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Improving Invasive House Mice Control and Eradication Strategies via More Effective Rodenticides

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ABSTRACT: In many cases, the control or eradication of invasive house mice has been problematic using rodenticide products that are currently registered. Our investigations using 12 commercial formulations confirmed that premise. In contrast, 11 of 12 of those commercial formulations were effective against Norway rats. Hence, we investigated 7 new rodenticide formulations to identify more effective alternative rodenticides (different formulation and/or different active ingredients). Several of the new formulations of rodenticides and new active ingredients were found to be relatively efficacious ($\geq 70\%$ mortality) and may warrant further investigation as potential control methods for invasive house mice. Additionally, a 2-active ingredient rodenticide, none of which are currently registered in the U.S., showed promise as a new house mouse control tool, and these may have some advantages over currently registered invasive house mouse rodenticides. Field trials with some of these new formulations are recommended as a next step in the research and pesticide registration process.

KEY WORDS: house mouse, invasive species, *Mus musculus*, Norway rat, *Rattus norvegicus*, rodenticide, wildlife damage

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INTRODUCTION

Originally from the Middle East and Asia, house mice (*Mus musculus*) have followed humans around the world and are now found worldwide (Long 2003, Witmer and Jojola 2006). In many situations they live in close commensal relationships with humans, but on many tropical islands and on portions of some continents, they are free-ranging and do not need the food and shelter provided incidentally by humans. House mice pose a threat to the native flora and fauna of islands (Burbidge and Morris 2002, Angel et al. 2009) and can cause significant damage to agricultural commodities and property (Timm 1994a, Long 2003). Most seabirds that nest on islands have not evolved to deal with predation and are very vulnerable to introduced rodents (Moors and Atkinson 1984). House mice are very prolific and populations have irrupted periodically to cause "plagues" in places such as Australia and Hawaii (Long 2003). There have been efforts to eradicate introduced house mice from some islands with some successes (e.g., Burbidge and Morris 2002). Successful eradication rates for house mice, however, have lagged behind rates for rats (MacKay and Russell 2007). Three APHIS pesticide registrations for rodenticide baits (two with brodifacoum and one with diphacinone) are now available to allow rodenticide baiting of islands to eliminate introduced rodent populations (Witmer et al. 2007a). Unfortunately, the diphacinone formulation has not proven very effective for house mouse control (Pitt et al. 2011). Studies in New Zealand have also shown that effective anticoagulant rodenticide formulations for house mice have proven elusive (Fisher 2005, Morriss et al. 2008).

Many commercial rodenticide baits are available on the market and many of these list house mice as a targeted species (Jacobs 1994, Timm 1994a, Timm 1994b). In the first part of this paper, we report on the findings of our earlier trials with various commercial products, relatively few of which were effective with wild-caught house mice. Pitt et al. (2011) found more of these same formu-

lations to be effective on wild-caught house mice from Hawaii, suggesting there may be regional differences in house mouse population susceptibility to rodenticides. It appears from a review of the literature that there has been little development of new formulations of house mouse rodenticides. Hence, efficacy trials with new formulations of acute and anticoagulant rodenticides for invasive house mice are warranted. Additionally, an efficacious rodenticide formulation could potentially replace the current, relatively ineffective APHIS diphacinone registration.

In the second part of this paper, we report on our trials with new active ingredients or formulations of rodenticides and their efficacy with wild house mice. This was determined using 2-choice trials because the new rodenticide bait(s) must be highly palatable even when the house mice have the choice of a food they are familiar with eating. We hypothesized that several of the test baits would exhibit a high ($\geq 80\%$) efficacy when presented to wild-caught house mice.

METHODS

Free-ranging house mice, live-trapped near Fort Collins, CO, were maintained in individual plastic shoebox cages within an animal room at the National Wildlife Research Center, Fort Collins, CO. The mice were provided with a commercial laboratory rodent chow (5001 Formulab Diet, PMI Nutrition International LLC, Brentwood, MO), an apple slice, and water *ad libitum*. Each cage had an absorbent ground cover, a cardboard tube for gnawing and housing, and cotton-like bedding material. The mice were quarantined for two weeks before the trial began. The mice were weighed and sexed before the start of a trial. Free-ranging Norway rats were trapped, processed, and maintained with similar procedures and materials (see Witmer 2007 for details).

