University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

USDA National Wildlife Research Center - Staff Publications

U.S. Department of Agriculture: Animal and Plant Health Inspection Service

2013

The effects of vitamin K1-rich plant foods on the efficacy of the anticoagulant rodenticides chlorophacinone and diphacinone, used against Montane Voles (*Microtus montanus*)

Gary W. Witmer
USDA-APHIS-Wildlife Services, gary.w.witmer@usda.gov

Nathan P. Snow
USDA/APHIS/WS National Wildlife Research Center, nathan.p.snow@aphis.usda.gov

Rachael S. Moulton
USDA/APHIS, Wildlife Services

Follow this and additional works at: https://digitalcommons.unl.edu/icwdm_usdanwrc

Part of the Life Sciences Commons

Taylor & Francis
Taylor & Francis Group

The effects of vitamin K1-rich plant foods on the efficacy of the anticoagulant rodenticides chlorophacinone and diphacinone, used against Montane Voles (*Microtus montanus*)

Gary W. Witmer*, Nathan P. Snow and Rachael S. Moulton

USDA/APHIS, Wildlife Services, National Wildlife Research Center, 4101 Laporte Avenue, Fort Collins, Colorado 80521-2154, USA

Voles can cause significant losses to agriculture and wood fibre production. California growers typically rely on baits containing chlorophacinone and diphacinone to reduce vole population densities, but the efficacy of those rodenticides has been decreasing. One hypothesis suggests that voles are consuming high levels of an antidote (vitamin K1) to the anticoagulants, contained within green leafy plants. We tested that hypothesis by first feeding Montane Voles (*Microtus montanus*) diets that were high in vitamin K1, and then providing the animals with either: (1) chlorophacinone-containing bait, (2) diphacinone-containing bait, or (3) a control diet. We found that the chlorophacinone-containing bait remained efficacious (100% mortality), whereas the diphacinone-containing bait had a much lower efficacy (60% mortality). When only the diphacinone-containing bait was presented, the efficacy was somewhat better (80%). We infer that a diet rich in vitamin K1 did not negate the effects of the chlorophacinone for voles, and so we recommend its continued use in California unless anticoagulant resistance is known to have developed in the vole population. We hypothesise that: (1) diphacinone has a relatively low efficacy against Montane Voles when compared to chlorophacinone, and (2) this lower efficacy could be further reduced by a vitamin K1-rich diet.

Keywords: anticoagulants; California; *Microtus*; rodenticide; vitamin K; wildlife damage

1. Introduction

Numerous species of microtine voles (subfamily *Microtinae*) occur throughout the northern hemisphere. Several of them are serious pests to agriculture and wood fibre production when they achieve high population densities (Nowak 1999). In North America, many of the pest species belong to the genus *Microtus*, commonly called voles or meadow mice (Clark 1984; Edge et al. 1995). Specifically, California Vole (*M. californicus* Peale) and Montane Vole (*M. montanus* Peale) cause significant agricultural damage in California (Clark 1994). California is the foremost agricultural state in the United States, generating nearly $39 billion worth of produce annually (Shwiff et al. 2009), yet in that state damage to crops from rodents (including voles) and birds consistently results in annual losses of jobs (2,106–6,317) and revenue ($168–504 million). Voles have been reported to cause significant amounts of damage to pastures and rangelands, orchards and nurseries, and a wide variety of field crops including alfalfa, grains such as oats and wheat, clover, potatoes, sugar beet, artichokes, carrots, Brussels sprouts, cauliflower, and tomatoes (Clark 1984; Hines 1993, 1997; O'Brien 1994; Hygnstrom et al. 1996, 2000; Hines and Hygnstrom 2000). In addition, most species of voles exhibit marked population cycling whereby they reach extremely high densities (> 1,000/acre) every 3–5 years (Ford and Pitelka 1984; Hornfeldt et al. 1986; Krebs 1996; Stenseth 1999; Ylonen et al. 2003). Severe damage to agriculture and forestry resources commonly occurs during the peak of the cycle (Witmer and VerCauteren

2001; Witmer et al. 2007). Therefore, many growers rely on baits containing the anticoagulants chlorophacinone and diphacinone to maintain low density populations of voles, and subsequently reduce the damage to crops (O'Brien 1994). This can be achieved in two ways: (1) routine use of rodenticides, or (2) rodent population monitoring, followed by rodenticide application when a notable increase in the populations occurs. Recently, however, observations have shown a reduction in the efficacy of those rodenticides in California (Salmon and Lawrence 2006a), thereby resulting in increased levels of damage.

