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Dr. Debbie Edwards  
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RE: Section 3 registration under the Federal Insecticide, Fungicide, and Rodenticide Act for Rozol and Kaput as rodenticides to control black-tailed prairie dogs.

Dr. Edwards:

The Central Mountains and Plains Section of The Wildlife Society (CMPS) is a private non-profit organization of approximately 1,400 wildlife professionals in seven member states, (CO, KS, NE, ND, SD, UT & WY), trained in various scientific disciplines who strive to further the conservation of natural resources within the Section and our Nation.

CMPS has members with extensive involvement in black-tailed prairie dog management, including use of lethal control measures to manage this species. We are aware that the Environmental Protection Agency (EPA) has recently approved Rozol (active ingredient: chlorophacinone) for use as a prairie dog rodenticide throughout 11 states in this species range and may also be considering registration of Kaput (active ingredient: diphacinone) for use as a prairie dog rodenticide. These anticoagulant rodenticides have been used in certain areas on prairie dogs under Section 24(c) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) through Special Local Needs registrations and have been documented to cause secondary poisoning to predators and scavengers (Klataske 2009, Lydick 2006, USFWS 2007). This new conditional Section 3 registration under FIFRA for Rozol and possible future registration of Kaput is expected to amplify secondary poisoning deaths to non target animals beyond the previous Special Local Needs registrations and kill avian and mammalian predators/scavengers throughout the 11 states where EPA's section 3 registrations could provide clearance for use of these products. The CMPS is providing this letter to EPA requesting withdrawal of the Rozol registration for prairie dog control and requesting EPA to not register Kaput for prairie dog control.

It is important to have an understanding of how anticoagulants kill prairie dogs in order to appreciate how interactions between prairie dogs and other wildlife species provide a conduit for anticoagulants to cause secondary deaths to predators/scavengers. Prairie dogs are colonial animals that serve as an important food source for a wide array of bird and mammal species that visit colonies and feed upon prairie dogs, many on a daily basis. This regular routine of animals visiting colonies and consuming prairie dogs is a key reason why the use of rodenticides with known secondary toxicity, can have large scale impacts to animal populations far beyond the footprint of the prairie dog colony.

Chlorophacinone (Rozol) and diphacinone (Kaput) act as blood thinners that depress the clotting capabilities of blood and increase the permeability of capillaries thereby predisposing the animals to widespread internal hemorrhaging (EPA 1991). Death occurs to poisoned animals over a period of days to weeks through loss of bodily fluids, mainly blood, through various orifices. In the final days or hours, the poisoned animal's skin membranes may also rupture, leading to fluid loss directly through body areas where no vents would otherwise exist. During the time between consumption of poison and death, prairie dogs move between their burrow systems and above ground habitats and over time become increasingly debilitated and susceptible to above ground predation. This extended period of morbidity before death, ensures that other animals, especially above ground predators and scavengers, are likely to consume poison laden prairie dogs before the anticoagulants are metabolized. Further, prairie dogs can continue to consume poisoned bait over this extended time period (EPA 1991) and if consumed by a predator may have high body burdens of these toxicants in their digestive, circulatory and muscle systems. Finally, there are significant data for other rodent species showing that even after death, there are sufficient levels of anticoagulants remaining in carcasses to secondarily poison the next animal that might consume the first (EPA 2004, EPA 2006, Erickson and Urban 2004, Fisher and Timm 1987). The overall effect of anticoagulant use for prairie dog control results in disoriented, moribund and dead prairie dogs on the surface of the colony that last for weeks with the unintended consequence of secondarily poisoning avian and mammalian predators/scavengers accustomed to feeding at prairie dog colonies. Descriptions of disoriented prairie dogs have been described by prairie dog control personnel, federal investigators and landowners that have returned to colonies days or weeks after anticoagulant applications (USFWS 2005, USFWS 2007).

### **Secondary Poisoning**

The risk of secondary poisoning to non-target wildlife from anticoagulants such as Rozol and Kaput is much higher than from zinc phosphide, the traditional choice for prairie dog control (Colvin et al. 1988, Erickson and Urban 2004). Several EPA documents note the risk from Rozol (EPA 2004, EPA 2006, Erickson and Urban 2004). The most recent document (EPA 2006) repeats a conclusion from Erickson and Urban (2004) that *use of chlorophacinone bait to control prairie dogs has a considerable potential for both primary and secondary risks to birds and nontarget mammals and possibly reptiles. Secondary risks, especially to mammalian predators and scavengers, are likely to be much greater for chlorophacinone than for zinc phosphide.* The secondary poisoning risks from Kaput are even greater than those from Rozol (Erickson and Urban 2004). Colvin et al. (1988) noted that anticoagulants can pose a substantial hazard to raptors and that has been borne out in recent years when raptor carcasses have been submitted for

analysis (Lydick 2006, FWS 2007). A study that evaluated the risks of 11 vertebrate pesticides (Littrell 1990) ranked both Rozol and Kaput as the second most hazardous pesticides. Strychnine was ranked as the most hazardous; zinc phosphide was ranked fifth.

