Final Report, QA-1304:

Efficacy of commercially available rodenticide baits for the control of wild house mice

Gary Witmer, Supervisory Research Wildlife Biologist
USDA/APHIS Wildlife Services
National Wildlife Research Center
4101 Laporte Avenue, Fort Collins, CO 80521-2154

June 4, 2007

ABSTRACT AND CITATION

Witmer, Gary. 2007. Efficacy of commercially available rodenticide baits for the control of wild house mice. Final Report, QA-1304. USDA/APHIS/WS National Wildlife Research Center, Fort Collins, CO 80521-2154. 16 pp.

House mice *(Mus musculus)* are now found worldwide. Most commonly, they are found in commensal situations with humans, but in some instances, they are free-ranging. Mice can cause extensive agricultural damage and pose a serious threat to native flora and fauna, especially on islands. Around the world, efforts have begun to eliminate introduced mouse populations from islands, with some successes. Currently, the most common method of eradicating rodents from islands in the U.S. has been the use of anticoagulant baits maintained in bait stations. However, many islands are simply too large, too rugged or too remote for this method to be feasible. Therefore, the U.S. EPA is considering the use of 0.005% diphacinone and 0.0025% brodifacoum bait pellets for aerial broadcast baiting to control rodents in conservation areas. There has been no recent work done with wild house mice, hence this study. Wild house mice were presented with 12 different rodenticides in two-choice feeding trials with 3-day and 7-day rodenticide exposure periods. The 3-day exposure period was chosen because this is considered the maximum time aerially broadcast bait on an island would remain available to rodents before being degraded by weather or being removed by non-target species. The 7-day exposure trial was conducted because of lower than expected efficacy rates during the 3-day trial. Each rodenticide was presented to 5 mice with 5 additional mice serving as controls. We examined first and second generation anticoagulants, as well as acute toxicants. During the 3-day trial, only 5 of the 12 rodenticides tested resulted in efficacy rates of 80% or higher. Because efficacy rates were lower than expected, 6 rodenticides were re-tested with naive mice and a 7-day exposure period. During the 7-day exposure period, only one rodenticide achieved an efficacy rate of 80%. Clearly, the eradication of house mice with current rodenticides will require the careful selection of one or more rodenticides and considerable effort to assure success.

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). In many situations they live in a close commensal relationship 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) and can cause significant damage to agricultural commodities and property (Long 2003, Timm 1994a). 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). The biology, ecology, and management of house mice was recently reviewed by Witmer and Jojola (2006). The Study Director recently conducted a site visit to Pennsylvania where house mice were posing a serious threat to the poultry industry, both by consuming and contaminating chicken feed and by the transmission of the bacterial disease, Salmonella. Despite the use of a variety of rodenticides by the poultry growers, mouse problems persisted. There has been a worldwide effort to eradicate introduced house mice from some islands with a few successes (e.g., Burbidge and Morris 2002, Howald et al. In Press). USDA Wildlife Services (WS) conducted a successful eradication of roof rats from Buck Island in the U.S. Virgin Islands for the National Park Service (Witmer et al. 2007), using a grid of elevated bait stations across the 200 ac island. Unfortunately, the house mice on Buck Island were not affected by the roof rat eradication strategy and have since come to dominate the island.

National registrations are being sought from the U.S. Environmental Protection Agency (EPA) to allow broadcast baiting of 0.005% diphacinone and 0.0025% brodifacoum bait pellets on conservation areas to eliminate introduced rodent populations (Witmer and Eisemann 2005). These registrations are being sought because on very large, or rugged, or remote islands bait stations would not be a practical approach to invasive rodent eradication. Unfortunately, these rodenticide baits have not been tested in recent years on wild house mice. The use of rodenticides to control or eradicate invasive rodents for conservation purposes was recently reviewed by Witmer et al. (In Press).

Many commercial rodenticide baits are available on the market and many of these list house mice as a targeted species (Jacobs 1994). It appears from a review of the literature, however, that there have not been recent efficacy trials of these baits using wild house mice and a standardized protocol across rodenticide bait types. Additionally, listings (e.g., Timm 1994b) of certain characteristics of the rodenticide active ingredients (such as LD50 values) do not always indicate whether wild or lab mice were used or how the trials were conducted. This study is designed to remedy these shortcomings.

