How to Spoil an Owl's Dinner

Photo © Mark Wilson

The Harmful Effects of Rodent Poisons on Birds of Prey

by Maureen Murray

I can still remember the first bird I diagnosed with rodenticide poisoning. A beautiful, well conditioned, adult female red-tailed hawk had been found on the ground, unable to fly. A Good Samaritan brought the bird to the Tufts Wildlife Clinic, part of the Cummings School of Veterinary Medicine at Tufts University in North Grafton, Mass. It was a weekend and I was the only veterinarian on duty, recently graduated from veterinary school and just starting my career as a wildlife veterinarian.

When I examined the bird, I found that an area on her right wing was severely bruised, and there was a small wound by her elbow that was bleeding. I took a radiograph (x-ray), certain I would find broken bones. But the radiograph proved me wrong – no broken bones. It seemed odd that the wing would be as swollen as it was without being fractured, but the clinic was busy and the bird was otherwise stable, so I bandaged the wound, gave the bird pain medication, and moved on.

When I checked on the hawk a little while later, the bandage I had placed on the wing was soaked through with blood. I removed the bandage and saw that this massive amount of blood was coming from that small wound over her elbow. Strange, I thought. It seemed as though the bird's blood was unable to clot. And that's when the list scrolled through my mind: the differential diagnoses list that 4 years of veterinary school trains you to produce for every case. What are all the possible reasons an animal's blood may not clot?

As I mentally went through this list I stopped on the explanation that made the most sense in this case: anticoagulant rodenticide (AR) poisoning. This bird had most likely ingested prey that had consumed a type of rodent poison that kills by preventing the blood from clotting, resulting in fatal hemorrhage. Veterinary students receive a good deal of training on this subject, as this type of poisoning can also happen in pet dogs.

I immediately gave the hawk an injection of vitamin K_1, which is the antidote for anticoagulant rodenticide poisoning. Because she had been found and received treatment before she lost too much blood, the hawk was able to make a full recovery. After four weeks of treatment with vitamin K_1, along with flight reconditioning to rebuild her fitness, the hawk

