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Submitted by email to opp-docket@epa.gov and white.kelly@epa.gov

Dear EPA Office of Pesticide Programs:

Thank you for the opportunity to comment on the revised comparative ecological risk assessment for the nine rodenticides currently being considered for re-registration (OPP-2004-0033). We, the undersigned organizations and individuals, welcome this opportunity to comment on this document and provide our input on what we consider to be a critically important issue – that being the continued widespread uses of these rodenticides in the environment.

As background, this rodenticide "cluster" RED, first released on 11 September 1998, includes nine compounds, including six anti-coagulant rodenticides (brodifacoum, bromadiolone, difenacoum, diphacinone, chlorophacinone, warfarin), one neurotoxic rodenticide (bromethalin), and two non-anti-coagulant rodenticides (zinc phosphide, cholecalciferol). It is clearly stated in this document that the EPA "is concerned about accidental poisonings of children by rodenticide products" and is also worried about incident data showing "potential problems involving non-target and secondary exposures to wildlife from the rodenticides". We agree wholeheartedly with those concerns.

All nine rodenticides are used against commensal rodents, but only 4 (warfarin, diphacinone, chlorophacinone, zinc phosphide) are used against field rodents. Overall, the EPA ranks its potential overall risk to birds and non-target mammals as follows: brodifacoum (1), zinc phosphide (2), difethialone (3), diphacinone (4) - all ranked HIGH. All 9 rodenticides are available to the public "over-the-counter" as grain-based food baits for control of commensal rats and mice (predominantly the Norway rat (*Rattus norvegicus*), roof or black rat (*R. rattus*), and house mouse (*Mus musculus*) in and around buildings, transport vehicles (mainly ships), and inside sewers. Four of these rodenticides – warfarin, diphacinone, chlorophacinone, and zinc phosphide – also are available for control of various rodents and other small mammal pests in the field and other outdoor settings. For example, zinc phosphide is a broad spectrum rodenticide that has a variety of agricultural uses including the control of jackrabbits and prairie dogs on rangeland. [ADD INFO on uses of the other three]

Our comments on the "Comparative Risks of Nine Rodenticides to Birds and Nontarget Mammals," will outline problems found with the current general use registration of these

rodenticides (business as usual), problems found with the comparative rodenticide risk assessment itself, include discussions of risk to wildlife species (including endangered species), domestic animals, and humans, and conclude by outlining a number of recommendations regarding the outcome of this re-registration process for these rodenticides. As far as the contents of the document itself, our comments do not include a critique of each point, rather we have focused on the larger issues as we see them. We have provided more content-related comments on previous versions of the rodenticide risk assessment (Jan 2003?) and defer to these for this purpose (see enclosed).

We point out here that a previous version of the rodenticide risk assessment, dated November 30, 2000, was peer reviewed by Dr. Elwood Hill, Dr. Raymond O'Connor and Dr. Charles Eason (Document ID OPP-2002-0049-0004). These three scientists are exceptionally qualified for this particular peer-review, and we defer to their critiques in matters relating to the toxicity, chemistry, and environmental hazards posed by the nine compounds investigated.

Inherent Problems with the Rodenticide Registration Notice Process

Initially, we have two major concerns regarding the way EPA has handled this rodenticide "cluster" re-registration process. First, we need to point out that the EPA conclusion that the rodenticides stated here were eligible for re-registration was reached in 1991, well before the mountain of evidence of widespread contamination and mortality/morbidity incidents of these rodenticides wherever they are used and regardless of labeling requirements. It is painfully obvious to us that the playing field today has changed drastically from what it was in 1991 with regard to rodenticides, yet EPA is still living in the past operating under a badly outdated conclusion. The data used to reach this conclusion should, in fact, be updated and revisited.

Second, we question EPA's chosen strategy of lumping all nine rodenticides together into a "comparative" risk assessment. This leaves the EPA without the ability to deal with one or more of the most egregiously hazardous rodenticides. By ranking the rodenticides by hazard for several different categories, it allows EPA an "out", where rodenticides can be ranked by some artificial method and compared and discussed instead of scrutinizing the hazards of each chemical individually. Consequently, through this method of comparative toxicity, close scrutiny of the most hazardous rodenticides is bypassed and the true hazards of individual rodenticides is obfuscated. We believe that this comparative method was not the proper way to conduct the risk assessment because the results will be of limited use for predicting the true environmental risks posed to animals and humans by the use of these rodenticides.

Problems with the Comparative Rodenticide Risk Assessment

The language in the EPA comparative risk assessment clearly points out their own concerns regarding the substantial risks to birds and non-target mammals. We could not agree more that the EPA should be concerned, extremely concerned. For one, the high acute toxicity of these rodenticide baits, particularly the 2nd generation anti-coagulants, is of major concern – they were designed to kill small mammals in rapid fashion; many are lethal following one exposure, some baits (commensal and field) contain ingredients that attract non-target animals, and predators and scavengers are then attracted to the dead or dying rodents or non-target organisms. For two, risk

estimates (based on available exposure and effects data) exceed the EPAs LOCs. For three, there is substantial mortality of birds and non-target mammals exposed to rodenticide baits or poisoned prey in controlled or uncontrolled settings as evidenced by controlled lab studies, field incident records, and lab and field observations. For four, retention times of residues in body tissues of primary consumers are of great concern because it is so high. And five, numerous reported incidents indicate that exposure is occurring in numerous non-target species, including avian and mammalian predators and scavengers.

