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Pen and field trials of flupropadine against the house mouse (*Mus musculus* L.)

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SUMMARY

Laboratory and field trials were conducted to determine the efficacy of the candidate rodenticide flupropadine against the house mouse (*Mus musculus* L.). In laboratory feeding tests, family groups of wild mice maintained in pens and conditioned to feeding on plain foods were offered flupropadine at either 0·10 %, 0·15 %, 0·18 % or 0·20 % in pinhead oatmeal bait. Overall mortalities in replicated 21-day treatments were 66/71 (93·0 %), 71/79 (89·9 %), 72/76 (94·7 %) and 69/75 (92·0 %) respectively.

In 17 field trials carried out against mice infesting farm buildings, flupropadine was used at 0·10 %, 0·15 % and 0·18 % in oatmeal bait. Mean treatment success, estimated from live-capture and mortality data, was 88·6 %, 96·2 % and 96·6 % respectively.

Flupropadine was found to be as near effective against mice as calciferol/warfarin and the second-generation anticoagulant rodenticides difenacoum, bromadiolone and brodifacoum. In further comparison with the anticoagulants, treatment with flupropadine bait achieved markedly quicker control.

INTRODUCTION

Recent work on alternative rodenticides in this laboratory has resulted in the evaluation of flupropadine, 1-(3,5-bistrifluoromethyl phenyl-3(4-tertbutyl piperidino)-prop-1-yne, as the hydrochloride (unpublished data). It was concluded from the results of feeding tests on wild house-mice (*Mus musculus* L.) that flupropadine would be most effectively employed at between 0·10 % and 0·20 % in poison-baiting treatments against mouse infestations.

In keeping with previous studies on promising new rodenticides (see, for example, Rowe & Bradfield, 1976; Rowe, Plant & Bradfield, 1981), the performance of flupropadine was subsequently examined in feeding trials carried out on confined and free-living populations of mice. The results of the pen and field trials are presented in this paper.

METHODS

Pen trials

Family groups of wild mice, consisting of sub-adult and adult animals, were used. Each group was reared in a breeding cage from stock known to be resistant to warfarin (Rowe & Bradfield, 1975) and the cage was transferred to the nesting area of a metal enclosure measuring 9·5 × 2·5 m when two or more litters had been raised. Trays containing plain food (whole wheat grain mixed with crushed laboratory Diet FFG(M), Dixon & Son's (Ware) Ltd) were placed on either side of the cage and drinking water was also provided *ad lib.* The mice were accustomed to the environmental conditions for 7 days before they were tested.

Flupropadine was used at 0·10 %, 0·15 %, 0·18 % and 0·20 % in cereal bait. Poison bait was prepared, the 0·18 % formulation excepting, by thoroughly mixing an appropriate amount of the pure compound in corn oil (5 %) with wholemeal flour (5 %) and pinhead oatmeal (to 100 %); the 0·18 % treated bait, a proprietary formulation (May & Baker Ltd), contained ondina oil (1·8 %) in place of corn oil and, also differently, a warning dye. In four trials with each concentration of the poison, bait was laid in open trays placed at eight sites outside the nesting area. The sources of plain food were maintained throughout the treatment period (21 days maximum duration) and the total amount of poison bait eaten was measured daily. The cage and pen were also searched daily to recover dead mice.

Field trials

In 17 field trials, flupropadine was used at either 0·10 %, 0·15 % or 0·18 % in oatmeal bait. The trials were carried out in isolated buildings (granaries, milling-sheds, dairy units, workshops and offices) located on mixed arable/dairy farms in Sussex and Surrey. A building was allocated for experimental use after an inspection for fresh rodent signs (faeces, runs, smears and holes) showed that it was infested by mice and not by rats (*Rattus norvegicus*).

A mark–release trapping programme was conducted in each building before poison bait was laid. For this purpose, Longworth live-traps (Chitty & Kempson, 1949) were set, for 4 days throughout the infested area. Mice were sexed, weighed and individually marked at first capture and recaptured animals were identified before their release, also at the point of capture.

The flupropadine treatment was begun 3 days later. Small covered containers were laid 1–2 m apart at sites different from those chosen for traps. The containers were initially supplied with 20–30 g of poison bait and, thereafter, they were regularly examined to ensure that surplus bait was always available. The total amount of poison bait eaten was measured on 4 days (Tuesday–Friday) and then over the next 3 days in each week of the treatment period. The treatment was terminated when the take of poison bait ceased; the containers and surplus bait were then removed.