Two 2-choice trials were conducted with commercial rodenticide products: one with a 3-day rodenticide exposure period, and one with a 7-day rodenticide exposure

period. Naïve mice were used for each trial. On Day 1 of the 3-day, 2-choice feeding trial, mice were randomly assigned to one of 12 treatment groups with each treatment group consisted of 5 mice; another 5 caged mice were assigned to the control group. All mice were at least 1 month of age and were deemed to be sexually mature by the start of the trial. An attempt was made to evenly distribute the sexes among the treatment groups. The control group continued to receive rodent chow, an apple slice, and water throughout the trial. The treatment groups received 15 g of rat chow supplemented with the assigned rodenticide bait and continued to receive water *ad libitum*. About 15 g of the rodenticide bait was added initially. In the case of the one liquid rodenticide formulation tested (Liqua-Tox[®] II, diphacinone; Bell Labs, Madison, WI), the liquid was prepared as per the label instructions and provided in the water bottles of that group of mice for 3 days before being replaced with regular water; although rodent chow was available *ad libitum*, this can be considered a no-choice test because the only water available contained a rodenticide. It should be noted, however, that house mice do not require free water to survive, obtaining adequate moisture from the metabolism of foods (Timm 1994a). Rodenticide bait and rodent chow were replenished as needed so that mice always had both types of food available.

A total of 12 rodenticides in 3 general categories were tested in the first trial: first-generation anticoagulants (diphacinone pellets, liquid diphacinone, chlorophacinone pellets, and warfarin blocks), second-generation anticoagulants (two different formulations of brodifacoum, difethialone pellets, and bromadiolone pellets), and acute toxicants (cholecalciferol, bromethalin, zinc phosphide on oats, and zinc phosphide pellets). Rodenticide bait consumption was monitored by weighing bait when the trial began, as bait was replenished, and the bait that accumulated on the floor of the cage at the end of the trial. All rodenticide bait was removed at the end of the 3-day exposure period in an effort to simulate the amount of time aerially-broadcast bait might be available to mice on an island before it is consumed by rodents and other animals (especially crabs and other invertebrates) or weathered and deteriorated.

All mice were examined daily and the condition of the individual mice and any mortalities were recorded. Dead mice were placed in a labeled zip-lock bag and refrigerated until necropsy and eventual incineration. The bag was labeled with the study number, date, cage/mouse number, and the final weight. After the rodenticide baits were removed, mice were then monitored daily for a 10-day post-exposure period. During this period, all mice were maintained on rat chow and water. Any mortalities that occurred during the post-exposure period were recorded and carcasses were processed as described above. The 3-day exposure trial with Norway rats was conducted with the same procedures and materials (see Witmer 2007 for details).

Because of lower-than-expected efficacy rates during the 3-day exposure trial with house mice, a second trial was initiated with a 7-day rodenticide exposure period. Six of the original rodenticides were re-tested during this second trial: diphacinone pellets, liquid diphacinone,

chlorophacinone, warfarin, bromadiolone, and cholecalciferol were all offered to naïve mice. All other procedures remained identical to the 3-day exposure trial. Because of the success of the 3-day exposure trial with Norway rats, a 7-day exposure trial was not conducted with Norway rats.

The trial with the new rodenticide formulations was conducted as a 2-choice, 7-day exposure trial. The methods were the same as for the 3-day and 7-day exposure trials described above. For this trial, the number of animals alive versus dead in each treatment group was compared to the number of animals alive versus dead in the control group using Fisher's exact test of independence. The bait consumption by acute toxicant versus anticoagulant-alone treatment groups was compared with a T-test. We considered a $P \leq 0.05$ to indicate a significant difference. We also compared the efficacy to the U.S. Protection Agency (EPA) standard of $\geq 80\%$ efficacy for rodenticides in cage trials.

RESULTS

3-Day Exposure Trial with Commercial Products

No first-generation anticoagulants tested resulted in more than 20% efficacy with house mice (Table 1). Diphacinone pellets did not kill any of the mice in the treatment group. Liquid diphacinone and chlorophacinone pellets both were 20% effective against wild house mice. Mean days to death were 5.0 days for the liquid diphacinone and 8.0 days for the chlorophacinone treatment group. Warfarin bait blocks resulted in no mortalities on wild house mice. The average mouse consumed 11.14 g of diphacinone pellets, 9.90 g of chlorophacinone pellets, and 8.30 g of warfarin. Amounts of liquid diphacinone consumed could not be accurately measured due to slight leakage of the water bottle used to dispense the anticoagulant liquid.

