Invasive Myna Control in American Samoa

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ABSTRACT: The common myna is an invasive species in Florida, Hawaii, and in numerous other locations around the world. It is native to southern and south-east Asia. Common mynas are considered pests to fruit crops in many locales, and they are predators on eggs of other birds. Since their introduction to American Samoa in the 1980s, mynas have become the most frequently observed avifauna in developed areas in the country. The American Samoa Department of Marine and Wildlife Resources (DMWR) is concerned that expanding myna populations will exert competitive pressures on native species such as the Samoan starling and white-collared kingfisher. Additionally, the mynas are increasingly becoming social nuisances through nesting, foraging, and vocalization behaviors. The government and general population of American Samoa would like to eradicate these birds before populations are too large to control. In partnership with DMWR, we conducted trials with captive mynas to determine sensitivity to the avian toxicant DRC-1339, and to evaluate a potential baiting strategy for applying this toxicant on American Samoa to reduce myna populations.

KEY WORDS: *Acridotheres tristis,* American Samoa, common myna, DRC-1339, invasive species, LD_{50}, lethal control, toxic bait

INTRODUCTION

The common myna (*Acridotheres tristis*) is an invasive species in Florida, Hawaii, and in numerous other locations around the world. It is native to southern and south-east Asia. Common mynas are considered pests to fruit crops in many locales and they are predators on eggs of other birds (Dawson and Bull 1970, Byrd 1979, Nagle 2006). Mynas were introduced in American Samoa in the 1980s, and they are now found commonly throughout developed areas in the country (Chen 2013). While the actual threat to ecological systems is currently unknown, the American Samoa Department of Marine and Wildlife Resources (DMWR) is concerned that expanding myna populations will exert competitive pressures on native species such as the Samoan starling (*Aplonis atrifusca*) and white-collared kingfisher (*Todiramphus chloris*). Additionally, the mynas are increasingly becoming social nuisances through nesting, foraging, and vocalization behaviors. The government and general population of American Samoa would like to eradicate these birds before populations are too large to control.

There have been many efforts to eradicate invasive myna populations (Parkes 2012). The techniques used have varied, but mainly include poisoning with DRC-1339, trapping, and shooting (Feare 2010, Canning 2011, Grarock et al. 2014). Some small colonizing populations have been eradicated using trapping alone, but in general a combination of methods is employed. After reviewing case studies of myna control efforts, Parkes (2012) concluded that sequential application of poisoning, trapping, and shooting, in that order, comprise the most appropriate approach.

DRC-1339 (active ingredient 3-chloro-*p*-toluidine HCL; CAS number 7745-89-3), commercially known as Starlicide®, is the avian toxicant usually associated with myna control programs. There is some evidence that

there are familial trends in the sensitivity of birds to DRC-1339 (Eisemann et al. 2003). Mynas and starlings are in the family Sturnidae. They are thus expected to be very sensitive to DRC-1339. The LD_{50} for the European starling (*Sturnus vulgaris*) is 3.8 mg/kg, with 95% confidence interval of 3.1-4.6 mg/kg (DeCino et al. 1966). Although we suspect that mynas are very sensitive to DRC-1339, this has never been formally documented. In the first part of this study, we determined the toxicity of DRC-1339 to mynas. We then developed and tested baits for effectively delivering the toxicant.

METHODS
Acute Oral Toxicity

We applied the up-and-down dosing procedure (UDP) as per the revised Environmental Protection Agency (EPA) acute oral toxicity testing guidelines (OPPTS 870.1100 Acute Oral Toxicity). These guidelines incorporate alternative test methods which provide for enhanced animal welfare by reducing the number of animals required during laboratory testing. According to the EPA, the new Acute Oral Toxicity guideline calling for use of the revised Up-and-Down Procedure should be used for acute oral toxicity studies initiated after December 2002.