One hypothesis put forward to explain the reduced efficacy of anticoagulant rodenticides in California is that voles are counteracting anticoagulants by inadvertently ingesting elevated levels of vitamin K along with their daily diet. Once ingested, vitamin K is physiologically processed for the production of blood clotting proteins, which gives vitamin K (in particular, Vitamin K1 and K2; Tasheva 1995) the ability to counteract anticoagulant compounds (e.g., Mackintosh et al. 1988; Tasheva 1995). Anticoagulant rodenticides irreversibly inhibit the enzyme vitamin K epoxide reductase that is necessary for post-translational carboxylation of the prothrombin group of serine protease coagulation factors; without the carboxyl group these factors do not assemble on cell surfaces to form active coagulation complexes (Rattner et al. 2011). There are several chemical formulations of vitamin K, all of which are required in metabolic activity in the liver where blood-clotting proteins are synthesized (Robbins 1993; Tasheva 1995). For example, vitamin K1

(phylloquinone or phytomenadione) is produced by green plants, and is ingested by most herbivores and omnivores through their daily diet (Robbins 1993). Vitamin K2 (menaquinones) is produced in the digestive system by microorganisms, but is thought to provide relatively little of the vitamin K needs of vertebrates (Booth and Suttie 1998). Vitamin K3 (menadione) is a synthetic chemical compound, but liver enzymes convert it to menaquinone; small amounts of vitamin K3 are commonly added to live-stock feed (Tasheva 1995). Vitamin K3 is not used as an antidote to anticoagulant poisoning (Tasheva 1995). The vitamin K requirements of vertebrates are met by: (i) their dietary intake of green plant materials together with microbial synthesis in the digestive system; (ii) "recycling" of vitamin K in the liver (Hadler and Buckle 1992; Robbins 1993). Vertebrates require a constant sup-ply of vitamin K. However, there is a limit to the amount to vitamin K an animal can utilize, and the excess will typically be excreted (Harrington et al. 2007).

The United States Department of Agriculture (USDA) provides a listing of the vitamin K content for a large num-ber of foods on the following website: (www.nal.usda.gov/fnic/foodcomp/Data/SR21/nutrlist/sr21w430.pdf). Vitamin K1-rich plants are readily available to voles in California and, in many cases; vitamin K1-rich plants are the particu-lar crop that growers are attempting to protect by using anticoagulant rodenticides. Some examples of vitamin K1-rich plants that are cultivated in California and are readily consumed by voles are collards (*Brassica Oleracae* L.), spinach (*Spinacea oleracea* L.), and sugar beet greens (*Beta vulgaris* L.; Clark 1994) among others. In addition, other non-cultivated plants (e.g., the common dandelion [*Taraxacum officinale* Weber]) also contains high amounts of vitamin K1 and have been introduced throughout the world (Gleason 1952), including California. The voles would normally encounter common dandelions away from agriculture fields. To our knowledge no studies have been aimed at assessing whether the efficacy of anticoagulant rodenticides can be reduced by vitamin K1-rich diets for voles, although this has been investigated in some other rodent species (Chaudhary et al. 2004; Witmer and Burke 2009).

There have been conflicting findings regarding whether diets enhanced with vitamin K can reduce the efficacy of anticoagulant rodenticides for rodents other than voles (e.g., Chaudhary et al. 2004; Witmer and Burke 2009). However, in these studies (which are dis-cussed below), the variations in the toxicants (i.e., active ingredients) and the species tested could account for the inconsistent findings. Witmer and Burke (2009) found that feeding green plants containing high levels of vitamin K1 (i.e., collard greens [0.62 mg vitamin K1 per 100 g food] and Brussels sprouts [0.19 mg/100 g]) to Norway Rat (*Rattus norvegicus* Berkenhout), Black Rat (*R. rattus* L.), and House Mouse (*Mus musculus* L.) did not seem to reduce the efficacy of brodifacoum or diphacinone antico-agulant rodenticides for any of the species. On the con-trary, Chaudhary et al. (2004), in a study with Indian Gerbil (*Tatera indica* Hardwicke), found a high dose of

vitamin K1 (i.e., 2 mg/kg body weight for 15 days via coated food provided after one day of rodenticide bait exposure) reversed the effects of difethialone, an antico-agulant rodenticide, whereas a smaller dose (i.e., 1 mg/kg body weight) did not. Furthermore, several researchers have noted that some feeds for commercial livestock are supplemented with vitamin K (Partridge 1980; MacNicoll and Gill 1993), and they have suggested that in livestock production facilities the availability of these vitamin K-enriched feeds to rodents may lessen the effectiveness of anticoagulant rodenticides.