Efficacy rates for second-generation anticoagulants ranged from 40% to 100% on house mice (Table 1). Two different formulations of brodifacoum resulted in 80% and 100% efficacy rates. The mean days to death for both formulations of brodifacoum tested were 9.0 days. Formulations of difethialone and bromadiolone tested killed mice with efficacy rates of 80% and 40%, respectively. Mean days to death for the difethialone formulation tested was 8.0 days, while the mean days to death for the bromadiolone treatment group was 6.5 days. Mean consumption rates were 8.62 g for the first formulation of brodifacoum tested and 8.76 g for the second formulation. The difethialone treatment mice consumed an average of 9.24 g of bait, while the mice fed bromadiolone consumed a mean of 9.84 g of the bait.

Of the 4 acute rodenticides tested, only one exhibited 100% efficacy with house mice (Table 1). The zinc phosphide on oats killed all mice in the treatment group with a mean days to death of 1.0 day. Zinc phosphide pellets killed 40% of the mice in the treatment group with a mean days to death of 2.0 days. Bromethalin killed 80% of mice with a mean days to death of 2.25 days. Cholecalciferol resulted in a 20% efficacy rate with mean days of death of 11.0 days. The mice in the cholecalciferol treatment group consumed an average of 2.82 g of the toxicant. Mice in the zinc phosphide on oats

Table 1. Average bait consumption (g), average days-to-death, and % mortality rate of wild house mice by treatment during a 3-day and 7-day rodenticide exposure period and for Norway rats during a 3-day exposure period using commercial rodenticide baits.

Treatment	House Mice, 3-Day Exposure			House Mice, 7-Day Exposure			Norway Rats, 3-Day Exposure		
	Bait Cons. g (S.D.)	Days-to-Death (S.D.)	Mortality Rate (%)	Bait Cons. g (S.D.)	Days-to-Death (S.D.)	Mortality Rate (%)	Bait Cons. g (S.D.)	Days-to-Death (S.D.)	Mortality Rate (%)
Diphacinone (L) 0.01%	N/A (liquid)	5 (0)	20%	N/A (liquid)	7.3 (0.9)	60%	N/A (liquid)	5.2 (0.8)	100%
Diphacinone (P) 0.005%	11.1 (2.7)	N/A	0%	16.3 (5.6)	6.5 (1.5)	40%	46.1 (4.9)	6.6 (1.1)	100%
Chlorophacinone (P) 0.005%	9.0 (1.4)	8 (0)	20%	21.0 (5.9)	9 (0)	40%	41.1 (7.4)	5.6 (0.5)	100%
Warfarin (P) 0.025%	8.3 (1.1)	N/A	0%	24.5 (7.4)	6 (0)	20%	19.5 (9.3)	5.25 (0.5)	80%
Brodifacoum (P) 0.005%	8.6 (3.6)	9 (4.1)	80%	–	–	–	45.0 (12.2)	6.2 (1.8)	100%
Brodifacoum (P) 0.005%	8.8 (1.0)	9 (1.5)	100%	–	–	–	42.0 (5.5)	5.6 (0.5)	100%
Difethialone (P) 0.0025%	9.2 (1.1)	8 (1.9)	80%	–	–	–	38.0 (8.3)	5.8 (0.8)	100%
Bromadiolone (P) 0.005%	9.8 (3.0)	6.5 (0.5)	40%	18.4 (2.9)	10.8 (2.6)	80%	41.4 (12.3)	4.8 (0.8)	100%
ZP on oats (2.0%)	0.26 (0.1)	1 (0)	100%	–	–	–	1.5 (0.7)	1 (0)	80%
ZP (P) 2.0%	2.0 (0.6)	2 (0)	40%	–	–	–	1.9 (0.6)	N/A	0%
Bromethalin (P) 0.01%	2.3 (0.4)	2.3 (0.8)	80%	–	–	–	9.1 (1.6)	1.8 (1.5)	80%
Cholecalciferol (P) 0.075%	2.8 (0.5)	11 (0)	20%	2.8 (0.4)	8 (0)	20%	12.3 (2.6)	2.8 (0.4)	100%
Control	N/A	N/A	0%	N/A	N/A	0%	N/A	N/A	0%

and bromethalin groups consumed an average of 0.26 g and 2.32 g of the toxicants, respectively. A mean of 1.96 g of zinc phosphide pellets were consumed by the mice in that treatment group.

