PREDICTING THE OUTCOME OF RODENTICIDE TRIALS AGAINST NORWAY RATS LIVING ON FARMS

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ABSTRACT: Difenacoum and bromadiolone treatments against Norway rats may fail because: 1) the animals eat little or no bait, 2) reinvasion rapidly offsets any success, or 3) the population contains resistant individuals. By monitoring bait takes and employing independent measures of rat activity such as tracking plates, it is possible to identify, often in the early stages of a treatment, patterns that indicate the contribution of each of these causes to the eventual outcome. If there is no bait take from the majority of bait points visited by rats in the first week then the treatment is unlikely to be successful, no matter how long it continues. Furthermore, treatments carried out on arable farms, where cereals are stored and the environment is relatively undisturbed, are likely to be less successful than those carried out on livestock farms, where alternative food may also be abundant but where the environment is less predictable. Bait takes that persist at the same bait points for longer than 16 days strongly suggest the presence of resistant rats, while immigration may be significantly affecting the treatment if takes recur at more than 30% of points after a period of seven days. Once a given problem has been identified remedial measures can be taken.

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INTRODUCTION

Control of Norway rats (*Rattus norvegicus*) in the UK is carried out mostly using anticoagulant rodenticides formulated into baits. These baits have to be sufficiently attractive and potent to ensure that rats are drawn away from their usual food sources and ingest a lethal dose. If a treatment, competently carried out, fails to deliver the expected reduction in numbers, rats may have survived because: 1) they ate little or no bait, 2) they are resistant to the active ingredient, or 3) although the treatment against the original population was successful, the effect was rapidly offset by immigration from populations outside the treated area. Methods of monitoring treatments that allow discrimination between these factors would be of benefit to pest control operators, so that remedial action can be taken at an early stage, and also in the evaluation of data on the efficacy of rodenticide formulations.

In the late 1970s there were reports of control problems in some parts of central-southern England when the then recently introduced second-generation anticoagulant, difenacoum, failed to give satisfactory results (Greaves, Shepherd and Gill 1982a). Field trials with other second-generation anticoagulants also gave rise to relatively poor population control in this area and resistance was viewed as a causal factor (Greaves, Shepherd and Quy 1982b). Greaves and Cullen-Ayres (1988) suggested that the degree of resistance possessed by difenacoum-resistant animals was insufficient, on its own, to account for the observed poor treatment outcomes. This view was confirmed by Quy, Shepherd and Inglis (1992b) who considered that reasons for failed treatments were more complex than resistance per se and that reinvasion and differential behavioral responses towards bait were also involved.

We have recently completed a series of trials on farms with either difenacoum or bromadiolone baits, with the primary aim of determining why rats survived specific treatments. In a first analysis of the data, the presence of difenacoum-resistant rats at a trial site was shown not to affect significantly the outcome of the treatment (Quy et al. 1992a). The presence of alternative food, particularly stored cereal, significantly reduced treatment effectiveness. About 90% of those rats that survived a seven-week poison treatment did so by consuming less than one g/rat/day. In this paper we consider the three factors which, alone or in combination, may reduce the efficacy of anticoagulant treatments, namely: 1) poor bait consumption, 2) resistance, and 3) reinvasion. Our aim is to identify patterns in bait takes and other measures of rat activity that predict the contribution of each of these factors to the eventual outcome of treatments. In addition, some possible remedies are suggested once a given problem has been identified.