Effective rodenticide baits are needed to control or eradicate introduced house mice. Rodents on islands often have a choice of food items, so an effective bait must be attractive and palatable as well as efficacious when presented with an alternative food type. Additionally, rodents may only have access to the bait for a short period of time. It

has been suggested that three days would be the maximum amount of time aerially broadcast bait on an island would remain available to rodents before being degraded by weather or being removed by non-targets species or other rodents.

METHODS

Free-ranging house mice, live-trapped near Fort Collins CO, were maintained in individual plastic shoebox cages within a room of the Animal Research Building at the National Wildlife Research Center (NWRC). The mice were provided with a commercial laboratory rat chow (5001 Formulab Diet, PMI Nutrition International, LLC, Brentwood, MO) and water (tap water treated and approved for human consumption) *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 the trial.

Two trials were conducted, one with a 3-day rodenticide exposure and one with a 7-day rodenticide exposure. Naïve mice were used for each trial. On day 1 of the 3-day, two-choice feeding trial, mice were randomly assigned to one of 12 treatment groups, 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 rat chow 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), 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 rat chow was available *ad libitum*, this can be considered a no-choice test because the only water available was treated. 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 rat chow were replenished as needed so that mice always had both types of food available. A total of 12 rodenticides in three 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). Food consumption was monitored by weighing food when the trial began, as food was replenished, and the food that accumulated on the floor of the cage at the end of the trial. All rodenticide bait was removed at the end of the trial 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 observation period. During the 10-day period, all mice were maintained on rat chow and water. Any mortalities that occurred during that 10-day observation period were recorded and carcasses were processed as described above. All carcasses from the study were refrigerated and eventually incinerated at the NWRC.

Because of lower than expected efficacy rates during the 3-day trial. A second phase 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 protocols remained identical to the 3-day exposure trial.

RESULTS

3-Day Exposure Trial

The overall trial results for individual mice are presented in Appendix 1.

First Generation Anticoagulants:

No first generation anticoagulants tested resulted in more that 20% efficacy (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.

Second Generation Anticoagulants:

Efficacy rates for second generation anticoagulants ranged from 40% to 100% on wild 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.

Acute Toxicants:

Of the 4 acute rodenticides tested, only one exhibited 100% efficacy (Table 1). The toxicant zinc phosphide on oats killed all mice in the treatment group with a mean days to death of 1.0 days. 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 and bromethalin groups consumed an average of 0.26 g and 2.32 g of the toxicants, respectively. A mean on 1.96 g of zinc phosphide pellets were consumed by the mice in that treatment group.

Control Group:

No mice in the control group died during this trial (Table 1).

7-Day Exposure Trial

The overall trial results for individual mice are presented in Appendix 2.

First Generation Anticoagulants:

All four first generation anticoagulants examined in the 3-day exposure trial were re-tested during the 7-day exposure trial. The diphacinone pellets and diphacinone liquid killed 40% and 60% of their treatment groups, respectively (Table 2). 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. Consumption rates for the liquid diphacinone could not be accurately calculated due to slight leakage of the water bottle used to dispense the anticoagulant.

Second Generation Anticoagulants:

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 2). Mean days to death was 10.8 days and mice consumed an average of 18.38 g of the rodenticide.

Acute Toxicants:

The only acute toxicant examined during the 7-day exposure trial was the toxicant cholecalciferol. During the 7-day exposure trial, 20% of the treatment group offered cholecalciferol died (Table 2). Mean days to death was 8.0 days and mice consumed an average of 2.84 g of the toxicant.

Control Group:

No mice in the control group died during the 7-day exposure trial (Table 2).

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. Efficacy rates 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 days. 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.

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 for. 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). 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. In Press). It is important that both compounds attain registration by the EPA for aerial broadcast baiting since they would both be ideally suited to different situations. In a similar study conducted on wild Norway rats with a 3-day rodenticide exposure, diphacinone pellets and brodifacoum pellets were equally effective, both resulting in 100% mortality rates (Witmer 2007). However, with only a 3-day exposure, diphacinone pellets were ineffective against wild house mice, failing to kill any mice in the treatment group; while the two formulations of brodifacoum tested resulted in 80% and 100% efficacy rates. 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 would allow for the use of either anticoagulant would allow 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. In Press). Each island situation is different and specific and appropriate eradication strategies must be developed. Because of the degradation of rodenticide pellets by weather and the consumption of rodenticide pellets by non-target animals such as crabs and ants (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. In Press, Witmer et al. In Press). 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). This study has demonstrated, however, that the eradication of house mice with current rodenticides will require the careful selection of one or more rodenticides and considerable effort to assure success.