In contrast to the house mouse trial results, 11 of 12 rodenticides were efficacious (80-100%) with Norway rats in the 3-day exposure trial (Table 1). Only one acute rodenticide, the ZP pellets, had a low efficacy (0%). Based on overall successful results of the Norway rat trial, a 7-day exposure trial with Norway rats was not conducted. No mice or rats in the control groups died during these trials (Table 1).

7-Day Exposure Trial with Commercial Products

All 4 first-generation anticoagulants examined in the 3-day exposure trial were re-tested during the 7-day exposure trial with house mice. The diphacinone pellets and diphacinone liquid killed 40% and 60% of their treatment groups, respectively (Table 1). Mean days to death for the diphacinone pellets was 6.5 days and 7.3 days for the liquid diphacinone. The anticoagulant chlorophacinone was 40% effective against wild house mice with a mean days to death of 9.0 days. The warfarin formulation tested resulted in only a 20% efficacy rate and an average of 6.0 days to death. On average, mice

consumed 16.28 g of diphacinone pellets, 21.02 g of chlorophacinone pellets, and 24.48 g of warfarin. Again, consumption rates for the liquid diphacinone could not be accurately calculated due to slight leakage of the water bottle used to dispense the anticoagulant liquid. Hence, while there was some improvement in efficacy with the 7-day exposure, no first-generation anticoagulants met the EPA efficacy standard of 80%.

Only one second-generation anticoagulant was tested during the 7-day exposure trial. Bromadiolone killed 80% of the wild house mice in the treatment group (Table 1). Mean days to death was 10.8 days and mice consumed an average of 18.38 g of the rodenticide. This was a substantial increase in efficacy over the 3-day exposure trial and now met the EPA standard of 80%.

The only acute toxicant examined during the 7-day exposure trial with house mice was the toxicant cholecalciferol. During the 7-day exposure trial, 20% of the treatment group offered cholecalciferol died (Table 1). Mean days to death was 8.0 days and mice consumed an average of 2.84 g of the toxicant. Hence, the 7-day exposure showed no increase in efficacy over the 3-day exposure trial. No mice in the control group died during this trial (Table 1).

Table 2. Average bait consumption and average days-to-death by treatment for wild-caught house mice that died during the 2-choice, 7-day exposure rodenticide trial using experimental formulations.

Formulation Code ^a	No. Dead Mice (% mortality)	Bait Consumption in g (S.D.)	Days-to-Death (S.D.)
B1	7/10 (70%)	12.7 (2.0)	8.9 (2.1)
B2	7/10 (70%)	15.4 (2.4)	8.6 (2.5)
B3	10/10 (100%)	7.3 (1.4)	6.4 (1.4)
LT	7/10 (70%)	15.9 (1.8)	7.6 (1.7)
ZP	7/10 (70%)	1.0 (0.6)	3.6 (2.4)
AD	6/10 (60%)	21.8 (4.6)	9.2 (2.5)
C+D	5/10 (50%)	10.3 (5.4)	7.4 (1.5)

^aB1 = Bell Labs formulation 1: 0.01% diphacinone (pellets)
 B2 = Bells labs formulation 2: 0.005% diphacinone (pellets)
 B3 = Bell Labs formulation 3: 0.03% cholecalciferol + 0.0025% brodifacoum (pellets)
 LT = LiphaTech formulation: 0.005% chlorophacinone (blocks)
 ZP = NWRC formulation: 0.5% zinc phosphide (coated grain)
 AD = Scimetrix formulation: 0.005% diphacinone (blocks)
 C+D = Connovation (NZ) formulation: 0.03% cholecalciferol + 0.005% diphacinone

New Formulations Trial with House Mice

All the new formulation rodenticides tested with house mice caused substantial mortality ($P \leq 0.0163$) in all cases when compared to the control group (0% mortality). However, we observed a wide range of efficacy levels from 100% down to 50% (Table 2). The most efficacious formulation at 100% efficacy was the B3 bait (0.03% cholecalciferol + 0.0025% brodifacoum pellets). This was followed by 4 formulations (B1, B2, LT, and ZP) all at 70% efficacy; see the Table 2 footnote for active ingredients and concentrations of these formulations. While only one formulation exceeded our expectation of $\geq 80\%$ efficacy in the cage trials, the 4 with 70% efficacy appear to have potential. Three of those formulations contain a first-generation anticoagulant active ingredient (diphacinone or chlorophacinone). Many of our wild-caught house mice came from a dairy that uses first-generation anticoagulants to control the mice population. This is often the case where mice populations are very large, such as at feedlots and dairies. When sizable numbers of wild house mice (or rats) are needed for research studies, these locations are often used to obtain adequate numbers of wild commensal rodents. However, this poses the potential that some of the rodents in the population have a genetic or behavioral resistance to anticoagulant rodenticides. Hence, our efficacy values may have been higher than 70% if we could have used wild house mice from populations where there was not a history of anticoagulant rodenticide use.