METHODS

A detailed description of the methods used can be found in Quy et al. 1992a. Briefly, a total of 48 trials were carried out on farms in two counties of central-southern England, 24 in Hampshire where difenacoum resistance was known to be widespread (Greaves et al. 1982a) and 24 in the adjacent county of West Sussex where such resistance has not been found. Before each treatment a sample of rats was live-trapped and tested for resistance in the laboratory. The trials were divided into eight replicates, each replicate consisting of six treatments, three in each county. The three treatments in each county were: 1) seven weeks baiting with 50 ppm difenacoum, 2) seven weeks baiting with 50 ppm bromadiolone, and 3) three weeks baiting with unpoisoned bait (to measure bait consumption in the absence of mortality due to consumption of rodenticide), followed by four weeks baiting with 50 ppm bromadiolone (hereafter referred to as a control treatment). All baits were formulated by mixing a liquid concentrate with pinhead oatmeal, corn oil and caster sugar. The baits also contained a chemical bait marker at 100 ppm: decachlorobiphenyl for the first three weeks then hexachlorobiphenyl for the next four weeks. Half the treatments began in the autumn (September-October)

and the other half in the spring (February-May). A surplus of bait was maintained in each container (usually a wooden box with a metal lid) and the bait was weighed on a Monday-Wednesday-Friday schedule and the amount taken by rats recorded at each visit.

Each farm was surveyed to determine the extent of the infestation based on rat signs such as tracks, droppings and burrows and a map was drawn of the farm buildings and the adjacent land. Superimposed on the map was a grid representing 10 x 10 m squares. In each square of the treated area the position of any bait point was marked and also the type of any food sources accessible to rats. The types of food were grouped into the following categories: 1) standing crops [wheat, barley, game-cover such as maize or kale]; 2) cereals [wheat, oats, barley]: a) stored in silos or clamps, b) spillage; 3) stored non-cereals [peas, beans, potatoes]; and 4) animal feedstuffs [commercial feeds, maize and apple silage, feeds made on the farm from crushed grain and nutritional additives]. Any change in the presence of such food that involved arrival or complete removal from any grid square during the trial period was recorded. Each farm was classified as either arable or livestock depending on the main activity. Of the 24 sites used in each of the two areas, arable farming was the main activity on 12 Sussex and 16 Hampshire farms; the remainder were classified as livestock farms. Animal feedstuffs were kept on 21/48 sites and cereals on 24 sites regardless of the main activity. Non-cereal seeds were stored on eight farms, although, in general, these were not often taken by rats. On eleven farms no food source in or around the buildings was identified: the attraction for rats on four of these appeared to be a nearby standing crop, on the other seven it was not obvious what the rats were feeding on.

The size of each rat population was assessed using a tracking plate method during the week before the bait was laid, the third and sixth week of the treatment, and the week following complete removal of the bait (Quy, Cowan and Swinney 1993). Daily estimates of the size of the population present on each farm were obtained by linear interpolation between each of the successive census estimates. On two non-consecutive days each week throughout the treatment, a visit to a bait point by rats was detected by footprints left on a tracking plate placed on one side of the container; each plate was scored as being marked or not. In this way bait points visited by rats but where no bait was taken, could be identified. Active bait points were defined as those visited by rats whether or not there was a take at anytime during the seven-week treatment. After the post-treatment census, any survivors were trapped, tested for anticoagulant resistance and analyzed for the presence of the chemical bait markers.

In the statistical tests all percentages or proportions were arcsine square-root transformed before analysis, but untransformed means and standard errors are given in the text. All significance levels are for two-tailed tests. In tests comparing populations before and after treatment, if the number of rats had increased, we assumed that 100% of the original population was left.

RESULTS

The mean estimated size of the initial rat population on the Sussex farms was 117.8 ± 21.6 (range 13 to 498), which did not differ significantly from 103.0 ± 15.0 (range 11 to 293) on the Hampshire farms ($t_{46} = 0.56$, NS). The mean estimated percentage of the original population remaining after the seven-week treatments was 34.6 ± 8.79 in Sussex and 73.0 ± 11.8 in Hampshire ($t_{46} = 2.55$, $p = 0.014$). Only five treatments (one Hampshire, four Sussex) were completely successful; in ten cases (six Hampshire, four Sussex) the population had apparently increased above its initial size by the end of the treatment.