Erickson and Urban (2004) synthesized various laboratory studies that evaluated feeding trails with captive animals of chlorophacinone (Rozol) killed rodents to birds and mammals. In addition to high mortality rates for species such as red fox, European ferrets, and mongooses, other species such as American Kestrels, Tawny Owls and White Storks survived consumption of chlorophacinone killed rodents but the exposure caused external bleeding, internal hematoma and increased blood coagulation time. These effects would likely prove fatal to animals living in the wild. Important to the causation of secondary mortality in these studies, was the level of chlorophacinone remaining in the rodent before it was fed to the test animals. Anticoagulants, such as chlorophacinone, are known to have a longer persistence in body tissues than zinc phosphide (Erickson and Urban 2004, Mendenhall and Pank 1980). A fundamental data gap with the recent EPA section 3 registration for Rozol are data documenting levels of chlorophacinone in prairie dogs after consuming Rozol and through the weeks until death. EPA should have directed some basic dose/body burden data be generated specific to prairie dogs to inform the issue "How toxic are prairie dogs that have consumed chlorophacinone?". Further, this study should have occurred prior to registration of this product for use on prairie dogs given that it is well known that poisoned prairie dogs will be consumed by predators and scavengers. With the multitude of animals secondarily poisoned after consuming Rozol poisoned prairie dogs, it appears prairie dogs carry higher body burdens of the product than many rodents that have already been tested and found to be lethal to the next animal that might consume it. Of note, is the extended time it takes a prairie dog to die after consuming Rozol which likely ensures that many poisoned prairie dogs are consumed by predators before actually dying from the product and thus may carry an even higher body burden of chlorophacinone than post mortem specimens.

Seery and Matiatos (2000) found ferruginous hawks are closely linked to prairie dogs and occur in higher numbers in areas where prairie dog concentrations occur. Proven secondary poisoning combined with some species propensity to feeding on prairie dog colonies, such as ferruginous hawks, makes it likely there will be disproportional effects to certain species by this EPA action to expand the use of Rozol for prairie dog control. Ferruginous hawks are in decline and on many conservation groups watch list and sensitive species lists (USFWS 2002).

In 2001, the President of the United States issued Executive Order (EO) 13186 which directs federal agencies proposing actions that may have measurable effects on migratory birds to develop a Memorandum of Understanding (MOU) with the US Fish and Wildlife Service to demonstrate how conservation of migratory birds will be promoted. This MOU is necessary due to the anticipated detrimental impacts to migratory birds from the registration of anticoagulants for prairie dog control and the resultant secondary poisoning that will occur. It is our understanding EPA has not completed this MOU. We believe authorization of Rozol and Kaput for prairie dog control has a high likelihood of adversely affecting ferruginous hawks and other raptors and scavenging birds at a measurable level. Accordingly, we request EPA undertake development of the MOU outlined in EO 13186.

## **Section 7 Endangered Species Act Compliance**

In 1991, EPA requested Endangered Species Act Section 7 formal consultation through publication in the federal register of “may affect” determinations for 31 pesticides (EPA 1991). Chlorophacinone and diphacinone were two of the 31 chemicals evaluated by that formal consultation, but none of the registered uses in 1991 of these anticoagulants were for prairie dog control. Regardless, when the US Fish and Wildlife Service issued a Biological Opinion in 1993, they determined the rather limited registered uses of chlorophacinone evaluated at that time would jeopardize the continued existence of 21 species listed under the Endangered Species Act (USFWS 1993). The Biological Opinion identified reasonable and prudent alternatives that if followed, should avoid jeopardizing the listed species for the registered product uses at that time. With the new 2009 registration by EPA to expand use of chlorophacinone and potentially diphacinone for use on black-tailed prairie dogs in 11 states, it would be expected the list of adversely affected species would be considerably greater than when the relatively constrained uses were the subject of formal Section 7 consultation in 1993. Additionally, there are twice as many listed species today than when EPA requested formal Section 7 consultation in 1991 when there were 639 versus 1,317 listed species in 2009 (USFWS 2003, USFWS 2009). The use of Rozol and Kaput for the control of prairie dogs constitutes new uses not evaluated in the 1993 Biological Opinion. EPA has been apprised of the need to reinitiate Section 7 consultation for this new use of Rozol on prairie dogs (EPA 2008, USFWS 2006a, USFWS 2006b, WAFWA 2008) but to our knowledge, EPA has not undertaken required Section 7 consultation for anticoagulant use on prairie dogs. Because additional formal Section 7 consultation has not occurred, we believe registration to allow the use of Rozol and Kaput for prairie dog control are outside regulatory parameters established under the Endangered Species Act and the Administrative Procedures Act and therefore should be revoked until consultation is completed. We also question whether EPA has relied upon the best available scientific information, as required under the Data Quality Act, in reaching the decision to register Rozol for use on prairie dogs in light of the significant concerns raised internally by EPA’s Environmental Fate and Effects Division and scientists with other agencies and organizations. Blatant disregard for the above federal laws in order to register these anticoagulants for prairie dog control, could call into question whether such products are indeed even legal for such use.