We want to point out numerous other problems with the comparative rodenticide risk assessment, as follows:

(1) there has been excessive undue influence from industry on the entire process; it seems that the RRTF (Rodenticide Registrants Task Force) has blocked, delayed, and essentially forced the EPA to re-write the rodenticide risk assessment. The RRTF has had numerous exclusive meetings with EPA since the initial 1998 risk assessment and the current risk assessment seems to reflect their undue influence over the entire process. And there has been little to no opportunity for any other stakeholder to provide input or even attend any of these meetings with EPA. In addition, the EPA would not allow any other stakeholder to view drafts of the rodenticide risk assessment as it progressed, yet willingly shared them with industry.

(2) there are incredibly few data for true toxicological tests that compare the toxicity and efficacy of these nine products side by side for a variety of species – this is a true weakness of the risk assessment.

(3) there is no consideration whatsoever of sublethal effects – the continued reliance by EPA on dead bodies is incredibly unrealistic and demonstrates that EPA is content with remaining in the dark ages of wildlife toxicology; in addition to liver pathology, there is some evidence to suggest that reproduction may be impacted by sublethal exposure to rodenticides – disruption of Ca mobilization and remobilization processes, eggshell production in birds and reptiles, ataxia, anorexia, dyspnea, and behavioral changes (lethargy, exercise intolerance) among others (Plumlee 2004).

(4) further, there is no consideration of the possible impact of prior exposure (tissue residues of one or more rodenticides) on subsequent exposure; there is some data to support the idea that non-target mammals already exposed to rodenticides have a greater susceptibility to subsequent exposure to rodenticides (Mosterd and Thijssen 1991); from the available data, it is apparent that the 2nd generation anti-coagulants are highly persistent in the liver and other tissues due to their high target binding capacity, so animals carrying around residue burdens of 2nd generation anti-coagulants may have increased susceptibility, a potentially critical factor.

(5) current rodenticide risk models are not at all realistic – they use data that are not indicative of reality (with very few high values, mainly low values), they use way too low of a number of % of rodents that will be exposed (i.e., 1%), there is a huge amount of uncertainty associated with these nine compounds, resistance issue is not being addressed,

(6) the EPA relies much too heavily on acute toxicity data for their comparative risk assessment; this is a problem because there are many reasons to be wary of acute toxicity studies of rodenticides; mortality is not a good endpoint because it is highly variable, tied to animal husbandry practices, the acute toxicity tests are too short a duration to account for all test mortality,

and for birds, the test birds are being provided the antidote (through their feed – soy and alfalfa often are high in vitamin K_1) at the same time they are being dosed, which really confounds matters.

(7) a significant source of uncertainty in the risk assessment is the fact that most of the laboratory studies have tested acute effects in species such as the northern bobwhite, mallard, laughing gull, ring-necked pheasant and domestic chicken. Secondary effects were tested primarily in barn owls, red-tailed hawks, Eurasian buzzard and laughing gulls. The incident data presented applies largely to great horned owls, screech owls, golden eagles, and red-tailed hawks. However, as indicated by a well-documented brodifacoum poisoning incident at the National Zoo, birds of much smaller body size, such as finches, thrushes and warblers, are also susceptible to secondary (and most likely primary as well) exposure to rodenticides. However, very little research has been presented to address either toxicity or exposure to small birds. As indicated in the critique by Dr. Woody Hill, "neither the 175-200 g quail nor the 1-1.2 kg duck is a proper representative (physiologically or toxicologically) of a 50 g bird even if the 50 g bird is a juvenile bobwhite or mallard. These sources of variation (error?) should be addressed in the narrative." Furthermore, the small size of these birds might well preclude them from being recovered and included in incident data. Many small birds may also face significant exposure. The ubiquity of birds such as robins, chickadees, finches and cardinals near urban and rural houses means that they could come into contact directly with baits placed outdoors, or secondarily by feeding on insects that have fed upon bait. We feel that the exclusion of data on small birds from consideration in either the laboratory studies or the incident data has significant potential to underestimate the overall risk to birds of these rodenticides.

(8) exposure as a component of risk - the 2002 rodenticide risk assessment document states that "Risk is a function of exposure and hazard (toxicity)." The assessment bases exposure estimates on the "amount of active ingredient available per kilogram of grain-bait formation," stating that more specific information about where and how much of each product is used is not available for the rodenticide compounds tested. The 2000 document, however, contains a significant amount of information and recommendations regarding use that have been deleted in the 2002 document.

(9) secondary risk to birds can be reduced by limiting the availability of the more highly toxic, persistent second-generation anticoagulents (e.g., brodifacoum, bromadiolone, difethialone) to certified applicators only. Zinc phosphide, chlorophacinone and diphacinone products for field use in orchards, range land and elsewhere must be applied by certified applicators, but products of all 9 rodenticides registered for rat and mouse control in and around buildings are available to anyone "over the counter." We believe that persons not trained or experienced in rodent control may be significantly more likely to intentionally or inadvertently misuse rodenticides.

(10) primary risk to birds can be reduced by making bait inaccessible to birds; for example, by applying bait in adequately designed bait stations. Current rodenticide labels require that bait for commensal rats and mice be placed in bait stations or areas inaccessible to non-target mammals, which should reduce primary exposure to birds. However, misuse may occur due to intentional or unintentional failure to comply with directions and restrictions. Further, this does nothing to address the continued problems of secondary poisoning.

(11) none of the scientists reviewing the 2000 document criticized that draft's comments and recommendations concerning reducing risk by altering use practices to minimize exposure. Yet this language has been entirely deleted in the 2002 document, and ingredient concentration in the

product is used as a proxy for exposure. This is not justified in light of the reviewers' comments, and the lack of attention to use as a factor in exposure also hampers the conclusions of the 2002 document. We request the agency please explain the deletion of the need to alter practices.