Bait consumption

The average consumption of bait per rat for each day during each treatment was calculated by dividing the total bait consumption recorded between visits by the number of days between visits and by the estimated size of the rat population on that day. On thirteen farms (one control, seven difenacoum, five bromadiolone) the mean estimated take of bait did not exceed 1.5 g/rat/day during any two week period of the treatment. This rate of bait consumption is unlikely to cause any significant mortality. For instance, a fully susceptible male rat weighing 250 g which consumed 1.5 g of bait per day for four days would not be exposed to an LD_{50} dose of either difenacoum or bromadiolone (Greaves and Cullen-Ayres 1988). The rat population on seven of these farms increased (five autumn, two spring treatments), on six it decreased (four spring, two autumn) although none went to extinction. This low average rate of bait consumption occurred on 12/28 (43%) arable farms but only 1/20 (5%) livestock farms (Chi-square 6.66, df 1, $p<0.01$). Furthermore, amongst the 32 treatments with either difenacoum or bromadiolone for seven weeks, this very poor rate of consumption occurred on 7/10 farms with cereals and no animal feeds, in contrast to only 1/7 farms with animal feeds and no cereals (Fisher statistic 4.87, $p = 0.027$).

For each farm the proportion of bait points visited by rats and from which there was a measurable take at any time was calculated (bait points where no take was recorded and there was no evidence of visits by rats at any time, were excluded from this and all subsequent calculations). There was a difference between the three treatments ($F_{2,45} = 5.68$, $p = 0.006$), derived solely from a difference between the control and either difenacoum or bromadiolone ($p < 0.05$, Tukey-B Multiple Range Test). The proportion of active bait points with a take was not affected by the location (county) of the treatment or the type of farm. However, on 16 farms where the main alternative food was cereals and no animal feedstuffs were present, there was a significant difference between the control treatments and the combined difenacoum/bromadiolone treatments: 89.3 ± 5.3% and 57.8 ± 7.9% of active bait points with a take respectively ($t_{14} = 2.65$, $p = 0.019$). In contrast, on 13 farms where animal feedstuffs were present and no cereals, the difference was negligible: 94.0 ± 2.8% control, 82.6 ± 6.8% difenacoum/bromadiolone ($t_{11} = 1.62$, $p = 0.18$).

The mean number of visits to check the bait before a measurable take was recorded at a point was 5.09 ± 0.38, equivalent to 11 to 12 days. The type of treatment, time of year, presence of resistance, type of farm or any other factor did not significantly influence this time. Omitting the control treatments, there was a positive correlation between the proportion of all points visited by rats during the first week of baiting on each farm for which no take was recorded and the proportion of the original rat population left at the end of the treatment (r_{30} = 0.601, $p < 0.001$) (Figure 1). The presence of stored cereal reduced the likelihood of a take occurring in the first week: 59.7 ± 7.0% of active points with no take in the presence of cereal compared with 39.3 ± 5.2% in the absence of cereal (t_{30} = 2.42, p = 0.022). However, any change in the distribution of the food supply increased the chance of getting a take during the seven-week treatment: 15.0 ± 7.7% of active points with no take and with change compared with 32.4 ± 4.7% of active points with no take and no change (t_{30} = 2.59, p = 0.015).