LITERATURE CITED

Advani, R. 1992. Field evaluation of three anticoagulant rodenticides against *Mus musculus* populations in apartment buildings in New York City. Proc. Vertebr. Pest Conf. 15:208-211.

Burbidge, A., and K. Morris. 2002. Introduced mammal eradications for nature conservation on Western Australian islands: a review. Pages 64-70 in C. Veitch and M. Clout, eds. Turning the tide: the eradication of invasive species. SSC Invasive Species Specialist Group, IUCN, Gland, Switzerland.

Donlan, C. J., G. Howald, B. Tershy, and D. Croll. 2003. Evaluating alternative rodenticides for island conservation: roof rat eradication from the San Jorge Islands, Mexico. Biological Conservation 114: 29-34.

Fisher, P. 2005. Review of house mouse susceptibility to anticoagulant poisons. DOC Science Internal Series 198. New Zealand Department of Conservation, Wellington. 19 pp.

Howald, G., C. Donlan, J. Galvan, J. Russell, J. Parkes, A. Samaniego, Y. Wand, D. Veitch, P. Genovesi, M. Pascal, A. Saunders, and B. Tershy. IN PRESS. Invasive rodent eradication on islands. Conservation Biology xx:xx-xx.

Jacobs, W. 1994. Pesticides federally registered for control of terrestrial vertebrate pests. Pages G-1 – G-22 in S. Hygnstrom, R. Timm, and G. Larson, eds. Prevention and Control of Wildlife Damage. University of Nebraska, Cooperative Extension Service, Lincoln, NE.

Long, J. 2003. Introduced mammals of the world. CSIRO Publishing, Collingwood, Victoria, Australia.

Moors, P.J., and I.A.E. Atkinson. 1984. Predation on seabirds by introduced animals, and factors affecting its severity. ICBP Technical Publication No. 2:667-690.

Parkes, J., and E. Murphy. 2003. Management of introduced mammals in New Zealand. New Zealand J. of Zoology 30:335-359.

Timm, R. 1994a. House Mice. Pages B-31 – B-46 in S. Hygnstrom, R. Timm, and G. Larson, eds. Prevention and Control of Wildlife Damage. University of Nebraska, Cooperative Extension Service, Lincoln, NE.

Timm, R. 1994b. Description of active ingredients. Pages G-23 – G-61 in S. Hygnstrom, R. Timm, and G. Larson, eds. Prevention and Control of Wildlife Damage. University of Nebraska, Cooperative Extension Service, Lincoln, NE.

Veitch, C.R., and M.N. Clout, eds. 2002. Turning the tide: the eradication of invasive species. IUCN SSC Invasive Species Specialist Group. IUCN, Gland, Switzerland and Cambridge, UK.

Witmer, G. 2007. Efficacy of commercially-available rodenticide baits for the control of introduced Norway rats. Unpublished Final Report, QA-1232. USDA/APHIS/WS-National Wildlife Research Center, Fort Collins, CO. 10 pp.

Witmer, G., F. Boyd, and Z. Hillis-Starr. 2007. The successful eradication of introduced roof rats *(Rattus rattus)* from Buck Island using diphacinone, followed by an irruption of house mice *(Mus musculus)*. Wildlife Research 34:108-115.

Witmer, G., P. Burke, S. Jojola, and P. Dunlevy. 2006. The biology of introduced Norway rats on Kiska Island, Alaska, and an evaluation of an eradication approach. Northwest Science 80:191-198.

Witmer, G., and J. Eisemann. 2005. An overview of the 2nd national invasive rodent summit. Proc. of the Wildlife Damage Management Conf. 11:102-111.

Witmer, G., J. Eisemann, and G. Howald. IN PRESS. The use of rodenticides for conservation efforts. Proc. Wildl. Damage Manage. Conf. 12.

Witmer, G., and S. Jojola. 2006. What's up with house mice?—a review. Proc. Vertebr. Pest Conf. 22:124-130.

Table 1. Average weight change, average chow and bait consumption, mean days to death (TD) and mortality rate of wild house mice by treatment during a 3-day rodenticide exposure period. Euthanized mice were excluded from days to death calculations.