Surviving mice were trapped-out, using Longworth traps and, at a later stage, when necessary, Little Nipper snap-traps. Removal was begun on the last day of each treatment and, after 2–3 days of trapping, numerous patches of fine dust, basic slag, were laid in areas where mice had been previously active. Trapping was continued until no mouse signs were found in the patches over 2 days.

Table 1. *The toxicity of flupropadine to penned groups of* Mus musculus *in 21-day 'choice' feeding tests*

Trial no.	Concentration (%)	Poison bait eaten (g) Days 1–7	Days 8–14	Days 15–21	Mortality	Mortality (%)	Days to death range	Days to death mean
1	0·10	55·0	0·5	0·0	15/16	93·8	4–12	6·5
2	0·10	43·0	1·2	0·3	14/15	93·3	4–9	5·8
3	0·10	42·3	3·5	0·7	22/24	91·7	3–28	10·3
4	0·10	25·7	2·0	0·3	15/16	93·8	6–10	7·6
5	0·15	52·2	0·5	0·0	14/19	73·7	5–12	7·8
6	0·15	90·0	0·0	1·5	32/33	97·0	4–14	6·6
7	0·15	28·3	0·0	0·0	11/12	91·7	4–9	6·6
8	0·15	27·7	0·0	0·0	14/15	93·3	3–12	5·0
9	0·18	17·1	3·9	0·0	15/19	79·0	3–11	6·0
10	0·18	16·6	—	—	19/19	100·0	3–6	4·4
11	0·18	27·0	0·0	—	22/22	100·0	3–9	5·9
12	0·18	18·2	—	—	16/16	100·0	4–7	5·3
13	0·20	22·1	2·3	0·5	19/21	90·5	3–31	9·4
14	0·20	32·5	0·4	1·0	19/20	95·0	5–19	8·3
15	0·20	33·8	0·0	—	17/17	100·0	4–8	5·5
16	0·20	20·9	0·0	—	14/17	82·4	3–37	9·2

Minimum percentage success in each treatment was estimated from counts of the number of mice known to have been present at pre-treatment (the number of marked and unmarked animals examined in the trial period) and at post-treatment.

Laboratory feeding tests

The live-caught survivors of the field trials were transferred to the laboratory. Some of the adult mice were paired for breeding purposes; the remaining animals were individually caged and maintained on Diet FFG(M) and water *ad lib.* After a rest period lasting 3 weeks or longer each mouse was given, without choice, the flupropadine bait tested against it in the field. The amount of poison bait eaten was measured daily until death.

RESULTS

Pen trials

The amount of flupropadine bait eaten by mice in the pens is shown in Table 1. In all 16 treatments, poison bait consumption was highest in the first week and in seven of the treatments there was no further take of bait. Dead mice were found from day 3 onwards and a high proportion (71·9 %) of the poisoned animals died within 7 days; the effects of flupropadine poisoning were delayed, however, in five mice that died several days after the end of a treatment, on days 28 (1), 30 (2), 31 (1) and 37 (1).

Overall mortality was high (278/301; 92·4 %). Mean treatment success in the trials of 0·10 %, 0·15 %, 0·18 % and 0·20 % flupropadine was 93·0 %, 89·9 %, 94·7 % and 92·0 % respectively.

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Table 2. *The results of flupropadine poison treatments against infestations of* Mus musculus

Trial no.	Concentration (%)	Pre-treatment number of mice	Poison bait eaten (g) Days 1–7	Days 8–14	Days 15–21	Post-treatment number of mice	Estimated success (%)
1	0·10	38	134	0	0	2	94·7
2	0·10	55	329	0	0	1	98·2
3	0·10	106	1249	0	0	7	93·4
4	0·10	14	98	0	0	1	92·9
5	0·10	76	34	12	15	17	77·6
6	0·10	88	147	38	21	15	83·0
7	0·15	71	109	9	0	4	94·4
8	0·15	67	470	9	0	8	88·1
9	0·15	148	250	0	0	4	97·3
10	0·15	77	482	0	0	0	100·0
11	0·15	32	178	0	0	0	100·0
12	0·15	27	161	4	0	0	100·0
13	0·18	72	590	51	51	3	95·8
14	0·18	30	101	79	0	2	93·3
15	0·18	41	60	0	0	2	95·1
16	0·18	48	168	12	0	0	100·0
17	0·18	137	364	56	26	4	97·1

Field trials

The results of the field trials are summarized in Table 2. As in the pens, most poison bait was consumed in the first week and no additional feeding occurred in 8 of the 17 treatments. Also similarly, the first dead animals were found on day 3.

