

Field trials of difenacoum against warfarin-resistant infestations of *Rattus norvegicus**

By B. D. RENNISON

Pest Infestation Control Laboratory, Ministry of Agriculture, Fisheries and Food, Hook Rise South, Tolworth, Surbiton, Surrey

AND M. R. HADLER

Sorex (London) Ltd, Research Department, Halebank Factory, Lower Road, Widnes, Cheshire

(Received 3 January 1975)

SUMMARY

The anticoagulant difenacoum was tested at two concentrations, 0.005 and 0.01 %, in bait against warfarin-resistant rat infestations in farm buildings. Twelve out of the 14 treatments in which the lower concentration of the anticoagulant was used resulted in complete control. One of the remaining two treatments was probably also completely successful, but in the other a few rats, that were not eating the poisoned baits, were still active after 30 days of baiting. All six treatments done using the stronger concentration of poison were completely effective.

Since it took as long to control infestations with 0.01 % as with 0.005 % difenacoum in treatments carried out under similar conditions, the lower concentration is recommended for use against warfarin-resistant rats.

INTRODUCTION

This paper describes the field trials that were carried out in Powys (Montgomeryshire) and Kent against *Rattus norvegicus* with the anticoagulant difenacoum [3-(3-*p*-diphenyl-1,2,3,4-tetrahydronaphth-1-yl)-4-hydroxycoumarin]. As in previous field trials of candidate rodenticides (Rennison, 1974*a, b*) the treatments were done in farm buildings that were infested by rats resistant to the commonly used anticoagulant warfarin. The two separate areas were chosen for the trials because the resistant rats in each probably represented different genetic strains.

METHODS

General

All the poison treatments described below were carried out using the method described by Drummond & Rennison (1973) for testing for anticoagulant resistance in the field. Infested farm buildings were surveyed and baiting points selected 2-4

* Crown copyright.

days before poison baiting began. Poison baits weighing 200–300 g. were laid on trays (for easy recovery) on a Monday and were thereafter inspected and, when necessary, replenished on successive Wednesdays, Fridays and Mondays until it was evident that the treatments had failed or succeeded. A treatment was deemed to have failed when the proportion of bait points with bait takes by rats had been significantly higher than expected for two successive visits, or to have succeeded when no bait takes had occurred and no other evidence of rats (active holes or foot-prints on trace patches) had been seen for the same length of time.

Powys

The presence of warfarin-resistant rats on the farms was first tested for and confirmed either by treating the infestations (unsuccessfully) with 0.025 % warfarin (farms 1–3) or by trapping samples of rats and then feeding them for 6 days in the laboratory on a diet of 0.025 % warfarin in medium oatmeal (farms 4–15).

In the treatments of the farms that followed, two concentrations of difenacoum, 0.005 % and 0.01 %, were tested, the former on nine farms (1–9) and the latter on six (10–15). Both concentrations had been effective against warfarin-resistant rats in laboratory tests (Hadler, Redfern & Rowe, 1975) but the baits containing the lower concentration had been marginally more palatable and for that reason could be more effective under practical conditions.

Stabilized medium oatmeal was used as bait in all treatments, including the warfarin test treatments on farms 1–3, except at those points that were sited, for example in granaries, milling sheds or animal food stores, where a dry bait would probably have been insufficiently attractive. In such situations soaked wheat was used instead.

The anticoagulant was added to the bait by mixing one part by weight of a fine oatmeal-based mastermix that contained, besides 0.5 % Chlorazol Sky Blue, 0.1 % difenacoum, into 19 (or 9 if 0.01 % difenacoum was required) parts by weight of cereal.

The first three farms were treated by one pair of Rodent Operators, the remaining 12 (4–15) by the same or by a second pair. Each pair treated a randomly chosen group of three of the latter farms at a time, and while one pair used 0.005 % the other used 0.01 % difenacoum and vice versa. The 12 treatments were completed in the same 32-day period.

