Anticoagulant Rodenticides: Implications for Wildlife Rehabilitation

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Introduction

Anticoagulant rodenticides (ARs) are commonly used to control rodents worldwide for the purposes of reducing disease transmission, agricultural losses, and damage to homes and property (Bradbury, 2008). In the 1970s and 1980s "second generation" anticoagulant rodenticides (SGARs) were developed to overcome resistance which had developed in some rodent species. SGARs are more acutely toxic and have half-lives which are substantially longer than first generation anticoagulant rodenticides (FGARs) (Thomas et al., 2011). As a consequence, SGARs are much more likely to bioaccumulate and biomagnify, leading to a substantially higher risk of secondary toxicity in non-target wildlife. Cases of apparently poisoned wildlife being brought to wildlife rehabilitation centres have already been documented (Grillo et al., 2016). Sub-lethal AR exposure may also increase the likelihood of wildlife entering care by increasing the probability of collisions with vehicles and anthropogenic structures (Albert et al., 2010; Mendenhall and Pank, 1980; Newton et al., 1990; Stone et al., 2003) and susceptibility to parasitism (Lemus et al., 2011; Riley et al., 2007; Serieys et al., 2018). Emerging evidence indicates that secondary AR toxicity is an important threatening process impacting wildlife across Australia (Lohr and Davis, 2018). The tendency of SGARs to biomagnify increases the likelihood of impacts on scavengers and carnivores in higher trophic levels. Species with long lifespans and low reproductive rates are more likely to suffer population-level impacts from AR toxicity (Rattner et al., 2014). Effective rehabilitation may be especially important in these instances.

Challenges in Treating Poisoned Wildlife

A number of challenges exist in treating wildlife exposed to ARs. Susceptibility to ARs varies dramatically both between and within species (Thomas et al., 2011). Treatment of AR poisoning in wildlife with an unknown history of exposure can be substantially more complicated than treatment of companion animals and humans where the type and quantity of the poison are often known. Unlike companion animals and humans, wildlife exposed through secondary poisoning are frequently exposed to multiple rodenticides (Christensen et al., 2012; Hughes et al., 2013; Murray, 2017; Walker et al., 2011). Little is known about effects of exposure to multiple ARs but some research suggests that the interactions between ARs may be synergistic rather than simply additive (Mosterd and Thijssen, 1991). Treatment is further complicated by a lack of species-specific guidelines for treating AR toxicity. The necessity of managing rodents within wildlife rehabilitation facilities can present additional challenges for wildlife rehabilitators. Understanding general principles relating to how ARs function may aid in successful diagnosis and treatment of poisoned individuals and help reduce the likelihood of unintentional poisoning during necessary rodent control activities.

ARs function by blocking the recycling of vitamin K in the liver. Vitamin K is required in the synthesis of several important blood clotting factors. Onset of symptoms is usually delayed for several days after ingestion of ARs because it takes several days to exhaust the body's reserves of vitamin K once recycling has been blocked. Symptoms of AR toxicity in wildlife

can include: anaemia, intramuscular or subcutaneous haemorrhage in the absence of physical trauma, pale mucous membranes, difficulty breathing (due to blood in the lungs), reduced activity, anorexia, bleeding from the mouth or nares, and blood in stool or droppings (Murray, 2017). Unfortunately, confirmation of the specific ARs involved in suspected poisonings can be difficult because ARs are only detectable in blood for a short period of time relative to other tissues (Erickson and Urban, 2004). As a consequence, negative test results can be misleading and should not be taken as an indication that poisoning has not occurred. Concentrations of ARs in plasma also do not correlate with the duration of treatment required in poisoned animals (Gunja et al., 2011). Accurate confirmation of the type and concentration of ARs involved in the poisoning requires analysis of liver tissue and is not appropriate for diagnosis of living animals.

