American Bird Conservancy ▪ Audubon of Kansas
Defenders of Wildlife ▪ Natural Resources Defense Council

September 24, 2012

Ms. Lois Rossi
Director, Registration Division
Regulatory Public Docket
Office of Pesticide Programs (OPP)
Environmental Protection Agency
1200 Pennsylvania Ave., N.W.
Washington, D.C. 20460

RE: Proposed Registration of Kaput-D (diphacinone) to control Black-Tailed Prairie Dogs (EPA-HQ-OPP-2012-0739)

Dear Ms. Rossi:

We understand the EPA is proposing to register Kaput-D for the control of black-tailed prairie dogs in Colorado, Kansas, Nebraska, New Mexico, North Dakota, Montana, Oklahoma, South Dakota, Texas and Wyoming. Kaput-D contains the anticoagulant rodenticide diphacinone, which is not selective and has a high probability of negatively impacting non-target wildlife including species protected under the Endangered Species Act, the Bald and Golden Eagle Protection Act, and the Migratory Bird Treaty Act. For this reason, and for the reasons described below, Defenders of Wildlife, American Bird Conservancy, Natural Resources Defense Council, and Audubon of Kansas oppose registration of Kaput-D for this use and urge EPA to reject the application for a conditional Section 3 registration under the Federal Insecticide Fungicide and Rodenticide Act.

In announcing its proposed registration decision, EPA leans heavily on the U.S. Fish and Wildlife Service's Biological Opinion for Rozol Prairie Dog Bait, which contains a different active ingredient, chlorophacinone. *See U.S. Fish and Wildlife Serv. Final Biological Opinion for Rozol Use on Black-tailed Prairie Dogs*, April 9, 2012. We acknowledge that the proposed label for this product is very similar to what was ultimately adopted for Rozol, after litigation by Defenders of Wildlife, Audubon of Kansas, and the Natural Resources Defense Council. We further understand that the manufacturer here, Scimetrics, has agreed to implement all mitigation and reasonable and prudent measures (RPMs) specified in that biological opinion, and that the label will specify that either Rozol or Kaput-D, but not both, could be used to control prairie dogs in a particular location at the same time. While we appreciate these steps, they are insufficient for EPA to meet its obligations under federal law.

First, it is not clear from the materials posted to the docket whether EPA intends to initiate and complete formal consultation under Section 7 of the Endangered Species Act with the U.S. Fish and Wildlife Service prior to registering this product. EPA cannot simply piggy-back onto another prior consultation for an entirely different active ingredient. While there may indeed be similarities between the two chemicals, registration of Kaput-D prior to completion of consultation violates the ESA. We strongly encourage EPA to complete Section 7 consultation

prior to registration both to avoid litigation risk and so that endangered species concerns may properly be analyzed and necessary use restrictions incorporated in the label and *Bulletins Live* prior to any field use.

In addition to these ESA concerns, we do not believe that expansion of the use of a first-generation anticoagulant like diphacinone for use on prairie dogs is appropriate because of its impact on other non-listed, non-target species.[1] Prairie dog colonies are used by many protected wildlife species that prey on or scavenge prairie dogs or use their burrows for shelter. The use of rodenticides in and around prairie dog burrows can have significant impacts to animal populations beyond the intended target. The proposed label change would make this product available for this use throughout the range of the prairie dog, an area covering some 2.4 million acres in the western United States. Numerous species will be impacted by this use.

Diphacinone causes internal hemorrhaging and damage to capillaries throughout the body. Affected animals exhibit differences in behavior or weakness prior to death, which makes them susceptible to predators that in turn are poisoned. Kaput is categorized as a "first-generation" rodenticide. First generation rodenticides are less acutely toxic and more rapidly metabolized and/or excreted than "second generation" rodenticides, thus they must be ingested at multiple feedings to administer a lethal dose. It can take several days for diphacinone to kill the target animal because it is tied to the metabolic turnover of Vitamin K. Because of this lag-time, prairie dogs may consume much more than a lethal dose. By the time the animal expires or is predated upon, it may be carrying in its system a "super dose" of the rodenticide, which can result in secondary poisonings of non-target species, including much larger animals such as eagles and badgers.

Birds and non-target mammals that feed on grain-based baits are also at risk of direct poisoning. Field applications put a broad spectrum of grassland birds, including prairie-chickens and sage-grouse, as well as songbirds like the western meadowlark, and shorebirds like upland plovers and mountain plovers, at risk of primary exposure. Misuse of the pesticide only heightens this risk. Prairie-chickens and sage-grouse are species of special concern that are being considered for possible ESA listing. Non-target predatory and scavenging species at risk of secondary poisoning include the highly-endangered black-footed ferret, as well as badgers, coyotes, foxes, raccoons, skunks, bald eagles, golden eagles, hawks, and owls.

