Sanchez-Paredes, Ciara M.; Burgess, Malcolm D.; Fryer, Michael; **Pereira, M. Glória**; Arnold, Kathryn E. 2025. **Predictive framework for estimating the risks to raptors from ecologically relevant exposure to second generation anticoagulant rodenticides**.

The definitive version was published in *Science of the Total Environment*, 992, 179903. 12, pp. https://doi.org/10.1016/j.scitotenv.2025.179903

The definitive version is available at https://www.elsevier.com/

Predictive framework for estimating the risks to raptors from ecologically relevant exposure to Second Generation Anticoagulant Rodenticides

Ciara M. Sanchez-Paredes*[a], Malcolm D. Burgess[b], Michael Fryer[c], M. Glória Pereira[d], Kathryn E. Arnold[a]

[a] Department of Environment and Geography, University of York, York, YO10 5DD, UK
[b]RSPB Centre for Conservation Science, The Lodge, Sandy, SG19 2DL, UK
[c]Health and Safety Executive, Mallard House, Kings Pool, 3 Peasholme Green, York YO1 7PX
[d]UK Centre For Ecology & Hydrology, Library Ave, Bailrigg, Lancaster LA1 4AP

* Corresponding author: cmsp501@york.ac.uk

Key words

Raptors, Anticoagulant Rodenticides, Breeding stages, Framework, Cumulative exposure

Abstract

Globally, populations of many raptor species are declining for reasons including exposure to chemical contaminants. Second Generation Anticoagulant Rodenticide (SGAR) residues are commonly found in the livers of raptors, indicating dietary exposure and potentially secondary poisoning. There is a gap in our understanding of SGAR exposure for raptors at different life stages and under different usage regimes.

We develop a new modelling framework for non-target species to refine the commonly applied risk assessment using the common kestrel (*Falco tinnunculus*) as a case study. Our scenarios simulate high and low rodent consumption by raptors with high and low SGAR usage, of four different rodenticides at different raptor life stages. The risk to raptors was evaluated in reference to sublethal and lethal effect thresholds used by regulators.

Our model suggests exposure to Brodifacoum exceeds the threshold for lethal and sublethal effects. Breeding adults and nestlings are at risk of sublethal effects from Difethialone and the mixture of Bromadiolone and Difenacoum exposure. This is concerning because Brodifacoum and Difethialone usage seems to be rising globally due to increased resistance of rodents to other SGARs. Difethialone represents cumulative risk in scenarios previously not a risk, reaching the threshold in a week Avoiding SGAR usage during the raptor breeding season

would reduce the likelihood and impact of secondary SGAR poisoning. Our framework can be used to assess different exposure scenarios for SGARs and raptors in ecological meaningful contexts to guide SGAR usage and regulation.

1. Introduction

Many raptor populations globally are in decline, for a variety of reasons. Climate change (Martínez-Ruiz et al., 2023), habitat change and degradation (Nemček, 2013; Butet et al., 2010; Butet, Rantier and Bergerot, 2022) and increases in exposure to chemical contaminants of top predators, such as raptors, can negatively affect individual fitness and so influence population abundance (Green et al. 2004; Berny et al. 2015; Méndez et al. 2021; Martínez-Padilla et al. 2017; Salim et al. 2014; Spadetto et al. 2024; Lemus et al. 2011). Second Generation Anticoagulant Rodenticides (SGARs), a class of biocide compounds used to control pests and protect public health (European Chemicals Agency, 2018), are commonly detected in raptor liver samples suggesting SGAR exposure via diet (Walker et al., 2016; Christensen, Lassen and Elmeros, 2012; Pay et al., 2021; Elliott et al., 2024).

Worldwide, SGAR exposure of raptors (Nakayama et al., 2019) has detrimental effects at individual and likely at population levels. SGAR poisoning at the individual level can cause impaired coagulation, internal haemorrhaging, disorientation, behavioural changes such as a decrease in feeding and alert behaviour, and in some cases death (Murray, 2018). In the United Kingdom (UK), SGAR poisoning has been confirmed as a cause of death in several raptor species (Walker et al. 2021a; Walker et al. 2017; Walker et al. 2021b; RSPB 2022; Smart et al. 2010; George et al. 2024; Molenaar et al. 2017; Broughton et al. 2022). SGAR exposure has also been theorised to have population effects via sublethal effects on fitness-related traits, including decreased parental care, bleeding during moulting, decreased body weight, and reduced growth rate (Martínez-Padilla et al., 2017; (Salim et al., 2014; Naim et al., 2011; Rattner et al., 2014). For the endangered Tasmanian wedge-tailed eagle (*Aquila*

audax fleayi), SGAR exposure is a negative factor implicated in the survival and conservation of the species (Pay et al., 2021).

