DOI: 10.7589/2014-04-116

Journal of Wildlife Diseases, 51(1), 2015, pp. 000–000

Anticoagulant Rodenticide Exposure and Toxicosis in Coyotes in the Denver Metropolitan Area

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ABSTRACT: Anticoagulant rodenticides are widely used in urban areas to control rodent pests and are responsible for secondary poisoning in many nontarget wildlife species. We tested the livers of five coyotes (*Canis latrans*) in the Denver Metropolitan Area, Colorado, USA, for anticoagulant rodenticides. All five livers were positive for brodifacoum, with values ranging from 95 ppb to 320 ppb, and one liver was positive for bromadiolone, with a value of 885 ppb. Both of these rodenticides are second-generation anticoagulants, which are more potent and more likely to cause secondary poisoning than first-generation anticoagulants due to their accumulation and persistence in the liver. We concluded that exposure to these rodenticides may have caused the death of at least two of the five coyotes, and urban coyotes in our study area are commonly exposed to rodenticides.

Key words: Brodifacoum, bromadiolone, *Canis latrans*, poison, second-generation, toxicant, urban.

Anticoagulant rodenticides are used extensively throughout urban areas to control rodent populations (Hosea 2000; Watt et al. 2005). These compounds act by interrupting the normal synthesis of clotting factors in the liver once bleeding commences, resulting in fatal hemorrhaging (Eason and Spurr 1995; Eason et al. 2002). Second-generation anticoagulants (e.g., brodifacoum and bromadiolone) are more potent than first-generation anticoagulants (e.g., warfarin and chlorophacinone) because they can effectively poison a rodent after only a single dose (Eason and Spurr 1995; Berny et al. 2006). Second-generation compounds also have slower elimination times from the liver (Eason and Spurr 1995; Erickson and Urban 2004). This persistence in the liver can lead to secondary poisoning of nontarget wildlife (Stone et al. 1999; Hosea 2000; Elliott et al. 2014), including coyotes (*Canis latrans*) in urban areas (Hosea 2000; Riley et al. 2003; Gehrt and Riley 2010). We report finding anticoagulant rodenticides in urban coyotes residing in the Denver Metropolitan Area (DMA) of Colorado, USA.

We captured 32 coyotes in the DMA using padded leg-hold traps and snares and fitted them with global positioning system radio collars from April 2012 to May 2013 as part of an ecological study of urban coyotes. Research protocols were approved by the National Wildlife Research Center, Institutional Animal Care and Use Committee (QA-1972). We monitored study animals with radio telemetry from April 2012 to June 2014. Collars were equipped with mortality sensors that alerted us when a coyote died. Thirteen collared coyotes died during the study. When cause of death was unknown, the coyote was necropsied at the Colorado Division of Parks and Wildlife, Wildlife Health Laboratory (Fort Collins, Colorado). Liver samples were submitted to the Texas A&M Veterinary Medical Diagnostic Laboratory (College Station, Texas) to be screened for anticoagulant rodenticides using high-performance liquid chromatography. Brodifacoum, bromadiolone, chlorophacinone, difenacoum, difethialone, diphacinone, and warfarin were included in the screening. We began testing liver samples from deceased animals only after a coyote was found dead

TABLE 1. Values of anticoagulant rodenticides in coyote livers in the Denver Metropolitan Area, Colorado, USA, 2012–2013.

Coyote ID	Date	Brodifacoum (ppb)	Bromadiolone (ppb)[a]
01M	January 2013	150	N/A
17M	February 2013	95	N/A
24M	March 2013	176	N/A
21M	April 2013	320	885
Uncollared	April 2013	95	N/A

[a] N/A indicates the compound was not found in the coyote liver.

with sarcoptic mange because of the relationship between mange and rodenticide poisoning discovered in bobcats (*Lynx rufus*) and mountain lions (*Puma concolor*) by Riley et al. (2007), Uzal et al. (2007), and Serieys et al. (2013). Thereafter, all necropsied coyotes were tested except for two coyotes that were too decomposed to obtain a valid liver sample. Hence, we only tested five coyote livers for rodenticide toxicosis. All five were positive for brodifacoum, with values ranging from 95 ppb to 320 ppb (Table 1). One coyote (the animal with the highest level of brodifacoum) also was positive for bromadiolone, with a value of 885 ppb (Table 1). No other compounds were found in the five liver samples.

Based on necropsy results, we concluded anticoagulant rodenticides contributed to the death of at least two of the five coyotes tested. The first case was a juvenile male (24M) found dead in open space, with no obvious external injuries or other signs of trauma. Upon necropsy, we found free blood in the abdominal cavity. A puncture wound was present on the left side of the body overlying the spleen but not penetrating the abdominal wall. The spleen was fractured and surrounded by clotted blood. We found no radiographic evidence of gunshot and no evidence of bite wounds. The interpretation for cause of death was acute severe hemorrhage, disproportionate to the amount of trauma observed. This coyote's liver was positive for brodifacoum (176 ppb; Table 1).