(12) a weakness pointed out by the peer-reviewers and addressed in the 2002 document is that missing data and other uncertainties about toxicity limit the predictive capabilities of the assessment. According to the 2002 document, data that would contribute to a better assessment of risk includes: chronic, secondary, sublethal and reproductive hazards, retention times in liver and blood, usage information, and differences between modes of action of the various types. We concur with this assessment; however, the need for such hazard data to improve decision-making should not outweigh the need to include considerations of exposure, particularly with respect to use. We are particularly concerned that the assessment fails to account for 1) the inability to enforce label guidelines on use, potentially leading to improper outdoor uses that increase exposure risk; and 2) the possibility that if one or more popular but high-risk compounds is restricted, the market for other compounds of equal hazard might expand, thus increasing potential exposure and therefore risk.

(13) the 2000 risk assessment document was much more straightforward in concluding that the risks of several posed by several of the rodenticides warrant measures to limit exposure:

Based on a "weight-of-the-evidence" approach and data evaluation by means of a decision table, the Agency concludes that there are major differences in the potential risks of these compounds.

The three rodenticides posing the highest primary risk to birds are brodifacoum, difethialone and zinc phosphide. Because brodifacoum and difethialone also exhibit high potential secondary risk to birds, reducing exposure to these compounds is essential.

Based on data evaluation by means of a decision table, brodifacoum and difethialone were identified as the two rodenticides posing the greatest overall risk to birds and nontarget mammals. Reducing exposure of wildlife to these two compounds is of utmost importance.

The 2002 document concludes that *brodifacoum poses the greatest potential overall risk to birds and nontarget mammals, followed by zinc phosphide, difethalione and diphacinone*, but offers no recommendations regarding exposure. This may be a function of having defined exposure so narrowly, but it detracts from the usefulness of the risk assessment.

Problems with the Continued General Use Registration of these Rodenticides (or business as usual)

The system of rodenticide registration and use as it is today in the United States is set up for failure - large numbers of mortalities/morbidities of not only wildlife but also domestic animals and humans as well are occurring each year across the United States. Sales and usage of these rodenticides is not followed by EPA, people generally are not responsible or knowledgeable enough to use rodenticides properly, and what ends up happening is that rodenticides are too widely broadcast - throughout buildings, around building perimeters, and across urban, suburban, and rural landscapes, and an unacceptable level of humans and animals are exposed and numerous mortalities/morbidities follow. As it currently stands, the system is broken and is in desperate

need of repair. Some additional observations are as follows:

(1) EPA has very little in the way of sales and usage data for rodenticides, although they do have this data for practically all other pesticides.

(2) people are just too careless and uninformed with regard to rodenticide usages, using them much more frequently than they are necessary; it is clear to us that the application of rodenticides cannot be left in the hands of the general public.

(3) there are just too many mortality/morbidity incidents for humans, domestic animals, and wildlife species (there is too much misuse, careless bait applications, etc.)

(4) even PCOs/certified applicators are not using rodenticides in a safe and effective manner in many cases – they just hire someone to apply it who may be miles away from the licensed person when they apply it; this needs to be addressed.

(5) there is scant evidence suggesting that we even need all of these 2nd generation anti-coagulant rodenticides in the US; remember, these compounds were introduced largely to deal with rodent resistance to warfarin; where is the data on warfarin resistance in the US that warrants this high level of use of the 2nd generation anti-coagulants? We are not aware of any data that clearly shows a measurable benefit to making 2nd generation anti-coagulant rodenticides as broadly available as they are at present. With the growing adoption of integrated pest management, due in large part to the efforts of the EPA, pest managers for urban and rural areas can be trained on effective non- and least-toxic methods and practices that of rodent control.

(6) these rodenticides kill way too many target and non-target field rodents, which then allows way too many secondary poisonings of predatory/scavenging species; these rodents are exposed to a palatable 2nd generation anti-coagulant bait and would be expected to eat as much of this as they would if exposed to a palatable 1st generation anti-coagulant bait, thereby accumulating a "super-lethal" dose. Do we have data on this?

(7) there seems to be some notion that tamper-resistant bait stations are the panacea for dealing with the excessive wildlife mortality incidents. However, tamper-resistant bait stations are not the answer because they do not even begin to address the entire secondary poisoning issue. Just because a bait station is tamper-resistant does not mean that the massive amount of secondary poisoning will decrease by even one animal.

(8) the EPA assumes that minor label amendments will solve the animal exposure problem; the EPA needs to check out the literature from other continents and see that exposure levels are as high in countries where 2nd generation anti-coagulant rodenticides are labeled for indoor use only (e.g., UK); also, regarding label issues, how will the EPA enforce a label change when a product has been used in the US for so many years? This would be an exceedingly difficult thing to do and in fact is unlikely to happen, particularly in light of the fact that the EPA has no concrete dataof how much of the product is being sold and how much is being used and where it is being used.

(9) the EPA fails to enforce the requirement that registrants provide information on the significance of the widespread contamination and mortality/morbidity caused by their products nor do they require the registrants to pay for all of the monitoring and analytical work necessary to track this widespread contamination.

