$501 \mu\text{g/g} \times 0.076 \text{ g/g}$ $7070 \mu\text{g/g}$	= 0.002
Dog	$501 \mu\text{g/g} \times 0.006 \text{ g/g}$ $200 \mu\text{g/g}$	= 0.15
Pig	$501 \mu\text{g/g} \times 0.04 \text{ g/g}$ $200 \mu\text{g/g}$	= 0.1
Cat	$501 \mu\text{g/g} \times 0.07 \text{ g/g}$ $200 \mu\text{g/g}$	= 0.18

pyrethrins is 0.002, well below the level of concern for endangered species (Table XIII). RQs were similarly calculated for dog, cat, and pig using the oral LD<sub>50</sub> values for the rat, 200 mg/kg (28) and literature referenced consumption rates for dog, cat, and pig (29). The resulting RQs ranged from 0.1 to 0.18 indicating that the potential secondary hazards for these species are minimal.

The same procedure was used to calculate RQs for rotenone and propoxur. The resulting RQs are summarized in Table XIV. For rotenone, the highest snake residue concentration of 61 μg/g was utilized. For crow, acute toxicity was estimated with the LC<sub>50</sub> (1608 μg/g) from ring-necked pheasant (29) to give an RQ of 0.003. For dog, pig, and cat, no rotenone LD<sub>50</sub>s were available, so the LD<sub>50</sub> for the rat (60 mg/kg) was used. The resulting RQs for the dog, pig and cat are 0.06, 0.04, and 0.07. These risk quotients suggest that the secondary hazards associated with the use of rotenone to control brown tree snakes on Guam are minimal.

To calculate the RQs associated with the use of propoxur, the highest tissue residue concentration of 141 μg/g was used. For crow, LC<sub>50</sub> for the house finch, 3.55 mg/kg was used (EPA data base). For dog, pig, and cat, the rat oral LD<sub>50</sub> of 41 mg/kg was used (RTECS)]. The resulting propoxur RQs ranged from 0.14 to 0.24 for mammals and 3.0 for the crow. The RQ of 3.0 for the crow triggers significant concern for secondary hazards, especially when an endangered species is potentially exposed.

**Table XIV. Risk Quotients for Oral Dosing**

<i>Toxicant</i>	<i>Crow</i>	<i>Pig</i>	<i>Dog</i>	<i>Cat</i>
Pyrethrin	0.002	0.1	0.15	0.18
Rotenone	0.003	0.04	0.06	0.07
Propoxur	3.0	0.14	0.21	0.24

Risk Quotients were also calculated using the highest rotenone concentration found in snakes dermally dosed at 10 and 20 mg/kg (Table XV). The resulting risk quotients were quite low for all species of concern, especially the crow. These data suggest that secondary hazards associated with dermal rotenone dosing to control brown tree snakes on Guam are minimal.

**Table XV. Rotenone Dermal Risk Quotients**

Animal	RQ	
	10 mg/kg	20 mg/kg
Crow	0.0002	0.0004
Dog	0.005	0.01
Pig	0.05	0.1
Cat	0.005	0.01

### Conclusions

When administered orally in solutions, the acute toxicity of the potential brown tree snake toxicants evaluated was rotenone > propoxur = pyrethrins. Incorporation of the pesticides into biological matrices (SPAM or quail chicks) reduced the acute toxicity of all the pesticides. The greatest reduction was noted for rotenone. When administered in fortified baits, the toxicity was propoxur > pyrethrins > rotenone. With respect to secondary hazards, the most favorable (least risk, lowest RQ) compound appears to be rotenone followed by pyrethrum. Secondary hazards associated with propoxur appear to be manageable for mammalian scavengers, but suggest high risk for birds.

Acute toxicity for the pesticides when administered in solutions via dermal application was rotenone > pyrethrins = propoxur. Acute toxicity of dermally applied pesticides was about half to one quarter of that observed for gavage. However, pesticide residues and associated secondary hazard risk quotients were significantly less for dermal application.

From a secondary hazard perspective, dermal application of rotenone appears to be a promising technique for the control of brown tree snakes on Guam. However, the development of an efficient and selective dermal application procedure for the brown tree snake is not available. Based on our findings with these toxicants, oral dosing in biological based baits appears to be a less promising approach due to decreased toxicity noted when the toxicant was combined with the bait matrix. However, in the absence of the biological matrix, oral application of rotenone is highly toxic to brown tree snakes. We are currently attempting to capitalize on these observations by developing a synthetic lure which will combine a brown tree snake attractant into a synthetic matrix that will not decrease the toxicity of the pesticides. If successful, a toxicant such as rotenone could be combined with the synthetic matrix to produce an efficacious oral bait for reducing brown tree snake populations. At this point, subsequent reevaluation of residues and risk quotients may be needed. Further work is being conducted to identify additional compounds with high toxicity to brown tree snakes, minimal secondary hazards to non-target species, and adequate efficacy when incorporated into biological matrices.

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