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[1] We also take issue with the premise of approving yet another product to eradicate the black-tailed prairie dog, which has suffered massive declines throughout its range due to poisoning and disease. The registration of Kaput-D is likely to result in expanded prairie dog poisoning, thus increasing the likelihood that the species will itself require federal endangered species protection in the future. EPA should also consider the impacts of an increasingly poisoned landscape on future black-footed ferret recovery efforts. The war on prairie dogs in the West, combined with the effects of plague on the species, has already dramatically reduced prairie dog populations and their geographic distribution. Elimination of more prairie dogs and their burrows from the landscape due to poisoning will undoubtedly diminish the future success of ferret recovery by reducing the number of suitable sites for reintroduction and restoration.

Recent studies, moreover, indicate that diphacinone may pose more significant risk to owls, hawks, and other non-target species than previously realized. *See, e.g.,* Rattner, et al. 2012. *Assessment of toxicity and potential risk of the anticoagulant rodenticide diphacinone using Eastern screech-owls (Megascops asio),* Ecotoxicology. Part of the earlier mortality underestimation derives from variations in species sensitivity that are not captured by traditional avian toxicity tests. Recent acute toxicity studies in the American kestrel (*Falco sparverius*), for example, demonstrate that these birds are 20 to 30 times more sensitive to diphacinone than the Northern bobwhites (*Colinus viginianus*) and mallards (*Anas platyrhynchos*) that are used for chemical registration toxicity studies. Rattner, et al. 2011. *Acute toxicity, histopathology, and coagulopathy in American kestrels (Falco sparverius) following administration of the rodenticide diphacinone.* Environ. Toxicol. Chem.

The EPA's Ecological Risk Assessment for Diphacinone for Use on Black-tailed Prairie Dogs (*Cynomys ludovicianus*) from June 2012, relies heavily on LD-50 and LC-50 levels in determining the effects on non-target wildlife. But recent research is challenging the applicability of standardized avian acute oral toxicity tests for first generation anticoagulant rodenticides. Work by Rattner shows that risk values derived from acute oral exposure studies can provide misleading data for ecological risk assessment and the interpretation of samples in forensic examinations. Rattner compares diphacinone acute oral toxicity studies and 7-day feeding trials: "….in diphacinone acute oral toxicity studies with Eastern screech-owls and American kestrels, the lowest doses evoking lethality were 171 mg/kg (retained dose) and 79 mg/kg, respectively. However, in 7-day dietary feeding trials in Eastern screech-owls, the lowest cumulative dose evoking lethality, 5.75 mg/kg, was more than an order of magnitude less than the lowest lethal dose in acute toxicity trials." (Rattner, et al. 2012).

A recent publication by Vyas and Rattner also considers the applicability of the testing methods and suggests that median lethal dose values derived from standardized acute oral toxicity tests underestimate the environmental hazard and risk of diphacinone and other first-generation anticoagulants. These products require multiple feedings over several days to achieve a threshold concentration and to cause adverse effects. The authors propose that testing the toxicity of these products may require a different exposure regimen than that used for acutely toxic rodenticides – the secondary anticoagulants and the non-anticoagulants: "…despite literature (as far back as 1986) documenting that the standardized acute oral toxicity test is not suited for FGARs [first generation anticoagulant rodenticides], the USEPA, USGS, US Fish and Wildlife Service, and the US Department of Agriculture continue to conduct standardized acute oral toxicity testing for FGARs and continue to use the results in their risk characterizations, and for planning and operational rodenticide applications." Vyas and Rattner also discuss data suggesting that the first-generation anticoagulant residue values in tissues derived from acute oral toxicity testing may not be appropriate benchmarks for confirming these rodenticides as the cause of death. Vyas NB, Rattner BA. 2012. *Critique on the use of the standardized avian acute oral toxicity test for first generation anticoagulant rodenticides*. Human and Ecological Risk Assessment in press. DOI:10.1080/10807039.2012.707934.

Incident data further indicate that diphacinone is harmful to birds. American Bird Conservancy's AIMS database (http://www.abcbirds.org/abcprograms/policy/toxins/aims/aims/login.cfm) includes reported bird

deaths from exposure to diphacinone from as far back as 1993. Species affected include the Turkey Vulture, Red-tailed Hawk, the Rock Pigeon, and the Snowy Owl. Many of the birds that could potentially be exposed to diphacinone are migratory birds, which are protected under the Migratory Bird Treaty Act, to which the EPA must adhere. These incidents indicate that secondary exposures are occurring following the use of diphacinone.