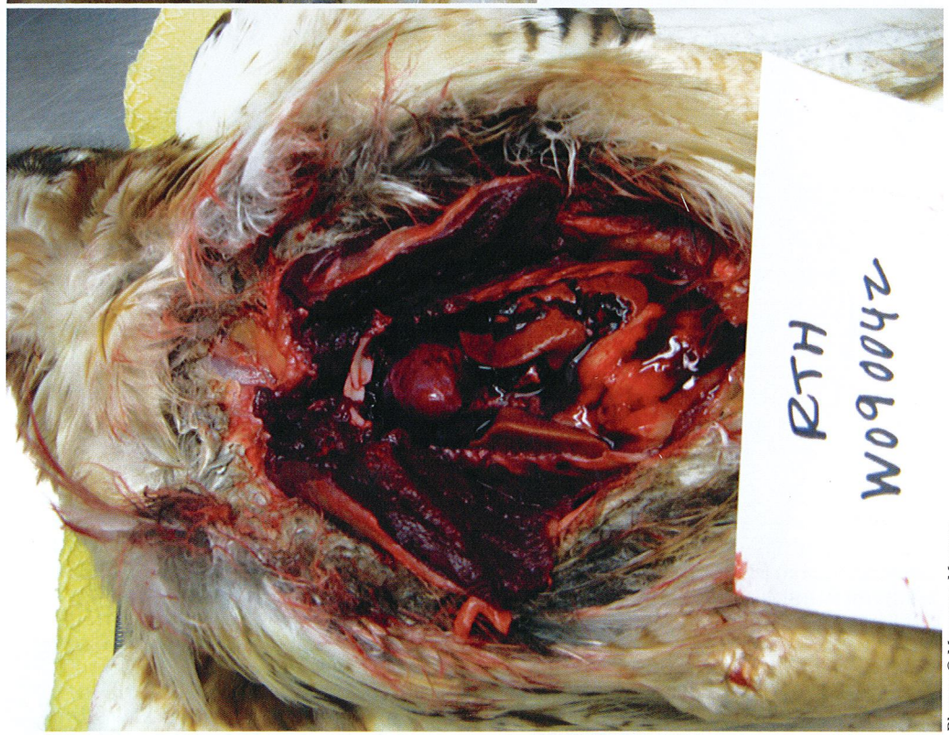
Photo © Andrew Cunningham

The author, a wildlife veterinarian whose research played a part in the EPA's restriction of some rodenticides due to their effects on raptors, examines a Barred Owl under anesthesia. Whenever a bird of prey is found to be in distress or flightless, rodenticide poisoning is always a possibility that must be investigated.

Looking as if it has been severely bruised, the exposed wing of a Great Horned Owl displays the purple, tell-tale hemorrhaging of blood into its wing muscles that is a typical symptom of rodenticide poisoning.

was released back to the wild. She was one of the lucky ones.

Since that first case 11 years ago, I have diagnosed many poisoned birds of prey: A female red-tailed hawk with an egg ready to be laid, bleeding from the enlarged blood vessels of her reproductive tract. An eastern screech-owl that bled into its lungs after a minor collision with a window. A great horned owl that was seen perched in the same spot high up in a tree for several days as it bled into



Photos © Maureen Murray

A necropsy photo of a Red-tailed Hawk reveals a puddle of blood filling the coelomic cavity surrounding its heart, liver, and other organs. This is how the anticoagulant properties of many rodenticides kill the target rodents, but unfortunately these chemicals can also result in the secondary poisoning of raptors and even mammals that prey or scavenge on the poisoned rodents.

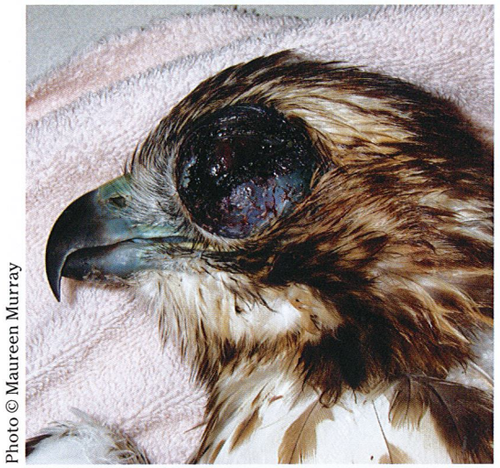


Photo © Maureen Murray

As this Red-tailed Hawk demonstrates, severe swelling and discoloration of the tissues around the eyes is one of the symptoms of rodenticide poisoning in these raptors. Because it takes 3-5 days for a poisoned rodent to die, the animal may consume the poison multiple times and develop a high concentration that may be debilitating or lethal to any raptor that feeds on the rodent.

the muscles of its wings and legs until it became so weak it finally fell to the ground. These birds died from ingesting poisoned prey. These are the effects of anticoagulant rodenticides.

The ability of rats and mice to flourish anywhere humans establish themselves is a constant throughout human history. The genetic origins of the modern domestic house cat may stem from wild cats that found a niche controlling rodents around early farming civilizations in the Near East at least 9,000 years ago. Given the detrimental effects of domestic cats on wildlife populations, particularly songbirds, it can be argued that the unintended environmental effects of rodent control on wildlife dates back this far as well. In recent decades rodent control has largely been achieved by the use of poisons. However, in recent decades there has also been an enormous increase in knowledge about the effects of various chemicals on ecosystem, wildlife, and human health. Consumer education about the potential harmful effects of chemicals used in the home is an important factor in reducing their negative impacts on the environment.

Public awareness of the risks to wildlife from anticoagulant rodenticides (ARs), while increasing, is lacking. Wildlife can be poisoned by ARs via two different pathways. The first, referred to as primary poisoning, occurs when an animal directly eats the bait. This route has been documented in species such as white-tailed deer and gray squirrels. The second, referred to as secondary poisoning, occurs when a predator or scavenger eats an animal that has consumed the bait. This route has been documented in birds of prey, crows, foxes, bobcats, raccoons, coyotes, and fishers, among other species. One property of the ARs that results in their ability to cause secondary poisoning is that they are not inactivated after they are ingested. They concentrate in the liver and retain their ability to cause poisoning further up the food chain.