Implementation of the UDP requires selection of an initial dose. The guidelines state: "The first animal is dosed a step below the toxicologist's best estimate of the LD_{50}." For the common myna, our best estimate of the LD_{50} is 3.8 mg/kg, based on starling toxicity (DeCino et al. 1966). In the test guidelines, several possible dose progressions are specified. In dose series 3, one step below our best estimate of 3.8 mg/kg is 1.75 mg/kg. Thus, we selected 1.75 mg/kg as our initial dose. The progression of doses (mg/kg) in series 3 is 0.175, 0.38, 0.81, 1.75, 3.8, 8.1, and 17.5 (USEPA 2002). Thus, if the first test bird survived the initial dose of 1.75 mg/kg, then

the second bird would receive a dose one step higher, 3.8 mg/kg. If the first bird died from the initial dose, then the second bird would be dosed one step lower, at 0.81 mg/kg. We followed the dose progression until we reached 1 of 3 stopping criteria specified in the test guidelines [USEPA 2002, section (3) (iv)].

We randomly selected test birds from the wild-caught (near Homestead, FL) captive population at the NWRC research field station in Gainesville, FL. We placed test birds in individual test cages (45 cm on each side) for at least 14 days prior to dosing to allow for acclimatization. On the day of dosing, we removed the test bird's food at 0700 (water was not withheld). Then, at approximately 0900, we weighed the designated test bird, determined the appropriate dose, and administered the test substance. We used a total of 6 birds in this test. We applied one-way analysis of variance (Minitab 2007) to examine differences in body mass and amount of toxicant ingested between birds at the 2 dose levels tested.

To administer a dose, one person held the bird steady and a second person opened the bill using a hemostat, the tips of which were covered by short segments of plastic tubing. The first person administered the dose by inserting the end of a blunt 17-gauge gavage tube into the bird's mouth and carefully down the esophagus, and then slowly releasing the aqueous solution from a 1-ml syringe. After dosing, we returned each bird its individual test cage.

Cage Test for Bait Efficacy
Test Procedure
We followed the EPA test guideline for determining efficacy of vertebrate control agents, avian toxicants (USEPA 1982). Common mynas in our captive population were caged individually ($1.2 \times 1.8 \times 1.2$-m) in an outdoor, roofed aviary. From the 17-bird captive population, we randomly assigned 10 birds to the treated group. The remaining 7 birds comprised the untreated group.

One week prior to the start of the feeding trial, we weighed each bird to determine its initial body mass. We removed each bird from its cage, placed it securely in a cloth bag, and weighed the total (bird plus bag) on an electric balance (Mettler P2010, Mettler Electronics Corp., Anaheim, CA). We recorded the total weight on a data sheet, and then released the bird back into its cage. We then weighed the bag separately and recorded that on the data sheet. The bird's body mass was the difference between the two values. We repeated this procedure for each of the 17 test birds.

On Test Day 1, we removed the previous day's maintenance food from each cage at 0700. At ~0900, in each cage, we presented 25 g of untreated cooked white rice in one food cup, and we provided a second cup containing 25 g of fresh maintenance diet. We placed each clear plastic food cup (8.2 cm diameter, 3.8 cm high) within its own plastic dish (25 cm diameter, 4 cm high) to catch food spilled by the birds. We also placed a control cup of both rice and maintenance feed in an unoccupied cage to measure mass changes due to moisture uptake or loss. The rice remained in each cage until 1200 when we retrieved it and weighed the balance remaining in the cup

and in the spill bowl. We also weighed the food remaining in the maintenance cup and spill bowl at this time. We then gave each bird a bowl of daily maintenance food. On Day 2, we repeated the procedure except that the 10 birds assigned to the treatment group received cooked white rice treated with DRC-1339. Thereafter, all 17 birds received only maintenance food and water.

To estimate each bird's daily consumption, we subtracted the amount of food remaining in the rice or maintenance food cup and the amount of spillage retrieved from the respective spill bowl from the weight of the food in the corresponding control cup. Any calculated values <0 were assumed to be 0. We used one-way analysis of variance to examine differences in bait consumption and loss of body mass between birds exposed to treated bait and those in the untreated control group (Minitab 2007).