As the U.S. FWS stated in the Rozol Biop, migratory raptors are particularly at risk:

> Migratory raptors are especially susceptible to secondary poisoning from anticoagulant use due to their propensity to feed in prairie dog colonies (Golden and Gober 2010). Raptors are believed to be especially susceptible to secondary poisoning from Rozol given the likelihood that they can spot dead or dying BTPDs [Black-Tailed Prairie Dogs] that are more difficult to see from a ground level perspective (Vyas 2010b) and raptors have been observed to be attracted to Rozol poisoned BTPD colonies (Vyas 2010a). The golden eagle, ferruginous hawk, and burrowing owl are among nine species with documented dependence on prairie dog colonies (Kotliar et al. 1999, Seery and Matiatos 2000). All three of these raptor species have been identified as "Species of Conservation Concern," defined as species that are likely to become candidates for listing under the ESA without additional conservation action (FWS 2008a). Further, bald and golden eagles are protected under the Bald and Golden Eagle Protection Act. In particular, ferruginous hawks and golden eagle populations appear to be experiencing declines throughout most of their range, and the availability of poisoned prey, which occurs when anticoagulants are used for prairie dog control, are expected to exacerbate population declines. Golden eagle populations may not be able to withstand additional loss of individuals (FWS 2009d, Golden and Gober 2010). Bald eagles have a kleptoparasitic association with ferruginous hawks (whereby eagles pursue ferruginous hawks and steal their prey) which are an efficient predator of prairie dogs (Jorde and Lingle 1988). Thus, both species may be particularly vulnerable to anticoagulants use to kill BTPDs (Golden and Gober 2010). This suspected vulnerability is further supported by the opportunistic recovery of two bald eagles killed from chlorophacinone exposure previously described and the abundance of dead ferruginous hawks reported by Audubon of Kansas from an area where Rozol was being used to poison prairie dogs. Migratory bird deaths attributed to chlorophacinone poisoning are not permitted or authorized under the Migratory Bird Treaty Act (MBTA).

USFWS Biological Opinion (Rozol) at 30-31.

We believe incidents such as these are significantly underreported. As noted, many carcasses are not found due to lack of monitoring, bodily decay, removal by scavengers, or dying in hard-to-see locations.[2] Poisoned birds may fly far from the site of application during the 4-6

[2] According to the U.S. FWS, "carcass-detection studies have found that even when searches are performed in areas known to contain carcasses, a significant percentage will never be found due to scavenging, size or coloration that renders the carcass inconspicuous, or field conditions such

days after they have been affected, prior to death. In addition, there is little evidence that label requirements for carcass searches are actually being followed in the field. Thus, the majority of incidents are probably not being reported to the appropriate authorities, and even when they are reported, those authorities may not have the funds to do analytical testing. The fact that there have already been numerous cases involving chlorophacinone and other anticoagulants provides compelling evidence that diphacinone can and will kill non-target organisms via secondary poisoning.

In the Rozol consultation and in prior correspondence with EPA over both Rozol and Kaput-D, the Fish and Wildlife Service identified multiple threats to non-target species that are not sufficiently abated by the current Rozol (and proposed Kaput-D) label. *See, e.g.,* USFWS Biological Opinion (Rozol) at 29-31. Indeed, FWS specifically stated that: "The current Rozol label and registration requirements are inadequate for addressing Migratory Bird Treaty Act and Bald and Golden Eagle Protection Act bird deaths that have previously occurred from Rozol use on prairie dogs and that are expected to continue under the proposed action." *Id.* at 2. Specifically, the USFWS found the label restrictions for Rozol inadequate: "The Service is gaining a better understanding of the Rozol label requirements regarding multiple return visits to retrieve dead and dying prairie dogs and exposed bait. Based on the information provided by the EPA and for reasons explained above, we believe the label requirements do not prevent exposure to migratory birds or may be impractical or not implementable." *Id.* at 31.

The fact that EPA is proposing "a one year time-limited registration" for Kaput D suggests that EPA will likely re-evaluate the registration based on its first-year experience. While we support FWS's admonition in the Rozol BO that the registration "be accompanied with detailed monitoring and field studies to abate Rozol secondary exposure and effects to raptors and other non-target animals," *Id.* at 2, and encourage this as well for Kaput-D, the unfortunate truth is that the proposed monitoring and mitigation measures may not be feasible for applicators. Simply put, the monitoring and carcass removal requirements for Rozol are out-of-touch with reality. Even though the labels require frequent searches and removal or burial of poisoned prairie dogs, in practice the amount of time and manpower this would entail is not feasible for applicators, and the required followup does not take place. This puts scavengers and predators at risk of consuming poisoned prairie dogs. (USFWS. 2012. *Anticoagulants: Rodenticide Use on Black-tailed Prairie Dogs and Unintended Consequences to Non-target Wildlife*).