We tested two new formulations that contained two active ingredients. No such rodenticides are currently registered for commercial use in the U.S. One of these (B3) contains cholecalciferol and brodifacoum; the other (C+D) contains cholecalciferol and diphacinone. Because of the concern about the future use of brodifacoum (a second-generation anticoagulant that is more toxic and more persistent in tissues than first-generation anticoagulants) with regard to the hazards posed to non-target animals, it would be of value to perform trials with a B3 formulation containing a lower concentration of brodifacoum. The B3 formulation contains 0.0025% brodifacoum, which is the concentration in most commercial one-active-ingredient brodifacoum rodenticides. While

the C+B bait was very efficacious (100%), we were somewhat surprised that the other two-active-ingredient formulation (C+D), which contains the anticoagulant diphacinone versus the brodifacoum in the B3 formulation, did not perform better (only 50% efficacy). This formulation was very efficacious with California voles (80% efficacy; unpubl.) and roof rats (100% efficacy; unpubl.). However, we know that there are differences in sensitivity to toxicants among rodent species. For example, in preliminary trials with a new potential rodenticide active ingredient, encapsulated sodium nitrite (QA-1752), a food bait appeared promising with several species of rodents but not with house mice (unpubl.).

The days-to-death varied by formulation (Table 2). As might be expected, those formulations containing a relatively acute toxicant (when compared to the chronic anticoagulant toxicants) tended to have shorter days-to-death. These included ZP (with zinc phosphide; 3.6 days-to-death), B3 (with cholecalciferol; 6.4 days-to-death), and C+D (with cholecalciferol; 7.4 days-to-death). The formulations containing only anticoagulant active ingredients had days-to-death ranging from 7.6-9.2. The days-to-death for formulations containing acute toxicants (average; 5.8 days-to-death) was significantly shorter ($P = 0.0440$) than for the anticoagulant formulations (average; 8.6 days-to-death). This may be a consideration when the humaneness of vertebrate toxicants is of concern.

The amount of rodenticide bait consumed varied by formulation (Table 2). The lowest amounts consumed were those containing the acute toxicants (zinc phosphide or cholecalciferol). These treatment groups consumed significantly less ($P = 0.007$) bait than the anticoagulant treatment groups. This could be expected because the adverse symptoms come on rather quickly with these toxicants versus anticoagulant toxicants. The lowest consumption amount was with the zinc phosphide formulation. That formulation achieved 70% efficacy, which was lower than expected. We used a 0.5% formulation, which is much lower than the commercially registered products that contain 2% zinc phosphide (like those used in the earlier commercial product trials). Katherine Horak (unpubl.) found 0.5% zinc phosphide formulation to be highly efficacious ($\geq 80\%$ efficacy) with

California voles. However, she used an encapsulated zinc phosphide formulation in her trials. We had hoped to use an encapsulated formulation in our house mouse trials, but none was available at the time. Because the zinc phosphide we used was not encapsulated, we may have had a “bait shyness” issue whereby the rodents consume a sub-lethal dose but become symptomatic and stop feeding on the bait. Typically, rodents will remember this experience and will not feed on that bait again.

Interestingly, in several cases, rodents that survived the 2-choice rodenticide trials consumed more bait than those that died. This occurred in both the cases of anticoagulant formulations and acute formulations. This may suggest a resistance to some anticoagulant rodenticides, variation in palatability by individual rodents, or just a greater tolerance in some individuals.