Code	Treatment	Wt. Change		Chow Cons		Bait Cons		Mortality Rate	Days TD	
		Mean	S.D.	Mean	S.D.	Mean	S.D.		Mean	S.D.
First Generation Anticoagulants										
LT	Diphacinone (liquid)	-3.10	3.8205	10.46	1.3440	N/A	N/A	20%	5	0.0
RG	Diphacinone (pellet)	-0.14	0.95415	2.74	1.484	11.14	2.6852	0%	N/A	0.0
RZ	Chlorophacinone	-0.70	1.7251	1.06	0.2728	9.90	1.4491	20%	8	0.0
MK	Warfarin	-0.56	1.5806	4.84	2.8274	8.30	1.0770	0%	N/A	N/A
Second Generation Anticoagulants										
CI	Brodifacoum	-2.08	1.1285	2.40	3.1010	8.62	3.5869	80%	9	4.0620
HA	Brodifacoum	-4.38	1.0419	1.06	0.2871	8.76	0.9952	100%	9	1.5492
GE	Difethialone	-3.76	1.8205	0.84	0.0800	9.24	1.0707	80%	8	1.8708
JB	Bromadiolone	-2.72	2.0653	1.08	0.5600	9.84	3.0454	40%	6.5	0.5000
Acute Toxicants										
ZO	ZP on Oats	-0.94	0.9728	1.16	0.3499	0.26	0.1020	100%	1	0.0
ZP	ZP pellets	-1.04	2.3602	7.62	4.2197	1.96	0.6829	40%	2	0.0
FT	Bromethalin	-2.46	0.8309	1.28	0.3311	2.32	0.4020	80%	2.25	0.8292
QT	Cholecalciferol	-3.68	2.6491	4.30	2.7871	2.82	0.4534	20%	11	0.0
Control Groups										
C	Rat Chow Only	-0.46	1.3291	12.66	2.4245	N/A	N/A	0%	N/A	N/A

Table 2. Average weight change, average chow and bait consumption, mean days to death (TD) and mortality rate of wild house mice by treatment during a 7-day rodenticide exposure period. Euthanized mice were excluded from days to death calculations.

Code	Treatment	Wt. Change		Chow Cons		Bait Cons		Mortality Rate	Days TD	
		Mean	S.D.	Mean	S.D.	Mean	S.D.		Mean	S.D.
First Generation Anticoagulants										
RG	Diphacinone (pellet)	-0.04	3.1532	2.46	2.2033	16.28	5.5848	40%	6.5	1.5000
LT	Diphacinone (liquid)	0.84	2.2069	25.90	6.1446	N/A	N/A	60%	7.3	0.9428
RZ	Chlorophacinone	0.80	1.8536	1.34	0.8523	21.02	5.9220	40%	9.0	0.0000
MK	Warfarin	2.12	3.6290	4.14	1.8543	24.48	7.3584	20%	6.0	0.0000
Second Generation Anticoagulants										
JB	Bromadiolone	0.70	1.8352	1.32	0.6493	18.38	2.8729	80%	10.8	2.5860
Acute Toxicants										
QT	Cholecalciferol	1.42	4.2054	17.38	7.0155	2.84	0.4409	20%	8.0	0.0000
Control										
C	Rodent Chow Only	3.62	3.4851	41.28	4.1658	N/A	N/A	0%	N/A	N/A

Appendix 1. House mouse treatments, sex, weight, food consumption, fate, and day to death (TD) for 3-day rodenticide exposure trial.