Treatment success in the six trials of 0·20 % and of 0·15 % flupropadine bait was estimated to be 88·6 % and 96·2 % respectively; the kill achieved in the five trials undertaken with the 0·18 % bait formulation was 96·6 %. Statistical analysis of the data given in Table 2, using the number of survivors as a percentage of the pre-treatment number of mice in an analysis of variance, indicated no difference in effectiveness between the three poison baits.

Laboratory feeding tests

The results of the 'no-choice' feeding tests on 29 survivors of the field trials are given in Table 3. Poison bait consumption fell after 2–3 days, the mice dying between days 3 and 7.

DISCUSSION

The flupropadine treatments in the pens followed the same general course. Examination of the amounts of poison bait eaten daily showed that consumption was highest on day 1, decreased steadily until day 3 or 4 and then almost ceased. These findings, coupled with the relatively early death of most animals, indicated that flupropadine is cumulatively toxic to *M. musculus*, about 1–2 days feeding on bait containing 0·1–0·2 % of the compound being sufficient to cause death.

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Table 3. *The results of flupropadine feeding tests on survivors of the field treatments*

Concentration (%)	Sex	Mortality	Lethal dose of active ingredient (mg/kg)	Days to death	
				Mean	Range
0·10	M	9/9	380–694	5·3	4–7
	F	4/4	417–635	5·5	5–7
0·15	M	1/1	882	5·0	—
	F	5/5	503–984	4·8	4–6
0·18	M	5/5	438–1120	4·6	3–7
	F	5/5	822–1447	5·4	4–7

The mouse populations in the experimental farm buildings were living under near optimum conditions. Food, mainly loose or crushed grain and concentrated feed, was available to them in excess and cover was more abundant than in the pens. Not surprisingly, in view of the more difficult baiting conditions and the larger numbers of mice, the farm populations were less readily diverted to feeding on flupropadine bait than were the family groups. Thus, the greatest take of poison bait in the buildings often occurred on day 2 or 3 and, in further comparison with the pen treatments, feeding was rather more protracted during the first week. The results of equivalent pen and field trials were, nevertheless, highly comparable.

All 23 survivors of the pen trials were female animals. No evidence of a sex difference in susceptibility to flupropadine was found in the laboratory cage tests, however, and both male and female mice were captured at post-treatment in the field. Aggressive interactions between males were occasionally observed in the pens and it is concluded that these had a deleterious effect on the take of bait by some of the females.

The laboratory feeding tests showed that the survivors of the field trials were susceptible to flupropadine poisoning. Their survival, and that of the females in the pens, must be presumed to have been due to a total lack of feeding on poison bait, or to inadequate feeding and the consequent ingestion of a sub-lethal dose of the poison. Flupropadine bait was well distributed for 3 weeks and this, together with the evidence of a persistent small take of bait in the last week of some of the pen and field trials, suggests that survival was most likely the result of under-feeding.

Assessment of the potential use of flupropadine as a poison against mice is best made by comparing its performance with other rodenticides tested in the field in recent years – calciferol combined with warfarin and the second-generation anticoagulant compounds, difenacoum, bromadiolone and brodifacoum. Four of the 17 flupropadine trials were completely successful and mean treatment success was 93·8%. In six field trials employing calciferol/warfarin in canary-seed bait, the control obtained ranged between 97·0% and 100%, mean 98·6% (Rowe, Smith & Swinney, 1974). Also similarly, there was marginal feeding on calciferol/warfarin bait after the first few days. The flupropadine and calciferol/warfarin treatments were near equally effective therefore in comparably short feeding periods.

Six field trials were also undertaken with each of the three anticoagulant

rodenticides (Rowe, Swinney & Plant, 1978; Rowe, Plant & Bradfield, 1981). Mean treatment success in the trials using difenacoum, bromadiolone and brodifacoum was estimated to be 96·0 %, 92·4 % and 98·8 % respectively. Nine of the 18 farm populations were eradicated—a higher success rate than that achieved using flupropadine. In further comparison with flupropadine, however, considerably more bait was consumed in the anticoagulant treatments, half of the latter having to be continued for 5 weeks or longer before satisfactory control was achieved.

It is concluded that flupropadine at between 0·1 % and 0·2 % in bait is an effective alternative poison for the control of house mice. The results of the present trials indicated the need to obtain high initial feeding on flupropadine bait, by the use of an attractive bait-base and adequate baiting points, in order to avoid sub-lethal dosing.

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