Kent

Laboratory injection tests (i.e. rats were injected with a dose of 200 mg./kg. of warfarin dissolved in dimethyl formamide) were used to confirm that samples of rats trapped on farms 16–20 included warfarin-resistant individuals. The treatments on the farms were conducted using 0.005 % difenacoum in stabilized medium oatmeal, mixed as in Powys. One of the authors (M.R.H.), assisted by a Rodent Operator from the Maidstone Division of MAFF, carried out the treatments.

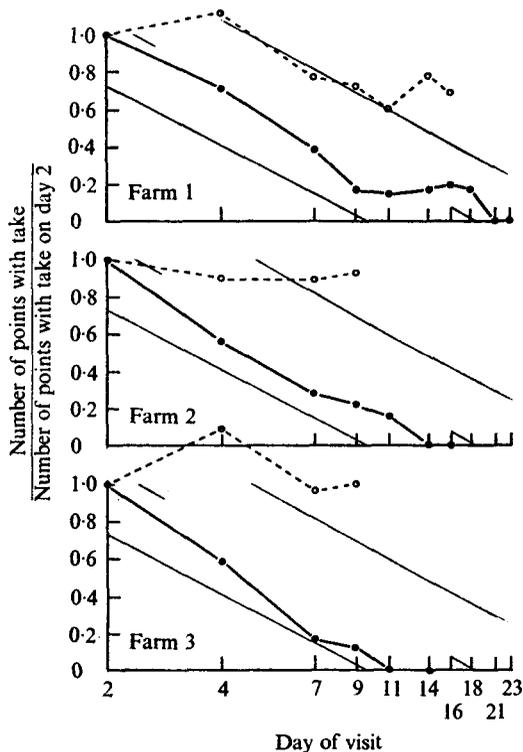


Fig. 1. The results of poison baiting with 0.025% warfarin (broken lines) and afterwards with 0.005% difenacoum (solid lines) on farms 1-3.

RESULTS

Powys

The results of the warfarin-resistance test treatments and of the re-treatments that followed with 0.005% difenacoum on farms 1-3 are shown in Fig. 1. The high proportions of warfarin bait points that had rat takes on day 4 on farms 1 and 3 probably occurred because, owing to the resistance of a proportion of the rat population to warfarin, feeding on the baits increased after the second day of baiting. The presence of resistant individuals was not considered to have been confirmed on any of the three farms, however, until the proportions of points with bait takes had clearly ceased to decrease and had remained above the upper (95% confidence) line of the graph for two consecutive visits. At that stage the warfarin baits were taken up and the three farms were left unbaited until the following Monday, when baits containing 0.005% difenacoum were laid, at the same bait points.

The difenacoum treatments were by contrast completely successful; on farms 2 and 3 in less than 18 days (the average length of time expected in a warfarin treatment against non-resistant infestations) and on farm 1 after 21 days. Birds were found to be responsible for some if not all of the later bait takes on farm 1 and it is possible that the rats were eradicated more quickly than the results in Fig. 1 suggest.

