

Anticoagulants

Rodenticide Use on Black-tailed Prairie Dogs and Unintended Consequences to Non-target Wildlife

Prairie dogs are considered a keystone species because they have a unique and substantial influence on plant and animal communities in the grassland ecosystem. Furthermore, prairie dogs themselves serve as prey for predatory mammals and birds, and their burrows provide shelter for many species (Power et al. 1996; Kotliar et al. 1999; Miller et al. 2000). Prairie dogs also are also regarded as competitors with livestock for forage on grasslands and efforts to eradicate or contain them have been an ongoing effort for decades (Derner et al. 2006, Forrest and Luchsinger 2006).

Most efforts to control or eradicate prairie dogs involve some type of poison. In 2005, the first anticoagulant-based rodenticide was registered for use in poisoning black-tailed prairie dogs (*Cynomys ludovicianus*). Although there is always some risk for unintended poisoning of non-target wildlife when using rodenticides, the use of anticoagulants can be especially harmful to animals that eat poisoned prairie dogs such as eagles, large hawks, and mammalian predators like swift fox and black-footed ferrets. Many of the non-target species that are unintentionally poisoned with anticoagulants aimed at prairie dogs are protected by wildlife conservation laws such as state and federal Endangered Species Act laws, the Migratory Bird Treaty Act, and the Bald and Golden Eagle Protection Act. Please contact local law enforcement if you suspect possible wildlife exposure to poisoned prairie dogs or observe dead or injured wildlife in the area where rodenticides have been used.

Black-tailed prairie dogs do not hibernate through the winter in most of their range and are active above ground when weather permits. This allows the use of certain anticoagulants registered

to control black-tailed prairie dogs to be used during the period of October 1 through March 15 of the following year. Anticoagulants are presented to prairie dogs in grain baits that must be placed at least 6 inches down in prairie dog burrows. Prairie dogs feed on the anticoagulant bait multiple times before death with symptoms (e.g. loss of attentiveness, lethargy, swollen or closed eyes) generally manifesting just days after ingestion (Yoder 2008). Unlike other poisons that act quickly, anticoagulants work slowly by preventing the production of clotting factors, leading to hemorrhaging, and death typically occurs one to three weeks after application.

Exposure of non-target animals to the anticoagulants occurs either by directly feeding on the bait or secondarily by consuming poisoned prey. Because of the duration of time it can take for a prairie dog to die, prairie dogs can accumulate the poison in their tissues from multiple feedings over time (Erickson and Urban 2004; Eason et al 2008). During this time, the prairie dogs often come above ground (Bruening 2007), but because of their debilitated state, they become easy targets for hungry predators. Additionally, some prairie dogs will die above ground which attracts scavengers.

In addition to the toxic effects from eating anticoagulants in food items, wildlife may also be negatively affected from prairie dog control when it results in loss of other species or the habitat that is linked to their survival. For example, the federally-endangered northern aplomado falcon (*Falco femoralis septentrionalis*) may become sick or die if it eats anticoagulant-poisoned prey. Furthermore, northern aplomado falcons do not build their own nests, but use nest sites constructed by other raptors or ravens that may



Meadowlark showing hemorrhaging after ingestion of chlorophacinone bait applied to a black-tailed prairie dog town, which lead to its death. / N. Vyas, USGS



Black-tailed prairie dog in a moribund condition after the ingestion of chlorophacinone bait. / N. Vyas, USGS



Dead black-tailed prairie dog on ground surface after the ingestion of chlorophacinone bait. / USFWS

also become depleted from exposure to anticoagulants, thereby reducing nests for alpmado falcons.

The number of non-target animals, such as raptors and predators, that die from anticoagulant poisons is believed to be much higher than what is located because of the extended time it takes for an animal to succumb to the poison (Vyas 1999; Colvin et al. 2008; Ruder et al. 2008). Prior to death, the non-target animal remains mobile, can continue consuming poisoned prey or bait, and often will seek a place to hide off-site, making detection of non-targets very difficult and unlikely (Colin et al. 1988; Vyas 1999; 2010). Also, even though the label requires frequent searches and removal or burial of any poisoned prairie dog carcass, applicators may not have the resources to do so, further increasing the likelihood that non-target animals will consume poisoned prairie dogs. To date non-target animals include badger, raccoon, bald eagles, hawks, owls, turkeys, meadowlark, coyotes, and kit fox (CAHFS 2009; USFWS 2007, 2009, 2011).

Although efforts to control prairie dogs may be warranted under some circumstances, we must ensure that the methods are used in the most responsible manner possible, including adherence to label instructions regarding bait placement, monitoring, and carcass removal. Smarter pest control tools that specifically target pest species specifically need to be implemented to reduce the number of non-target animal deaths. We encourage prairie dog control to include an Integrated Pest Management approach to prevent unnecessary applications when good alternatives to pesticides exist (see <http://www.fws.gov/contaminants>)



Turkey vulture scavenging on a chlorophacinone-poisoned black-tailed prairie dog town. / N. Vyas, USGS.

Bald eagle that succumbed to chlorophacinone poisoning. / USFWS



Prairie grassland ecosystems are one of the most biologically productive ecosystems and one of our Nation's most splendid assets. / USFWS

U.S. Fish & Wildlife Service
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