The ARs are divided into two types, referred to as first generation and second generation anticoagulant rodenticides (FGARs and SGARs, respectively). Both FGARs and SGARs work in the same way: they prevent the blood from clotting normally. To understand the relative threats the two categories of ARs present to wildlife – as well as the rationale behind recent US EPA regulation changes regarding the sale of ARs (discussed later in this article) – it is necessary to understand the history of and the differences between FGARs and SGARs.

Warfarin, the original FGAR, was first registered for use in 1950. After many years of use, some rodent populations, primarily in Europe, were noted to develop resistance to the effects of FGARs. In response, the SGARs, often referred to as "super warfarins" were developed. One commonly used SGAR, brodifacoum, was registered for use in 1979. Over the following decades and until very recently, SGARs, particularly brodifacoum, have been the most commonly used rodenticides in the U.S., available to pest control professionals and homeowners alike.

While FGARs and SGARs cause death in the same way, by impairing blood

Photo © Bill Byrne

Consider the possibility that you could be responsible for inadvertently poisoning wildlife – and especially certain birds of prey such as this soaring Red-tailed Hawk – if you choose to use a rodenticide to control a rodent problem. Snap traps are a more humane (though more labor intensive) option, but if a rodenticide is used, check the ingredients to avoid brodifacoum, a poison so dangerous to children and wildlife that it is being phased out by the EPA for general consumer sales.

clotting, there are important differences between the two categories. Firstly, SGARs persist in the body, stored in the liver, for a longer time than FGARs. While this property does not offer SGARs an advantage over FGARs for controlling targeted rodents, the longer persistence of SGARs does make them more dangerous to wildlife. The SGARs have been shown to accumulate in the liver over time with repeated feeding. For a red-tailed hawk hunting on its home territory where SGARs are in use, repeatedly feeding on prey containing sub-lethal amounts of SGARs places that bird at risk of accumulating a lethal amount over time.

Secondly, SGARs are more potent than FGARs, meaning that rodents have to consume less of the poison to suffer its effects. More than one feeding of an FGAR is required for a rodent to ingest a fatal dose. With SGARs, a single feeding is likely to be fatal, and this property has been marketed to general consumers as a benefit of these products. However, this increased potency of SGARs compared to FGARs also means that wildlife or any "nontarget" species also need to consume less to suffer symptoms of poisoning. Neither FGARs nor SGARs kill immediately. It takes about 3-5 days for a poisoned animal to die. During this time, until the animal becomes affected by the poison, it can return to the bait, continue to feed, and accumulate high levels of poison. In the case of SGARs, repeated feeding results in prey items that can deliver a highly concentrated amount of a very potent poison to a predator or scavenger.

Back in 2003, when I diagnosed my first case of AR toxicosis, I searched the scientific literature on this topic and found a study published that same year reporting the number of birds of prey examined by New York State's Department of Environmental Conservation that had been exposed to ARs. In this study, liver tissue from 265 raptors of 12 different species was tested for residues of ARs. The results showed that 49% of these birds were positive, with 84% of the positive birds having residues of the SGAR brodifacoum. Given that I was seeing and treating poisoned birds at Tufts Wildlife Clinic, I believed it was likely that our populations of raptors in Massachusetts were equally exposed. The study from New York was the only one of its kind in the U.S. at the time, so I decided that I would conduct a similar study at Tufts.

I collected liver samples from four species of birds of prey from 2006 through 2010. The results of this study were published in the *Journal of Zoo and Wildlife Medicine* in 2011. The birds I sampled had been admitted to the Tufts Wildlife Clinic and either died or required humane euthanasia due to the severity of their injury or illness. The species included were red-tailed hawks (*Buteo jamaicensis*), barred owls (*Strix varia*), eastern screech-owls (*Megascops asio*), and great horned owls (*Bubo virginianus*). In total, I sampled 161 birds over these 4 years.

