Literature Review on Residues of Anticoagulant Rodenticides in Non-Target Animals

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Preface

Anticoagulant rodenticides are the principal means of controlling pest rodents in the Nordic countries. Due to the intrinsic properties of second generation anticoagulants, i.e. extremely slow elimination from the body and high toxicity, they are prone to accumulate in the non-target species which consume poisoned rodents. Despite wide use there are no published studies on occurrence of residues of anticoagulant rodenticides in the non-target animals in the Nordic countries. This review of publicly available studies was aimed to find out which anticoagulant substances are found and in which species. The concentrations are reported as well as the proportion of exposed animals. We have further compiled a list of species that could potentially be exposed to anticoagulant rodenticides in the Nordic countries. The review shows that anticoagulant residues have been found everywhere they have been measured and secondary exposure to second generation anticoagulants is common among certain avian and mammalian predators. The results call for initiation of measurements of anticoagulant rodenticides also in the Nordic countries.

Information on residues is important for the consideration of appropriate risk mitigation measures for the second generation anticoagulants. Several risk mitigation measures are already applied, such as restriction to indoor use or restriction to professional use only. The information on residue levels and the extent of exposure would help to judge whether the current risk mitigation measures are sufficiently effective or whether further measures or even restrictions should be considered.

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Summary

Residues of second generation anticoagulant rodenticides in wildlife have been found all around the world where the occurrence of residues has been investigated. Second generation anticoagulants, brodifacoum, bromadiolone, difenacoum, difethialone and flocoumafen, have been gradually introduced after the appearance of resistance to warfarin and other first generation substances. They are much more toxic and in particular more persistent in rodents and hence potent in accumulating in non-target species that feed on target and non-target rodents. Despite wide use of anticoagulants, there are relatively few studies where anticoagulant residues have been measured in non-target animals. More systematic incident studies have been done only in Britain and in some states of the US. In addition to Britain, published data on anticoagulant residues is available only in France in Europe. In Britain anticoagulant residues have been studied in particular in barn owl (*Tyto alba*) and in polecats (*Mustela putorius*). About one third of studied carcasses contained residues. In most cases, the residues were expected to be sublethal. No studies on the effects of the sublethal residues have been found. In the UK incident program other commonly exposed species were buzzard (*Buteo buteo*), red kite (*Milvus milvus*), and fox (*Vulpes vulpes*). In the US the commonly exposed species were great horned owls (*Bubo virginianus*), red-tailed hawks (*Buteo jamaicensis*), coyotes, foxes and raccoons. Anticoagulants are the dominant rodenticides in the Nordic countries, but no information is available on residues in non-target species. Information on residues is needed in order to judge whether the currently used risk mitigation measures are effective and whether further measures or restrictions should be considered.

1 Introduction

Use of anticoagulant rodenticides is the dominating way to control undesired rodent species. The effectiveness of anticoagulants is due to the delayed mode of action which prevents rodents to connect the poisoning symptoms to the bait they have fed a few days ago. This is important in the control of rats which are cautious and avoid food which makes them ill. Anticoagulant rodenticides were introduced for the control of harmful rodents, but unfortunately non-target species are affected too, either directly through consumption of poisoned baits or indirectly through consumption of contaminated prey animals (secondary poisoning) (Lambert *et al*. 2007). The indirect poisoning, i.e. secondary poisoning threatens birds and mammals that feed on living or dead rodents. Secondary poisoning is most commonly associated with the second generation anticoagulants (Berny *et al.* 1997; Shore *et al.* 2003; Stone *et al.* 2003; Fournier-Chambrillon *et al.* 2004).

The aim of this literature review is to find out which substances tend to accumulate in wildlife and which species are most likely exposed to anticoagulant rodenticides, and further serve as a preliminary study for the survey of anticoagulant residues in non-target species in the Nordic countries.

2 Second generation anticoagulant rodenticides

2.1 General information

First generation anticoagulants were introduced as rodenticides in the late 1940's. The appearance of resistance to warfarin and other first generation substances led to the development of more potent, second generation anticoagulants (IPCS Environmental Health Criteria 175, Anticoagulant Rodenticides). The first generation anticoagulants, e.g. warfarin, sodium warfarin, chlorophacinone and coumatetralyl, are effective first after several repeated ingestions. The newer, second generation anticoagulants bromadiolone, brodifacoum, difenacoum, difethialone and flocoumafen may cause an effect already after a single ingestion and are toxic at a much lower dose than the first generation anticoagulants (Berny 2007).

Active substances used in rodenticides have been included in the review program of existing biocidal active substances (Directive 98/8/EC, Commission Regulation No 1451/2007). Nine anticoagulant rodenticides are included in the review programme and most of them are on the market in the Nordic countries (Table 1). Denmark, Finland, Norway and Sweden have been the Rapporteur Member States for coumatetralyl, difenacoum, difethialone and bromadiolone, respectively. Anticoagulants are the dominant active substances used for rodent control and in Finland they are the only rodenticides on the market. The summaries of the risk assessments (assessment reports) for anticoagulants are available on the Internet, and can be accessed from the EU Commission's web site on biocides or from the CIRCA database:

http://circa.europa.eu/Public/irc/env/bio_reports/library?l=/assessement_directive&vm=detailed&sb=Title (Ref. 21.10.2009).

In the risk assessment all second generation anticoagulants were identified as potential PBT substances, i.e. substances which are persistent, bioaccumulating and toxic. Such substances are problematic when released to the environment because they will stay there for a long time and could accumulate in animals. According to the Technical Notes for Guidance (TNsG) on Annex I inclusion, substances fulfilling the PBT criteria shall not be included in Annex I unless releases to the environment can be effectively prevented.

Annex I inclusion has been proposed for the evaluated anticoagulants under the directive 98/8/EC, in spite of the identified PBT properties and the risk for primary and secondary poisoning of non-target animals. Annex I inclusion has been suggested because anticoagulant rodenticides are

regarded as necessary for public health reasons and so far equally useful, effective and less hazardous alternative substances do not exist. The main target organisms in Europe are brown rat (*Rattus norvegicus*), black rat (*Rattus rattus*), and house mouse (*Mus musculus* or *Mus domesticus*). These species have been identified as target species in the review programme. In the UK the grey squirrel (*Sciurus carolinensis*) is also controlled with anticoagulants (McDonald *et al.* 1998). Anticoagulant rodenticides are also used against voles (*Microtus sp*. and *Arvicola sp.*) in agriculture (Directive 91/414/EEC).

Table 1. Anticoagulant rodenticides and the Rapporteur Member States (RMS) in the EU review programme of existing biocidal active substances. Anticoagulants used in the Nordic countries are given according to Lodal and Hansen (2002).

Anticoagulant	CAS No	RMS	Denmark	Finland	Iceland	Norway	Sweden
Warfarin	81-81-2	IE				x	x
Warfarin sodium	129-06-6	IE					x
Coumatetralyl	5836-29-3	DK	x	x		x	x
Chlorophacinone	3691-35-8	ES			x		
Difenacoum	56073-07-5	FI	x	x	x	x	x
Bromadiolone	28772-56-7	SE	x	x	x	x	x
Brodifacoum	56073-10-0	IT	x	x		x	x
Flocoumafen	90035-08-8	NL	x	x		x	x
Difethialone	104653-34-1	NO	x	x		x	

The rodenticides fumarin, diphacinone and pindone, which are also mentioned in this report, are not included in the EU review programme. Only substances in the review programme and accepted in Annex I of the Directive 98/8/EC can be legally used in the EU/EEA.

2.2 Mode of action, symptoms and toxicity

Anticoagulant rodenticides have a common mode of action. These substances are vitamin K antagonists. They inhibit the vitamin K-dependent steps in the coagulation cascade in the liver, disrupting normal blood-clotting mechanisms and causing death by haemorrhage. The first generation substances are not sufficiently toxic to rodents to cause death after a single feeding, and repeated exposure is needed. Second generation products have a greater affinity to binding sites in the vertebrate liver and consequently display greater accumulation and persistence in the body (Parmar *et al*. 1987, in Eason *et al*. 2002). Elimination half-lives of 118–220 days from the livers of rats have been reported for brodifacoum, bromadiolone, difenacoum and flocoumafen (Erickson & Urban 2004). The superiority of anticoagulants compared to acute rodent poisons previously used is the delayed mode of action, rendering the target rodents unable to connect the poisoning symptoms to the bait.

The symptoms appear 3–4 days after ingestion of the bait (Lodal & Hansen 2002). In general death results from haemorrhage 4 to 10 days after

the intake of the bait. Anticoagulant poisoning is expressed as severe haemorrhage with massive bleeding and poor coagulation. Bleeding may be observed externally but is usually internal. Signs associated with blood loss such as anemia, pale mucous membranes, weakness, hypothermia and tachycardia, can be observed. Evidence of massive bleeding is noted at necropsy and the lack of coagulation indicates exposure to anticoagulant rodenticides. Confirmation of poisoning is obtained from analysis of blood or liver samples for the presence of coumarines (Berny 2007).

Published investigations have shown that many mustelids have detectable residues of anticoagulants in their liver, although death has been attributed by some other cause, e.g. trauma or infectious disease (Shore *et al.* 2003; Fournier-Chambrillon *et al.* 2004). Exposure to low levels of anticoagulants may have behavioural or pathological effects, and in most cases exposed animals are weak, have slower reactions and movements and are more susceptible to accidents, predation or infection (Fournier-Chambrillon *et al.* 2004). Repeated sub-lethal exposures, even intermittent ones, may be expected to eventually cause fatal haemorrhage.