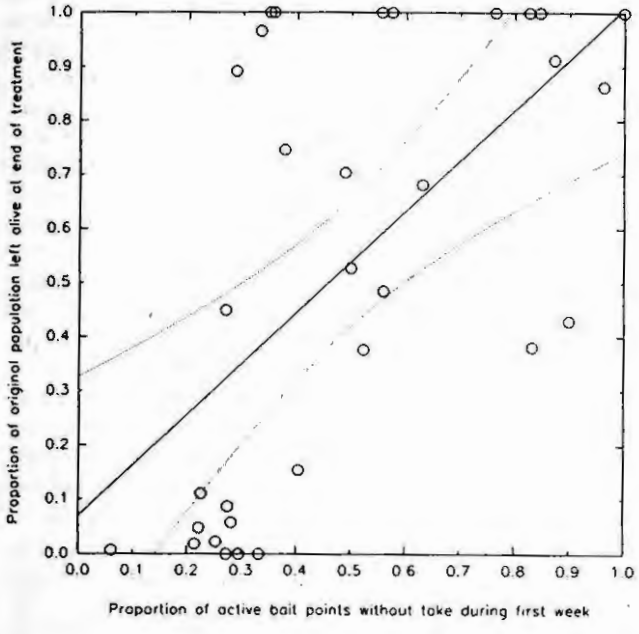

Figure 1. The relationship between the proportion of active bait points (i.e., those visited by rats) during the first week of poision treatments for which no take was recorded in relation to the estimated proportion of the original population left alive at the end of the seven-week treatment (y=0.93x+0.07, r=0.601, p<0.001).

The estimated mean amount of poison bait ingested by each rat present in the first week of poisoning was significantly different between the control treatments (9.6 ± 1.65 g) and difenacoum (3.36 ± 1.11 g) or bromadiolone (3.59 ± 0.92 g) ($F_{2,47}$ = 7.78, p = 0.001), presumably due to what was effectively prebaiting in the control treatments. However, by the second week of poisoning, there was no significant difference in the estimated amount eaten between the treatments (mean = 2.68 ± 0.56 g, range = 2.41-3.52) (F < 1.0). Although the estimated mean amount of unpoisoned bait eaten by each rat on the last day was not significantly different to the estimated mean for the first day's take of poison bait, there was a consistent fall in the amount of bait eaten at the first visit after poison bait had been laid (paired t15 = 4.48, p < 0.001). The greater quantity of poison bait (bromadiolone) consumed in the control treatments led, not surprisingly, to a significantly greater reduction in the rat population (29.0 ± 5.48% remaining) after the second week of poisoning compared with the unprebaited bromadiolone treatments (57.5 ± 7.35% remaining) (t30 = 3.11, p = 0.004). (For this comparison the difference between the second and third census for the control treatments was compared with the difference between the first and second census for the unprebaited bromadiolone treatments.)

Resistance

Warfarin-resistant rats (Martin et al. 1979) were found on all the Hampshire trial sites and on five Sussex sites. Difenacoum resistance (Gill et al. 1992) was found on 21/24 Hampshire sites, but on none of the Sussex farms. The presence of resistant rats might be expected to lead to persistent bait takes from some bait points. Such patterns were looked for on those farms where there were at least two bait points with a take in the first week; the control treatments were omitted. The mean number of consecutive visits where a take was recorded and where difenacoum-resistant rats were found was 2.73 ± 0.29 (equivalent to 6 to 7 days)(n = 11), which was different from the mean 2.00 ± 0.14 (4 to 5 days) consecutive visits where difenacoum resistance was absent (n = 16) (t_{25} = 2.47, p = 0.021). It is apparent from Figure 2 that bait points showing more than seven consecutive visits with a bait take were only found on farms where resistance was present.

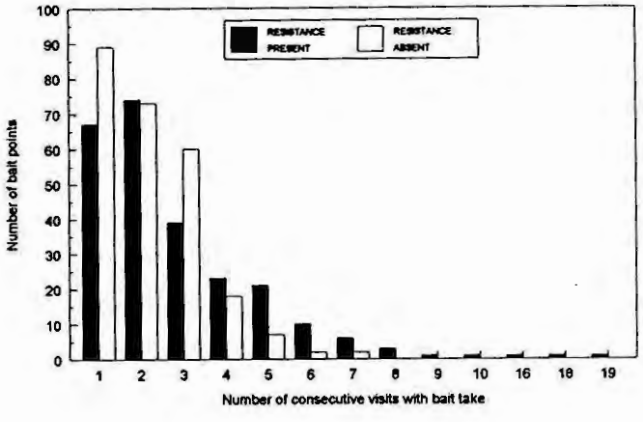

Figure 2. The number of consecutive visits to specific bait points for which bait take was recorded on farms where difenacoum resistant animals were present and farms where there was no evidence of difenacoum resistance.

