Resistance as a factor in environmental exposure of anticoagulant rodenticides - a modelling approach

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Abstract

Anticoagulant rodenticide (AR) resistance in Norway rat populations has been a problem for fifty years, however its impact on non-target species, particularly predatory and scavenging animals has received little attention. Field trials were conducted on farms in Germany and England where resistance to anticoagulant rodenticides had been confirmed. Resistance is conferred by different mutations of the VKORC1 gene in each of these regions: tyrosine139cysteine in Germany and leucine120glutamine in England. A modelling approach was used to study the transference of the anticoagulants into the environment during treatments for Norway rat control. Baiting with brodifacoum resulted in lower levels of AR entering the food chain via the rats and lower numbers of live rats carrying residues during and after the trials due to its lower application rate and efficacy against resistant rats. Bromadiolone and difenacoum resulted in markedly higher levels of AR uptake into the rat population and larger numbers of live rats carrying residues during the trials and for long periods after the baiting period. Neither bromadiolone nor difenacoum provided full control on any of the treated farms. In resistant areas where ineffective compounds are used there is the potential for higher levels of AR exposure to non-target animals, particularly predators of rats and scavengers of rat carcasses. Thus, resistance influences the total amount of AR available to non-targets and should be considered when dealing with rat infestations, as resistance-breakers may present a lower risk to wildlife.

Keywords: anticoagulant rodenticides, environmental exposure, residues, resistance

Introduction

Anticoagulant rodenticides (ARs) are used globally to control pest rodent infestations. Resistance to ARs first appeared against warfarin and diphacinone in Scotland in 1958 (Boyle, 1960). In the wake of this, more potent ARs were produced but resistance has since developed to some of these (RRAG, 2010). Monitoring residues in carcasses of predators and scavengers in the UK has shown that ARs may affect a range of non-target species, including some of high conservation value (Burn and Carter, 2002; Shore et al., 2005). The risk to wildlife depends on several factors, including AR loading in individual rats and total AR residue in rat populations. The impact of resistance on these factors is less well understood. Resistant rats are able to consume some rodenticides without dying and so may carry body burdens of active substance at higher levels and for longer than rats that are susceptible to the poisons (Atterby et al. 2005; Brakes and Smith, 2005). Using data from field trials against resistant rats and modelling the movement of active substance in the rat population we show that this is indeed the case and rodenticide-resistant areas could be a particular hazard with regard to non-target exposure.

Materials and methods

Field trials of three ARs (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. AR baits were applied according to product labels, pulsed-baiting was used for brodifacoum and surplus baiting for bromadiolone and difenacoum (Buckle, 1994). All baits contained 0.005% w/w of the respective active substances. A model was created utilizing the data obtained from these trials to predict the amounts of the AR active substances in different environmental compartments through the course of the trials.

Results

Across all trials brodifacoum was the most effective compound reducing rat populations to less than 1% of their original size. Bromadiolone and difenacoum had lower rates of success as would be expected in

resistant areas (29% reduction to population growth and 13-84% reduction per farm respectively). Our model showed that bromadiolone and difenacoum use in these trials resulted in much higher levels of AR entering the food chain via the rats than the use of brodifacoum (Figure 1). It also showed that AR 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 live rats carrying residues of AR were present for more than 10 times as long for bromadiolone and difenacoum trials than for brodifacoum trials.

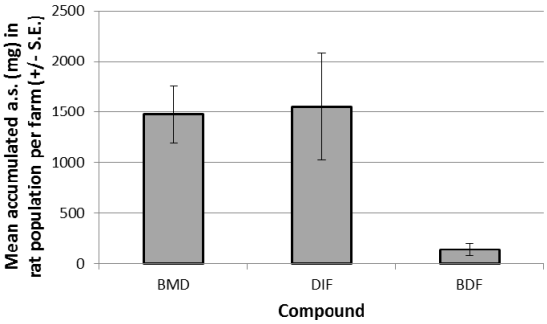

Fig. 1 Mean amounts of anticoagulants taken up by rat populations baited with each compound: bromadiolone (BMD, n=6 trials), difenacoum (DIF, n=4) and brodifacoum (BDF, n=2), over the whole farm. BMD and DIF trials showed much higher uptakes over the course of the trials than BDF trials.

Discussion

The use of potent AR 'resistance-breakers' (including brodifacoum and flocoumafen) is sometimes avoided due to their higher toxicity and potential to be more hazardous in the environment (Eason et al., 2002). 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 to enter the environment. The higher quantities of AR used show that using ineffective compounds may extend both the period and severity of exposure to non-target animals to ARs. Conversely the use effective use of resistance-breakers to control anticoagulant rodenticide-resistant rat populations results in lower environmental exposure of ARs for non-targets. Of course, the relative toxicity of the different ARs will also play an important part in overall risk assessments.

Acknowledgements

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