Effects of Rodenticides on Wildlife

It is difficult to ascertain to what degree rodenticides injure wildlife throughout the United States, in addition to the rodents that are directly targeted. We know that vast numbers of wildlife are accumulating rodent poisons in their blood, their livers, their fat. Most thoroughly studied in that respect have been the rodenticides that prevent coagulation, that promote internal bleeding.

A 2015 article in the Tufts Veterinary Magazine stated, "So-called “second-generation” anticoagulant rodenticides, or SGARs, became popular because even a single feeding easily kills mice or rats. However, because it takes several days for the poisoned rodents to bleed out, they can continue to feed on the poison. When they do die, their carcasses can contain residues that are lethal for hawks, owls and other animals that often make a meal of rodents, living and dead."

"SGARs also can accumulate in liver tissue, so an animal that repeatedly feeds on prey containing nonlethal amounts can store up a deadly dose over time. [Maureen] Murray has been studying rodenticide poisoning in birds of prey for years and published research in 2011 that the EPA has cited frequently. That study, published in the Journal of Zoo and Wildlife Medicine, found anticoagulant rodenticide residues in 86 percent of 161 birds that were tested over five years at the Tufts Wildlife Clinic. Murray examined four species of birds—red-tailed hawks, barred owls, Eastern screech owls and great horned owls—and found that of those that tested positive, 99 percent had residues of the SGAR brodifacoum, one of the most widely used rodent poisons in the world." <http://sites.tufts.edu/vetmag/winter-2015/safe-rodent-control/>

In California, in 2013, the Department of Pesticide Regulation collated data about rodenticide residues in 492 non-target animals, including 194 birds (primarily raptors) and 298 mammals (primarily San Joaquin kit fox, bobcats, mountain lions, coyotes, and foxes). "The livers (and/or blood, in a few cases) of each animal were analyzed for at least six anticoagulant rodenticides. Of the 492 non-target animals analyzed, approximately 75% had residues of one or more rodenticide, approximately 73% (359) had residues of at least one second generation anticoagulant rodenticide, and approximately 25% (124) were negative." <http://humanepestcontrol.com/docs/brodifacoum_final_assess.pdf>

It's hard to know how accurately this sample reflects larger populations. Or to gauge just how deleterious sublethal doses may be. An EPA study examined the latter issue: "Potential Risks of Nine Rodenticides to Birds and Nontarget Mammals: a Comparative Approach", July 2004, Office of Pesticides Programs, Environmental Fate and Effects Division. And stated:

"The 'threshold of toxicity' concept (Kaukeinen et al. 2000, Anonymous 2001) also assumes that mortality is the only endpoint of concern. A sublethal dose of anticoagulant can produce significant clotting abnormalities and some hemorrhaging (Eason and Murphy 2001), and such effects might be especially detrimental if combined with other stressors that have additive or synergistic adverse effects... Others have speculated that birds exposed to anticoagulants may become more susceptible to environmental stressors, such as adverse weather conditions, food shortages, and predation (Hegdal 1985, Hegdal and Colvin 1988, LaVoie 1990). Newton et al. (1999) have speculated that sublethal levels of rodenticide might predispose death from other causes (e.g., collisions with automobiles, starvation) or may reduce the chance of recovery from accidents.

[...]
"Papworth (1959), in discussing the mechanism of anticoagulant toxicity, speculated that a slight scratch, bruise, or even a minor internal injury might lead to death from hemorrhage if clotting is inhibited over an injured surface. Some incidents reported to the Agency suggest that raptors exposed to anticoagulants can be in danger of excessive bleeding from minor wounds caused by their prey. Such wounds, not normally life-threatening, may cause prolonged bleeding and mortality when blood-clotting mechanisms are disrupted."
<www.pesticideresearch.com/site/docs/bulletins/EPAComparisonRodenticideRisks.pdf>

And according to the 2013 California Department of Pesticide Regulation memorandum:
"The data also show that exposure of wildlife to second generation anticoagulant rodenticides can lead to sub-lethal effects. Multiple studies have shown that sub-lethal doses can cause lethargy, shortness of breath, anorexia, bloody diarrhea, and tenderness of the joints. Riley et al's (2007) study of bobcats is an example of sub-lethal effects. Mortality in bobcats due to notoedric mange had not previously been reported as a significant pathogen in wild felid; mange has been strongly correlated to brodifacoum ($p<0.05$), but has not been shown to be caused by rodenticides. This shows that even sub-lethal exposures to anticoagulants may contribute to the ill thrift of the animal and hence the mortality in a wild animal... The sub-lethal effects of rodenticides reduce the biological fitness of wildlife."

Besides the anticoagulants, the other most commonly used household rodenticides, bromethalin and cholecalciferol, appear to present less risk of secondary poisoning of predators who consume carcasses of dead or dying mice and rats (though there is for example "a risk of hypercalcaemia and adverse effects on target organs such as the kidney in dogs repeatedly eating carcasses of animals poisoned with cholecalciferol baits.") One reason for the lesser secondary poisoning risk is that rodents tend to lose their appetite as cholecalciferol or bromethalin is taking effect and therefore don't keep multiplying the amount ingested as is often the case with anticoagulants.

In respect to cholecalciferol, there is great variability of susceptibility among non-target wildlife who ingest the bait directly. Possums and rabbits are especially sensitive to its toxic effects and rock squirrels, gophers and ground squirrels are all considered highly susceptible, cats somewhat less so, but that is variable, with some cats dying from doses a quarter of what others survive. "The sensitivity of different bird species to cholecalciferol varies considerably, and care must be taken to minimise exposure to birds when baiting in the field." "Some fish-eating marine mammals, such as seals, which are exposed to high dietary levels of cholecalciferol, are quite resistant" (Keiver, 1988 in www.kurataauriverwines.co.nz/journal/53/nzpp_532990.pdf).

As for the neurotoxin bromethalin, which causes lethal swelling of the brain, according to the EPA's Environmental Fate and Effects Division, Office of Pesticide Programs: it "is very highly toxic to birds on both an acute oral and a subacute dietary exposure basis, and is very highly toxic to mammals on an acute oral exposure basis. No data are available to assess the chronic toxicity of bromethalin to birds."
<http://www.epa.gov/espp/litstatus/effects/redleg-frog/2011/bromethalin/assessment.pdf> There is not a lot of research data on the impact of bromethalin on wildlife; this is likely largely due to its being less common and the lesser risk it presents of secondary poisoning, but also partly due

to the difficulties diagnosing animals that it may have affected. Not only is it hard to diagnose, but there’s no known antidote.