SGARs are used in different ways globally to control rodents, but their usage is generally differentiated between “indoors and around buildings” and “outdoors”. Germany, The Netherlands, Belgium and the United States of America (USA) only allow the use of SGARs outdoors by trained professionals and limit the access of all SGARs to the general public (Van Den Brink et al., 2018, chap.11). In contrast, in the UK SGARs are used by amateurs (e.g. householders, smallholders) and professionals (e.g. pest controllers, farmers and gamekeepers) inside and around the immediate vicinity of buildings (Van Den Brink et al., 2018, chap.12; CRRU, 2021; European Chemicals Agency, 2018). The usage outdoors and around buildings can provide an ongoing source of SGARs into the environment, exposing wildlife through food chains (Van Den Brink et al., 2018, chap.2). The risk of exposure to SGARs is evaluated by an Environmental Risk Assessment (ERA) performed by regulators (eg. in the USA, Australia, the European Union) where all SGARs on the market fail the ERA worst-case scenario (100% of prey is contaminated) assumptions (Van Den Brink et al., 2018, chap.2; Pay et al., 2021). However, in light of observed declines in some raptor populations and known interspecies variation in foraging behaviour, it would be valuable to implement refinements on the model parameters (diet, prey residue concentration, body weight) to develop a more fine-scale understanding of risks that top predators face under different SGAR exposure scenarios (Aagaard et al., 2023). Raptors forage across different habitats with different degrees of human usage (Rattner et al., 2014), so could potentially be exposed to multiple sources of SGARs, highlighting a need to predict the risks from exposure to mixtures of SGARs given that they have similar modes of action and likely additive risk (Rattner and Mastrota, 2018).

In this study, we develop a predictive framework for estimating the exposure of raptors at three life stages to different SGARs under different ecologically relevant scenarios. We illustrate the

use of the modelling framework for populations of common kestrels (hereafter kestrel) exposed to Brodifacoum, Bromadiolone, Difenacoum and Difethialone which are currently approved for use in Europe (Van Den Brink et al., 2018, chap.2). Kestrels were chosen because the European population is in decline, and high SGAR liver residues have been reported in kestrel carcasses, implicating SGARs as a plausible cause of the population decline (Roos et al., 2021). Our scenarios mimic different stages of rodent population cycles (high and low population sizes and thus inclusion in raptor diet) and associated SGAR usage (high and low usage). For each scenario, we first investigated the exposure to each of the four SGAR compounds, and secondly a mixture of SGARs. Specifically, for breeding male and female adults, non-breeding adults, and nestlings, we:

1) Predict the exposure of kestrels to four individual SGARs under four scenarios:
    a) High SGAR usage and high percentage of rodents in diet (HS-HD).
    b) High SGAR usage and low percentage of rodents in diet (HS-LD).
    c) Low SGAR usage and high percentage of rodents in diet (LS-HD).
    d) Low SGAR usage and low proportion of rodents in diet (LS-LD).
2) Calculate whether exposure at any life stage (breeding male and female adults, non-breeding adults, and nestlings) exceeds the expected lethal or sublethal threshold to each SGAR under the four scenarios.
3) Predict whether the risk of harmful effects increases with exposure to mixtures of the two most commonly used SGARs (Difenacoum and Bromadiolone).
4) Estimate the number of days before lethal and sublethal thresholds of exposure of each SGAR are reached.

2. Methods

Our predictive framework integrates data on different levels of SGAR contamination of rodents in kestrel diet which likely reflects local SGAR usage, with proportional estimates of rodents in raptor diet, quantity of food consumed in each life stage, and body weight. The framework predicts the lethal and sublethal risk of SGAR exposure by comparing the predicted daily oral

intake of each SGAR to a toxicity threshold obtained from toxicity studies on mortality and reproductive effects from surrogate species.

Our modelling framework integrates, but extends, the tiered approaches detailed in the European Chemical Agency (ECHA) emission scenario guidelines for product type 14 (Rodenticides), which includes SGARs (European Chemicals Agency, 2018), and the European Food and Safety Agency (EFSA) 2023 (Aagaard et al., 2023) guidelines on pesticide risk assessment for birds and mammals. From ECHA, the tier-two secondary exposure was chosen, and from EFSA, the tier-one and tier-two were combined. ECHA tier 2 assesses the amount of SGARs consumed by a generic predator species based on fixed values, with diet parameters that can be refined based on specific species studies. Similarly, tier 1 from EFSA uses a generic species under worst-case scenarios, but EFSA tier 2 allows refinements for specific species. Therefore, we calculated the risk for lethality and sublethal effects based on the inputs defined in Table 1.