We tested whether, in controlled laboratory trials, a diet rich in vitamin K1 could reduce the efficacy of two commonly used first-generation anticoagulant rodenti-cides (i.e., 0.005% chlorphacinone and 0.005% diphaci-none) for montane voles (*M. montanus*). The United States Environmental Protection Agency (EPA) standard for the desired level of efficacy of rodenticides in a two-choice laboratory trial is 90% mortality (Schneider 1982). The EPA standard was used as our guideline for acceptable efficacy in our trials. To make the study realis-tic for conditions in California, we used vitamin K1-rich plants that were commonly cultivated in that state as the maintenance food, and alternative two-choice diet. We predicted that both anticoagulant rodenticides would have lower than acceptable efficacy when fed to voles that were maintained on diets rich with vitamin K1.

2. Material and methods

We live-trapped 40 wild, montane voles from areas that we confirmed anticoagulant rodenticides were likely never used in the past. Thus, our results were unlikely to be confounded by voles that had developed genetic resistance to anticoagulant rodenticides (Salmon and Lawrence 2006a).

2.1. Pre-study feeding trial

Voles were maintained in individual cages in an animal research room. Each cage contained corn cob floor cover-ing, a den tube, and burlap squares for bedding. The room was maintained at 21 °C, 40% relative humidity, and a 12 h/12 h light–dark cycle. The voles varied in mass from 20 to 36 g. Voles were held in quarantine for 2 weeks before trials began. If a female vole had a litter, the young were euthanized before the female was used in a trial. Animals were maintained and used in compliance with the requirements of the United States' Animal Welfare Act. We conducted a pre-study feeding trial to identify which vitamin K1-rich plants were highly palatable to our voles. For two days, three groups of three voles were each fed either collard greens (0.62 mg of vitamin K1 per 100 g food), spinach leaves (0.54 mg/100 g), or beet greens (0.48 mg/100 g). We measured how much of each food item was consumed each day (i.e., percentage con-sumed), adjusted for water loss from evaporation. This water loss adjustment was determined by weighing plant material maintained in cages without voles at the start and

end of each trial. We conducted analysis of variance (ANOVA) (Proc GLM, SAS Institute, Cary, North Carolina) procedures to compare the average percentage of plant material consumed. When the pre-study feeding trial was completed, we used the vitamin K1-rich plants that were most preferred by voles in the subsequent trials. We surmised it would be best to offer more than one type of vitamin K1-rich plant in case of any individual variability in taste-preference. Because collard greens and spinach leaves had the highest vitamin K1 content, we offered a mixture of those two plants for the vitamin K1-rich efficacy trial.

2.2. Vitamin K1-rich plant food trial

We randomly assigned 3 groups of 10 wild-captured voles (5 males and 5 females) to a 0.005% chlorophacinone rodenticide (Rozol pellets, LiphaTech, Inc., Milwaukee, Wisconsin), a 0.005% diphacinone rodenticide (Ramik Brown nuggets, Hacco, Inc., Randolph, Wisconsin), or control (rodent chow: Formulab 5008, PMI Nutrition International, Inc., Brentwood, Missouri) treatments. To ensure the voles in each treatment group were similar, we conducted an ANOVA to compare the average weights among the groups. We fed all voles *ad libitum* a mixture of rodent chow and the preferred vitamin K1-rich plants for a period of 10 d, pre-treatment. The rodent chow did contain a small amount of vitamin K3 (menadione; 0.32 mg vitamin K3 per 100 g food), but we surmised it was not enough to confound our results. On day 11, we replaced the rodent chow with the respective rodenticide bait (except for the control group) for 10 additional days. We recorded the amount of rodenticide bait that had been consumed by each vole over the 10-d period. After the 10-d exposure period, we returned all voles to the pre-treatment diet for 10 more days. Thereby, voles received the vitamin K-rich plants throughout the entire 30-d trial (i.e., including pre-exposure, rodenticide exposure, and post-exposure periods). We monitored voles for mortality throughout the trial, checking each animal twice per day. All voles that died or were euthanized at the end of the trial were dissected and examined for signs of haemorrhaging that would indicate anticoagulant poisoning (Stone et al. 1999).