The second case was a juvenile male coyote (21 mo) found dead on a two-lane road, with minor evidence of skin tearing over the ventral neck and chest. Necropsy findings indicated additional moderate tearing of the muscle in the region overlying the thoracic inlet, although injuries did not penetrate the chest cavity. The chest was filled with blood. The interpretation for cause of death was severe acute hemorrhage, disproportionate to the mild to moderate trauma received from being hit by a vehicle. We suspected rodenticide toxicosis, and the liver was positive for brodifacoum and bromadiolone (Table 1).

In two additional cases, we found hemorrhage into body cavities with severe lesions to explain the hemorrhage, but also evidence of rodenticide exposure. An adult male coyote (01M) had severe lesions of sarcoptic mange, a gunshot through the chest from a pellet rifle, and free blood in the chest cavity. The liver was positive for brodifacoum (150 ppb; Table 1). A juvenile male coyote (17M) had severe crushing lesions to the head and body from being run over by a vehicle and free blood in the chest and abdomen. The liver was positive for brodifacoum (95 ppb; Table 1). One additional coyote (uncollared male) that we captured for our study was euthanized due to self-inflicted trap-related injuries, but the liver also was positive for brodifacoum (95 ppb; Table 1). Causes of death for nine collared coyotes that were not tested for rodenticide toxicosis-included vehicle collision (five coyotes), gunshot (one coyote), conflict resolution (one coyote removed from Denver International Airport), and undetermined (two coyotes).

Our findings suggest anticoagulant rodenticides likely contributed to at least two of the five mortalities, triggered by mild to moderate trauma resulting in fatal internal hemorrhaging. The detection of anticoagulant rodenticides in coyotes in the DMA indicates exposure to these poisons, either directly or secondarily. Because coyotes are omnivores, they could have ingested poisoned rodent bait (Hosea 2000). However, Elliot et al. (2014) determined that targeted rodents are more likely to provide the exposure pathway of anticoagulant rodenticides to secondary consumers. Small rodents are generally an important food source and the dominant animal prey for coyotes in urban areas (Morey et al. 2007; Lukasik and Alexander 2012), resulting in a high probability that repeated consumption of poisoned rodents leads to rodenticide toxicosis in urban coyotes.

The residue values of brodifacoum in our study coyotes were generally lower than those found in other coyote studies. The acute oral LD_{50} value of brodifacoum in dogs ranges from 250 ppb to 1,000 ppb (Stone et al. 1999). In a study conducted near Boston, Massachusetts, Way et al. (2006) found brodifacoum values of 733 ppb and 542 ppb in two coyotes that were presumably directly poisoned. Hosea (2000) identified values up to 500 ppb of brodifacoum in coyotes in California. Erickson and Urban (2004) described coyotes with values of brodifacoum up to 930 ppb. In our study, the two coyotes for which we interpreted exaggerated hemorrhage were also the two cases with the highest values of brodifacoum in their livers, although these values were still lower than the highest values found in other studies. The lower values are not surprising, however, considering both cases had readily observable mild to moderate trauma to initiate excessive bleeding. Nevertheless, our results indicated that poisoning at a lower level may be enough to contribute to fatal hemorrhaging in these carnivores.

Only one coyote was positive for bromadiolone. The acute oral LD_{50} value of bromadiolone in dogs ranges from 11,000 ppb to 15,000 ppb (Stone et al. 1999); the value in our study animal was 885 ppb. Both Erickson and Urban (2004) and Hosea (2000) reported values of bromadiolone in coyotes up to only 460 ppb. Our study coyote also was positive for brodifacoum, and other investigators also have identified coyotes with both of these rodenticides in liver tissue (Hosea 2000; Erickson and Urban 2004). Overall, brodifacoum appears to be more prevalent and of higher concern in the DMA than other rodenticides, although our results indicated that multiple toxicants may be in use throughout our study area.

In addition to the five coyotes in the DMA, we also tested the liver of another coyote carcass found in rural Colorado (Huerfano County) showing signs of hemorrhage. The most likely cause of death was trauma, but a definitive interpretation was limited by advanced decomposition. We found no evidence of any rodenticides in the liver, indicating that rodenticide toxicosis may not always occur in coyotes. To further understand the effects of anticoagulant rodenticides on coyotes, future studies should compare the values of these poisons in coyote livers across urban and rural systems.

Our findings are consistent with those of other studies that have determined anticoagulant rodenticides are contributing to mortality in urban wildlife (Hosea 2000; Riley et al. 2007). The exposure of all five tested coyotes to rodenticides, especially brodifacoum, indicates the ubiquity of these toxicants in the urban landscape and their ability to reach higher levels in the food chain (Riley et al. 2007). One coyote liver contained more than one rodenticide (both brodifacoum and bromadiolone), and multiple compounds have been found in wildlife species in other studies (Stone et al. 1999; Hosea 2000; Erickson and Urban 2004). The effects of

exposure to multiple anticoagulant rodenticides in urban wildlife species should be a focus of future research to increase our understanding of these toxicants and their population effects on urban carnivores.

We thank J. Brinker, J. Kougher, S. Koyle, D. Lewis, and F. Quarterone for assistance with trapping and handling coyotes. We also thank Texas A&M Veterinary Medical Diagnostic Laboratory for testing liver samples. Funding was provided by the U.S. Department of Agriculture, Wildlife Services, National Wildlife Research Center, and Colorado Parks and Wildlife.

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Submitted for publication 22 April 2014.
Accepted 3 July 2014.