Table 1. Parameter values used in calculating the risk of lethal and sublethal effects and their sources. The parameter "Rodent whole body concentration of each SGAR (ng/g)" is the geometric mean of all the values reported.

Variable	Model term	Values used in the model				References
		Non-breeding Adult	Breeding Male	Breeding Female	Nestling	
Body mass (g)	BW	244	192.5	271	100	1
Daily food intake (g wet weight)	FIR	57.3	114.6	85.95	57.54	1, 2, 21
Proportion of rodents in diet	PT	Low proportion of rodents in diet (LD) = 30%				3
		High proportion of rodents in diet (HD) =78%				
Percentage of contaminated rodents in diet	PD	Low proportion of contaminated rodents in diet (LS) = 8%				3
		High proportion of contaminated rodents in diet (HS) = 23%				6
	C	Brodifacoum = 68.82				4, 5, 6, 7, 8,19

Mean rodent whole body concentration (ng/g) (Geometric mean)		Bromadiolone = 6.28	7, 8, 9,20
		Difethialone = 1.09	8
		Difenacoum = 0.72	7, 8
Lethal deterministic threshold (ng/g)		Brodifacoum = 0.55	10
		Bromadiolone = 60.67	11
		Difethialone = 0.87	12
		Difenacoum = 0.58	10
Oral dose - lethal probabilistic threshold (ng/g)		Brodifacoum = 14.28	18
		Bromadiolone = 22.85	18
		Difethialone = 11.424	18
		Difenacoum = 22.85	18
Oral dose - sublethal threshold (ng/g)		Brodifacoum = 0.01	13
		Bromadiolone = 0.38	11
		Difethialone = 0.038	12
		Difenacoum = 0.33	14
Excretion rate (given in percentage)		Brodifacoum = 17.8	15
		Bromadiolone = 53	15
		Difethialone = 25	16
		Difenacoum = 30	17

References; 1 (Masman et al., 1986); 2 (Village, 1990b); 3 Our study, see Table SM5; 4 Our study, values used to calculated "Mean rodent whole body concentration (ng/g)" are in Table SM 1 and pilot monitoring rodenticide burden is in table SM 2; ,; 5 (Geduhn et al., 2016); 6 (Geduhn et al., 2014); 7 (Tosh et al., 2012); 8 (Berny, Fourel and Lattard, 2014); 9 (Hernández et al., 2013); 10 (Newton, Wyllie and Freestone, 1990), 11 (Biocidal Products Committee, 2010a); 12 (Biocidal Products Committee, 2007); 13 (Biocidal Products Committee, 2010b), 14 (Biocidal Products Committee, 2016), 15 (Horak, Fisher and Hopkins, 2018), 16 (Prat-Mairet et al., 2017), 17 (Larsen, 2003), 18 this study, calculation of oral dose for lethal probabilistic threshold in Table SM5; 19 (Walther et al, 2021); 20 (Elmeros et al, 2019); 21 (Weathers, 1992)

The model parameterisation for each step and the details of each equation are detailed below.

2.1 Basic risk characterisation approach

The model considers secondary SGAR exposure risk for adult and nestling raptors from consuming contaminated rodents and compares this daily oral dose with toxicity thresholds for lethal and sublethal effects to derive a risk characterisation ratio (RCR).

Exposure considers the consumption by an individual kestrel of each SGAR in one day. The overall daily exposure to each SGAR is calculated using equation (1).

$$E = \left(\frac{FIR \times C \times PD}{BW}\right) * PR \quad (1)$$

Where: E is the overall daily oral intake in ng/g of a kestrel of body weight/day, FIR is the daily kestrel food intake rate in g fresh weight; BW is body weight in g; C is the SGAR concentration per fresh diet fraction expressed in ng/g; PD is the proportion of contaminated food in the kestrel diet and PR is the percentage of rodents contained in the diet (Aagaard et al., 2023; European Chemicals Agency, 2018).