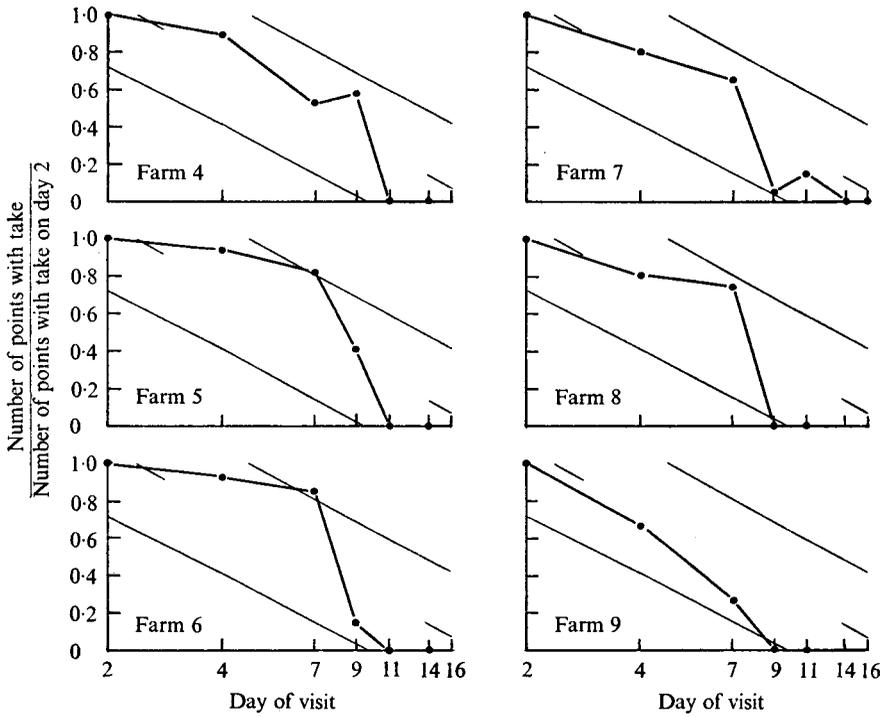


Fig. 2. The results of poison baiting with 0.005 % difenacoum on farms 4-9.

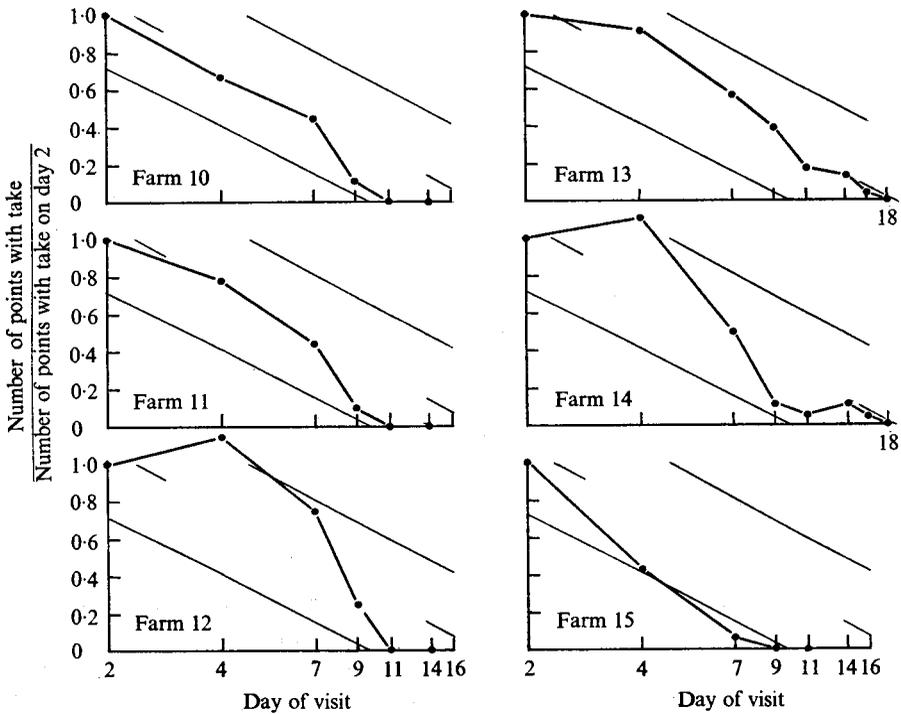


Fig. 3. The results of poison baiting with 0.01 % difenacoum on farms 10-15.