Reinvasion

Reinvasion by rats living outside the treated area was suspected at several sites. The most common observation was that new takes were recorded in areas thought to have been cleared of rats. To examine this, the farms were divided into two groups: 1) where the population estimate fell below its original size and never increased, and 2) where the population estimate fell below its original level but was then subsequently increased by at least three rats according to the census data, suggesting that reinvasion had occurred. The control treatments were omitted as were any farms where the population never fell below the initial estimate or where census data were missing. A bait point was classed as reactivated if, between recorded takes, there were at least three consecutive visits (i.e., seven days) where no take was recorded. Intermittent takes of within a week are likely to be attributable to the same individuals (Buckle, Odam and Richards 1987). Only data from the first five weeks of the treatment were considered. The mean percentage of bait points that were reactivated on those farms where the census data did not indicate reinvasion was 12.3 $\pm$ 3.8% (n = 8), which differed from 30.4 $\pm$ 4.5% (n = 13) on those farms where reinvasion was suggested by the census data (t_{19} = 2.56, p = 0.019). The mean percentage of reactivated points for Sussex farms did not differ from that for Hampshire farms (t_{19} = 1.49, p = 0.154).

DISCUSSION

It has long been appreciated by pest controllers that effective control is more difficult in places where there are plentiful supplies of food. Yet it is obvious that any rat population cannot exist without an adequate supply of food and if, for any reason, that source is removed, the animals will be forced to move or starve. Simply denying rats access to a food supply may solve many control problems without the need to use any toxic preparations. Yet, realistically and practically, control of rats on farms is going to rely heavily on the use of poison baits and these baits will have to compete for the rats' attention alongside stored grain or livestock feed. The problem can be divided into the likelihood of rats taking any bait and then subsequent amounts they consume. On average the first measurable take at a bait point occurred during the second week of treatments. Such a delay is not surprising since rats are known to be wary of new objects such as bait containers and novel foods such as bait (Shepherd and Inglis 1987). Thus a delay of up to two weeks before any bait is consumed at some points should not cause concern. However, if no bait is taken from more than half of those bait points visited by rats during the first week, then the prospects of a successful treatment are poor. Furthermore, the average time to first consumption is apparently not a good predictor of the eventual outcome of treatments. Thus monitoring takes alone is insufficient: instead it is necessary to also place some tracking device next to each bait container.

If stored cereal is present on a farm, the likelihood of takes occurring is reduced and, if most of the infested buildings are used to store grain, any treatment relying on attracting rats to grain-based baits is unlikely to give acceptable results. On this basis an alternative method of control might be considered from the outset. An absence of grain, for example, on a farm exclusively rearing livestock, improves the likelihood of rats consuming bait. The difference between the two farm types cannot be solely due to the cereal being a competing attraction. Livestock farms often have abundant foods accessible to rats just as stored cereals often are. The key component and difference between livestock and arable farms to a rat may be the relative lack of disturbance in cereal stores, often for many weeks, whereas the movements of farm animals and high turnover of feeds mean that rats are faced with almost constant change. Interestingly, change in the distribution of the food supply, such as removing some, but not necessarily all, cereals increases the likelihood of a take. Unsettling rats by reducing the predictability of their habitat may be sufficient to turn a predicted poor treatment into a successful one.