Toward a Treatment Protocol

In humans and companion animals, prothrombin time and other measurements of clotting times are typically used in the diagnosis of AR toxicity (Rattner et al., 2014). Clotting times which are more than 25% longer than baseline values for the relevant species suggest potential AR poisoning and are likely to be useful in evaluating wildlife with suspected AR exposure (Rattner et al., 2014). While no formal treatment protocols appear to be available specifically for wildlife exposed to ARs, a treatment regime used successfully in dogs diagnosed with AR poisoning involved an initial oral administration of 5 mg/kg of vitamin K_1 split into two or three doses throughout the day (Robben et al., 1998). Vitamin K administration was repeated daily at this dosage until the condition of the animal stabilised and prothrombin time returned to normal (Robben et al., 1998). Daily dosage of vitamin K_1 was then reduced by between 30% and 50% on each subsequent day unless prothrombin time increased (Robben et al., 1998). Treatment was ceased when prothrombin times did not increase after two to four days without vitamin K_1 supplementation. Duration of treatment in this study lasted for up to 30 days (Robben et al., 1998) but treatment of some human cases of SGAR poisoning has required up to six months of treatment (Gunja et al., 2011). Necessary treatment durations for exposed wildlife are likely to vary substantially dependant on exposure levels, type of ARs involved, individual genetic factors, and susceptibility of the species involved. It is also important to keep in mind that due to long persistence time in liver tissue, SGARs will still be present and partially blocking vitamin K recycling even after prothrombin times and associated blood clotting return to normal levels. Animals with residual SGARs in their livers will be vulnerable to future exposure at lower doses relative to animals with no ARs accumulated in liver tissue. At present, no practical solution to this problem exists.

Managing Rodents at Wildlife Care Centres

Facilities engaged in the rehabilitation of wildlife face additional practical difficulties in managing rodents, due, in part, to the availability of spilled or uneaten food provided to animals in care. In most wildlife rehabilitation facilities, commensal rodents present an unacceptable risk of disease transmission, infrastructure damage, and predation of smaller species in care. However, in several instances, raptors in rehabilitation facilities have been lethally poisoned with ARs as a consequence of poisoned rodents entering their enclosures (Mooney, 2017). Careful consideration needs to be given to the management of rodent pests within wildlife rehabilitation facilities to ensure the safety of animals in care.

In some instances, rodent abundance and associated negative outcomes can be sufficiently reduced through non-lethal means. When practical, reduction of rodent food sources by prompt removal of uneaten food provided to injured wildlife, use of feeders which prevent spillage, and picking up fallen fruits in landscaped areas can aid in reducing rodent numbers. Sealing holes and other potential entry points in buildings and enclosures can also reduce rodent access to food resources while simultaneously excluding them from areas where they are likely to cause damage. Reducing available rodent habitat by cleaning up brush piles and rubbish can aid in reducing activity and abundance of commensal rodents. Replacing dense introduced vegetation – especially palms – with native plants reduces rodent nesting habitat and provides better habitat for native avian predators which help control rodents.

Lethal control methods can also be helpful in reducing rodent numbers once the factors driving rodent abundance have been addressed to the degree practicable. A wide variety of lethal traps are readily available for control of commensal rodents. Careful positioning of the traps is necessary to ensure efficacy at capturing rodents and reduce harm to non-target species. These considerations will vary dramatically with trap design. If rodenticides are used, baits containing the FGARs warfarin and coumatetralyl are substantially less likely to cause secondary toxicity than SGARs, due to their relatively short half-life and lower toxicity (Erickson and Urban, 2004). However, resistance to FGARs may reduce their utility in rodent control in some areas. The distribution and prevalence of resistance to FGARs among commensal rodent species in Australia is poorly known but has been documented in Sydney as early as 1978 (Saunders, 1978). If resistance is suspected, baits containing the active ingredient cholocalciferol may be helpful. Cholecalciferol is not an anticoagulant and is effective at controlling rodents which have developed resistance to ARs. It is also substantially less likely than SGARs to cause secondary poisoning in native wildlife but does carry a limited risk of secondary toxicity which likely varies by species (Eason et al., 2000).

Conclusion

While many obstacles to effective treatment of AR toxicity in Australian wildlife exist at present, several factors may help to reduce the incidence and severity of such events and improve treatment outcomes in the future. The Australian Pesticides and Veterinary Medicines Authority is currently reviewing the scheduling of SGARs (Australian Pesticides and Veterinary Medicines Authority, 2015) and recommendations suggesting more stringent regulation of SGARs have been published (Lohr and Davis, 2018). Ongoing research into the ecology of AR exposure may also improve future treatment protocols. Future work assessing exposure rates and sensitivity to ARs across a wide variety of Australian wildlife species will be contribute substantially to our knowledge of the probability of AR exposure and allow more rapid assessment of animals admitted to rehabilitation centres. Research into patterns in spatial distribution of AR exposure will also allow faster identification of individuals at high risk of poisoning and potentially improve treatment outcomes.

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