Another point that needs to be raised here is that, as part of this comment period, the EPA is requesting a lot of basic information on these rodenticides that they should already possess. We point out here that this information that EPA is requesting is, by and large, does not include information that it would request for any other group of pesticides. Therefore, the EPA should re-examine its regulatory program for rodenticides and explain why certain information, that is otherwise standard, is not requiredfor rodenticides. Examples of this are as follows:

* the EPA does not keep track of sales and usage data for rodenticides – these data should already be required of all manufacturers, sellers, and PCOs.
* the EPA does not keep track of, or assess the problems of, resistance to rodenticides. In fact, the only reason the 2nd generation anti-coagulant rodenticides are being used is supposed to be that there is widespread resistance to the 1st generation anti-coagulants such as warfarin; however, the fact that the EPA does not keep track of resistance has resulted in the widespread use of the 2nd generation anti-coagulants that we see today.
* the EPA is asking for examples of commensal rodent control programs in urban areas and IPM programs targeting any rodents –
* the EPA is asking for wholesale and retail prices of rodenticide baits – this information should already be required of all rodenticide sellers.
* the EPA is asking for importance of rodenticide baits in relation to non-chemical control methods
* the EPA is asking for detailed estimates of types of damage caused by rodents in the US and economic loss resulting from such damage

Rodenticide risks to wildlife species

Risks to Endangered Species

San Joaquin kit fox (*Vulpes macrotis mutica*) – the San Joaquin kit fox is listed as federally endangered, and there is a clear record of mortality of the San Joaquin kit fox in CA from anti-coagulant rodenticides. At present, the EIIS database contains records for 32 San Joaquin kit foxes, including 27 for brodifacoum, 2 each from bromadiolone and chlorophacinone, and one for diphacinone. The incidents and their trends resulting from brodifacoum poisoning are troubling.

These incidents have increased in number from 4 each in 1999, 2000, and 2001 to 14 in 2002. So far, there is one record from 2003, and it contained the highest level of brodifacoum ever seen in kit foxes (11 ppm in the liver). San Joaquin kit fox carcasses have been piling up in California, with approximately 40 foxes showing rodenticide poisoning (many already listed in the EIIS database) and a freezer full of dead foxes waiting for residue analysis that will likely show additional rodenticide poisoning. We should point out here that this residue analysis is contingent on generating funds to conduct it which are not available at present and therefore this data unfortunately may not be available for some time. In addition to high liver residues of brodifacoum as well as other rodenticides in the tissues of these animals, necropsies are revealing large amounts of free blood in abdominal cavities, meaning that the likely cause of death was rodenticide poisoning. It is uncommon for brodifacoum to be the sole rodenticide present – there are usually multiple rodenticide residues found upon analysis, including other 2nd generation anti-coagulants such as bromadiolone. We note here that at least 5 kit foxes have recently been found with residues of coumatetralyl, an anticoagulant rodenticide not even registered in the

United States! Finally, it is ironic to note here that the finding that San Joaquin kit foxes were susceptible to rodenticides was published 30 years ago by Schitoskey (1975), who reported that the San Joaquin kit fox was susceptible to both primary and secondary poisoning from rodenticides (sodium monofluoroacetate, strychnine, zinc phosphide) contained in poisoned kangaroo rats.

One further note on San Joaquin kit fox - we note that the USFWS Biological Opinion (1993) stated that the San Joaquin kit fox was a species for which brodifacoum "is not likely to jeopardize" their continued existence. Their reasonable and prudent alternatives/measures for the species was that incidental take can be minimized by requiring that outdoor applications be made in tamper-resistant bait boxes placed in areas not accessible to wildlife. We point out here that this assessment is egregiously erroneous in the sense that tamper-resistant bait boxes will have absolutely no effect on the probability of kit foxes dying from secondary poisoning. So, this needs to be taken into account if and when the EPA ever decides to take a closer look at this Biological Opinion.

Bald eagle (*Haliaeetus leucocephalus*) – bald eagles are federally listed as threatened in the contiguous lower 48 states, and there are two records of bald eagles killed by brodifacoum and one record of a bald eagle killed by warfarin in the EIIS database.

Spotted owl (*Strix occidentalis*) – spotted owls are federally listed as endangered, and there is at least one case of a spotted owl being killed by brodifacoum in the EIIS database.

San Joaquin antelope ground squirrel (*Ammospermophilus nelsoni*) – the San Joaquin antelope ground squirrel was an ESA Category I Candidate Species in 1995, but subsequently relegated to a Species of Concern in 1996. This species, endemic to the San Joaquin Valley, has also suffered poisoning from rodenticides – an unknown number of mortalities.

In 1993, the USFWS published a Biological Opinion "Effects of 16 Vertebrate Control Agents on Threatened and Endangered Species" dealing with the 1991 ESA Section 7 consultation with EPA. This Biological Opinion included jeopardy determinations for mammals, birds, and reptiles potentially exposed via primary or secondary exposure to 8 of the 9 rodenticides (the other one, difethiolone, was not registered for use until 1995). Unfortunately, the EPA chose to totally ignore this Biological Opinion, and as a result, numerous birds, non-target mammals, and other wildlife species, including endangered and threatened species, had to pay the price ever since. The fact that the taxpayers spend millions of dollars annually on the San Joaquin kit fox recovery as well as the recovery of many other T&E species seems to be lost on the EPA as they continue to allow the San Joaquin kit fox and other T&E species such as the bald eagle and spotted owl to perish from rodenticide poisoning each year.

Incident Data

The EPA's EIIS database reveals at least 358 wildlife mortality incidents in which one or more of the rodenticides was detected in birds or non-target mammals. This includes 255 incidents for brodifacoum alone, including 58 owls, 72 diurnal raptors, 18 corvids, 4 other birds, 48 wild canids, 5 wild felids, 10 other carnivores, 5 white-tailed deer, 33 rodents/lagomorphs, and 2 opossums. Other incident totals include bromadiolone (40), zinc phosphide (25), diphacinone (20), chlorophacinone (13), warfarin (4) and difethiolone (1), with none for bromethelin or

cholecalciferol.