Mouse No.	Sex	InWt.	OutWt.	Wt.Ch.	BaitC	ChowC	Fate	DaysTD	Trt
GJ17	M	25	22.8	-2.2	9.8	0.9	Dead	6	CI
GJ42	F	20	18.6	-1.4	10	0.7	Dead	4	CI
GJ65	F	16	12	-4	8.8	0.9	Dead	13	CI
GJ68	M	14	13.4	-0.6	1.9	8.6	Dead	13	CI
GJ37	M	22	19.8	-2.2	12.6	0.9	Euth.	N/A	CI
GJ30	F	20	18.5	-1.5	none	12.7	Euth.	N/A	C
GJ51	M	28	30.1	2.1	none	15.2	Euth.	N/A	C
GJ63	F	19	17.5	-1.5	none	12.2	Euth.	N/A	C
GJ66	F	20	19.3	-0.7	none	14.8	Euth.	N/A	C
GJ67	M	22	21.3	-0.7	none	8.4	Euth.	N/A	C
GJ7	M	21	17.4	-3.6	2.5	1.4	Dead	2	FT
GJ24	M	18	15.9	-2.1	2.1	1.1	Dead	3	FT
GJ49	M	19	15.8	-3.2	2.9	1.3	Dead	3	FT
GJ58	F	13	11.7	-1.3	1.7	0.8	Dead	1	FT
GJ33	F	24	21.9	-2.1	2.4	1.8	Euth.	N/A	FT
GJ8	M	23	15.7	-7.3	8.7	0.8	Dead	11	GE
GJ9	F	17	14.5	-2.5	8.2	0.8	Dead	7	GE
GJ56	F	18	14.4	-3.6	9	0.8	Dead	8	GE
GJ61	M	22	19	-3	9	1	Dead	6	GE
GJ36	M	22	19.6	-2.4	11.3	0.8	Euth.	N/A	GE
GJ12	M	27	22.6	-4.4	10.5	1.6	Dead	8	HA
GJ13	F	15	12.5	-2.5	8.5	0.8	Dead	8	HA
GJ27	M	22	16.3	-5.7	7.4	0.9	Dead	8	HA
GJ45	F	19	14.3	-4.7	8.7	0.9	Dead	12	HA
GJ55	M	17	12.4	-4.6	8.7	1.1	Dead	9	HA
GJ10	M	20	16.9	-3.1	8.6	0.8	Dead	7	JB
GJ70	M	39	32.4	-6.6	15.7	2.2	Dead	6	JB
GJ34	F	16	14.9	-1.1	8.3	0.8	Euth.	N/A	JB

GJ46	M	15	13.7	-1.3	7	0.8	Euth.	N/A	JB
GJ62	M	17	15.5	-1.5	9.6	0.8	Euth.	N/A	JB
GJ43	F	23	12.4	-10.6	Liq.	12.4	Dead	5	LT
GJ15	F	23	21.7	-1.3	Liq.	10.4	Euth.	N/A	LT
GJ16	F	17	17	0	Liq.	8.6	Euth.	N/A	LT
GJ26	M	21	19.7	-1.3	Liq.	11.4	Euth.	N/A	LT
GJ44	M	22	19.7	-2.3	Liq.	9.5	Euth.	N/A	LT
GJ14	M	23	21.9	-1.1	8.6	8.5	Euth.	N/A	MK
GJ22	M	20	16.9	-3.1	7.9	3.8	Euth.	N/A	MK
GJ50	M	14	14.8	0.8	7.8	2	Euth.	N/A	MK
GJ59	F	16	15.2	-0.8	7	2	Euth.	N/A	MK
GJ69	F	16	17.4	1.4	10.2	7.9	Euth.	N/A	MK
GJ18	M	16	9.5	-6.5	3	1.4	Dead	11	QT
GJ5	M	24	22.6	-1.4	2.1	9.5	Euth.	N/A	QT
GJ19	M	22	15.1	-6.9	2.7	2.6	Euth.	N/A	QT
GJ31	F	22	18.7	-3.3	3.5	3.6	Euth.	N/A	QT
GJ40	M	16	16.3	-0.3	2.8	4.4	Euth.	N/A	QT
GJ6	F	13	13	0	7.7	1.6	Euth.	N/A	RG
GJ25	F	21	22.4	1.4	15.8	5.3	Euth.	N/A	RG
GJ28	F	21	21.2	0.2	11.7	3.1	Euth.	N/A	RG
GJ52	M	19	17.9	-1.1	10.8	1	Euth.	N/A	RG
GJ60	M	13	11.8	-1.2	9.7	2.7	Euth.	N/A	RG
GJ32	M	22	18.6	-3.4	10.1	1.3	Dead	8	RZ
GJ1	F	18	17	-1	8.7	0.7	Euth.	N/A	RZ
GJ11	F	16	14.8	-1.2	9.8	0.8	Euth.	N/A	RZ
GJ29	M	21	22.8	1.8	12.5	1.4	Euth.	N/A	RZ
GJ41	M	13	13.3	0.3	8.4	1.1	Euth.	N/A	RZ
GJ20	F	19	21.3	2.3	2.2	4.2	Dead	2	ZP
GJ57	F	14	12.6	-1.4	1.2	1.7	Dead	2	ZP
GJ4	M	25	23.6	-1.4	2.7	13.7	Euth.	N/A	ZP
GJ21	M	25	20.1	-4.9	1.1	9.2	Euth.	N/A	ZP
GJ39	M	19	19.2	0.2	2.6	9.3	Euth.	N/A	ZP