2.3. No-choice rodenticide trial

If a rodenticide had less than 80% efficacy in the previous trial, we tested its efficacy in another trial, where voles were given no choice of foods, that is, just the rodenticide bait (no vitamin K-rich plant foods or rodent chow). We randomly assigned groups of 10 voles (mixed sexes with 4–6 individuals of each sex) to the rodenticide. For a period of 10 d pre-treatment, the voles were fed a maintenance diet, *ad libitum*, that had low vitamin K content. The diet consisted of the rodent chow and vegetables or fruit very low in vitamin K: carrots (0.01 mg vitamin K per 100 g food), potatoes (0.01 mg/100 g), and apples (0.002 mg/100 g). On day 11, we replaced the maintenance diet with the appropriate rodenticide for 10 d. We recorded the amount of rodenticide bait that had been consumed by the voles. After the exposure period, we returned the voles to the maintenance diet, and monitored them for 10 more days. We monitored voles for mortality throughout the trial, checking each animal twice per day. As with the previous trial, all voles that died or were euthanized at the end of the trial were dissected and examined for signs of haemorrhaging that would indicate anticoagulant poisoning (Stone et al. 1999).

2.4. Comparing vitamin K1-rich plant and no-choice rodenticide trials

We compared the weights of voles between the two trial-types by ANOVA. We also compared the average amounts of rodenticide bait that were consumed, respectively, between the trials. We compared the efficacy (i.e., percentage mortality) between the trials for each respective rodenticide using Fisher's Exact test (Proc Freq). Finally, using Student's *t*-test, we compared the amount of diphacinone rodenticide bait consumed by voles that lived versus that consumed by voles that died.

3. Results

3.1. Pre-study feeding trial

We found that voles consumed similar amounts of all three types of vitamin K1-rich plants that they were offered ($F_{14} = 0.19$, $P = 0.825$). On average, they consumed 74% of all plant material (adjusted for water evaporation losses) they were offered, generally leaving the stem of the leaves untouched.

3.2. Vitamin K1-rich plant food trial

We observed that every vole in the trial consumed all, or nearly all, of the vitamin K1-rich plants it was offered during the study. We found that the average weights of voles were similar among the two treatment and control groups ($F_{27} = 3.00$, $P = 0.066$). Regardless of the vitamin K1-rich diet, the efficacy of chlorophacinone was high (100% mortality), whereas the efficacy of diphacinone (60% mortality) was lower than the acceptable EPA standard (Table 1). Interestingly, the voles in the diphacinone treatment group that lived consumed significantly more ($t = 2.72$, $P = 0.0238$) of the diphacinone bait (mean = 24.6 g) than the voles in that group that died (14.3 g). This result may reflect our observation that rodents feeding on anticoagulant baits tended to slow or stop feeding as they became sick and eventually died. The voles that lived presumably continued to feed on the rodenticide bait for the entire 10 d of exposure. The numbers of voles showing signs of internal hemorrhaging in the trials is presented in section 3.4 below. No control animals died during this phase of the study.

Table 1. Percentage mortality, bait consumption, and days until death for four groups of voles to test the effects of ingesting high amounts of vitamin K-rich food on the efficacy of two anticoagulant rodenticides.

Treatment group	*n*	Deaths	Percent mortality	Bait consumption		Days until death		Evidence of haemorrhaging
				Mean (g)	SE	Mean	SE	
Vitamin K								
Chlorophacinone	10	10	100	15.6	3.4	7.9	0.8	Yes
Diphacinone	10	6	60	18.2	2.5	8.2	0.8	Yes
Control (rodent chow)	10	0	0	N/A	N/A	N/A	N/A	No
No-choice (no vitamin K)								
Diphacinone	10	8	80	24.3	4.2	9.8	1.9	Yes

SE = standard error; N/A = not applicable.

