Resistence in mice

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Since the introduction of anticoagulant rodenticides in the 1950s control of mice, particularly in the inner cities, has met with varying degrees of success. As the house mouse possesses a degree of natural resistance to anticoagulant rodenticides, these products have always proven to be less effective against house mice than they are against Norway rats (Rattus norvegicus).

True resistance to anticoagulants, conferred by genetical mutation, has been known among house mice in the UK since the 1960s. However, in comparison to the Norway rat, little research has been carried out to investigate the number and spread of these mutations.

In a German study using the new system of DNA sequencing for the detection of anticoagulant resistant mutations, more than 90% of the mice examined carried genetical resistance mutations and resistance was found at 29 of the 30 locations sampled. The two resistant house mouse strains found in the German study are also known to be present in the UK, so there is little to suggest that a similar situation does not exist here.

Resistance to first-generation anticoagulants in mice has for many years been widely accepted in most parts of the UK, but in an increasing number of cases difenacoum and bromadiolone have failed to provide complete control. This has been demonstrated in laboratory and field trials where the tests also showed that control was likely to be more problematic in the case of bromadiolone than difenacoum.

Resistance does seem to be increasing

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The genetics of anticoagulant resistance in the house mouse is not well understood – it is complicated by the involvement of several genes and, perhaps, even several different resistance mechanisms. Using a method which examines the genetic make-up of individual rodents to discover whether they possess mutated genes that might confer anticoagulant resistance has resulted in two different genetic mutations being found:

- **Cambridge strain** – leucine128serine mutation, or it may be referred to by its abbreviated name L128S. It is likely that this mutation occurs widely in the UK, as it does in Germany.

Not all treatment failures are caused by resistance. As this bait box shows there is plenty of mouse activity but the mice are going over the box and not into it.
Resistance is not the sole reason for failure

Resistance to the anticoagulants however cannot be accepted as the sole reason for treatment failure. The mouse’s habit of feeding from a large number of locations in a night has always provided a challenge to ensuring the consumption of a lethal dose of active ingredient. The phenomenon of ‘behavioural resistance’, as it became known, has further complicated mouse control strategies. Some mice are known to refuse to eat traditional cereal-based baits in preference to a diet of protein-based foods.

Of greater concern however, is the apparent suspension of the mouse’s inquisitive nature, leading it to avoid entering bait boxes – or at least not going as far in as would permit it to eat the bait, or be caught in a trap. The bait box on the left clearly shows this behaviour. It has been passed over several times resulting in heavy smearing but no smears can be seen on the entrance lip of the box.

RRAG mouse control recommendations

Recently published information from the Rodenticide Resistance Action Group (RRAG) recommends the use of anticoagulants rodenticides against resistant house mice as follows:

- **The first-generation anticoagulants**
  It has long been a regulatory policy that anticoagulants such as warfarin, chlorophacinone, diphacinone and coumatetralyl should not be used for the control of house mice in the UK. Consequently, there are no approvals for the use of rodenticide products carrying these active ingredients for mouse control.

- **Bromadiolone and difenacoum**
  Y139C (the Reading strain) shows a significant degree of resistance to bromadiolone. There are also many anecdotal reports of the failure of bromadiolone to control house mice. Some infestations may be controlled, at least in part, by bromadiolone. But the use of this active substance against house mice in the UK is not recommended as it may not result in an adequate level of control and will exacerbate resistance problems.

  Difenacoum is widely used in successful mouse control treatments. Yet, mice carrying the Y139C mutation possess a degree of resistance to difenacoum.

- **Brodifacoum and flocoumafen**
  Brodifacoum and flocoumafen are the most potent active substances against susceptible house mice.

  There is good evidence from early field studies that brodifacoum and flocoumafen are effective against anticoagulant-resistant house mice.

  Laboratory studies conducted on mice carrying the Y139C mutation at the University of Reading have confirmed that brodifacoum baits are effective against this type of resistant house mouse.

  The advantage for resistant house mouse control is that only small quantities of bait are required to achieve a lethal dose, even of resistant mice, thus capitalising on their sporadic feeding behaviour.

  When using brodifacoum and flocoumafen for house mouse control pest controllers should be on the alert for infestations that are more difficult to control than normal using products that contain these active substances. Should difficulties occur, these should be reported to RRAG.

- **Diffethialone**
  Baits containing the second-generation anticoagulant difethialone are new to the market in the UK. At present, RRAG is unaware of any difethialone field trials conducted in the UK against resistant mice.

The information on resistance in this article is based on the Rodenticide Resistance Action Group’s RRAG House Mouse Resistance Guideline produced by Dr Alan Buckle of the University of Reading and chairman of RRAG.

Downloaded the full document at www.bpca.org.uk/rrag/documents.html