GJ2	M	20	20.1	0.1	0.3	1.8	Dead	1	ZO
GJ3	F	24	22.8	-1.2	0.1	1.1	Dead	1	ZO
GJ23	F	18	16.8	-1.2	0.4	0.8	Dead	1	ZO
GJ38	M	19	19.1	0.1	0.3	1.2	Dead	1	ZO
GJ47	M	19	16.7	-2.5	0.2	0.9	Dead	1	ZO

Appendix 2. House mouse treatments, sex, weight, food consumption, fate, and days to death (TD) for 7-day rodenticide exposure trial.

Mouse No.	Sex	In Wt.	Out Wt.	Wt. Change	Bait Cons	Chow Cons	Fate	Days TD	Trt
HE-33	F	20	23.6	3.6	N/A	40.5	EUTH	N/A	C
HE-37	F	19	22.8	3.8	N/A	44.5	EUTH	N/A	C
HE-38	M	15	20.3	5.3	N/A	34.1	EUTH	N/A	C
HE-39	F	8	16	8	N/A	46.2	EUTH	N/A	C
HE-40	F	25	22.4	-2.6	N/A	41.1	EUTH	N/A	C
HE-47	F	22	21.9	-0.1	19.3	1.8	DEAD	7	JB
HE-49	F	16	16.6	0.6	14.5	0.6	DEAD	10	JB
HE-52	M	15	13	-2	17.9	1.2	DEAD	14	JB
HE-54	M	10	13.6	3.6	17	2.3	DEAD	12	JB
HE-45	F	18	19.4	1.4	23.2	-0.7	EUTH	N/A	JB
HE-59	F	17	14.5	-2.5	LT LIQ	21.7	DEAD	8	LT
HE-64	M	15	18.7	3.7	LT LIQ	16.7	DEAD	6	LT
HE-66	M	20	19.6	-0.4	LT LIQ	26.2	DEAD	8	LT
HE-57	F	19	19.7	0.7	LT LIQ	33.0	EUTH	N/A	LT
HE-60	F	21	23.7	2.7	LT LIQ	31.9	EUTH	N/A	LT
HE-51	F	18	13.9	-4.1	15.5	4.6	DEAD	6	MK
HE-53	F	22	23.4	1.4	36.9	5.2	EUTH	N/A	MK
HE-55	F	15	17.1	2.1	20.5	1.9	EUTH	N/A	MK
HE-56	M	13	19.7	6.7	27.9	2.2	EUTH	N/A	MK
HE-58	M	18	22.5	4.5	21.6	6.8	EUTH	N/A	MK
HE-62	F	19	15.3	-3.7	2.3	14.6	DEAD	8	QT
HE-61	F	19	19.1	0.1	3.5	22.2	EUTH	N/A	QT
HE-63	F	22	22.2	0.2	2.6	20.5	EUTH	N/A	QT
HE-67	M	8	17.1	9.1	3.2	24.6	EUTH	N/A	QT
HE-69	M	20	21.4	1.4	2.6	5.0	EUTH	N/A	QT
HE-65	F	21	19.4	-1.6	14.1	2.1	DEAD	8	RG
HE-72	M	13	14.7	1.7	9.7	0.1	DEAD	5	RG
HE-68	F	21	20.7	-0.3	12.2	5.9	EUTH	N/A	RG
HE-70	F	19	14.3	-4.7	20.6	-0.3	EUTH	N/A	RG
HE-71	M	14	18.7	4.7	24.8	3.9	EUTH	N/A	RG
HE-44	F	12	11.6	-0.4	14.4	-3.0	DEAD	9	RZ
HE-48	M	22	20.9	-1.1	24.3	0.9	DEAD	9	RZ
HE-42	F	18	17.4	-0.6	14.5	-1.0	EUTH	N/A	RZ
HE-43	F	12	15.2	3.2	29.8	1.2	EUTH	N/A	RZ
HE-46	M	13	15.9	2.9	22.1	0.6	EUTH	N/A	RZ