3.3. No-choice rodenticide trial

Because the efficacy of the diphacinone rodenticide bait when presented with the vitamin K1-rich plant food (60% mortality) was below the 90% efficacy standard of the EPA, we conducted the no-choice diphacinone rodenticide bait trial for that bait only. The efficacy of the diphacinone rodenticide bait in the no-choice trial was 80%. This was somewhat higher (80% versus 60% mortality) than when the rodenticide bait was presented with the vitamin K1-rich plant foods, but still lower than the acceptable EPA standard of 90% (Table 1). Consistent with the two-choice diphacinone trial, the voles given the no-choice diphacinone treatment that lived consumed more rodenticide bait (mean = 37.6 g) rodenticide bait than the voles that died (18.4 g); however, in this case, the difference was not significant ($t = 2.47$, $P = 0.0690$), probably because of the wide variation in the amount consumed by the larger number of voles that died (range = 9.1–32.7 g).

3.4. Comparing vitamin K1-rich plant and no-choice rodenticide trials

Overall, we found the average weights between voles in the vitamin K1-rich plant food trials and the no-choice rodenticide trials to be similar ($F_{38} = 1.38$, $P = 0.247$). Also, the voles ate similar amounts of the rodenticide bait in the chlorophacinone trial ($F_{13} = 3.71$, $P = 0.076$) and both diphacinone trials ($F_{13} = 1.73$, $P = 0.211$). We found that the efficacy did not differ between the vitamin K1-rich plant food and no-choice rodenticide trial for the diphacinone rodenticide bait ($\chi_1^2 = 0.95$, $P = 0.588$). We were able to find obvious signs of internal and/or external haemorrhaging in approximately 80% of all voles found dead during the trials. Interestingly, we found only one vole that survived until it was euthanized at the end of the study (i.e., from the no-choice, diphacinone rodenticide group) that showed obvious signs of being affected by the rodenticide (e.g., internal haemorrhaging), but did not die from the poisoning. All other voles that survived did not show obvious signs of being affected.

4. Discussion

We determined in the vitamin K1-rich plant food trial that the efficacy of chlorophacinone rodenticide was not reduced by feeding voles a diet of vitamin K1-rich plants. This result was similar to those obtained for commensal rodents that were fed anticoagulant rodenticides (i.e., diphacinone and brodifacoum) along with vitamin K1-rich plants (Witmer and Burke 2009). The result was also similar to that obtained with low levels of vitamin K1 given to Indian Gerbils (Chaudhary et al. 2004). All of these findings would suggest that growers do not need to be concerned about the potential of reduced efficacy when applying chlorophacinone to, or near, fields where they grow vitamin K1-rich crops. In general, very high doses of vitamin K1 or K2 given over lengthy periods of time (usually through intravenous administration) are required to counteract the adverse effects of poisoning by anticoagulants (Tasheva 1995).

The use of chlorophacinone in numerous field studies has demonstrated its effectiveness for various species of voles (Byers and Young 1975; Byers, 1978, 1979, 1981; Hunter et al. 1987; Bryson 2004). Because we did not find evidence to suggest vitamin K1-rich plant food reduced the efficacy of chlorophacinone in this study, we infer that chlorophacinone is effective for controlling voles, particularly where populations are not genetically resistant to anticoagulants (Salmon and Lawrence 2006a).

Compared to the EPA standard of 90% efficacy for cage efficacy trials, the efficacy of the diphacinone rodenticide was lower than acceptable in both the vitamin K1-rich plant food and no-choice rodenticide trials. Hence, we suspect that diphacinone may be less effective than chlorophacinone regardless of vitamin K1-rich diets for voles. Efficacy was somewhat lower (60% versus 80%) in the vitamin K1-rich plant food trial, possibly suggesting that a diet containing high levels of vitamin K1-rich foods might reduce somewhat the efficacy of diphacinone for controlling vole populations. However, this difference does not support the conclusion with any certainty in this preliminary study. Our results were similar to those which have reported that diphacinone is only marginally effective for control of vole populations

(Byers 1978, 1979). In fact, one study showed that diphacinone rodenticide was only as effective as chlorophacinone rodenticide for two species of voles after a 10-fold increase in the diphacinone concentration (Byers and Carbaugh 1987).