Preparation of Rice Bait
Based on information provided by colleagues in American Samoa (Josh Seamon, DMWR, Honolulu, HI, unpubl.), we selected cooked white rice as the bait. We prepared rice (350 g rice plus 400 ml water and 5 ml canola oil) in a standard rice cooker. When the rice was cooked, we rinsed it with cold water to remove starch and reduce stickiness. The rice air-dried 4 hours in an air-conditioned lab. We placed then put the cooked rice in a closed container and refrigerated it overnight. The next day, when we weighed out rice for the feeding trial, we also removed ~200 g of rice, sealed it in a plastic bag, and refrigerated it for chemical analysis.

To prepare treated rice, there was one extra step: after the rice was air-dried, and before it was refrigerated overnight, we placed 400 g of the air-dried rice in a rotating mixer, and we added 40 ml of the aqueous toxicant solution (100 mg DRC-1339 in 50 ml distilled water) as the rice rotated slowly in the mixer. We also added 5 ml of canola oil to the rotating rice to aid in adherence of the toxicant to the rice particles. After 15 minutes, we stopped the rotating mixer and transferred the treated rice to a closed container and refrigerated it overnight. The next day, when we weighed out treated rice for the feeding trial, we also collected 6 samples of ~20 g each from the container. We sealed the 6 samples in plastic bags and refrigerated them for chemical analysis to determine DRC-1339 content.

Observations of Test Subjects
On Days 1 and 2, we monitored the behavior of 4 randomly chosen birds in the treatment group at their food cups (0900-1200) with surveillance cameras. On Day 2, after the treated bait was removed, we adjusted the cameras so that all of the birds in the treatment group were in view and monitored them until 1900 to detect indications of distress or intoxication (fluffed feathers, inability to remain upright, tremors, etc.). On Day 3, monitoring of the treatment group birds resumed at 0700 and continued until 1900. We continued to monitor the condition of the birds through Day 7 when the trial ended. We weighed each dead bird, recorded time of death, and stored the carcass in a freezer until disposal. The surviv-

ing birds were weighed and returned to holding cages on Day 8.

RESULTS
Acute Oral Toxicity

The first bird died from a dose of 1.75 mg/kg. The second bird survived a dose of 0.81 mg/kg. We repeated the progression, up and down between 0.81 and 1.75 mg/kg with consistent results (Table 1). After 6 birds were tested, a stopping criterion was met (5 reversals in 6 trials) and the trial ended. The estimated LD_{50} was 1.19 mg/kg (95% confidence interval = 0.81-1.75).

Table 1. Summary of common myna dosing with DRC-1339 using the up and down procedure as specified in USEPA acute oral toxicity test guideline OPPTS 870.1100.

Test Order	Dosage mg/kg	Dosage mg/bird	Body Mass (kg)	Result
1	1.75	0.153	0.087	Died
2	0.81	0.086	0.106	Lived
3	1.75	0.186	0.106	Died
4	0.81	0.094	0.116	Lived
5	1.75	0.205	0.117	Died
6	0.81	0.083	0.103	Lived

There was no difference ($F_{1,4} = 0.27$, $P = 0.634$) in body mass between birds given the 0.81 mg/kg dose (mean = 108.3 g, SE = 4 g) and those dosed at 1.75 mg/kg (mean = 103.4 g, SE = 9 g). Birds at the higher dose level received greater ($F_{1,4} = 36.19$, $P = 0.004$) absolute amounts of toxicant (mean = 0.181 mg DRC-1339/bird, SE = 0.015 mg) than did those at the lower dose level (mean = 0.088 mg DRC-1339/bird, SE = 0.003 mg).

Each of the 3 birds dosed at 1.75 mg/kg appeared to be normally active and alert until 30-33 hours post-treatment. After that, they were noticeably quieter and less active than previously, and each of them stayed on the floor of the cage, on the right side, near the water cup. Each of these birds died during the second night post-treatment, 36-46 hours after dosing.