The LD50 value is the dose of a substance which kills 50% of the test animals. LD50 values are used to describe the acute toxicity of a substance. An overview of the LD50 values of second generation anticoagulants for target and non-target animals are given in Table 2. Howald *et al.* (1999) noted that assuming an LD50 for a raven or crow of 0.56 mg brodifacoum/kg body weight and a total-body burden (meaning whole-carcass anticoagulant residues) in a rat of 1.4 mg, one poisoned rat could contain 2 to 3 times the LD50 dose for a raven or crow. A bald eagle would need to eat about 3.2 rats to obtain an LD50 dose. Rats which had consumed on average brodifacoum bait at 10.7 g (range 7.3–13.9 g), containing 0.54 mg of brodifacoum (a lethal dose), died 4–6 days after poisoning (Hooker & Innes 1995).

Liver concentrations of 1.0 to 1.9 mg/kg brodifacoum have commonly been found in rats. Concentrations between 2 to 5 mg/kg occur with a lower frequency, but even concentrations of over 7 mg/kg of brodifacoum in rat liver have been detected (Eason *et al.* 1999). Repeated dose studies in rats with flocoumafen have demonstrated that concentrations in the liver of rats can reach 3–5 mg/kg when lethal anticoagulant effects occur in most animals (Huckle *et al.* 1988).

Table 2. LD50 values of second generation anticoagulants to target rodents and some non-target animals.

Common name	Scientific name	Anticoagulant	LD50 mg/kg	Reference
Pig	*Sus domestica*	Brodifacoum	0.1	Eason & Spurr 1995
Rat	*Rattus norvegicus*	Brodifacoum	0.24–0.39	Eason et al. 2002; Stone et al. 1999; Erickson & Urban 2004
Dog	*Canis lupus familiaris*	Brodifacoum	0.25–3.5	Eason & Spurr 1995; Erickson & Urban 2004
Mouse	*Mus musculus*	Brodifacoum	0.4	Erickson & Urban 2004 EU risk assessment[1]
Laboratory rat	*Rattus norvegicus*	Brodifacoum	0.42–0.56	Erickson & Urban 2004 EU risk assessment[1]
Raven	*Corvus corax*	Brodifacoum	0.56	Howald et al. 1999
Mink	*Mustela lutreola*	Brodifacoum	9.2	Erickson & Urban 2004
Australasian harrier	*Circus approximan*	Brodifacoum	10	Eason & Spurr 1995
Cat	*Felis catus*	Brodifacoum	25	Stone et al. 1999; Erickson & Urban 2004
Rat	*Rattus norvegicus*	Bromadiolone	0.55–1.25	Stone et al. 1999; Eason et al. 2002; Newton et al. 1997
Laboratory rat	*Rattus norvegicus*	Bromadiolone	0.56–0.84	Erickson & Urban 2004 EU risk assessment[1]
Mouse	*Mus musculus*	Bromadiolone	0.99–1.75	Stone et al. 1999; Newton et al. 1997
Laboratory mouse	*Mus musculus*	Bromadiolone	1.75	Erickson & Urban 2004
Dog	*Canis lupus familiaris*	Bromadiolone	8.1–15	Stone et al. 1999; Erickson & Urban 2004
Cat	*Felis catus*	Bromadiolone	>25	Stone et al. 1999; Erickson & Urban 2004
Rat	*Rattus norvegicus*	Difenacoum	0.29–1.8	Eason et al. 2002; Erickson & Urban 2004
Mouse	*Mus musculus*	Difenacoum	0.47	Erickson & Urban 2004
Laboratory rat	*Rattus norvegicus*	Difenacoum	0.55	Erickson & Urban 2004
Laboratory mouse	*Mus musculus*	Difenacoum	1.29	Erickson & Urban 2004
Rat	*Rattus norvegicus*	Difenacoum	1.8–2.6	EU risk assessment[1]
Pig	*Sus domestica*	Difenacoum	2–3	Erickson & Urban 2004
Dog	*Canis lupus familiaris*	Difenacoum	4–11.8	Erickson & Urban 2004
Cat	*Felis catus*	Difenacoum	>16	Erickson & Urban 2004
Rat	*Rattus norvegicus*	Difethialone	0.4–0.8	EU risk assessment[1]
Dog	*Canis lupus familiaris*	Difethialone	11.81	EU risk assessment[1]
Rat	*Rattus norvegicus*	Flocoumafen	0.13–0.5	EU risk assessment[1]
Rat	*Rattus norvegicus*	Flocoumafen	0.25	Eason et al. 2002
Mouse	*Mus musculus*	Flocoumafen	1.13	Newton et al. 1997

[1]http://circa.europa.eu/Public/irc/env/bio_reports/library?l=/assessement_directive&vm=detailed&sb=Title

2.3 Accumulation and elimination

The greater potency of second generation anticoagulants compared to first generation anticoagulants is related to accumulation and slow elimination from the body after absorption. The anticoagulants accumulate in the liver which is the target organ for their action. Eason *et al.* (2002) present a following synthesis of the literature on the relative retention of anticoagulants in the liver after sub-lethal exposure:

- *low potency anticoagulants,* warfarin and pindone: 0.5–1.0 month
- *moderate potency anticoagulants,* coumatetralyl, chlorophacinone and diphacinone: 3–6 months
- *high potency anticoagulants,* difenacoum, bromadiolone, flocoumafen, brodifacoum and difethialone: 6–12 months.

Pindone and diphacinone are not legal in the EU/EEA. A summary of body half-lives and retention times of some second generation anticoagulants in the livers of different animals are given in Table 3.

Brodifacoum has a very long body half-life (150–200 days). It is extremely persistent in liver and, to a lesser extent, in muscle tissue (Erickson & Urban 2004; Eason *et al.* 1999). Elimination from liver is slow and biphasic with a very prolonged terminal phase. After administration of brodifacoum and bromadiolone to rats in a single oral dose of 0.2 mg/kg the half-lives in liver were 63 days for brodifacoum and 17 days for bromadiolone in the first elimination phase (28 days), and 282 and 318 days in the terminal phase, respectively. Mean liver concentrations of brodifacoum were significantly higher compared to bromadiolone throughout the study (Hawkins *et al.* 1991, in Erickson & Urban 2004). In the study by Batten and Bratt (1987, in Erickson & Urban 2004), 21 to 34% of brodifacoum in livers of rats was still detected after 13 weeks from dosing, and more than 11% after 104 weeks. Rats also displayed signs of internal haemorrhage.

Murphy *et al.* (1998a) detected brodifacoum in the livers of 17 of 25 sampled rats at Waipapa, New Zealand. The proportion of rats which contained poison residues was 75% during the poison campaign. Three months after removal of baits still 61% of the rats captured contained poison residues. During the poisoning operation, the mean residue level of brodifacoum in poisoned rats was 0.87 mg/kg. The mean concentration of brodifacoum in rats captured in the post-poisoning period was 0.17 mg/kg, and the last rat containing poison residue (0.18 mg/kg) was captured 13 weeks after all baits were removed from the bait stations.

Sub-lethal doses of brodifacoum can persist in the liver of sheep for over 16 weeks (Eason & Spurr 1995). Bromadiolone was detected for 256 days in the liver of sheep that received a sub-lethal dose of 2 mg/kg (Erickson & Urban 2004). Substantial amounts of brodifacoum were found in the livers of sub-lethally dosed possums (*Trichosurus vulpecula*) eight months after exposure, with little decline after the first week (Eason *et al.* 1996).

Bromadiolone persists very long in the liver, up to 270 days (Giraudoux *et al.* 2006). Erickson & Urban (2004) have reported half-lives in the liver between 170–318 days. In livers of rats fed for 1 day with feed containing 50 mg/kg bromadiolone, concentrations were 2.08 mg/g after 1 day and 0.6 mg/g after 3 days. Even after a single oral exposure of 0.2 mg/kg bromadiolone, liver concentrations of 0.3 mg/g were detected after

200 days (Fournier-Chambrillon *et al.* 2004). In the study of Giraudoux *et al.* (2006), total residues in field voles (*Microtus agrestis*) exposed to baits containing 150 mg/kg bromadiolone reached 1.22 mg/kg on average and almost all of the animals caught had detectable bromadiolone residues in all tissues. Concentrations were at a maximum from 3.3 to 6.5 days after treatment for water voles (*Arvicola terrestris*), and after 1.3–3.7 days for common voles (*Microtus arvalis*). Predators may have access to a large proportion of contaminated voles, each of them containing on average 93.5 µg (including guts) or 57.6 µg (without guts) of bromadiolone. After 135 days, eight out of the ten water voles and one of the two common voles still had detectable residues. This indicates that although the risk of secondary poisoning is at a maximum during the first 15–20 days of the control campaign when the rodent densities remain high, exposure conditions are maintained at least for 135 days.

The half-life of flocoumafen in the liver is estimated to be between 100 and 220 days (Huckle *et al.* 1989; Newton *et al.* 1994). Sub-lethal doses of flocoumafen can accumulate and persist in rats for 14 weeks (Eason & Spurr 1995).

Many rat and mice populations have developed resistance against the first generation anticoagulants. This was also the reason for the introduction of the second generation substances. Resistance against the second generation anticoagulants is not very common, but is observed in some areas. Resistant rodents are assumed to pose a greater risk to predators than susceptible rats because they have higher body burdens and are expected to be available for predation for a longer time compared to non-resistant rats, which die faster (Atterby *et al.* 2005). Atterby *et al.* (2005) described a feeding test with difenacoum at 25 mg/kg for 5, 10, or 20 days in anticoagulant-resistant or susceptible rats. Whole-carcass difenacoum residues reached values around 0.6 mg/kg, resistant rats having higher residues than the susceptible ones (0.679 mg/kg compared to 0.519 mg/kg after five days).

Table 3. Half-lives and elimination times of anticoagulants in the livers of target and non-target animals.