The scope of the problem with wildlife mortalities due to rodenticides is readily apparent in the few states that actively monitor. In New York, Stone et al. (2003) reported on 80 incidents involving raptors exposed to anti-coagulant rodenticides, mainly brodifacoum (84%). Stone et al. (1999) previously reported on 55 incidents involving wildlife species exposed to anti-coagulant rodenticides in New York. Brodifacoum was implicated in 80% of the incidents. Secondary poisoning of raptors, mainly great-horned owls and red-tailed hawks, comprised 50% of all cases. Gray squirrels, raccoons, and white-tailed deer were the most frequently poisoned non-target mammals.

Of course, this is just the tip of the proverbial iceberg, as carcasses in the field last but a very short amount of time (usually a matter of just days) and there are very few individuals or local, state, or federal agencies actually looking for carcasses. Two states (New York and California) make up well over 90% of the records cited above, as the majority of states do not have monitoring efforts. However, even when there are monitoring efforts, in many instances, carcasses may not be detected. Further, death may be attributed to natural causes, as rodenticide-poisoned animals do not appear to be anything but natural. And many incidents that could be added to the database may simply go unreported for any number of reasons. Therefore, the large number of incidents that actually found their way into the EPA EIIS database provides substantial evidence of a much larger problem as a direct result of the present system of rodenticide use.

Risk to raptors

Rodenticides pose a substantial risk to both diurnal and nocturnal raptors, particularly from secondary poisoning. In addition to the extensive wildlife incident record, there are many published studies dealing with the impact of rodenticides on raptor species. Applications of brodifacoum in apple orchards resulted in the deaths of radio-marked screech-owls (*Otus asio*) (Hegdal and Colvin 1988). Mendenhall and Pank (1980) documented secondary poisoning of owls by anticoagulant rodenticides (36 barn owls - bromadiolone, brodifacoum, diphacinone were lethal, difenacoum was sublethal; 3 GHOWs and 1 NSWO fed diphacinone-killed mice – 3 of them died 7-14 days following exposure). Mendenhall and Pank (1980) make a good point that susceptibility to rodenticides can be exacerbated by stress, changes in diet, increased activity, and minor injuries (even if injury preceeds exposure by many days).

Newton et al. (1990) – rodenticides in barn owls from the UK

Berny et al. (1997) – bromadiolone was detected in livers of 15/16 dead Eurasian buzzards (*Buteo buteo*), 5/5 black kites (*Milvus migrans*), and 1/1 harrier examined.

Saucy et al. (2001) – reported deaths of numerous raptors (Eurasian buzzads, black kites) and carrion crows following a mechanical application of bromadiolone bait (150 ppm) to underground burrows for water vole control in Switzerland.

Sheffield (1997) conducted a thorough review of pesticide impacts on owls, and found that many of the papers in the published literature dealt with rodenticide impacts.

Townsend et al. (1981) assessed the secondary poisoning hazard of warfarin to tawny owls.

In New York, 50% and 77% of asymptotic red-tailed hawks and great-horned owls, respectively, tested positive for all rodenticides combined (Stone et al. 2003).

Mineau (unpublished data) assessed a random sample of red-tailed hawks and great-horned owls found dead from 1995-2001 in Ontario and Manitoba for rodenticides (using LCMS/MS) – found that 57% of RTHA (n=30) and 87% of GHOW (n=84) had rodenticide residues, and that more owls had two or more rodenticides more commonly than either 0 or 1; RTHA had 40% brodifacoum and 50% bromodiolone, GHOW had 75% brodifacoum and 67% bromadiolone, but others found included warfarin, diphacinone, chlorphacinone, and difethiolone.

Risk to other bird species

Rodenticides have been also been shown to pose substantial hazards to other birds species. Ramey and Sterner (1995) found that death due to zinc phosphide poisoning occurred in 18/26 (69%) of pheasants (*Phasianus colchicus*) exposed in 0.2 ha enclosures planted in alfalfa; sublethal effects were seen in some pheasants (ataxia, lethargy, hypoactivity – took 7 days for them to move normally again and 14 days to fully recover); 94% of the mortalities occurred within 24 hrs of bait application; none of the 26 Calif quail used in the study ended up dying from zinc phosphide exposure.

Risk to non-target mammalian species

Non-target mammalian species are common victims of both primary and secondary poisoning from rodenticides. There are a high number of mortality incidences due to rodenticides not only for wild mammals but also for domestic mammals (dogs, cats, farm animals, etc.). Mammalian carnivores seem to be the most common victim of rodenticide poisoning,

Saucy et al. (2001) reported the mortality of 38 wild mammals, mainly red foxes and weasels, and 18 cats and dogs, following mechanical application of bromadiolone bait (150 ppm) in underground burrows for control of water voles in Switzerland.

Alterio (1996) found secondary poisoning of stoats (*Mustela erminea*), feral ferrets (*Mustela furo*), and feral house cats (*Felis catus*) occurred following exposure to brodifacoum; Alterio and Moller (2000) secondary poisoning of stoats (*Mustela erminea*) in a South Island podocarp forest, New Zealand: implications for conservation.

Townsend et al. (1984) found that least weasels (*Mustela nivalis*) suffered secondary poisoning following exposure to warfarin.

McDonald et al. (1998) reported that residues of one or more anti-coagulant rodenticides were found in the livers of stoats (*Mustela erminea*) and weasels (*M. nivalis*); residues were found in 9 of 40 stoats (23%) and 3 out of 10 weasels (30%); most common rodenticides involved included the 2nd generation anti-coagulants brodifacoum and bromadiolone; concluded that weasels were victims of secondary poisoning on these estates through consumption of non-target species (rodenticides used widely away from buildings).

Evans and Ward (1967) found that nutria (*Myocastor coypus*) killed with anti-coagulant rodenticides were responsible for secondary poisoning of mink (*Mustela vison*) and dogs (Canis familiaris).