Bait Efficacy

Rice consumption during Day 1 was virtually identical between the treated and untreated groups (Figure 1). On Day 2, however, when 10 birds received DRC-1339-treated rice, consumption by the 2 groups diverged markedly. In particular, consumption of rice by the treated group averaged 2.1 g/bird (SE = 0.4 g/bird) compared to 3.8 g/bird (SE = 0.7 g/bird) in the untreated cages ($F_{1, 15} = 4.96$, $P = 0.042$). Total food consumption (maintenance food plus rice) followed a similar pattern, with no difference between groups (P = 0.43) on Day 1. On Day 2, total food consumption by the treated group averaged 3.8 g/bird (SE = 0.4) compared to 5.9 g/bird (SE = 0.5) for the untreated group ($F_{1, 15} = 12.43$, $P = 0.003$). Nine of the 10 birds in the treatment group died during the trial, from 12 to 84 hours post-treatment. Generally, time to death varied inversely with the bird's rice consumption (Figure 2). We observed no unusual behaviors or signs of distress in any test bird. Birds that died (n = 9) lost an average of 14.6% of their initial body mass compared to

average losses of 9.1% and 6.8% for surviving birds in the treated (n = 1) and untreated group (n = 7), respectively ($F_{2, 14} = 4.33$, $P = 0.034$; Figure 3).

There was a mean concentration of 124 ppm DRC-1339 on the samples of treated cooked white rice submitted for analysis. Based on this concentration, the calculated dose of DRC-1339 received by birds in the treatment group ranged from 0.769-4.749 mg/kg (Table 2). The surviving bird ate the least and consumed the lowest estimated dose.

DISCUSSION

Although other investigators have used DRC-1339 in efforts to reduce myna populations from islands (Millet et al. 2004, Nagle 2006, Feare 2010, Parkes 2012), there has been no formal attempt to determine the degree of toxicity of this chemical to mynas, or to use such information as a basis for designing and implementing an eradication program. In the present study, we determined the median lethal dose of DRC-1339 for mynas and we incorporated that information into a baiting method with the intention that it be applied to the invasive myna situation on American Samoa.
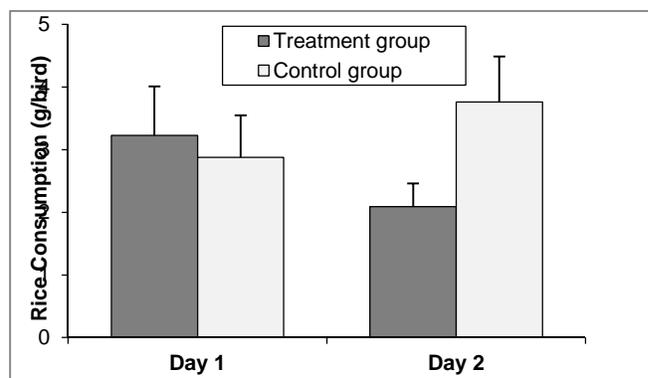

Figure 1. On Day 1, when each bird received untreated rice, mean rice consumption did not differ between the treatment and control groups. On Day 2, when the treatment group was given rice treated with DRC-1339, mean rice consumption by birds in the treatment group was suppressed relative to the control group. Capped vertical bars denote 1 SE.
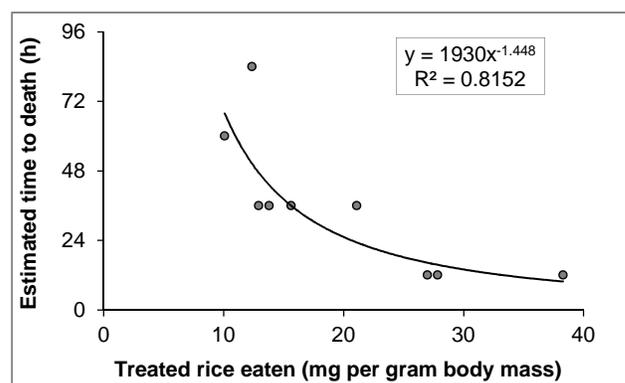