Common name	Scientific name	Anticoagulant	Dose mg/kg	Elimination time, days	t½ days	Reference
Rat	*Rattus norvegicus*	Brodifacoum	0.35		130	Erickson & Urban 2004
Rat	*Rattus norvegicus*	Brodifacoum	0.25		150–200	Erickson & Urban 2004
Rat	*Rattus norvegicus*	Brodifacoum	0.2		282–350	Erickson & Urban 2004, EU risk assessment[1]
Rat	*Rattus norvegicus*	Bromadiolone	0.93		170	Erickson & Urban 2004
Rat	*Rattus norvegicus*	Bromadiolone	0.2		318	Erickson & Urban 2004, EU risk assessment[1]
Rat	*Rattus norvegicus*	Difenacoum			108–120	Eason et al. 2002
Rat	*Rattus norvegicus*	Difenacoum			118	EU risk assessment[1]
Rat	*Rattus norvegicus*	Difethialone	0.5		126	Erickson & Urban 2004, EU risk assessment[1]
Rat	*Rattus norvegicus*	Flocoumafen			215	EU risk assessment[1]
Rat	*Rattus norvegicus*	Flocoumafen			220	Eason et al. 2002
Barn owl	*Tyto alba*	Flocoumafen		>100		Eason et al. 2002
Dog	*Canis lupus familiaris*	Flocoumafen		>300		Eason et al. 2002

[1]http://circa.europa.eu/Public/irc/env/bio_reports/library?l=/assessement_directive&vm=detailed&sb=Title

Giraudoux *et al.* (2006) calculated daily intakes of bromadiolone for different animal species feeding on water voles (*Arvicola terrestris*), assuming that voles contain on average 93.5 mg (including guts) or 57.6 mg (without guts) of bromadiolone, as mentioned above. Adult red foxes (*Vulpes vulpes*) eat between 0.3 and 0.6 kg of food daily. This means that if they feed on water voles exclusively (which is realistic during high-density peaks) they may eat between four and eight individual voles and thus ingest 364–728 mg of bromadiolone per day on average. This corresponds to a daily acute dose of 60–121 mg of bromadiolone/kg bw/day.

Stoats (*Mustela erminea*) usually discard the digestive tract, but not the liver, when they feed on small mammals. Stoats weigh 180–330 grams (depending on sex), and their daily intake ranges between 0.37 and 0.62 g/g. This means that the food consumption may range between 67 and 204 g/day, which corresponds to between one and more than three individual water voles/day. Thus, the bromadiolone daily intake would range between 58–173 mg. A minimum value of bromadiolone (58 mg) could lead to an acute dose of 174–320 mg/kg, according to stoat body weights.

Common buzzards (*Buteo buteo*) weigh between 0.5–1.35 kg depending on the sex. Daily food intake varies between 40–170 grams, pointing to an average intake ranging between 48.8 and 207.4 mg of bromadiolone. The average minimum dose (large animals eating a low vole num-

ber) would reach 36 mg/kg but may be closer to 153 mg/kg. No data on LD50 are available on birds of prey for bromadiolone, but a daily dose of 279 mg/kg/day of difenacoum has been shown sufficient to kill a barn owl (*Tyto alba*, Gray *et al.* 1994).

Eason and Murphy (2001) emphasize that the risk of anticoagulant rodenticides is magnified by their persistence, which could lead to accumulation in cases of repeated exposure. A compound that is rapidly metabolized or excreted from a primary consumer may result in a lesser risk than one that bioaccumulates with repeated sub-lethal exposure, even if repeated exposure occurs weeks or even months after the initial exposure.

2.4 Secondary exposure of non-target species

2.4.1 Laboratory studies on secondary poisoning

The target rodents form a variable proportion of the diet of many predatory and scavenging species such as the weasel (*Mustela nivalis*), barn owl (*Tyto alba*), long-eared owl (*Asio otus*), short-eared owl (*Asio flammeus*) and tawny owl (*Strix aluco*). Generalists, such as the red fox, polecat (*Mustela putorius*), buzzard and red kite (*Milvus milvus*), rely less on small mammals and change their feeding habits depending on available prey (Shore *et al.* 1999; Berny *et al.* 2007). Kestrels, stoats and weasels are specialist predators of non-target small mammals, e.g. woodmouse (*Apodemus sylvaticus*), bank vole (*Clethrionomys glareolus*) and field vole (*Microtus agrestis*), which have shown to feed on rodenticide from bait boxes during rat control campaigns (Brakes & Smith 2005).

Anticoagulants are toxic to all vertebrates. Invertebrates are less susceptible as their blood clotting systems differ from that of vertebrates (Shirer 1992, in Eason & Spurr 1995), but it has been suggested that invertebrates have a potential to carry poison to vertebrates (Stephenson *et al.* 1999, in Eason *et al.* 2002). The potential exposure routes could be the consumption of rodent faeces, the consumption of rodent carcasses, ingestion of soil-bound residues by e.g. earthworms, and direct consumption of poison baits (Dowding *et al.* 2010). Shrews are insectivores and are not expected to feed on grain-based bait, yet several shrews were found dead with dyed faeces in traps (Brakes & Smith 2005). (Baits are dyed as birds are assumed to avoid coloured baits (Moran 2001) and dyes can also reveal anticoagulant poisoning of non-target animals.) Residues of bait have been found in shrews, indicating that shrews may have eaten grain bait opportunistically or they may have consumed contaminated invertebrates or carrion. Also insectivorous birds have been found dead after eating ants and cockroaches that have fed on brodifacoum baits (Brakes & Smith 2005). Dowding *et al.* (2010) have recently shown that 66.7% of 120 analysed

individuals of the European hedgehog (*Erinaceus europaeus*) contained residues of first and second generation anticoagulants.

Mendenhall and Pank (1980) compared secondary hazards of 3 second generation and 3 first generation anticoagulants to barn owls. Six owls (per rodenticide) were exposed to rats fed with brodifacoum (20 ppm bait), bromadiolone (50 ppm bait) or difenacoum (50 ppm bait) and two owls (per rodenticide) were exposed to rats fed with diphacinone (50 ppm bait), chlorophacinone (50 ppm bait) or fumarin (250 ppm bait). Six of the 18 owls (33%) exposed to second generation anticoagulants died, whereas none of the 6 owls offered first-generation anticoagulant-poisoned rats exhibited any signs of intoxication. Brodifacoum-fed rats accounted for 5 of the 6 owl deaths. The other mortality occurred in 1 of 2 owls exposed to bromadiolone-fed rats for 10 days. Sub-lethal haemorrhage occurred in owls fed difenacoum-fed rats.

Newton *et al.* (1990) gave mice feed containing 20 ppm brodifacoum or difenacoum. The mice died after 2–11 days, and mean residues in the whole carcasses (35 g) were 10.17 µg for difenacoum and 15.36 µg for brodifacoum. Six barn owls fed with difenacoum-poisoned mice survived after consuming 101.7 µg of difenacoum (on average) and their blood coagulation time returned to near normal in 5–23 days. Of six barn owls fed with brodifacoum-dosed mice, four died 6–7 days after the treatment. In average, the amount of brodifacoum in these owls was 46.07 µg (0.15–0.182 mg/kg) and the liver residues ranged from 0.63 to 1.25 mg/kg.

2.4.2 Factors influencing primary and secondary exposure

In the risk assessment of anticoagulant rodenticides, primary and secondary poisoning has been identified as a major concern for the environment. Exposure calculations according to the emission scenario for rodenticides show that feeding on the poisoned target rodent results in secondary poisoning (Larsen 2003). In nature, accumulation of anticoagulants to birds and mammals from non-target rodents may be an even more important exposure route. It has been noticed that non-target rodents feed on baits even during a properly performed control campaign. Findings of residues in birds which seldom prey on target rodents also support the observation that there is accumulation via non-target rodents. In general, voles are the preferred food of many raptorial bird species and target rodents form only a minor proportion of the raptors' diet under normal conditions.

The following factors have been noticed to influence the exposure of non-target animals:

- the wildlife species found in and around treatment areas, since the composition of the species community contributes to the main prey

species of predators in a certain area, and thereby to the risk of secondary poisoning
- the species' food habits and foraging behaviour, e.g. if a predator is a specialist or generalist, and whether it eats the whole carcass or only parts of it, *et cetera*.
- home range, some predators may live in a wide area and therefore prey on animals from several treatment areas
- propensity to feed in and near human buildings, since these are the main focus areas of poisoning
- bait availability (e.g., quantity, and how, where and when applied), since there is a clear correlation between the amount of baits, duration of poisoning operations, and exposure of non-target animals

Primary and secondary poisoning of non-target animals can be reduced by various measures. Use of anticoagulants could be restricted to in and around buildings or to indoor use only. Specific product design can make baits less accessible, in particular to birds and domestic animals, and can thus reduce the exposure. For example, the inclusion of a blue dye is believed to render the product unattractive to birds, and non-dusting formulations prevent the rodenticide from spreading to the environment. Bait boxes may be useful to prevent access by other than target animals, and baits should also be secured so that they cannot be dragged away. Unfortunately, bait boxes do not prevent non-target animals which are smaller than or of equal size to the target rodents from getting to the bait. Searching for and removing dead rodents during treatment is also recommended, at least as often as baits are checked and/or replenished. Disposing of dead rodents in accordance with local environmental requirements could also reduce the risk of secondary poisoning. However, this may not be a very effective measure as only a small proportion of the controlled rodent population is found dead during or after the control campaign. After treatment all remaining baits should be removed and disposed of

(http://ec.europa.eu/environment /biocides/pdf/anticoagulants.pdf Ref. 1.10.2009).