There are also State listed species throughout the CMPS and other States, such as swift fox, that these anticoagulants are expected to secondarily poison. There appears to have been no consideration given to these State listed species prior to the EPA Rozol registration on May 13, 2009. This omission is unacceptable given the input provided to EPA by the Western Association of Fish and Wildlife Agencies in an August 18, 2008 comment letter (attachment) expressing concerns by State Wildlife Managers on the use of anticoagulants for prairie dog control (WAFWA 2008).

A specific example involving the Special Local Needs registration for Rozol in Nebraska illustrates the frustration of the conservation community with EPA on these anticoagulants. In 2006, the Nebraska Department of Agriculture began accepting comments on a proposed Special Local Needs registration under Section 24 (c) of FIFRA to use Rozol for prairie dog control. During the comment period the Nebraska Game and Parks Commission and US Fish and Wildlife provided information showing secondary poisoning to non target animals were likely,

that there is already a wide array of other products available for prairie dog control and requested EPA undertake Section 7 consultation under the Endangered Species Act. That information was also provided to EPA, including a reminder that in 2005, EPA had investigated Rozol use in South Dakota on a prairie dog colony that resulted in extensive numbers of dead and dying prairie dogs above ground which had been widely scavenged by other wildlife. Further, that pattern would be expected to be repeated in Nebraska because the slow acting nature of Rozol results in large availability of poisoned prairie dogs on the surface of a prairie dog colony. It was imperceptible that EPA did anything with the information or that Section 7 consultation for Rozol use on prairie dogs was undertaken. The Special Local Needs registration became effective October 1, 2006. By December of 2006 a dead bald eagle was retrieved close to a prairie dog colony that had recently been poisoned with Rozol and laboratory analysis indicated the eagle died of chlorophacinone (Rozol) poisoning (USFWS 2007). At the time, the bald eagle was still listed and protected under the Endangered Species Act, the Bald and Golden Eagle Protection Act and the Migratory Bird Treaty Act. Since that time, additional animals have been collected and documented to have been secondarily poisoned by Rozol, EPA has still not conducted formal Section 7 consultation and then on May 13, 2009 EPA registered Rozol for use on prairie dogs in 11 states beginning in the fall of 2009.

### **Previous Actions**

In 2008, the Western Association of Fish and Wildlife Agencies requested EPA to rescind Special Local Needs Registrations for Rozol and Kaput for prairie dog control because of impacts to non target species, lack of consultation and inadequate label restrictions to protect grassland species (WAFWA 2008). Those concerns, which have yet to be addressed, are now amplified by EPA's recent registration of Rozol for rangewide prairie dog control.

In South Dakota, a Special Local Needs Registration for Rozol use on prairie dogs was rejected in 2005 by the South Dakota Department of Agriculture based on four factors:

- 1) **Legality** - with other products already registered for prairie dog control, they believed it was questionable whether this product met the definition under FIFRA for a "special local needs" exemption.
- 2) **Efficacy** – the data presented show a lower efficacy than other readily available products, and Rozol requires multiple feedings and possible follow up treatments.
- 3) **Environmental Hazards** – significant concerns were raised for secondary poisoning of animals that feed on prairie dogs, how to dispose of poisoned prairie dogs that are retrieved as well as the multiple return trips to a colony to collect dead and dying prairie dogs outweigh any perceived advantage of Rozol over zinc phosphide.
- 4) **Cost** – estimated costs would be at least 50% higher than using zinc phosphide. The expense of multiple return trips to a colony to collect dead and dying prairie dogs outweigh any perceived savings from the need to only place bait once with Rozol versus a need to pre bait with zinc phosphide.

The environmental hazards, especially concerns about secondary poisoning, have been shown to be prophetic, given the non target animals found since 2005 when the original request to South Dakota was received and other states authorized anticoagulants for prairie dog control under Special Local Needs registrations. Also, the level effort to retrieve and dispose of disoriented, moribund and dead prairie dogs on the surface to prevent exposure to non target predators and scavengers would require much more extensive search efforts than the two searches recommended on the label. However, even those minimal label search requirements appear to not be followed based on EPA and USFWS investigations into Rozol use on prairie dog colonies (USFWS 2005, USFWS 2007).

### **Conclusion**

The CMPS requests EPA rescind the new registration for Rozol granted May 13, 2009 and not register Kaput for use on prairie dogs. Further, we request EPA require studies to determine body burden levels of these anticoagulants in prairie dogs at specific periods post consumption including prairie dogs still alive and undertake studies to ascertain impacts of secondary poisoning to non target species. Finally, we request EPA complete their statutory responsibilities under the Federal Endangered Species Act including formal Section 7 consultation and comply with the various State Endangered Species Acts as applicable, prior to registration of these anticoagulants that are documented to secondarily poison species listed under those Acts when used according to labels. Should EPA choose to disregard these comments, we ask that EPA prepare an Environmental Impact Statement to assess the environmental impact of and alternatives to this major federal action that will significantly affect the environment.

Sincerely,

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