Overall, we conclude that voles are unlikely to ingest enough vitamin K1-rich plants to counteract the effects of the anticoagulant rodenticide chlorophacinone; thus, we propose chlorophacinone as a useful tool for protecting crops from vole damage in California. Because there has been some evidence of resistance to chlorophacinone for voles in California (Salmon and Lawrence 2006a), we suggest also using another rodenticide in combination (e.g., zinc phosphide; see Salmon and Lawrence 2006a, 2006b) as the most effective method of controlling vole populations. We found slight evidence to suggest that voles may be able to ingest enough vitamin K1-rich plant foods to counteract the effects of the anticoagulant rodenticide diphacinone; however, additional studies would be needed to confirm this. Combining that finding with the fact that diphacinone was only marginally efficacious for the montane voles in our trials; we recommend that managers use alternative rodenticides to diphacinone (e.g., chlorophacinone, zinc phosphide) for voles wherever possible. We caution that our results with montane voles may not translate to other voles species; additional trials would need to be done before that would be known. The continued effective management of vole populations and crop and forest damage may require the development of additional rodenticide products (Eason et al. 2010).

Acknowledgements

References

Booth SL, Suttie JW. 1998. Dietary intake and adequacy of Vitamin K1. J Nutr. 128:785–788.

Bryson DT. 2004. Montane vole control with Rozol® paraffinized pellets in orchards of the Pacific Northwest. Proc Verteb Pest Conf. 21:145–149.

Byers RE. 1978. Performance of rodenticides for control of pine voles in orchards. J Am Soc Hortic Sci. 103:65–69.

Byers RE. 1979. Meadow vole control using anticoagulant baits. HortScience. 14:44–45.

Byers RE. 1981. Pine vole control with anticoagulant baits in apple orchards. J Am Soc Hortic Sci. 106:101–105.

Byers RE, Carbaugh DH. 1987. Efficacy of diphacinone for control of orchard voles. HortScieice. 22:46–48.

Byers RE, Young RS. 1975. Pine vole control with anticoagulant baits. J Am Soc Hortic Sci. 100:691–694.

Chaudhary V, Tripathi R, Poonia FS. 2004. Effect of supplementary feeding of vitamin K on difethialone treated Indian gerbils in the laboratory. Indian J Exper Biol. 42:297–302.

Clark JP. 1984. Vole control in field crops. Proc Verteb Pest Conf. 11:5–6.

Clark JP. 1994. Vertebrate pest control handbook, 4th ed. Sacramento (CA): Div Plant Industry, California Department of Food and Agriculture. p. 350.

Eason CT, Fagerstone KA, Eisemann JD, Humphrys S, O'Hare JR, Lapidge SJ. 2010. A review of existing and potential New World and Australasian vertebrate pesticides with a rationale for linking use patterns to registration requirements. Int J Pest Manage. 56:109–125.

Edge WD, Wolff JO, Carey RL. 1995. Density-dependent responses of gray-tailed voles to mowing. J Wildl Manage. 59:245–251.

Ford RG, Pitelka FA. 1984. Resource limitation in populations of the California vole. Ecology. 65:122–136.

Gleason H. 1952. Illustrated flora of the Northeastern United States and adjacent Canada. Lancaster (PA): Lancaster Press.

Hadler MR, Buckle AP. 1992. Forty-five years of anticoagulant rodenticides – past, present, and future trends. Proc Verteb Pest Conf. 15:149–155.

Harrington DJ, Booth SL, Card DJ, Shearer MJ. 2007. Excretion of Vitamin K by young adults responds to changes in dietary phylloquinone and dihydrophylloquinone intakes. J Nutr. 137:1763–1768.

Hines RA. 1993. Prairie vole damage control in no-till corn and soybeans. Proc Great Plains Wildl Damage Control Workshop. 11:134–147.

Hines RA. 1997. Rodent damage control in no-till corn and soybean production. Proc Eastern Wildl Damage Manage Conf. 7:195–201.

Hines RA, Hygnstrom SE. 2000. Rodent damage control. In: Reader R, editor. Conservation tillage systems and management. Ames (Iowa): Midwest Plan Service, Iowa State University. p. 167–176.

Hornfeldt B, Lofgren O, Carlsson BG. 1986. Cycles in voles and small game in relation to variations in plant production indices in northern Sweden. Oecologia. 68:496–502.

Hunter RE, Askham LR, Godfrey MER. 1987. The effectiveness of five rodenticides and three application procedures in controlling Microtine voles (*Microtus montanus*) in Washington State apple orchards. Crop Prot. 6:277–279.