Despite these actions, there is no doubt that many birds and non-target mammals are attracted to, and will consume grain-based foods. These exposed non-target animals are then eaten up by predators and scavengers. Poisoned rodents may also leave trails of blood, stray away from cover and have slower reactions than unpoisoned ones, making them more vulnerable to predation (Murphy *et al.* 1998b). Up to 73% of dead poisoned muskrats (*Ondatra zibethicus*) were detected above ground, increasing the risk of secondary poisoning (Tuyttens & Stuyck 2002). According to Sage *et al.* (2008) storing of baits by water voles increases the persistence of bromadiolone even up to 10 times. This could also lead to a delayed exposure of rodent predators during a possible re-colonization by voles. Determining the relationship between an exposure

and the resulting dose in target tissue is therefore a critical issue encountered in quantitative risk assessment, since the mechanism of anticoagulant metabolism and excretion is poorly known. While primary exposure of larger non-target animals may be reduced by the use of bait stations, secondary exposure is more difficult to manage due to the prolonged effect of the anticoagulant (Eason *et al.* 2002).

Predators and scavengers are not expected to only consume contaminated animals, and therefore the estimation of degree of exposure is difficult. The risk of secondary poisoning of avian predators and scavengers from anticoagulant-poisoned prey is related to exposure factors such as the behaviour of the target species during the latent (pre-mortality) period, the location of carcasses (above or below ground), the anticoagulant residue loading in the target species, as well as the behaviour and diet of the non-target species (Howald *et al.* 1999). Newton *et al.* (1990) note that there remains the possibility that sub-lethal levels of rodenticides may predispose to death from other causes, or reduce the chance of recovery from accidents. The relationship between usage patterns and occurrence of liver residues in predators is also complex and potentially affected by physiological, ecological and anthropomorphic factors (Shore *et al.* 2003, 2006 in Walker *et al.* 2008a).

3 Review on studies on anticoagulant secondary poisoning in wildlife

3.1 United Kingdom

Secondary poisoning of mustelids and raptors by anticoagulant residues in Britain has been demonstrated by e.g. Barnett *et al.* 2003, 2004; Newton *et al.* 1990; Shore *et al.* 1996, 2003; McDonald *et al.* 1998 and Walker *et al.* 2008a. Anticoagulant residues have been found in e.g. barn owl, buzzard, red kite, crow, fox, kestrel and polecat. More than 30% of studied barn owls and polecats contained difenacoum residues (Newton *et al.* 1997; Shore 2003). In general, the residue levels are low and are not considered to be the primary cause of death. We have not found studies where effects of sublethal residue levels have been examined. However, in some incidents the animals have been determined to having died of haemorrhages.

In the UK the Wildlife Incident Investigation Scheme (WIIS) investigates deaths of wildlife where there is evidence to suggest that pesticide poisoning may be involved. Most sampling is done by the public, who find casualties and suspect that pesticides may have played a role in harming them. The carcasses are examined and chemical analyses are done on tissues. The pesticide incidents are assigned to one of four categories: approved use, misuse, abuse (deliberate poisoning attempts) and unspecified use where the cause could not be assigned to one of the above categories. Exposure of non-target animals to anticoagulant rodenticides is likely more widespread than the number of incidents suggests. Due to delayed effect and the living habits of predators the carcasses may usually not be detected (Birks *et al.* 1998). It is also noteworthy that small mammals like mice and voles are underrepresented in these incidents, since people do not deliver these animals for analyses. Either they are not discovered when dead or the carcasses are ignored.

The suspected secondary poisoning incidents, belonging to classes of approved use or unspecified use and involving birds of prey or carnivorous mammals (cats and dogs excluded) are summarized in table 4. According to WIIS data from 2004, rodenticide residues were found in every third of the buzzards investigated. The residue concentrations have not been given in the reports, and therefore only the concerned substance and species are given.

Table 4. Number of non-target species with brodifacoum, bromadiolone or difenacoum residues in the UK in 1998–2006. In addition there was one incident where flocoumafen was found in tawny owl. From Barnett et al. 1999–2007 (WIIS data).

Common name	Scientific name	Number of incidents 1998–2006		
		Bromadiolone	Difenacoum	Brodifacoum
Buzzard	*Buteo buteo*	30	42	2
Red kite	*Milvus milvus*	32	41	7
Fox	*Vulpes vulpes*	32	6	4
Barn owl	*Tyto alba*	11	9	3
Sparrow hawk	*Accipiter nisus*	5	5	0
Tawny owl	*Strix aluco*	3	5	3
Badger	*Meles meles*	4	4	1
Kestrel	*Falco tinnunculus*	3	3	0
Otter	*Lutra lutra*	3	2	0
Peregrine falcon	*Falco peregrinus*	1	0	1
Eagle owl	*Bubo bubo*	0	1	0
Golden eagle	*Aquila chrysaetos*	1	0	0
Marsh harrier	*Circus aeruginosus*	0	2	0
Stoat	*Mustela erminea*	1	0	0

Use of rodenticides on farms is widespread in the UK. In 2000, 91% of farms used rodenticides (McDonald & Harris 2000). Difenacoum is reported to be the most widely used rodenticide on arable farms and game estates. Surveys of rodenticide contamination in kestrel (Shore *et al*. 2003), stoat and weasel (McDonald *et al*. 1998) have all demonstrated significant rodenticide residues. Because of their heavy predation on rodents, mustelids may be at high risk for secondary poisoning by anticoagulant rodenticides, like several birds of prey such as barn owls and red kites (Shore *et al*. 2003).

Shawyer (1987, in Newton *et al*. 1990) reported suspected poisonings in barn owls associated with the use of brodifacoum (four cases), difenacoum (four cases) and bromadiolone (one case) between 1982 and 1985. Subsequently, about 10% of 145 barn owls found dead in Britain between 1983 and 1989 were found to contain detectable (> 0.005 ppm) levels of brodifacoum. In the study by Walker et al. (2008), 33 (19.2%) of the 172 tawny owl livers analysed contained detectable concentrations of one or more rodenticides. The occurrence of individual rodenticides was 11.6%, 5.8%, and 4.7% for bromadiolone, difenacoum, and brodifacoum, respectively. Tawny owl carcasses were collected through the Predatory Bird Monitoring Scheme, a long-term UK chemical monitoring program.

Newton *et al*. (1997) reviewed the mortality causes in British barn owls. Of 557 birds examined during 1983–1994, 132 (24%) contained residues of rodenticides, either difenacoum, brodifacoum, bromadiolone, flocoumafen or more than one of these compound (Table 5). 10% of 145 barn owls found dead in the period 1983–1989 contained residues of difenacoum (0.005–0.106 mg/kg bw) or brodifacoum (0.019–0.515 mg/kg bw) (Newton *et al.*, 1990). The proportion of birds in which residues were detected increased over the years, reaching 32% in 1993–1994. Eight birds were concluded to having died of rodenticide poisoning (Table 6), meaning that no other cause of death was found. Except for in

these eight birds, the residues were considered to be at sub-lethal level, and most deaths resulted directly from collisions with cars and trucks or starvation. However, the proportion of deaths due to rodenticides may have been underestimated, since death from anticoagulant exposure is delayed and preceded by lethargy, and most victims are likely to die in their roosts where they are not likely to be found. According to Newton *et al.* (2003), barn owls appear to be one of the most susceptible species to rodenticides.

Table 5. Percentage of barn owls with anticoagulant residues in the UK (Newton et al. 1997).

Period	Number with residues/ Number of owls analyzed	Percent containing residues
1983–1984	1/18	6
1985–1986	9/75	12
1987–1988	8/61	13
1989–1990	31/133	23
1991–1992	41/139	29
1993–1994	42/131	32

Table 6. Concentrations of residues in barn owls that were diagnosed as having died to anticoagulant poisoning in the UK between 1983 and 1994. (Newton et al. 1997)

Specimen number	Compound	Concentration mg/kg
1	Bromadiolone	0.13
2	Bromadiolone	0.05
	Flocoumafen	0.003
	Brodifacoum	0.02
3	Difenacoum	0.17
4	Bromadiolone	1.07
5	Brodifacoum	0.87
6	Bromadiolone	1.72
	Brodifacoum	0.07
7	Bromadiolone	0.33
8	Brodifacoum	0.42

McDonald *et al.* (1998) detected anticoagulant rodenticides in 23% (9 of 40) of stoats and 30% (3 of 10) of weasels received from gamekeepers in the UK between August 1996 and March 1997. The most frequently found substance was coumatetralyl which was found in six stoats and three weasels in concentrations ranging from 0.0046 to 0.06 mg/kg. Bromadiolone was found in three stoats in concentrations of 0.04–0.38 mg/kg and one weasel (0.25 mg/kg). Brodifacoum was found in one stoat (0.12 mg/kg).

A dataset of 100 polecats collected throughout the 1990's from across the whole of their prevailing range was created by combining three different studies. Shore *et al.* (1996) analyzed livers of 24 road-killed polecats and found anticoagulant rodenticides in 31% of the animals. A second study by Shore *et al.* (1999), in which the livers of another 26 adult animals were analyzed for the same compounds, was carried out in 1999. In 2003, Shore *et al.* reported measurements of rodenticide concentrations in the livers of 50 polecats. At least one rodenticide was detected in the

livers of 13 out of 37 (35.1%) male and 5 out of 13 (38.5%) female polecats (Shore *et al.* 2003).

Overall, 31% of the analysed polecats contained residues (Table 7). Bromadiolone (0.034–0.217 mg/kg) and difenacoum (0.005–0.917 mg/kg) were found most frequently. Brodifacoum was found in three animals. Flocoumafen was not detected, probably because it is not commonly used in Great-Britain. Results indicate that exposure of polecats to second generation rodenticides may be common, as a matter of fact around a third of adult polecats are exposed to second generation rodenticides in the western England. Shore *et al.* (2003) also note that it is possible that this may even be an underestimate because analysis of road-kills may involve a bias against detection of pesticide residues and animals in which no residues were detected may have eliminated any earlier contamination. However, the frequency with which residues were detected in polecats was very similar to that in barn owls, another rodent predator about which there has been concern over secondary exposure to second generation rodenticides.

Table 7. Residues of anticoagulant rodenticides in polecats in the UK (Shore et al. 1996, 1999 & 2003).