The results of the treatments on farms 4–9, in which 0.005 % difenacoum was used, and those of the treatments on farms 10–15 with 0.01 % difenacoum are given respectively in Figs. 2 and 3. All 12 were successfully completed in 18 days or less. On two of the farms (8 and 9) that were treated with 0.005 % difenacoum and on one (15) treated with 0.01 % difenacoum the infestations were controlled in significantly ($P < 0.05$) less than the average time (18 days) that is recorded on the graph for warfarin against non-resistant rats. However, the three infestations were relatively small ones and since there was also little farm stock the rats had little or no food to eat other than the bait. The significantly slow rate at which the numbers of takes decreased between days 2 and 7 on farms 5 and 6 and the significantly high proportions that were recorded on farms 12 and 14 on day 4 indicate that warfarin-resistant rats possibly feed longer than might be expected on difenacoum before succumbing to the poison.

Kent

The trials in Kent were, unfortunately, disrupted during the first week of baiting by exceptionally heavy rain. Consequently, it was impossible on farms 17 and 20 to judge how many of the wet or washed out bait points had been visited by rats on either day 4 or 7. The bait takes on the two farms could not, therefore, be satisfactorily recorded until day 9 and so the first week's results were disregarded and each treatment was re-started and monitored from the beginning of the second week. Fig. 4 shows the results of all five treatments and the true time scale of the two in question can be obtained by adding 7 days to the values shown on the abscissae of the graphs.

The treatments on farms 16–18 were completely effective within 23 days. No baits were touched by rats on farm 19 between day 21 and 23 but on day 28 (the farm was not visited on day 25) a take was recorded at a point at which the bait had been untouched by rats since day 7. Since none of the blocked holes in the vicinity of the point had been reopened, the single take can probably be attributed to a rat or rats from outside the treated area. The significantly large numbers of takes that were recorded on this farm during the first week may have been a consequence of warfarin resistance, but it is also likely that the rats behaved abnormally in the exceptionally wet weather.

Some rats were also still active on farm 20 after 30 days of baiting (Fig. 4; day 23). In this case the activity was confined to the area of the grain silos on the farm where spillage provided abundant attractive food for the rats. Because of the available grain, the recorded takes of oatmeal bait in that area were never large and for the last week of the treatment they amounted, when they occurred, only to foot-prints or tail swipes on the bait. A more attractive bait, such as soaked wheat, could possibly have been used to overcome the baiting problem and deal with the few surviving rats, but it was not possible in the limited time to continue the treatment. As it was the authors were satisfied that the maximum control had been achieved with the oatmeal bait.