DISCUSSION

Invasive rodents have been extremely detrimental to the flora and fauna of islands worldwide. Rodenticide baits that effectively eliminate invasive rodents over a short exposure period are required for successful eradication programs on large and remote islands. A worrisome result of this study was that a number of commercially-available rodenticide baits in the U.S. were not effective against wild house mice with only a 3-day exposure period. In sharp contrast, almost all the rodenticides were effective with wild Norway rats. Efficacy rates with wild house mice improved with a 7-day rodenticide exposure; however, this might be a moot point if aerially broadcast rodenticides on islands would only be available to rodents for 3-4 days (e.g., Berentsen et al. 2014). The bright spot is that a 3-day exposure of some rodenticides did result in acceptable efficacy rates (80-100%), specifically the brodifacoum formulations, as well as difethialone and zinc phosphide on oats. It is possible that the long-term use of first-generation anticoagulants (mainly chlorophacinone and diphacinone) on the U.S. mainland has resulted in a resistance or tolerance to those rodenticides in some mainland house mouse populations.

Although the first trials demonstrated that many of the commercially-available rodenticides tested for wild-caught house mice from the Fort Collins, CO, area were ineffective, the next trial identified several new formulations of rodenticides that may warrant further investigation as potential control methods for invasive house mice. Additionally, a two-active-ingredient rodenticide, none of which are currently registered in the U.S., showed much promise as new house mouse control tool. These may have some advantages over currently registered invasive house mouse rodenticides. For example, there may be increased efficacy and reduced concentrations of active ingredients over those currently being used in single-active-ingredient rodenticides; that may result from a synergistic effect of the two chemicals. It has also been suggested that the acute toxicant, because of its more rapid “knock down” time, might result in sickened animals retreating to burrows or other refugia prior to the anticoagulant causing their death. This could potentially reduce the risk of predators and scavengers having access to poisoned carcasses. Field trials with these formulations are recommended as a next step in the

research process.

The eradication of invasive rodents from islands poses many challenges. In many cases on large or remote islands, a single aerial bait drop may be all that limited resources will allow. To be effective an eradication strategy must be able to put all individuals at risk, animals must be removed from the population faster than they can reproduce, and there must be no risk of new individuals immigrating into the area (Parkes and Murphy 2003). The relatively recent registration from the EPA for the aerial broadcast baiting of brodifacoum and diphacinone anticoagulant rodenticide pellets should set the stages for successful eradications of invasive rodent on islands of the U.S. and its territories (Witmer et al. 2007a). Our study demonstrated why there may be greater difficulty with house mouse eradications versus rat eradications. Advani (1992) and Fisher (2005) also noted the difficulty of killing house mice with first-generation anticoagulants. Witmer et al. (2006), in a field trial on Kiska Island, Alaska, found that a single hand-broadcast application of diphacinone pellets greatly reduced Norway rat activity and sign. When compared to diphacinone, brodifacoum presents a higher risk of primary poisoning to non-target species; also, animals fed brodifacoum retain higher anticoagulant residue levels in their body tissues (Donlan et al. 2003). These higher residue levels translate into a greater risk of secondary poisoning to animals that might feed on the carcasses of poisoned rodents, including raptors. Having a registration that allows for the use of either anticoagulant allows managers to tailor the bait to be used to the target species, while still being able to weigh possible secondary hazards and environmental risks. In situations where the elimination of Norway rats is the goal, and where concerns over non-target and secondary species poisoning need to be addressed, diphacinone might be the preferred alternative. However, if house mice were the target of eradication and non-target species are not as great of a concern, brodifacoum might be the more appropriate choice. Fisher (2005) also suggested that second-generation anticoagulants be looked at more closely for mice eradications rather first-generation anticoagulants.

Even with aerial broadcast baiting as an option, bait stations may still be preferred in some situations for various reasons (e.g., the presence of highly valued non-target species) on smaller islands, and these products will still allow for that (Witmer et al. 2007b). Each island situation is different, and specific and appropriate eradication strategies must be developed (see Witmer et al. 2014). Because of the degradation of rodenticide pellets by weather and the consumption of rodenticide pellets by non-target animals such as crabs and ants (which are not affected by the anticoagulant baits), the strategy must include ways to mitigate these adverse effects. Compressed pellets, pellets coated with paraffin wax, higher application rates, and the use of insecticides or insect anti-feedants are some of the techniques that could be employed.

Island resources typically recover very quickly after the successful eradication of invasive rodents (Howald et al. 2007, Witmer et al. 2007b). As methods of invasive rodent eradication continue to improve and to be refined,

we can expect many more successful events from around the world (Veitch and Clout 2002, Veitch et al. 2011). This study has demonstrated, however, that the eradication of house mice with current rodenticides will require the careful selection of one or more efficacious rodenticides and considerable effort to assure success.

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