Based on the results of the study from New York, I hypothesized that my results would show that roughly half of the birds would have residues of ARs in their liver tissue. As my results started coming in, however, showing one positive bird after another, I realized that the proportion of positive birds would be much higher than 50%. Upon final analysis, 86% of the 161 birds I tested (all four species combined) had AR residues in their liver tissue. Of these positive birds, 99% had residues of the SGAR brodifacoum, which could be found in various products available to general consumers.

During the course of the study I diagnosed nine birds (6%) as having died due to AR poisoning. These birds showed various signs of severe hemorrhage, including massive bleeding into muscles, body cavities, lungs, and the lining around the heart. The next logical question is: If only a small number of birds in the study died of poisoning, what does the high number of exposed birds mean? Is there a threat to their health from exposure that did not result in bleeding? In thinking about this question, it is helpful to recall the properties of SGARs previously described. Remember that SGARs persist and accumulate in the liver over time.

Summary of Rodenticides

Type of rodenticide	Examples	Status under 2008 EPA decision
SGAR	brodifacoum difethialone bromadiolone difenacoum	Prohibited for residential consumer purchase; available to licensed professionals and agricultural users
FGAR	chlorophacinone diphacinone	Approved for residential consumer use (must be enclosed within a bait station)
Non-anticoagulant	bromethalin	Approved for residential consumer use (must be enclosed within a bait station)

It is best not to use a rodenticide, but if you must, please use an FGAR listed in the table above. These poisons do not persist as long in the bodies of animals that consume sub-lethal amounts. The SGARs are more likely to move up the food chain.

Therefore, an exposed bird represents a bird that could potentially reach a lethal dose of an SGAR when it ingests its next contaminated meal.

What this high percentage of exposed birds further shows is that SGARs, in particular brodifacoum, are widespread in the prey of these four species. The exact route SGARs take through the food chain is not understood. Are each of these four species – which have varied diets in relation to each other – all being exposed through pest mice and rats? Or are these poisons making their way into other natural prey items of these birds?

It is known that insects – a large component of the eastern screech-owl's diet – will feed on rodenticide baits and can accumulate the poisons without being affected by them. In addition, AR residues have been detected in songbirds, as well as in birds that prey on songbirds, such as Cooper's hawks (*Accipiter cooperii*). Tertiary poisoning is also possible with SGARs, meaning that a great horned owl could be exposed by eating an animal that became contaminated by eating another contaminated animal.

Photo © Andrew Cunningham

The author, left, shown here examining a Red-tailed Hawk safely restrained by a veterinary technician, tested 161 birds of 4 species (Red-tailed Hawk, Barred Owl, Great Horned Owl, and Screech Owl) that died or had to be euthanized at the Wildlife Clinic from 2006 to 2010. She found that 86% of them had anticoagulant rodenticides in their liver tissue. Of these, 99% had residues of the SGAR brodifacoum, indicating its sale to the general public presents a significant problem for raptors. While the EPA recently banned the sale/use of this chemical for all but agriculture and professional pest control use, producers are allowed to sell their inventories until the end of this year, and products containing it may still be on store shelves until well into 2015.

Since the 2011 publication of my study, another study of red-tailed hawks and great horned owls in New Jersey between 2008 and 2010 found that 81% of 127 tested birds were positive for SGARs in liver tissue, with the most frequently detected compound being brodifacoum. Despite uncertainty about which prey items are delivering SGARs to birds of prey, the evidence is clear that there's a lot of brodifacoum out there and it is unquestionably making its way up the food chain.

The next question for anyone concerned about wildlife is whether anything is being done to address this widespread exposure of birds of prey – and other wildlife species – to these poisons. The answer is yes. The U.S. Environmental Protection Agency (EPA) began taking steps to impose certain restrictions on SGARs in 2008 when it issued its "Risk Mitigation Decision for Ten Rodenticides." Due to the risk of poisoning from SGARs in children as well as in wildlife, this decision, which was to take effect in June 2011, banned the sale of SGARs through the general consumer market – meaning homeowners would no longer be able to purchase brodifacoum or other SGARs. However, the decision still allows licensed pest professionals and agricultural users to employ SGARs.