Year (Number of animals)	Average residues (range) mg/kg		
	Difenacoum	Bromadiolone	Brodifacoum
1993 (5)	0.319 (0.016–0.581)	-	0.07
1994 (7)	0.13 (0.005–0.321)	0.125 (0.039–0.217)	0.008
1995 (1)	-	0.095	-
1996 (2)	0.016	0.016	-
1997 (7)	0.182 (0.019–0.319)	0.074 (0.018–0.095)	-
1998 (7)	0.179 (0.03–0.397)	0.064 (0.034–0.094)	-
1999 (2)	0.649 (0.381–0.917)	0.186	0.052

During the period 1998–2001 14 red kites died due to anticoagulant rodenticide poisoning and one or more residues of brodifacoum, bromadiolone and difenacoum were detected in the carcasses (Barnett *et al.* 2000–2002). In 1994–2005, exposure to anticoagulant rodenticides was widespread among kites, since 73.9% of birds analyzed had detectable residues in their livers (Table 8). Two or three different rodenticides were found in the livers of almost half of the birds (Walker *et al.* 2008b). This suggests that multiple exposure events are relatively common.

Table 8. The occurrence and concentrations of second generation anticoagulant rodenticides in the livers of 23 red kites found dead in the UK between 1994 and 2005 (Walker et al. 2008b).

Substance	No of birds with detectable residues	Mean liver concentration (range)
Difenacoum	11 (47.8%)	0.052 (0.04–0.067)
Bromadiolone	12 (52.2%)	0.073 (0.056–0.094)
Flocoumafen	1 (4.3%)	0.015
Brodifacoum	7 (30.4%)	0.125 (0.071–0.222)
≥ One substance	17 (73.9%)	0.166 (0.137–0.2)
Two substances	6 (26.1%)	
Three substances	4 (17.4%)	

3.2 France

Berny *et al.* (1997, 2007, 2008), Lambert *et al.* (2007) and Fournier-Chambrillon *et al.* (2004) have investigated rodenticide poisonings in France and found that secondary poisoning is highly common, due to abundant use of rodenticides.

According to Berny *et al.* (1997), anticoagulant poisoning is among the most common causes of poisoning in domestic and wild animals in France. They present the result of a 4 year survey in France based on the activity of a wildlife disease surveillance network (SAGIR). Foxes, buzzards, one heron (*Ardea cinerea*) and one stoat were found dead with signs of internal haemorrhages in the vicinity of rodenticide-treated areas. All these species are carnivorous and potential consumers of field voles. Bromadiolone was detected in the livers of 22 of 31 red foxes, 15 of 16 buzzards, 5 of 5 kites, 2 of 13 rabbits (*Oryctolagus cuniculus*), 2 of 14 hares (*Lepus capensis*), 3 of 6 wild boars (*Sus scrofa*), 2 of 2 roe deers (*Capreolus capreolus*) and 2 of 2 stone martens (*Martes foina*). In addition, bromadiolone was found in one lynx (*Lynx lynx*), badger (*Meles meles*), harrier (*Circus pygargus*), mallard (*Anas platyrhynchos*), swan (*Cygnus sp.*) and heron (*Ardea cinerea*). Based on the species involved, secondary poisoning seems to have been the predominant route of exposure. Liver concentrations in the most commonly exposed foxes were 1.5 mg/kg and in buzzards 0.4 mg/kg.

Fournier-Chambrillon *et al.* (2004) estimated the exposure of 122 dead riparian mustelids of four species, European mink (*Mustela lutreola*), American mink (*M. vison*), polecat (*M. putorius*) and European otter (*Lutra lutra*) collected between 1990 and 2002 in south western France: bromadiolone was found in all species and in 9% of the collected carcasses (one of 31 European mink, three of 47 American mink, five of 33 polecats, and two of 11 European otters). Liver concentrations ranged from 0.6 mg/kg to 9.0 mg/kg. Chlorophacinone was found in two species and in 4% of the collected carcasses, with liver concentrations ranging from 3.4 mg/kg to 8.5 mg/kg. Two polecats and one American mink had lesions and liver residues indicating that bromadiolone was directly responsible for their deaths.

Anticoagulant rodenticides were detected or quantified in the liver of 22 of 30 raptors (73%) and 4 of 28 water birds (14%) (Lambert *et al* 2007). Bromadiolone was detected in the liver of 15 birds, difenacoum in the liver of 8 birds and brodifacoum in the liver of 4 birds. The most contaminated bird species was buzzard. Saucy *et al.* (2001) also reported deaths of numerous birds, mostly Eurasian buzzards but also kites and carrion crows, 38 wild mammals, mostly red foxes and mustelids, and 18 cats and dogs, after bromadiolone bait (150 ppm) was mechanically applied in underground burrows for water vole control in Switzerland.

Over the period 1992–2002, 62 red kites were found dead and poisoning was the confirmed cause of death in over 80% of these cases (Berny & Gaillet 2008). The major toxicants were anticoagulant rodenticides used to control water vole populations.

3.3 United States

According to Erickson and Urban (2004), brodifacoum and difethialone stand out as the two rodenticides posing the greatest potential overall risk to birds and non-target mammals in the USA, followed by bromadiolone and diphacinone. The avian species most commonly exposed to anticoagulant rodenticides in the USA are great horned owls (*Bubo virginianus*) and red-tailed hawks (*Buteo jamaicensis*). Multiple poisoning incidents have also been reported for golden and bald eagles (*Aquila chrysactos*, *Haliaeetus leucocephalus*), corvids (*Corvus spp.*), barn owl, eastern screech owl (*Otus asio*), northern spotted owl (*Strix occidentalis*), Cooper's hawk (*Accipiter cooperi*), red-shouldered hawk (*Buteo lieatus*), sharp-shinned hawk (*Accipiter striatus*), peregrine falcon (*Falco peregrinus*), American kestrel (*Falco sparverius*), and vultures (*Cathartes aura*, *Coragyps atratus*). Carnivores for which incidents have been reported include coyotes, foxes, raccoons, bobcats, skunks, mountain lions and a weasel. Difenacoum and flocoumafen were not included in the risk assessment. The reason might be that these substances were not on the market on that time in the US.

The data used in the risk assessment of rodenticides made by the US EPA (Erickson and Urban 2004) consists of more than 300 incidents in which one or more rodenticides were detected in birds or non-target mammals. Brodifacoum was detected in more than 244 of incidents and bromadiolone in 39 incidents (Table 9). Brodifacoum residues have been detected in liver tissue of 27 of 32 endangered kit foxes screened for rodenticide residues between 1999 and 2003. Birds in which rodenticides are most frequently detected include owls, hawks, eagles, and corvids; mammals include wild canids and felids, tree squirrels, raccoons and others. Traces of difethialone were detected in one bobcat. In 11 secondary hazard studies where 149 raptors or scavengers were exposed to brodifacoum-poisoned prey, 42% of exposed birds died. Many survivors had signs of intoxication, including haemorrhage (Erickson & Urban 2004).

Table 9. Liver residues of brodifacoum and bromadiolone in avian and mammalian predators and scavengers in the US. The number of animals with residues is given in parenthesis after the range (Erickson & Urban 2004).

Common name	Scientific name	Liver residues ppm	
		Bromadiolone (n)	Brodifacoum (n)
Great horned owl	*Bubo virginianus*	0.065–0.8 (9)	0.01–0.84 (41)
Long-eared owl	*Asio otus*	-	0.30 (1)
Eastern screech owl	*Otus asio*	0.05–4.29 (2)	0.16–0.91 (7)
Northern saw-whet owl	*Aegolius acadicus*	0.43 (1)	-
Barn owl	*Tyto alba*	0.31–0.38 (3)	0.03–0.85 (7)
Barred owl	*Strix varia*	-	0.04 (1)
Northern spotted owl	*Strix occidentalis*	-	0.05–0.1 (2)
Golden eagle	*Aquila chrysaetos*	-	0.03–0.85 (13)
Bald eagle	*Haliaeetus leucocephalus*	-	Detected (2)
Red-tailed hawk	*Buteo jamaicensis*	0.08 (1)	0.003–1.6 (43)
Red-shouldered hawk	*Buteo lineatus*	0.28 (1)	0.01–0.23 (3)
Cooper's hawk	*Accipiter cooperii*	0.24–0.6 (2)	0.03–0.37 (6)
Sharp-shinned hawk	*Accipiter striatus*	-	0.023–0.17 (2)
Black vulture	*Coragyps atratus*	-	0.13 (1)
Turkey vulture	*Cathartes aura*	-	0.26 (1)
Raven	*Corvus corax*	-	1.04 (1)
American crow	*Corvus brachyrhynchos*	-	0.07–1.9 (13)
Fish crow	*Corvus ossifragus*	2.1 (1)	-
Crow	*Corvus corone*	-	0.14–1.67 (4)
Coyote	*Canis latrans*	0.07–0.46 (5)	0.03–0.66 (21)
San Joaquin kit fox	*Vulpes macrotis mutica*	0.14–0.72 (2)	0.007–11.0 (27)
Red fox	*Vulpes vulpes*	-	0.04–4.01 (5)
Grey fox	*Urocyon cinereoargenteus*	-	0.02–0.35 (3)
Bobcat	*Felis rufus*	0.11 (1)	0.018–0.07 (4)
Mountain lion	*Puma concolor*	-	0.52 (1)
Raccoon	*Procyon lotor*	0.41–1.1 (2)	0.011–5.3 (9)
Long-tailed weasel	*Mustela frenata*	-	0.07 (1)
Striped skunk	*Mephitis mephitis*	0.08–0.29 (3)	0.3–1.05 (2)

From 1971 to 1997, Stone *et al.* (1999) documented 51 cases of poisoning of non-target wildlife in New York State with anticoagulant rodenticides (Table 10). Brodifacoum was detected in 80% (41) of the incidents and bromadiolone in three cases (once in combination with brodifacoum). The most commonly poisoned species were great horned owl (13 cases) and red-tailed hawk (7 cases). For the period 1998–2001, anticoagulant rodenticides were detected in 49% of the 265 raptors analyzed in New York State. Brodifacoum was detected in 84% of the positive cases and bromadiolone in 22%. Brodifacoum and bromadiolone were found in combination in 15 birds. Great horned owl was again the most frequently poisoned species. Among positive cases, brodifacoum levels were on average 0.18 mg/kg (range 0.005–1.28 mg/kg) and bromadiolone levels on average 0.31 mg/kg (range 0.03–1.08 mg/kg). Anticoagulants were detected in great horned owls (81%), red-tailed hawks (58%), Cooper's hawks (36%), and screech owls (45%) (Table 11). Based on lethal blood loss in the absence of severe injury or other factors, anticoagulant-facilitated haemorrhage was considered the cause of death in 28 cases (Stone *et al.* 2003).