Littrell (1988) reported deaths of a raccoon and a mountain lion in northern CA resulting from diphacinone poisoning.

Shore et al. (1996) found that polecats (*Mustela putorius*) in the UK were frequent victims of secondary poisoning by 2nd generation anti-coagulant rodenticides by hunting around farm buildings and feeding on rodents mainly in winter. Residues were found in 7 of 24 livers (29%) and in 2 of 5 stomachs (40%); difenacoum was detected most frequently, but bromadiolone and brodifacoum were also detected; most carcasses were found along roadsides; results indicated that exposure of polecats to 2nd generation anti-coagulant rodenticides may be common.

Hill and Carpenter (1982) – found that Siberian ferrets consuming rodents killed by zinc phosphide learned to avoid eating the GI tracts of the rodents, thereby minimizing the toxicity; zinc phosphide has an emetic action, so after one incident, the ferrets learned to avoid; however, the ferrets suffered sublethal effects, including significant decreases (18-48%) in Hb, increases of 35-91% in serum iron, and elevated levels of serum globulin, cholesterol, and triglycerides; Hb/Fe, urea nitrogen/creatinine, and albumin/globulin ratios also were altered by the secondary poisoning; also, 19 of the 20 ferrets lost body mass.

Schitoskey (1975) – reported that the San Joaquin kit fox was susceptible to both primary and secondary poisoning from rodenticides (sodium monofluoroacetate, strychnine, zinc phosphide) contained in poisoned kangaroo rats.

Savarie et al. (1979) – orally dosed 10 wild coyotes with diphacinone (doses ranged from 0.31 – 5 mg a.i./mg); radiocollars were attached to the coyotes and they were released back into the wild; Seven of 10 (70%) coyotes died within 7 – 16 days, with an average time to death of 9.6 days.

Concerns regarding brodifacoum

Brodifacoum accounts for 30% of all rodenticide active ingredients.

(Conservation usage of brodifacoum)

Brodifacoum is such a highly hazardous chemical to animals and humans that we feel that its continued uses should be severely restricted. However, having said that, one use that we agree should continue to be allowed is for conservation purposes uses on islands. Introduced commensal rats (*Rattus* spp.) are a major contributor to the extinction and endangerment of island plants and animals. We believe that the use of the rodenticide brodifacoum to completely eradicate rats from islands is a powerful conservation tool. However, brodifacoum is highly toxic to animals other than rats and its continued use for this purpose should be tightly controlled. Further, on some islands, its use may not be feasible without prohibitively expensive mitigation.

Donlan et al. (2003) experimentally tested brodifacoum and two less toxic rodenticides, diphacinone and cholecalciferol, in eradicating *Rattus rattus* from three small islands in the northern Gulf of California, Mexico. All three rodenticides were successful in eradicating rats,

suggesting that the less toxic diphacinone and cholecalciferol may be useful alternatives to brodifacoum for some island eradication programs. However, the choice of rodenticide must be balanced between efficacy and the risks to non-target species. Applied field research is needed on less toxic rodenticides, as well as improving palatability of baits. This may prove invaluable in preventing extinctions and in restoring larger and more diverse island ecosystems (Donlan et al. (2003).

Howald et al. (1999) - Langara Island, at the northwestern tip of British Columbia's Queen Charlotte archipelago, was once nesting grounds for an estimated 500,000 seabirds. However, infestations of Norway rats (*Rattus norvegicus*) and their predation of eggs and breeding adults have caused extirpation or serious declines of all seabird species. By 1993, the breeding population of ancient murrelets (*Synthliboramphus antiquus*) had declined to 10% of its historical size. Successful eradication of rats on smaller New Zealand islands using the anticoagulant brodifacoum prompted its application on Langara Island. The island is also home to breeding bald eagles (*Haliaeetus leucocephalus*), peregrine falcons (*Falco peregrinus*), and other wildlife. In 1994 and 1995 they initiated a two-year study into the risk of secondary poisoning to non-target species. During 1994, rat carcasses were laid out with motion sensor cameras to identify potential scavengers. Ravens, northwestern crows and bald eagles were photographed at carcasses, and therefore at risk of feeding on rats that die above-ground. During the baiting program, 19 rats were radio-tagged to determine the proportion dying above-ground, and thus available to predators/ scavengers. Ravens were found poisoned both from feeding directly on the bait, and predating/scavenging poisoned rats. Bald eagles were trapped and blood sampled for brodifacoum residue analysis and prothrombin time evaluation; 15% of the sampled population showed detectable residues but no adversely affected birds were found. They concluded that the use of brodifacoum for rat removal on seabird islands poses a clear risk of secondary poisoning to avian scavengers, which must be weighed against the benefit of rat removal programs.

Eason et al. (1999) - the field use of brodifacoum baits to control brushtail possums (*Trichosurus vulpecula*) has increased in recent years. This has raised concerns of secondary and tertiary poisoning, resulting from the transfer of this toxicant through the food chain. In New Zealand, feral pigs (*Sus scrofa*) are known to scavenge possum carcasses and may also gain access to bait stations containing possum baits. We have determined the concentrations of brodifacoum in muscle and liver tissue from captive pigs after primary and secondary poisoning. Highest concentrations were found in the liver. Pigs eating 500 to 1776 g of brodifacoum bait containing 20 mg/kg had muscle concentrations ranging from 0.02 to 0.07 mg/kg and liver concentrations ranging from 0.72 to 1.38 mg/kg. Both appeared to be independent of the amount of bait eaten. Possums fed 400 g of bait had similar liver concentrations (0.52-1.20 mg/kg). Pigs that had eaten the soft tissue from eight poisoned possums had brodifacoum concentrations of 0.32 to 0.80 mg/kg present in the liver and the concentration increased in a dose-dependent manner. Brodifacoum was detected in muscle from only one of these animals. In a preliminary field survey, 11 of 21 wild pigs sampled from areas where possum control had been undertaken were contaminated with brodifacoum concentrations in the liver ranging from 0.007 to 1.7 mg/kg. In view of the potential impact on pig hunters and dogs consuming wild pig meat and offal, some restrictions on the wide-scale field use of brodifacoum baits appears to be warranted.

