Anticoagulant rodenticide uptake in resistant rat populations

Anticoagulant rodenticide uptake in resistant rat populations

Laura Daniells[1], Pernille Thorbek[2], Alan Buckle[1], Mark Greener[2] and Colin Prescott[1]

[1] University of Reading, School of Biological Sciences, Harborne Building, University of Reading, Berkshire, RG6 6AS, UK
[2]Syngenta, Jealott's Hill International Research Centre, Bracknell, Berkshire, RG42 6EY, UK
E-mail contact: l.j.daniells@pgr.reading.ac.uk

1. Introduction

Anticoagulant rodenticides are used globally to control pest rodent infestations due to their efficacy and ease of use. Resistance to anticoagulant rodenticides first appeared against warfarin and diphacinone in Scotland in 1958 [1]. In the wake of this, more potent anticoagulant rodenticides were produced but resistance has since developed to some of these [2]. Monitoring of residue levels in predators and scavengers in the UK has shown that anticoagulant rodenticide exposure affects a range of non-target species, including some of high conservation value [3]. The risk to a non-target species will depend on its exposure to anticoagulant rodenticides in the diet and its susceptibility to the anticoagulant rodenticide. However, the impact of resistance on dietry exposure is not well understood, although it is predicted to increase exposure to non-target species [4]. The aim of our work was to explore the level of anticoagulant rodenticide uptake into rodenticide-resistant rat populations during rodent-control trials in Germany and the UK using three different anticoagulant rodenticide compounds, and to assess the relative risks to non-target species as a result. We show that rodenticide-resistance can affect both population control and bait uptake and that rodenticide-resistant areas could be a particular hazard with regard to non-target exposure.

2. Materials and methods

Field trials of three anticoagulant rodenticides (brodifacoum, bromadiolone and difenacoum) were conducted on farms in North-west Germany and Southern England between 2005 and 2010 where resistance to bromadiolone and difenacoum in Norway rats (*Rattus norvegicus*) was confirmed. Rat population censuses were carried out pre- and post-baiting. Anticoagulant rodenticide baits were applied according to product labels, pulsed-baiting was used for brodifacoum and surplus baiting for bromadiolone and difenacoum [5]. All baits contained 0.005% w/w of the respective active substances. Models were created utilising the data obtained from these trials to predict the amounts of the anticoagulant rodenticide active substances in different environmental compartments through the course of the trials. The daily uptake of bait was modelled along with the predicted rat population size and thus the level of active substance in different sectors of the rat population could be predicted. Based on the natural history of the species of interest, the relative exposure to different compounds in non-target species as a result of these field trials was quantified.

3. Results and discussion

3.1. Comparing trial efficacy

Across all trials brodifacoum was the most effective compound reducing rat populations to less than 1% of their original size on all farms. Bromadiolone and difenacoum had lower rates of success as would be expected in resistant areas. Control was inversely proportional to the amount of active substance (a.s.) entering the food chain.

3.2. Anticoagulant rodenticide levels in the rat population

Our model showed that bromadiolone and difenacoum use in these trials resulted in much higher levels of a.s. entering the food chain via the rats than the use of brodifacoum (Fig. 1). It also showed that a.s. continued to enter the rat population at a much higher rate throughout the trials where control was less effective. The model predicted that after the end of baiting, rats carried residues of bromadiolone and difenacoum until the end of their natural life. However, no brodifacoum baited rats were predicted to be alive at the end of the trials.

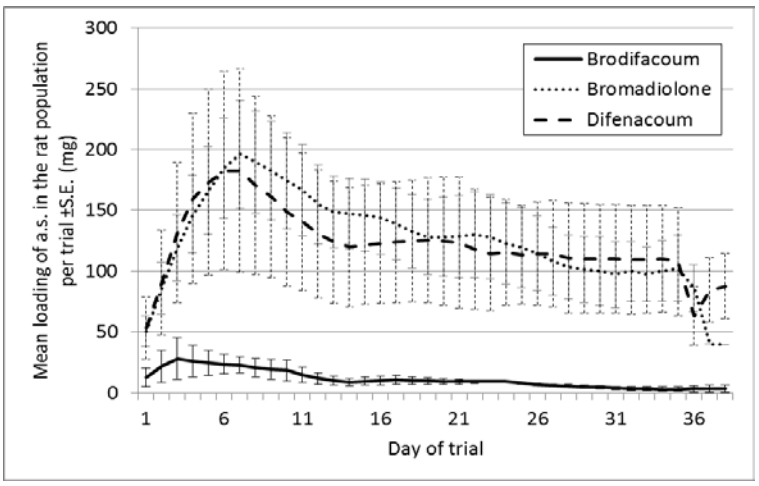

Figure 1.
Mean (± S.E.) loading of active substance present in the rat population over the whole farm throughout the trials. Baiting was with either: bromadiolone (n=6), difenacoum (n=4) or brodifacoum (n=2). Bromadiolone and difenacoum trials showed much higher uptakes over the course of the trials than brodifacoum trials.

3.3. Anticoagulant rodenticide levels in the non-target population

Modelling secondary non-target uptake in these scenarios shows that higher residue levels in the rat population and higher numbers live rats in the environment increase exposure and increase the chance of lethality to the non-target.

4. Conclusions

The use of potent anticogulant rodenticide 'resistance-breakers' is avoided due to their higher toxicity and potential to be more hazardous in the environment [6]. However, in areas where practitioners seek to control resistant rodent infestations, their use may pose less of a risk than applications of ineffective baits. Compounds to which rodents are resistant to, do not provide effective control and create a long-term source of AR in the environment. The higher quantities of anticoagulant rodenticide used show that using ineffective compounds may extend both the period and severity of exposure to non-target animals to anticoagulant rodenticides. Conversely the effective use of resistance-breakers to control anticoagulant rodenticide-resistant rat populations results in lower environmental exposure of anticoagulant rodenticides for non-targets. Of course, the relative toxicity of the different anticoagulant rodenticides will also play an important part in overall risk assessments. However, this can be outweighed by the relative exposure to different anticoagulant rodenticides in such situations.

5. References

[1] Boyle CM. 1960. Case of Apparent Resistance of *Rattus norvegicus* Berkenhout to Anticoagulant Poisons. Nature 188: 517
[2] RRAG. 2010. Anticoagulant resistance in the Norway rat and guidelines for the management of resistant rat infestations in the UK. http://www.bpca.org.uk/rrag/downloads/RRAG_Resistance_Guideline.pdf. Accessed 28/11/2011
[3] Shore RF, Malcolm HM, Wienburg CL, Turk A, Walker LA and Horne JA. 2005. Wildlife and pollution: 2001/02 Annual report.
[4] Burn AJ, Carter I and Shore RF. 2002. The threats to birds of prey in the UK from second-generation rodenticides. Aspects of Applied Biology 67: 1-10.
[5] Buckle AP. 1994. Control Methods: Chemical. Wallingford, Oxon, UK,, CAB International. 127-160.
[6] Eason CT, Murphy EC, Wright GRG and Spurr EB. 2002. Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. Ecotoxicology 11: 35-48.

Acknowledgement - The authors thank RRAC and Nicole Klemann for carrying out and reporting on the German field trials, staff at the University of Reading that conducted the UK field trials and staff at Syngenta and Jon Stark for advice on the model construction.