Hygnstrom SE, VerCauteren K, Ekstein J. 1996. Impacts of field-dwelling rodents on emerging field corn. Proc Verteb Pest Conf. 17:148–150.

Hygnstrom SE, VerCauteren KC, Hines RA, Mansfield C. 2000. Efficacy of in-furrow zinc phosphide pellets for controlling rodent damage in no-till corn. Int Biodeterior Biodegrad. 45:215–222.

Krebs CJ. 1996. Population cycles revisited. J Mammal. 77:8–24.

Mackintosh CD, Laas FJ, Godfrey MER, Turner K. 1988. Vitamin K1 treatment of brodifacoum poisoning in dogs. Proc Verteb Pest Conf. 13: 86–90.

MacNicoll AD, Gill JE. 1993. Vitamin K3 in feedstuffs: antidotal effects in captive anticoagulant-resistant rats and mice. J Wildl Manage. 57:835–841.

Nowak R. 1999. Voles, or meadow mice. In: Nowak R, editor. Walker's mammals of the world. 6th ed, vol. II Baltimore (MD): Johns Hopkins University Press. p. 1470–1474.

O'Brien J. 1994. Voles. In: Hygnstrom S, Timm R, Larsen G, editors. Prevention and control of wildlife damage. Lincoln (NE): Cooperative Extension Division, University of Nebraska. p. B–177–B-182.

Partridge GG. 1980. Vitamin K deficiency syndromes in warfarin-resistant brown rats (*Rattus norvegicus*). Lab Anim. 14:193–195.

Rattner BA, Horak KE, Warner SE, Day DD, Meteyer CU, Volker SF, Eisemann JE, Johnston JJ. 2011. Acute toxicity, histopathology, and coagulopathy in American kestrels (Falco sparverius) following administration of the rodenticide diphacinone. Environ Toxicol Chem. 30:1213–1222.

Robbins CT. 1993. Vitamins. In: Cunha TJ, editor. Wildlife nutrition and feeding. 2nd ed. New York: Academic Press. p. 81–109.

Salmon TP, Lawrence SJ. 2006a. Anticoagulant resistance in meadow voles (*Microtus californicus*). Proc Verteb Pest Conf. 22:156–160.

Salmon TP, Lawrence SJ. 2006b. Zinc-phosphide treated bracts as an alternative rodenticide in artichoke fields for meadow vole (*Microtus californicus*) control. Proc Verteb Pest Conf. 22:161–165.

Schneider BA. 1982. Pesticide assessment guidelines – Subdivision G: product performance. Washington (DC): US Environmental Protection Agency. p. 422.

Shwiff SA, Gebhardt K, Kirkpatrick KN. 2009. The economic impact of bird and rodent damage to California crops. USDA APHIS Wildlife Services National Wildlife Research Center Report. p. 35.

Stenseth NC. 1999. Population cycles in voles and lemmings: density dependence and phase dependence in a stochastic world. Oikos. 87:427–461.

Stone WB, Okoniewski JC, Stedelin JR. 1999. Poisoning of wildlife with anticoagulant rodenticides in New York. J Wildl Dis. 35:187–193.

Tasheva M. 1995. Anticoagulant rodenticides. Environmental Health Criteria 175. Geneva (Switzerland): World Health Organization.

Witmer GW, Burke PW. 2009. Influence of vitamin K-rich plant foods on anticoagulant baiting efficacy in wild House Mice, Norway Rats, and wild Black Rats. Pac Conserv Biol. 15:87–91.

Witmer GW, VerCauteren KC. 2001. Understanding vole problems in direct seeding – strategies for management. In: Veseth R, editor. Proc of the Northwest Direct Seed Cropping Systems Conference. Pasco (WA): Northwest Direct Seed Conference. p. 104–110.

Witmer GW, Sayler RD, Huggins D, Capelli J. 2007. Ecology and management of rodents in no-till agriculture in Washington, USA. Integr Zool. 2:154–164.

Ylonen H, Eccard JA, Sundell J. 2003. Boreal vole cycles and vole life histories. In: Singleton GR, Hinds LA, Krebs CJ, Spratt DM, editors. Rats, mice, and people: rodent biology and management. ACIAR Monograph No. 96. Canberra (Australia): Australian Centre for International Agricultural Research. p. 137–142.