Eason et al. (2002) - the risks to non-target birds and other wildlife from the use of vertebrate pesticides, including anticoagulant rodenticides, are determined to a significant extent by species' intrinsic susceptibility, and the toxicokinetics of the compounds used. Brodifacoum is highly toxic to birds and mammals. The acute toxicity of brodifacoum to birds in New Zealand varies

from <1 mg/kg in pukeko (*Porphyrio p. melanotus*), the native swamp hen, to >20 mg/kg in the paradise shelduck (*Tadorna variegata*). Like other second-generation anticoagulants, brodifacoum is strongly bound to vitamin K epoxide reductase and will persist, apparently for at least 6 months, in organs and tissue containing this enzyme (e.g., liver, kidney, and pancreas). The unique toxicokinetics of this class of compound exacerbates the risk of primary and secondary poisoning of non-target species. Vertebrate pest control programmes in New Zealand using bait containing brodifacoum have resulted in the primary and secondary poisoning and sublethal contamination of non-target species. These include native raptors, such as the Australasian harrier (*Circus approximans*) and morepork (*Ninox novaeseelandiae*), other native birds such as the pukeko, weka (*Gallirallus australis*), southern black-backed gull (*Larus dominicanus*), and kiwi (*Apteryx* spp.), and introduced mammals, including game animals. There are increasing numbers of reports worldwide of wildlife contamination and toxicosis after the use of second-generation anticoagulants.

MBTA and BGEPA implications

We need to add in here some language on the implications of re-registering these nine rodenticides in light of the Migratory Bird Treaty Act (MBTA) and Bald and Golden Eagle Protection Act (BGEPA). Specifically, these nine rodenticides are KNOWN to kill migratory birds and both bald and golden eagles, and the MBTA and BGEPA are both STRICT LIABILITY STATUTES.

Currently, there are 11 golden eagles and 2 bald eagles that were poisoned by brodifacoum included in the EIIS database (and 11 golden eagles and 3 bald eagles total for all nine rodenticides).

There are also 152 migratory birds that were poisoned by brodifacoum included in the EIIS database (and 207 total for all nine rodenticides)

Rodenticide Risks to Domestic Animals

The Animal Poison Control Center (APCC) in Urbana, IL reports 2,334 cases (2,685 individual animals) of domestic animal poisoning with rodenticides (brodifacoum – 1,161; bromadiolone – 511; zinc phosphide – 218; diphacinone – 206; bromethalin – 66; difethialone – 48; warfarin – 48; chlorophacinone – 42; cholecalciferol - 34) between November 2001 and June 2003 (US EPA unpubl). Most of these cases involved domestic dogs. The number of incidents has been increasing steadily since the 1st 3 years of the APCC (1978-1981), when only 4.4% of the incidents were related to anti-coagulant rodenticides. By 1982, percentage had almost doubled to 8% of the incidents, and in 1984, it had doubled once again to 17% of the incidents, ranking anti-coagulant rodenticides as the number one cause of animal poisoning incidents.

Table 1. Ounces of Rodenticide Bait LD50s for Pets.

Rodenticide	Dog 10 lbs.	Dog 22 lbs.	Dog 30 lbs.	Cat 4.4 lbs.
Warfarin	13	28	38	8
Bromadiolone	35	77	105	35
Diphacinone	3	6	8	7
Chlorophacinone	160	353	481	-
Cholecaliferol	19	42	57	-
Bromethalin	8	16	22	1

Zinc phosphide	0.16	0.35	0.48	0.06

From: 1998. Rodecticide Risk to Dogs and Cats. *Techletter: For Pest Control Technicians.* Vol. 4, no. 23.

Of note is a case in which a female dog gave birth to litter of puppies where two of them died neonatally from brodifacoum poisoning from placental exposure; the mother had no symptoms and no known recent exposure to rodenticides (Munday and Thompson 2003). This gives a clear indication of the scope of the problem with brodifacoum.

In most cases, domestic animals are dying following a single exposure. Boermans et al. (1991) gavaged six horses with a commercial brodifacoum-containing bait (Talon) at a dosage of 0.125 mg brodifacoum/kg BW. The horses showed weight loss, severe hypocoagglability and hemogram alterations. The data indicate that a single exposure of horses to brodifacoum has the potential of causing clinical illness and possibly death.[which data??]

Numerous mortalities have also been reported from captive animals in zoological parks. Borst and Counotte (2002) found that 2nd generation anticoagulant rodenticides can give rise to unexpected casualties in nontarget species in zoos. The first two offspring of a pair of turkey vultures (*Cathartes aura*) died of brodifacoum toxicosis. The adult birds fed rodenticide-killed mice to their offspring. There are previous case reports of small carnivorous birds (*Dacelo novae-guinae* and *Tockus deckeni*) killed eating poisoned (difenacoum and brodifacoum) mice. Even a granivorous species (*Rollulus roulroul*) died, probably by contamination of its food by cockroaches that transported the rodenticide. In addition, there have been numerous records of captive animals dying from rodenticide poisoning at the National Zoo in Washington, DC.