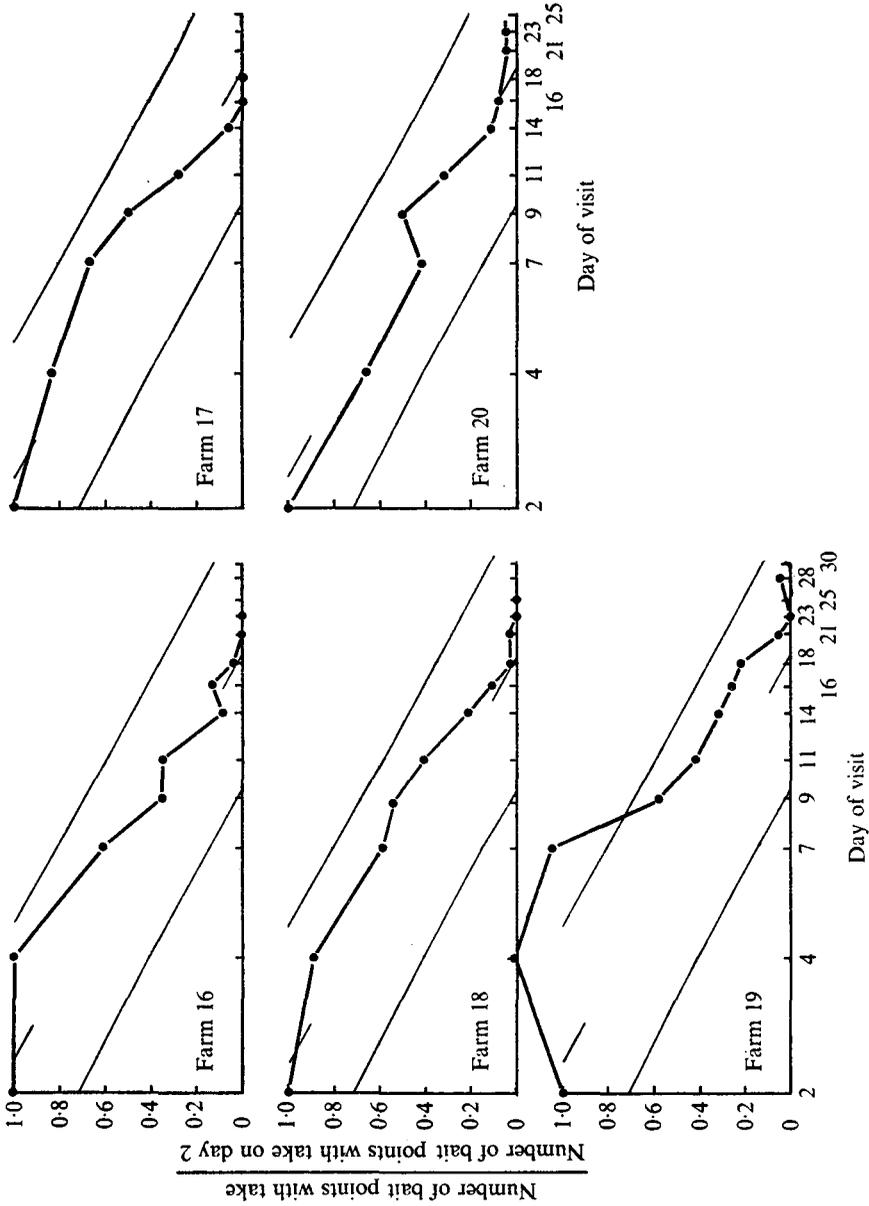


Fig. 4. The results of poison baiting with 0.005% difenacoum on farms 16-20.

CONCLUSIONS

The field trials confirm that difenacoum is an effective rodenticide for the control of warfarin-resistant common rats. Since there is no evidence in the trials to suggest that the treatments that are carried out using the anticoagulant at 0.005% will take longer or be less effective than would those in which twice the optimal concentration is used, both cost and safety dictate that use of the lower concentration should be recommended.

It is possible that in practice a lower concentration of difenacoum than 0.005% would also prove to be effective against warfarin-resistant rats. However, baits would then probably be so ineffective against warfarin-resistant mice, against which 0.01% difenacoum proved to be more effective than 0.005% in pen trials (Rowe & Bradfield, 1975) that it would not be possible to control both rodents satisfactorily with the same concentration of poison.

The authors wish to acknowledge with thanks the important parts played by Mr F. Pritchard, Mr E. Jones, Mr G. Long and Mr E. Pugh in Powys and the assistance of Mr E. Link in Kent; also the help received from Mr N. J. Wallace and Mr E. J. Wilson in the former county.

REFERENCES

- DRUMMOND, D. C. & RENNISON, B. D. (1973). The detection of rodent resistance to anti-coagulants. *Bulletin of the World Health Organization* **48**, 239-42.
- HADLER, M. R., REDFERN, R. & ROWE, F. P. (1975). Laboratory evaluation of difenacoum as a rodenticide. *Journal of Hygiene* **74**, 441.
- RENNISON, B. D. (1974a). Field trials of the rodenticide 5-*p*-chlorophenyl silatrane against wild rats (*Rattus norvegicus* Berk.). *Journal of Hygiene* **73**, 45-8.
- RENNISON, B. D. (1974b). Field trials of calciferol against warfarin resistant infestations of the Norway rat (*Rattus norvegicus* Berk.). *Journal of Hygiene* **73**, 361-7.
- ROWE, F. P. & BRADFIELD, A. (1975). Comparative acute and chronic toxicity tests on confined colonies of wild house mice (*Mus musculus* L.). *Proceedings of the 4th British Pest Control Conference, Jersey*, April 1975, to be published.