Table 10. Anticoagulant poisonings in wildlife in New York and adjoining states in the period 1989–1997 (Stone et al. 1999). Only poisonings caused by the second generation anticoagulants are reported here.

Common name	Scientific name	No. of animals	Liver residues mg/kg	
			Brodifacoum	Bromadiolone
Common crow	*Corvus corone*	1	1.34	
Eastern chipmunk	*Tamias striatus*	1	3.8	
Golden eagle	*Aquila chrysaetos*	1	1.04	
Gray squirrel	*Sciurus carolinensis*	5[1]	1.68 (0.7–4.1)	
Great horned owl	*Bubo virginiatus*	13	0.33 (0.01–0.73)	0.14
Opossum	*Didelphis virginiana*	1	0.18	0.8
Raccoon	*Procyon lotor*	6	2.69 (0.32–5.3)	
Raven	*Corvus corax*	1	1.04	
Red fox	*Vulpes vulpes*	2	2.67 (1.32–4.01)	
Red-tailed hawk	*Buteo jamaicensis*	7	0.65 (0.23–1.6)	
Screech owl	*Otus asio*	2	0.57 (0.34–0.8)	
Skunk	*Mephitis mephitis*	3		0.13 (0.02–0.28)

[1]One concentration from colon, 25.8 mg/kg.

Stone *et al.* (2003) note that anticoagulants appear to be present in the majority of great horned owls, in about half of the red-tailed hawks, and probably in substantial proportions of other raptors in New York State. The poisonings in the red foxes, skunks (*Mephitis mephitis*), opossums (*Didelphis virginiana*) and the other raccoons could have been primary or secondary in nature.

Table 11. Range of liver residues of brodifacoum and bromadiolone in four raptor species analyzed by Stone et al. (2003).

Common name	Scientific name	Liver residues ppm	
		Bromadiolone	Brodifacoum
Red-tailed hawk	*Buteo jamaicensis*	0.08–0.50	0.006–1.28
Great horned owl	*Bubo virginiatus*		0.007–0.97
Screech owl	*Otus asio*		0.007–0.47
Cooper's hawk	*Accipiter cooperii*	0.04–0.60	0.008–0.22

Hegdal and Colvin (1988) examined the risk of secondary poisoning to eastern screech owls during experimental vole control baiting in orchards during the fall and winter of 1981–82. The study indicated a considerable risk to screech owls and possibly other raptors that feed on voles baited with 10 ppm (mg/kg) brodifacoum bait (baits registered for rat and mouse control are normally 50 ppm). Liver residue analyses were conducted on 16 owls, and brodifacoum residues were detected at levels ranging from 0.3 to 0.8 mg/kg in 9 owls.

Howald *et al.* (1999) examined effects of brodifacoum baiting on avian scavengers during a rat control program on Langara Island, Canada, and concluded that there is a risk of secondary poisoning for some predators and scavengers. The impact on ravens may have been severe. Of an island population of about 20–72 individuals, 13 ravens were found dead

after 12 to 47 days from beginning of baiting. All dead ravens had brodifacoum residues ranging from 0.98 to 2.52 mg/kg in their livers. Ravens were likely exposed from eating the bait as well as secondarily feeding on intoxicated prey. Brodifacoum was also detected in a pooled sample of 3 north western crows (*Corvux caurinus*) collected 12 days after the start of baiting.

Riley *et al.* (2007) tested bobcats (*Lynx rufus*) for a disease, notoedric mange, and found out that anticoagulants were present in 35 of 39 (90%) tested bobcats. Multiple compounds were present in 27 of these animals (77%). Brodifacoum levels in the livers ranged up to 0.56 mg/kg, bromadiolone 0.82 mg/kg and difethialone < 0.25 mg/kg. 19 of 19 (100%) bobcats that died with mange were also exposed to the rodenticides brodifacoum and bromadiolone.

3.4 New Zealand

In New Zealand anticoagulants have been used in the elimination of introduced rodents on islands and other areas inhabited by indigenous species found only in New Zealand. These pest control programmes have resulted in the primary and secondary poisoning and sub-lethal contamination of non-target species (Alterio *et al.* 1996, 1997, 2000; Eason *et al.*1995, 1999, 2002; Murphy *et al*.1998a, 1998b).

Birds of at least five indigenous species have been reported killed and lethal or sub-lethal effects have been documented in 26 bird species, as a result of secondary poisoning with brodifacoum after rat-, rabbit- and opossum-poisoning operations (Eason & Spurr 1995; Hoare & Hare 2006). Mortality of radio-tagged stoats, ferrets (*Mustela furo*), weasels, and cats was reported to be even 100% after brodifacoum application, and secondary adverse effects on Australasian harriers (*Circus approximans*), New Zealand falcons (*Falco novaeseelandiae*), rails, brown skuas (*Catharacta skua*), gulls, and owls (morepork, *Ninox novaeseelandiae*) have also been reported (Alterio *et al.* 1997; Eason & Spurr 1995).

Residues of brodifacoum were detected in 78% of 40 stoats, 71% of 14 weasels, and 56% of 16 ferrets trapped after a rat- and opossum-poisoning operation (Murphy *et al.* 1998a). Residue levels in stoats were greater during the three months after the removal of baits than during the poison operation. The mean residue level in the livers of weasels was 1.26 mg/kg and 1.01 mg/ kg in the livers of ferrets. In the study by Alterio (1996), all three radio-tagged stoats and five ferrets, and two of three radio-tagged cats, died shortly after brodifacoum-poisoning in the study area. Liver residues were 0.94–1.72 mg/kg in stoats, 1.47–1.97 mg/kg in ferrets and 2.71–3.73 mg/kg in cats. The last cat was trapped, and had brodifacoum residues of 2.7 mg/kg in the liver. At least six stoats are also likely to be killed because of the poisoning. In 2000, Alterio & Moller

reported deaths of nine of ten radio-tagged stoats after a poisoning operation with brodifacoum. Six of the stoats died 1–2 weeks after scavenging on poisoned carcasses.

In a pen trial, one out of four Australasian harriers died after eating rabbits poisoned with brodifacoum (Godfrey 1985, in Eason & Spurr 1995). Eason and Spurr (1995) also note that bats that are primarily insectivorous may be at risk from both primary and secondary poisoning. Besides of eating baits, they eat carrions and might feed on brodifacoum-poisoned insects or carcasses.

4 Conclusions and implications to the Nordic countries

The existence of substantial incident data along with liver-residue analyses confirms that birds and non-target mammals are being exposed to anticoagulant rodenticides all around the world. The fact that numerous species of birds and mammals, including predators and scavengers have been exposed to these substances indicates that both primary and secondary exposure is occurring. Exposure of non-target animals is likely to be more widespread than the number of reported incidents suggests. Most surveys have been based on the activity of the general public who has sent carcasses to analysis when they have suspected poisoning. In many situations carcasses might not be detected, death may be attributed to natural mortality, or an incident may not be reported for a variety of reasons. In conclusion, studies made on secondary exposure reveal only the top of the iceberg. Residues found in predators and scavengers show that predominantly the second generation substances, i.e. brodifacoum, bromadiolone, difenacoum, flocoumafen and difethialone, cause secondary exposure. There were not many incidents where first generation anticoagulants were involved. The frequency of incidents is assumed to correlate to the use volumes of the substances. Unfortunately we have not found published data on sale volumes that could be compared to the number of incidents in the UK and US where most incident data are available. The statistics on sale volumes or use frequencies would enable the comparison of the likelihood or potency of the substances in causing the secondary poisoning. Due to lack of resources and/or ignorance of the potential risk measurements have been made only in few countries, and the incidents are likely to be much more widespread than reported here.

The available data show that more attention should be paid to the use of second generation anticoagulant rodenticides. Several risk reduction measures have been suggested (see 2.4.2), but it is uncertain how efficient these measures are or to what extent these measures are applied in practice. Maybe more education on potential risks and risk management should be directed to the users of anticoagulants. The use of second generation anticoagulants should be restricted to situations where they cannot be replaced by first generation substances or alternative methods. In general, use of mechanical traps and other non-biocidal methods should be prioritized in homes where mice are the main target animals. It is imperative that the use of second generation anticoagulants is restricted to well organised, strictly time-limited campaigns where the number of target animals is carefully monitored in order to obtain optimal eradication and

minimise the treatment time period. Extended use beyond the time needed for optimal effect must be avoided. Control of larger rat infestations is challenging and should be left to professional pest control operators. Residue-monitoring programmes could be established in those countries where anticoagulants are used in the field or extensively around farm buildings.