Figure 2. For the mynas that died from eating rice treated with DRC-1339, time to death was inversely related to consumption.
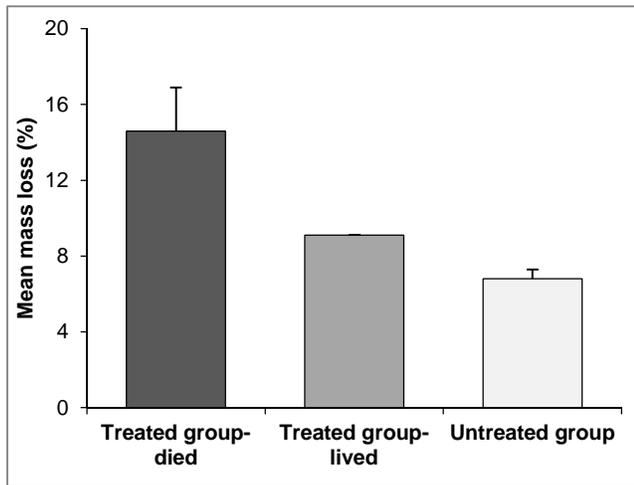
Figure 3. Loss of body mass by 9 mynas in the treatment group that died averaged twice the loss of body mass by birds in the untreated control group (n = 7). Body mass loss by the lone bird in the treatment group that survived was intermediate.

Table 2. Consumption of rice treated with DRC-1339 was lethal to all birds except bird 247, which survived a calculated dose of 0.769 mg/kg. For comparison, the LD$_{50}$ of DRC-1339 for common mynas is 1.19 mg/kg (95% confidence interval 0.81- 1.75 mg/kg). Measured rate of DRC-1339 treatment on rice was 0.0124%.

Bird	Treated Rice Eaten (g)	Body Mass (kg)	DRC-1339/Body Mass (mg/kg)
204	4.45	0.1162	4.749
248	3.40	0.1221	3.453
214	2.88	0.1067	3.347
206	2.27	0.1075	2.618
256	1.61	0.1030	1.938
238	1.65	0.1196	1.711
281	1.35	0.1043	1.605
213	1.47	0.1189	1.533
225	1.23	0.1219	1.251
247	0.62	0.1000	0.769

We recorded a substantial decrease in rice consumption from untreated to treated rice, but even so, 9 of the 10 birds in the treatment group died from ingesting treated bait. We noted no discoloration or off-odor that has sometimes been associated with DRC-1339-treated baits in other applications (Cummings et al. 2003, Pipas et al. 2004). Also, other investigators have suggested that birds do not like the taste of bait treated with DRC-1339. Feare (2010) included table sugar in his cooked rice formulation "as a sweetener to negate the bitterness of Starlicide." In discussing the potential use of DRC-1339 for myna management, Parkes (2006) stated, "The toxin can be surface coated on boiled rice, and it helps to add a little icing sugar to the mix to mask any taste – the toxin is bitter to mammals although birds are not supposed to be able to taste it." Thus, other researchers have presumed

that this toxicant has an unpalatable taste to mynas and that the supposed bitterness should be masked with sugar. We can find no documentation to support these presumptions, however.

An alternative explanation for less-than-ideal bait acceptance by mynas is the DRC-1339 concentration on the rice bait. Feare (2010) mixed 3 g of DRC-1339 with 3 kg of cooked white rice, a concentration of 0.1%. In contrast, we combined 80 mg of DRC-1339 with 400 g of cooked white rice to produce a nominal concentration of 0.02%, 5 times less than Feare (2010) used. The measured concentration on our bait was actually less, 0.0124% (Hulslander 2012). Feare (2010) provided no analysis of the actual concentration on the white rice bait, but in all likelihood his was much greater than our treatment. We detected no hesitancy to eat the treated rice bait by the birds we observed via camera. Thus, we feel the treatment level we used did not inhibit feeding and would be an appropriate level for field use. The decline in rice consumption relative to pre-treatment levels on Day 1 was likely due to onset of illness rather than taste characteristics of the bait. The presumption that bait acceptance observed by Feare (2010) was adversely affected by elevated DRC-1339 concentration is further supported by the fact that he recorded most myna mortality within 24 hours. Time to death with DRC-1339 is dose-related and normally, as in our trial, the affected birds succumb over a period of days.

We do not foresee that DRC-1339 alone will eradicate mynas from American Samoa. Successful management of the myna problem will require a long-term commitment of personnel and funding, and development of a strategy that incorporates several techniques and methods (Parkes 2012).

ACKNOWLEDGMENTS

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