The chlorophacinone product labels include stipulations that applicators return to the colonies to retrieve and dispose of dead and dying animals. On August 12, 2010, the USFWS and EPA/North Dakota personnel held a meeting with ranchers, producers, and North Dakota

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as remote, inaccessible areas, that impede searches (Vyas 1999). In the case of anticoagulants, the delayed toxicity can temporally or geographically distance the carcass from the application area (Colvin et al. 1988). In addition, exposure to chlorophacinone may result in sub-lethal effects that occur at concentrations below a diagnostic threshold for lethality, masking their role in mortality incidents where acute lethal hemorrhage is not the proximal cause of death and may be attributed to causes such as trauma or disease (Stone et al. 1999)." USFWS Biological Opinion (Rozol) at 30.

Agricultural Extension personnel. Participants were uniform in their indictment of this label requirement as unrealistic and impractical. They indicated that they do not have the time, resources, or inclination to search for dead prairie dogs. (USFWS. 2012. *Letter from Scott Larson, Field Supervisor, South Dakota Field Office to Dr. Debbie Edwards, US EPA re: Proposed chlorophacinone use on California ground squirrels*).

Threats to non-target wildlife are simply not addressed by the protections recommended for ESA listed species. Given the magnitude of the impact to non-target wildlife, which will only be magnified with the proposed registration of another first generation anti-coagulant for use on prairie dogs, and given that use of this product will almost certainly result in take in violation of the Migratory Bird Treaty Act and the Bald and Golden Eagle Protection Act, we believe it is impermissible for EPA to register this product under FIFRA's no unreasonable adverse environmental effects standard.

Furthermore, EPA cannot lawfully issue a conditional registration for a compound for which it lacks essential data. The Rozol biological opinion identifies multiple research gaps which should be addressed to ensure that this product is safe for wildlife. Studies which EPA required be prepared for Rozol need to be in hand and analyzed by EPA prior to any final decision to register Kaput-D, because they bear directly on the statutorily required reasonableness finding. Registration of Rozol prior to preparation and review of those studies does not justify registering Kaput-D, since EPA wrongly failed to make a reasonableness determination of Rozol. While EPA does not elaborate on its reasons for limiting Kaput-D registration to one year, to the extent that lack of data is part of that reason, a time limit is not a legally sufficient basis for registration in the absence of information needed for a reasonableness determination.

Among other things, we request that EPA require new data from the manufacturers for avian toxicity studies using a songbird model. Songbirds are more sensitive to acute exposure and may also be more sensitive to chronic exposure during the breeding season. Young of altricial species (e.g. sparrows) are less developed at hatching, having lower liver and kidney metabolic capabilities than precocial species (e.g. mallard and quail), which may render them more sensitive to pesticide exposure. EPA should require that all studies recommended in the FWS's General Conservation Measures and Reasonable and Prudent Measures and Conservation Recommendations specified in the Rozol BiOP. Additional studies may be identified during consultation for Kaput-D, which is another reason why completion of consultation prior to registration is essential.

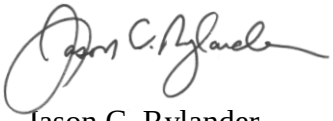
Conclusion

In conclusion, the undersigned organizations continue to have serious concerns both about the impacts of Kaput-D on threatened and endangered species and, more broadly, EPA's commitment to ensuring that registration of pesticides like Rozol and Kaput-D does not harm imperiled wildlife or unreasonably impact the environment. Any action by EPA to register this product for field use on prairie dogs requires initiation and completion of consultation with the U.S. FWS for risks to endangered species prior to registration of Kaput-D. We again stress that while adoption of conservation measures in the Rozol Biop may address some concerns for ESA-

listed species, these measures will not eliminate risk to species protected under the Bald and Golden Eagle Protection Act and the Migratory Bird Treaty Act. Finally, EPA is basing its risk assumptions on inappropriate acute toxicity tests, lacks essential information on secondary toxicity, and cannot demonstrate that Kaput-D will not cause unreasonable environmental harm as is required by FIFRA.

The risks are too great and the benefits too little to justify approving this use. Accordingly, EPA should deny this registration.

Respectfully submitted,



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