In the UK, the only EU country where residues of anticoagulant have been more systematically measured, the most commonly indirectly exposed species were red kite, fox and buzzard (Table 12). Secondary exposure to anticoagulants does most likely occur also in the Nordic countries, although there are hardly any incident studies to be found. In Denmark, anticoagulant rodenticide residues were detected in 4 of 5 red kites, 11 of 22 barn owls and 1 of 2 common kestrels (Laursen 2008, unpublished master study). Bromadiolone and brodifacoum were most frequently detected in red kites and barn owls, one red kite having detectable residues from three and two barn owls having residues of two different anticoagulant rodenticides. The data indicate that anticoagulant residues may occur in a great proportion of raptors, although the sample size was too small for making statistically valid conclusions.

The most commonly exposed predatory species in the UK (Table 12) occur also in the Nordic countries and would be appropriate candidates to start the measurements of anticoagulant residues, if a monitoring project would be initiated. The species potentially exposed either directly or indirectly to anticoagulants in the Nordic countries are given in Attachment A. The species were selected by the experts from the Nordic countries.

Table 12. The most commonly exposed non-target species in the UK and percentage of animals with residues.

Species	Percentage with residues	No. of individuals studied	Reference
Red kite	74	23	Walker et al. 2008b
Fox	57	23	WIIS
Buzzard	37	61	WIIS
Polecat	31	100	Shore et al. 1993, 2003
Weasel	30	10	McDonald et al. 1998
Barn owl	24	557	Newton et al. 1990
Stoat	22	40	McDonald et al. 1998

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Sammanfattning

Antikoagulant rodenticider har hittats i icke-måldjur i alla delar av världen där man har mätt rester. Brodifakum, bromadiolon, difenakum, flokumafen och difetialon betraktas som andra generationens antikoagulant rodenticider. De har kommit ut på marknaden sedan målgnagarna hade blivit resistenta mot warfarin och övriga första generationens anticoagulant rodenticider. Andra generationens antikoagulanter är giftigare och mera persistenta i gnagare jämfört med första generationens medel. Därför har de en tendens att ackumuleras i icke-måldjur som äter gnagare. Trots omfattande användning av antikoagulanter för bekämpning av råttor och möss, finns det relativt få publikationer om antikoagulater i icke-måldjuren. Resterna har studerats systematiskt bara i Storbritannien och i vissa delstater i USA. I Europa har antikoagulantrester mätts förutom i Storbritannien bara i Frankrike. I Storbritannien, har antikoagulantresterna studerats framförallt i tornuggla (*Tyto alba*) och i iller (*Mustela putorius*). Ungefär en tredjedel av de studerade exemplaren innehöll rester. Antikoagulanthalterna har oftast varit subletala. Vi vet inte hur sådana låga halter påverkar djuren. I Storbritannien har man också hittat rester av antikoagulanter bl.a. i ormvråk (*Buteo buteo*), röd glada (*Milvus milvus*), och räv (*Vulpes vulpes*). I USA har man hittat antikoagulanter i virginauv (*Bubo virginianus*), rödstjärtad vråk (*Buteo jamaicensis*), prärievarg, räv och tvättbjörn. Antikoagulanter är de mest använda rodenticiderna i de nordiska länderna, men det finns ingen publicerad information om rester av antikoagulanter i icke-måldjur. Information om halterna behövs för att kunna värdera effektiviteten av de metoder som används för att minska riskerna som är förbundna med rodenticider och för att kunna avgöra om det behövs ytterligare åtgärder.

Attachment A

Table 1. List of birds in the Nordic countries which may potentially be exposed to anticoagulant rodenticides. The information on diet is obtained from Perrins (1987).

Scientific name	English name	Swedish name	Main food	Country
Accipiter gentilis	Goshawk	Duvhök	Medium-size birds and mammals	D, F, N, S
Accipter nisus	Sparrowhawk	Sparvhök	Small birds, to a lesser extent small mammals	D, F, N, S
Aquila chrysaetos	Golden eagle	Kungsörn	Hare, gamebirds, carcasses	D, F, N, S
Buteo buteo	Buzzard	Ormvråk	Small mammals, frogs, snakes, lizards	D, F, N, S
Buteo lagopus	Rough-legged buzzard	Fjällvråk	Lemmings, voles	D, F, N, S
Circus aeruginosus	Marsh harrier	Brun kärrhök	Water voles, frogs, young of water birds	D, F, N, S
Falco peregrinus	Peregrine falcon	Pilgrimsfalk	Birds	D, F, N, S
Falco tinnunculus	Kestrel	Tornfalk	Predominantly small rodents	D, F, N, S
Haliaeetus albicilla	White-tailed eagle	Havsörn	Fish, water birds, carcasses	D, F, N, S
Milvus milvus	Red kite	Röd glada	Fish, carcasses, insects	D, N, S
Aegolius funereus	Tengmalm´s owl	Pärluggla	Predominantly small mammals	D, F, N, S
Asio otus	Long-eared owl	Hornuggla	Small rodents	D, F, N, S
Bubo bubo	Eagle owl	Berguv	Mammals, birds, frogs	D, F, N, S
Bubo scandiacus (Nyctea scandiaca)	Snowy owl	Fjälluggla	Lemmings, voles, willow grouse	F, N, S
Strix aluco	Tawny owl	Kattuggla	Small mammals, birds, insects	D, F, N, S
Tyto alba	Barn owl	Tornuggla	Small mammals, frogs, insects	D, N, S
Corvus corone cornix	Hooded crow	Kråka	Waste, dead animals	D, F, N, S
Corvus corax	Raven	Korp	Omnivorous, carcasses in winter	D, F, N, S
Corvus monedula	Jackdaw	Kaja	Small animals	D, F, N, S
Pica pica	Magpie	Skata	Omnivorous	D, F, N, S
Larus argentatus	Herring gull	Gråtrut	Dead fish, young of water birds, waste	D, F, N, S
Larus canus	Common gull	Fiskmås	Dead fish, insects, waste	D, F, N, S
Larus marinus	Great black-backed gull	Havstrut	Young of water birds, fish, mussels	D, F, N, S
Larus ridibundus	Black-headed gull	Skrattmås	Insects, earthworms, molluscs,	D, F, N, S
Carduelis chloris	Greenfinch	Grönfink	Seed	D, F, N, S
Columba livia	Rock dove	Tamduva	Seed, grain, food waste	D, F, N, S
Emberiza calandra	Corn bunting	Kornsparv	Small seeds, insects	D, N, S
Emberiza citrinella	Yellow-hammer	Gulsparv	Seed, grain, insects	D, F, N, S
Galerida cristata	Crested lark	Tofslärka	Insects, small seeds	D, N, S
Passer domesticus	House sparrow	Gråsparv	Seed, food waste	D, F, N, S
Passer montanus	Tree sparrow	Pilfink	Omnivorous, predominantly seed	D, F, N, S
Streptopelia decaocto	Collared dove	Turkduva	Seed, grain, berries	D, F, N, S

Table 2. List of mammals in the Nordic countries which may potentially be exposed to anticoagulant rodenticides. The information on diet is obtained from Koivisto (1983).

Latin name	English name	Swedish name	Main food	Country
Apodemus flavicollis	Yellow-necked mouse	Större skogsmus	Seed, grain	D, F, N, S
Apodemus sylvaticus	Wood mouse	Mindre skogsmus	Seed, grain	D, F, N, S
Arvicola terrestris	Water vole	Vattensork	Seed, grain	D, F, N, S
Clethrionomys glareolus	Bank vole	Ängsork	Seed, grain	
Microtus agrestis	Field vole	Åkersork	Seed, grain	D, F, N, S
Mustela erminea	Stoat	Hermelin	Small rodents	D, F, N, S
Mustela nivalis	Least weasel	Snövessla	Small rodents	D, F, N, S
Mustela putorius	Polecat	Iller	Small rodents, fish, frogs, birds	D, F, N, S
Mustela vison	American mink	Mink	Fish, small rodents, frogs, crayfish, birds, carcasses	D, F, N, S
Martes martes	Pine marten	Mård	Squirrel, hare, small mammals, muskrat, gamebirds, insects, berries	D, F, N, S
Meles meles	Badger	Grävling	Omnivorous, small vertebrates	D, F, N, S
Lutra lutra	Otter	Utter	Fish, small mammals, birds	D, F, N, S
Vulpes vulpes	Fox	Räv	Small rodents, carcasses, waste	D, F, N, S
Nyctereutes procyonoides	Raccoon dog	Mårdhund	Small mammals, fish, frogs	F, N, S
Sciurus vulgaris	Squirrel	Ekorre	Seed, grain, berries, birds and eggs	D, F, N, S

Attachment B

Table 1. Number of species with anticoagulant residues. WIIS and EIIS data are excluded from the summary.