The average amount of bait taken by each rat was clearly insufficient to reduce the population on at least 43% of arable farms and 5% of livestock farms. In practice, whereas the majority of points had no take at all, some points had good takes and a few rats may have succumbed, although the overall effect was insignificant. The likelihood of rats taking bait appeared to be greater if the unpoisoned bait was present even though cereals were available. This may be a spurious result, because in the presence of unpoisoned bait rats can visit several points over several days, whereas the effects of a poison bait might prevent rats visiting as many points. However, there was no difference between the baits when animal feeds were present; a difference here might be expected since there is usually better control. If it is clear that rats are not starting to eat poison baits within two weeks, replacing the baits with unpoisoned formulations until such time as the rats are willing to take the bait has the advantage of eliminating environmental risks. Action can then be taken to undermine the predictability of the environment to enhance the likelihood of rats consuming bait.

Although difenacoum resistance was found on most of the Hampshire farms used in this study, its impact on the overall efficacy of the treatments was apparently minimal. However, bait takes at specific points that persist for longer than seven visits (i.e., for over two weeks) should arouse strong suspicion that some animals are surviving exposure to the anticoagulant due to resistance. This concept is similar to that involved in the use of the warfarin sampling graph (Drummond and Rennison 1973) to detect anticoagulant resistance. It is, however, more sensitive given that it is based on monitoring consecutive takes at the same bait points and is thus less prone to confounding factors such as reinvasion. Where takes persist, an alternative method of control is required. In this study trapping was employed, but for normal pest control operations this would be labor intensive and therefore expensive, particularly if the residual population was large. In such cases, where the rats are already being attracted to baits, poisons with different modes of action could be tried; calciferol and zinc phosphide are currently available and have the advantage of not selecting for anticoagulant resistance. It is important that such steps are taken when a problem is identified. Otherwise selection pressure favoring anticoagulant resistance will be imposed by partially successful treatments. Such

selection could ultimately lead to more severe practical control problems in the future if it is overlooked.

In the part of England where these trials were carried out, there is a thriving game-rearing interest. It is quite common to find extensive field infestations of rats associated with cover crops such as maize, or pheasant (*Phasianus colchicus*) feeding stations. Often it is impracticable to extend the treated area to include these places and, therefore, inevitably treatments fail or are prolonged. If more than 30% of bait points become reactivated after one week of no takes, immigration should be suspected even if large numbers of rats have been killed. Treatments carried out following the end of the shooting season (February) may be more successful; the cover crops are usually cut and ploughed in or fed to livestock and the feeding stations are empty.

To summarize, in evaluating anticoagulant rodenticide treatments, we recommend that takes from individual bait points should be monitored simultaneously with some means of assessing rat activity that is independent of bait take. With the use of such methods, it is possible to monitor and modify the control strategy accordingly, with various options available if problems are encountered (Figure 3). These problems will not always be clear cut; for example, rats may be attracted to baits quite easily in some parts of a farm, while in other parts they are not. Nevertheless, the additional cost of careful monitoring may well be justified if prolonged and ineffective treatments are avoided. Such monitoring could also be invaluable in carrying out comparative evaluations of anticoagulant formulations while allowing for those factors that influence the outcome of treatments.

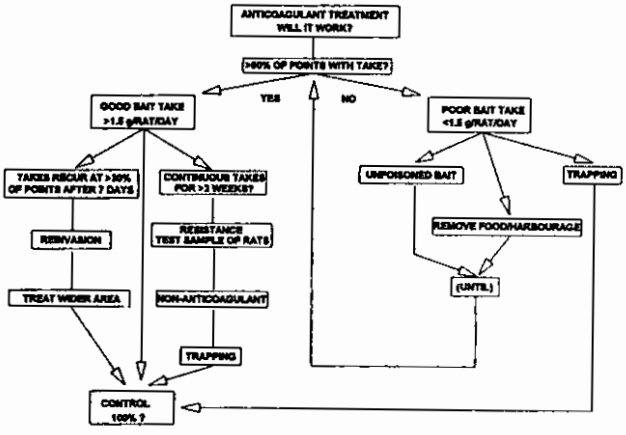

Figure 3. A flow diagram illustrating the use of monitoring techniques to identify the factors reducing treatment effectiveness and the remedial steps available.

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