In response to the EPA decision, most rodenticide manufacturers discontinued production of SGARs for residential consumers and brought alternative products to the market by the June 2011 deadline. These products contain the FGARs chlorophacinone and diphacinone, as well as a non-anticoagulant, bromethalin, which affects the nervous system. The new EPA regulations also prohibit certain forms of bait – such as pellets, which are easily scattered over a large area and can easily be ingested by a child – and require that all baits be enclosed within bait stations. While the words *safe* and *poison* inherently do not go together, the EPA's aim is to replace SGARs, for which ample evidence exists demonstrating their risk to wildlife, with products that may pose less risk.

However, if you went to your local big box store now, you still might find brodifacoum on the shelf. Despite the EPA's decision, the multinational corporation Reckitt Benckiser, manufacturer of the d-Con brand, refused to discontinue selling its SGAR-containing and other noncompliant products by the June 2011 deadline and challenged the EPA's authority to revoke its product registrations. The resulting regulatory and legal action stretched into 2014. During this time, 12 noncompliant d-Con products remained on store shelves. In May 2014, Reckitt Benckiser finally reached an agreement with the EPA. Under the terms of this agreement, Reckitt Benckiser will stop production of its noncompliant products by December 31, 2014, and will cease all distribution of these products by March 31, 2015 – although stores can continue selling them until their stocks run out.

Consequently, at the time of publication of this article, there are more types of rodenticides available to consumers than ever before. It is of the utmost importance that consumers are educated about these products before deciding to purchase a poison to use in or around their homes or to hire a pest control company. Following are questions to ask yourself before you decide whether or not to use a rodenticide:

DO I NEED TO USE A POISON?

Before resorting to a poison, have you taken all steps you possibly can to rodent-proof vulnerable areas in and around your home? Can you eliminate or better contain potential food and water sources for rodents? Have you looked for and patched potential entry sites into your home? Have you considered alternatives such as snap traps? (While people often comment that they feel poisons are more humane than snap traps, what most don't realize is that animals poisoned

with ARs bleed to death – a process that is neither quick nor especially humane.)

WHAT'S IN IT?

If you feel you need to use a poison, check the active ingredient before you buy. Through March 2015, SGAR-containing products will still be available to residential consumers (refer to the table Summary of Rodenticides). SGARs have been deemed highly dangerous to wildlife by the EPA, a conclusion well backed by research. With regard to the SGAR-replacement products on the market, know that less risk is not no risk. Research has shown that birds of prey are more sensitive than other bird species to FGARs, and there is little research about the possibility of the neurotoxin bromethalin to cause secondary poisoning in wildlife.

WHAT ARE THE PROFESSIONALS USING?

Know that if you employ a pest control company, they are likely using SGARs – hence it is important to question what poisons the company uses and to question assertions that these products are "safe" for wildlife. Also, be sure the pest control company employs integrated pest management techniques – a strategy that uses multiple approaches to pest control without relying solely on poisons.

The struggle to control mice and rats is usually viewed in terms of humans vs. rodents. Wildlife such as the raptors and mammalian predators are not recognized by most as players in this battle. When I talk about my research, many people are genuinely surprised to learn that the mouse poison they have been using in their basement can kill the great horned owl they hear calling outside in the dark of a January night. The most important steps you can take to reduce the impact of rodent poisons on wildlife are to arm yourself with information and to explore alternative strategies for rodent control rather than immediately declaring full-scale poison warfare on rodents in and around your home. And then spread the word.

So if you find yourself needing to address a rodent problem during the coming winter months, take a moment to think of the barred owls calling to each other with their characteristic phrase – who-cooks-for-you, who-cooks-for-you-all – and ask yourself this question as you decide on a course of action: Am I going to spoil their dinner?