Scientific name	Common name	Substance	No. with residues	Range of residues in liver, mg/kg	Reference
Accipiter cooperii	Cooper's hawk	Bromadiolone	5	0.04–0.6	Stone et al. 2003
Accipiter cooperii	Cooper's hawk	Brodifacoum	12	0.008–0.22	Stone et al. 2003
Accipiter striatus	Sharp-shinned hawk	Brodifacoum, bromadiolone	1		Stone et al. 2003
Aegolius acadicus	Saw-whet owl		1		Stone et al. 2003
Aquila chrysaetos	Golden eagle	Brodifacoum	1	1.04	Stone et al. 1999
Ardea cinerea	Heron	Bromadiolone	1	0.2	Berny et al. 1997
Asio otus	Long-eared owl		2		Stone et al. 2003
Asio otus	Long-eared owl	Brodifacoum	1		Hegdal & Colvin 1988
Bubo virginianus	Great-horned owl		56		Stone et al. 1999, 2003
Buteo buteo	Eurasian buzzard	Bromadiolone	15	0.2–1.3	Berny et al. 1997
Buteo buteo	Eurasian buzzard	Brodifacoum	1		Lambert et al. 2007
Buteo buteo	Eurasian buzzard	Bromadiolone	5		Lambert et al. 2007
Buteo buteo	Eurasian buzzard	Difenacoum	3		Lambert et al. 2007
Buteo buteo	Eurasian buzzard	Brodifacoum	1		Shore et al. 2006
Buteo buteo	Eurasian buzzard	Bromadiolone	2		Shore et al. 2006
Buteo buteo	Eurasian buzzard	Difenacoum	13		Shore et al. 2006
Buteo buteo	Eurasian buzzard	Flocoumafen	1		Shore et al. 2006
Buteo jamaicensis	Red-tailed hawk		45		Stone et al. 2003
Buteo jamaicensis	Red-tailed hawk	Brodifacoum	7	0.23–1.6	Stone et al. 1999
Cathartes aura	Turkey vulture	Brodifacoum, bromadiolone	2		Stone et al. 2003
Circus approximans	Australasian harrier	Bromadiolone	1		Berny et al. 1997
Circus approximans	Australasian harrier	Brodifacoum	2	0.61–0.66	Eason et al. 2002
Circus pygargus	Harrier (suohaukka)	Bromadiolone	1	6.1 [2]	Berny et al. 1997
Corvus caurinus	Northwestern crow	Brodifacoum	1	0.048	Howald et al. 1999
Corvus corax	Raven	Brodifacoum	1	1.04	Stone et al. 1999
Corvus corax	Raven	Brodifacoum	13	0.98–2.52	Howald et al. 1999
Corvus corone	Common crow	Brodifacoum	1	1.34	Stone et al. 1999
Falco peregrinus	Peregrine falcon	Brodifacoum, bromadiolone	1		Stone et al. 2003
Falco tinnunculus	Kestrel		49		Walker et al. 2008b
Falco tinnunculus	Kestrel		24		Shore et al. 2001
Falco tinnunculus	Kestrel	Brodifacoum	2		Lambert et al. 2007
Falco tinnunculus	Kestrel	Bromadiolone	2		Lambert et al. 2007
Felis catus	Cat	Brodifacoum	2	0.39–1.4	Murphy et al. 1998
Felis catus	Cat	Brodifacoum	3		Alterio 1996
Felis catus	Cat	Brodifacoum	57	0.078–1.84	Eason et al. 2002
Haliaeetus leucocephalus	Bald eagle	Brodifacoum	3	0.037–1.74 [1]	Howald et al. 1999
Haliaeetus leucocephalus	Bald eagle		1		Stone et al. 2003
Lutra lutra	European otter	Bromadiolone	2	6.0–7.1	Fournier-Chambrillon et al. 2004
Lynx lynx	Lynx	Bromadiolone	1	1.3 [2]	Berny et al. 1997
Lynx rufus	Bobcat		54		Riley et al. 2007
Martes foina	Stone-marten	Bromadiolone	2	0.6–1.0 [2]	Berny et al. 1997

Scientific name	Common name	Substance	No. with residues	Range of residues in liver, mg/kg	Reference
Meles meles	Badger	Bromadiolone	1	0.9	Berny et al. 1997
Mephitidae sp.	Skunk	Bromadiolone	3	0.02–0.28	Stone et al. 1999
Milvus migrants	Black kite	Bromadiolone	5	0.3–0.6 [2]	Berny et al. 1997
Milvus milvus	Red kite		23		Walker et al. 2008b
Mustela erminea	Stoat	Brodifacoum	31	0.05–1.52	Murphy et al. 1998
Mustela erminea	Stoat	Bromadiolone	3	0.04–0.38	McDonald et al. 1998
Mustela erminea	Stoat	Brodifacoum	1	0.12	McDonald et al. 1998
Mustela erminea	Stoat	Brodifacoum	9		Alterio 1996
Mustela erminea	Stoat	Brodifacoum	11		Alterio et al. 1997
Mustela erminea	Stoat	Brodifacoum	98	0.008–1.32	Eason et al. 2002
Mustela furo	Feral ferret	Brodifacoum	9		Murphy et al. 1998a
Mustela furo	Feral ferret	Brodifacoum	5		Alterio 1996
Mustela lutreola	European mink	Bromadiolone	1	5	Fournier-Chambrillon et al. 2004
Mustela nivalis	Weasel	Brodifacoum	10		Murphy et al. 1998a
Mustela nivalis	Weasel	Bromadiolone	1	0.25	McDonald et al. 1998
Mustela nivalis	Weasel	Brodifacoum	1		Alterio et al. 1997
Mustela putorius	Polecat	Bromadiolone	5		Fournier-Chambrillon et al. 2004
Mustela putorius	Polecat	Difenacoum	1	1.4	Fletcher et al. 1994
Mustela putorius	Polecat	Difenacoum	2	0.3–1.4	Birks 1998
Mustela putorius	Polecat		31		Shore et al. 1996; 1999; 2003
Mustela vison	American mink	Bromadiolone	3	1.9–4.2	Fournier-Chambrillon et al. 2004
Otus asio	Screech owl	Brodifacoum	9	0.007–0.8	Hegdal & Colvin, 1988
Otus asio	Screech owl	Brodifacoum	2	0.34–0.8	Stone et al. 1999
Otus asio	Screech owl		10		Stone et al. 2003
Puma concolor	Mountain lion		4		Riley et al. 2007
Procyon lotor	Raccoon	Brodifacoum	6	0.32–5.3 [2]	Stone et al. 1999
Strix aluco	Tawny owl		33		Walker et al. 2008
Strix aluco	Tawny owl	Bromadiolone	2		Lambert et al. 2007
Strix varia	Barred owl		3		Stone et al. 2003
Sus scrofa	Wild boar	Bromadiolone	3	0.4–3.6	Berny et al. 1997
Sus scrofa	Wild boar	Brodifacoum	21	0.007–1.78	Eason et al. 2002
Tyto alba	Barn owl		124		Walker et al. 2008
Tyto alba	Barn owl	Brodifacoum	15	0.019–0.515	Newton et al. 1990
Tyto alba	Barn owl	Difenacoum	15	0.005–0.106	Newton et al. 1990
Tyto alba	Barn owl		132		Newton et al. 1997
Tyto alba	Barn owl		187		Newton et al. 1999
Tyto alba	Barn owl	Bromadiolone	4		Lambert et al. 2007
Tyto alba	Barn owl	Difenacoum	5		Lambert et al. 2007
Tyto alba	Barn owl	Difenacoum	16		Shore et al. 2006
Tyto alba	Barn owl	Bromadiolone	15		Shore et al. 2006
Tyto alba	Barn owl	Brodifacoum	3		Shore et al. 2006
Vulpes vulpes	Red fox	Bromadiolone	22	0.8–6.9 [2]	Berny et al. 1997
Vulpes vulpes	Red fox	Brodifacoum	2	1.32–4.01	Stone et al. 1999

[1]Residue from plasma

[2]Whole-carcass residues

Table 2. Secondary exposure studies made in controlled circumstances. Time after exposure stands for the time after which animals have died in poisoning. Also survivers displayed signs of haemorrhage in majority of studies.

Scientific name	Common name	No dead / No tested	Substance	Time after exposure, days	Reference
Aquila chrysaetos	Golden eagle	0 / 4	Brodifacoum	4	Marsh & Howard 1978
Arvicola terrestris	Water vole	1	Bromadiolone	135	Sage et al. 2008
Buteo buteo	Eurasian buzzard	4 / 5	Brodifacoum	6	Lutz 1987
Buteo buteo	Eurasian buzzard	3 / 40	Bromadiolone	3	Grolleau et al. 1989
Buteo buteo	Eurasian buzzard	3 / 4	Bromadiolone	10	Lutz 1986
Buteo jamaicensis	Red-tailed hawk	4 / 4	Brodifacoum	4	Marsh & Howard 1978
Buteo lineatus	Red-shouldered hawk	2 / 2	Brodifacoum	4	Marsh & Howard 1978
Canis latrans	Coyote	2 / 7	Bromadiolone	5	Marsh & Howard 1986
Canis lupus familiaris	Dog (domestic)	1 / 6	Brodifacoum	1–4	Erickson & Urban 2004
Circus approximans	Australasian harrier	1 / 4	Brodifacoum		Godfrey 1985; Williams et al. 1986
Corvux caurinus	Nothwestern crow	3	Brodifacoum	12	Howald et al. 1999
Falco sparverius	American kestrel	9 / 18	Brodifacoum	5–6	Savarie & LaVoie 1979; LaVoie 1990
Herpestes auropunctatus	Mongoose	1 / 4	Brodifacoum	10	Pank & Hirata 1976
Herpestes auropunctatus	Mongoose	3 / 4	Bromadiolone	6	Pank & Hirata 1976
Larus atricilla	Laughing gull	5 / 5	Brodifacoum	5	Erickson & Urban 2004
Larus atricilla	Laughing gull	4 / 5	Brodifacoum	5	Erickson & Urban 2004
Mustela erminea	Stoat (ermine)	1 / 11	Bromadiolone	3–5	Grolleau et al. 1989
Mustela erminea	Stoat	11	Brodifacoum	6–9	Alterio et al. 1997
Tyto alba	Barn owl	4 / 8	Bromadiolone	5–7	Lee 1994, 1995
Tyto alba	Barn owl	3 / 4	Flocoumafen		Lee 1995
Tyto alba	Barn owl	1 / 9	Bromadiolone	6–10	Mendenhall & Pank 1980; Wyllie 1995
Tyto alba	Barn owl	9 / 12	Brodifacoum	3–8	Mendenhall & Pank 1980; Newton et al. 1990
Tyto alba	Barn owl	4 / 8	Brodifacoum	5–7	Lee,1994, 1995
Tyto alba	Barn owl	1 / 4	Brodifacoum	15	Gray et al. 1994
Tyto alba	Barn owl	1 / 6	Difenacoum	6–10	Mendenhall & Pank 1980
Vulpes vulpes/ Urocyon cinereoargenteus	Red fox and grey fox	2 / 5	Brodifacoum	4	Erickson & Urban 2004