Rodenticide Risks to Humans

Perhaps the most distressing portion of the EPAs push for re-registration of these nine rodenticides is the fact that in excess of 20,000 people, mainly children ages 5 and under, are suffering exposure and effects from these rodenticides in the United States each year (Litovitz et al. 1999). And of these cases, 30-40% of them are requiring either a visit to a physician or a hospital (or both). Anti-coagulant rodenticides are responsible for a vast majority (>90%) of these cases. Data from 2002, 1998 and 1995 from the American Association of Poison Control Centers (AAPCC) can be compared as follows:

Year	# exposures (total)	# exposures (< 6yrs)	treated in health care facility	deaths
2002	18,144	16,000	5,476	3
1998	17,724	15,854	5,882	1
1995	14,710	13,167	5,479	1

Data from the AAPCC indicates that the number of exposures (total and those <6 yrs of age) is actually increasing over the past 9 years and also since the time EPA issued the rodenticide RED in 1998. And, when the data for these 9 years are summed, the total number of people exposed to rodenticides was >150,000 (150,132), the number of children <6 years of age was 133,685, the total number of cases serious enough to require medical treatment was 48,837, and the number of deaths was 17. These data are indicative of the scope of the problem at hand. The situation at

present requires immediate action on the part of the EPA to address this serious problem. Therefore, we call on the EPA to immediately return to their 1998 policy that recognized that rodenticides are an unreasonable health risk in violation of FIFRA and not approve the re-registration of these rodenticides unless manufacturers include two safety measures to protect children: a dye that would make it more obvious when a child had ingested a rodenticide, and a taste aversion ingredient that would discourage children from ingesting rodenticides. The 2001 EPA decision to revoke these safety regulations was incorrect and must be rectified immediately.

Other Aspects that Need to be Considered

(1) Rodenticide sales and usage data

Directly related to the comments herein, we want to take this opportunity to point out that there is a serious paucity of both sales and usage data for rodenticides in the United States. As an example, the most recent EPA pesticides sales and usage report (Kiely et al. 2004) does not include rodenticides as a separate category and lumps them in with "other". Both sales and usage data for rodenticides is exceedingly difficult to find in both the US and Canada and it is imperative that the EPA begin to address this by requiring both manufacturers as well as retailers to keep records of their sales. And need we remind you that EPA is the regulating agency responsible for administering FIFRA and has the regulatory power in which to require sales and use data from registrants. The EPA routinely requires sales and use data for insecticides, herbicides, and fungicides (among other pesticides), so now is the time to include rodenticides to this list. Following the necessary restriction of these rodenticides by EPA, professional pesticide applicators should keep close track of rodenticide usage and both sales and usage should be reported in BEAD's (EPA) annual report.

(2) Alternatives to rodenticides

We feel strongly that it is not enough to restrict the use of these nine rodenticides, but the EPA must insist on alternative uses to rodenticides whenever and wherever feasible, especially the use of non-chemical alternatives when it comes to rodent control, of which there are many that have been proven effective. The EPA should be reminded that its role in advocating integrated pest management that educates people on exclusion, rodent-proofing, habitat modification, proper storage and containment, and other methods, is undone and contradicted with the continued registration of hazardous rodenticides.

For purposes of rodent control that involves public health, we agree with the criteria outlined by Frantz (2004) for selecting rodenticides for use in an IPM program for rodents, including the following:

(1) the rodenticide should be the least toxic product that will be effective on the targeted species, and,

(2) the rodenticide must have a highly efficacious and readily available antidote that typically can be administered in time to save an accidentally intoxicated human or animal.

(3) Consultations with the "Services"

The "Update to the Overview of the Rodenticide Comparative Ecological Assessment" dated 9 Sept 2004 states that the EPA is working with the USFWS and NMFS to further evaluate the potential effects of rodenticides on endangered species. Given the recent EPA Counterpart regulations, we wonder if this is really happening and, if so, what the timetable is for this?

We are aware of the formal consultation conducted with the USFWS in 1991 under Section 7 of the ESA, and of the USFWS's Biological Opinion issued in 1993 titled "Effects of 16 Vertebrate Control Agents on Threatened and Endangered Species." This Biological Opinion included jeopardy determinations for mammals, birds, and reptiles potentially exposed via primary or secondary exposure to 8 of the 9 rodenticides (the other one, difethiolone, was not registered for use until 1995).

We strongly believe that it is imperative for EPA to reinitiate consultation with the Services to supplement and re-evaluate the 1993 Biological Opinion for the very reasons given in the "Update", including that the original consultation did not include difethiolone, additional species have been listed since 1991, and many carcasses of endangered and threatened species have been found to contain one or more of the rodenticide residues.

We feel that the EPA should move immediately to implement the 1993 Biological Opinion as an interim measure in advance of re-consultation with the Services.

Conclusions

The current rodenticide cluster RED as presented succeeds in some areas and lacks inothers. Collectively, these nine rodenticides present a serious level of environmental contamination with consequences that are largely unknown or understood. We therefore request that the EPA rework their RED for each of the nine rodenticide chemicals so that it finally restricts all current usages of the nine rodenticides. Continued usage for all nine rodenticides should be limited to professional pest control operators (PCOs) only and concomitantly, EPA needs to require all PCOs and all of their hired applicators to undergo a training program specifically for rodenticide use to minimize environmental hazards. Additionally, we request that the EPA cancel uses for rodenticides that involve field rodents (not commensals) that are not public health-related, that the EPA actively promote non-chemical alternatives to rodenticides, and that the EPA go back to, and enforce, their 1998 regulations that would insist on industry adding the two safety measures to all rodenticides (dye, taste aversion agent). Thank you for the opportunity to provide comments on this document and on this very important issue in general.

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Other sign-ons

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