For More Information

Information regarding EPA regulations on rodenticides, as well as tips on managing rodents and guidance on disposing of unwanted rodent poisons can be found on the following EPA websites:

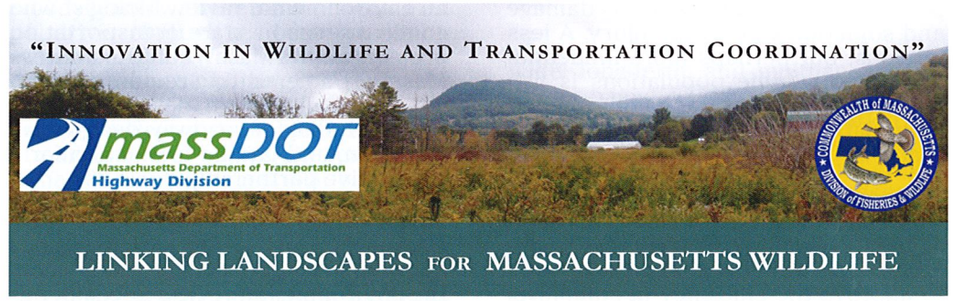
http://www2.epa.gov/rodenticides

www.epa.gov/pesticides/mice-and-rats/

Photo © Andrew Cunningham

Maureen Murray, DVM, DABVP, shown here examining the eye of a Bald Eagle, is a Clinical Assistant Professor at the Cummings School of Veterinary Medicine at Tufts University in North Grafton, Mass. Her continuing research aims to evaluate the effectiveness of new EPA regulations in decreasing exposure to rodenticides in birds of prey.

To support research at Tufts Wildlife Clinic on the effects of rodenticides in birds of prey, go to tuftsgiving.org. Under "Select a School" choose "Cummings Veterinary"; under "Select an Area" choose "Other" and type in "Ruby Memorial Research Fund".



LINKING LANDSCAPES FOR MASSACHUSETTS WILDLIFE

by David Paulson and Tim Dexter

Starless and moonless beneath a blanket of storm clouds, the night was as dark and featureless as a black bear's hide. A car navigated a winding, rain swept highway. A huge, frightening shape suddenly loomed on the roadside. The driver slammed on the brakes, skidded dangerously, but somehow managed to bring the vehicle to a stop just short of a seemingly unconcerned bull moose that ambled across the road inches from the front bumper. Pale and full of adrenaline, the driver let out a long breath, relieved that what would have been a very serious accident had been avoided.

Why does wildlife cross the road? It's an age-old question that begs new and possibly more important questions: Can wild animals cross our roads without jeopardizing both their safety and that of the public? Does anybody aside from a startled but very relieved driver even care? Indeed, here in Massachusetts your state wildlife agency, the Division of Fisheries and Wildlife (MassWildlife), and your state highway agency, the Department of Transportation Highway Division (MassDOT), do care. They are partnering in innovative initiatives with goals to provide safe passage for both wildlife and people while addressing the conservation needs of rare fish and wildlife listed under the Massachusetts Endangered Species Act (MESA).

Where survey data indicates high road mortality for turtles and salamanders, a good option for replacing a corrugated pipe culvert is a wide design like this with a natural floor that invites the passage of turtles and salamanders.

At first glance, a partnership between a state transportation agency and a state wildlife agency would appear unlikely; however, by working together, both agencies have found they can improve how well they meet their respective mandates and exceed expectations. The location and design of transportation infrastructure such as roadways, bridges, and culverts has an impact on wildlife. The reality facing the two agencies is the fact that nearly 12,000 miles of state highways and major roads and 24,500 miles of local roads lace their way throughout the Commonwealth. A glance at a state road map shows road densities are highest in the eastern part of the state; a veritable web of roadways radiates out from Boston, Worcester, and the highly developed coastal communities. Other areas of high road density include portions of the Connecticut River Valley in Franklin, Hampshire, and Hampden counties.

With 6 million people sharing 5 million acres of water and land with wildlife, these roadways impact (no pun intended) both people and wildlife. The most obvious impacts are vehicle collisions, usually resulting in wildlife mortality and

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