The daily oral dose is compared with a toxicity threshold for lethal and sublethal effects to derive a risk characterisation ratio (RCR) (Equation 2); this ratio determines the potential risk of adverse lethal or sublethal effects following SGAR exposure. .

$$RCR\ lethal = \frac{E_i}{lethal\ threshold} \quad (2)$$

$$RCR\ sublethal = \frac{E_i}{sublethal\ threshold} \quad (3)$$

Where:

$RCR\ lethal$ is the risk characterisation ratio for lethal exposure; E is kestrel exposure to the SGAR in ng/g bw/d (equation 1). The lethal threshold (ng/g body weight/d) is derived from endpoints obtained from short-term studies (see Table SM3). A $RCR\ lethal$ value above 1 indicates that SGAR exposure represents a lethal risk under the model assumptions.

$RCR\ sublethal$ is the risk characterisation ratio for sublethal effects; E is the kestrel SGAR exposure expressed in ng/g body weight/d. The sublethal threshold (ng/g body weight/d) is derived from endpoints, for example, growth or reproductive studies (see Table SM4).

2.1.1. Predicting whether the risk of harmful effects increases with exposure to mixtures of SGARs.

Bromadiolone and Difenacoum are rodenticides with similar toxicity (Biocidal Products Committee, 2016), globally account for 56% of SGAR exposure in wild animals (Nakayama et al., 2019) and have a similar mode of action.(McGee, McGilloway and Buckle, 2020) Thus,

exposure to both rodenticides combined is predicted to increase the risk of lethal and sublethal effects. The risk for this mixture was evaluated using the concentration addition approach, which is used as an efficient approximation tool for compounds with a similar mechanism of actions like SGARs (Backhaus and Faust, 2012; Backhaus et al., 2004; Rattner et al., 2014; Rattner and Mastrota, 2018). The $RCR_{mixture}$ is the risk for the mixture of SGARs is calculated as follows:

$$RCR_{mixture\ lethal} = \sum_{i=1}^{n} RCR_{lethal} \quad (4)$$

$$RCR_{mixture\ sublethal} = \sum_{i=1}^{n} RCR_{sublethal} \quad (5)$$

Where $\sum_{i=1}^{n} R_{mixture\ lethal}$ is the sum of Bromadiolone and Difenacoum R_{lethal}. Similarly, $\sum_{i=1}^{n} R_{mixture\ sublethal}$ is the sum of Bromadiolone and Difenacoum $R_{sublethal}$.

2.2 Model parameterisation and scenarios for the common kestrel

The exposure and risk models were parameterised for the common kestrel for three life stages exposed to the four SGARs. Some parameters shown in Table 1 were obtained from a single study, and for others we used the geometric mean from the range of values from different sources because geometric mean is not driven by extreme values (Aagaard et al., 2023; European Chemicals Agency, 2018, 2017).

The quantity of SGARs in rodent prey items was estimated based on the concentration detected in the liver of rodent species (Table SM1) with values used from field studies prioritised over lab studies because they represented real-case scenarios. Only wild studies undertaken in Europe were considered, under the assumption that all the countries complied with the European regulation (European Union, 2012).

The toxicological dose descriptors used to build the threshold for lethal and sublethal effects (European Chemicals Agency, 2017), e.g. LD50 and NOEL, were extracted from assessment regulator reports (https://echa.europa.eu/information-on-chemicals), the ECOTOXicology Knowledgebase: A Curated Database of Ecologically Relevant Toxicity Tests to Support

194 Environmental Research and Risk Assessment (Olker et al., 2022; U.S. Environmental
195 Protection Agency, 2024), and from the literature (Tables SM3 and SM4). Avian toxicity test
196 studies for SGARs are limited in the literature with only three studies fulfilling the criteria of
197 control and treatment groups stated by the guidelines for avian toxicity tests (OECD, 2010,
198 1984a, 1984b).

199 2.2.1 Kestrel diet, food intake and body weight

200 *Adult food intake and body weight*

201 The kestrel is an opportunistic forager, with diet varying spatially and temporally according to
202 prey abundance, however small rodents consistently represent a large proportion of kestrel
203 diet (Village, 1990a). Females whether breeding or not weigh more than males, but both
204 consume more rodents than during the non-breeding stage, because of increases in energy
205 expenditure needed during breeding (Masman et al., 1986). Therefore, in our model female
206 and male food consumption and body weight were estimated separately. Kestrel body weight
207 and the wet weight of food consumed per day were obtained from the literature (see Table 1).

208 *Nestling food intake and body weight*

209 Here, we model the dietary uptake of 8 day old nestlings (Sylvie et al., 2002), where the
210 maximum weight gain occurred (Massemin et al. 2002). At this stage, raptors cannot
211 thermoregulate and direct all food to growth and development (Village, 1990b). There are few
212 studies evaluating kestrel food intake but there are studies evaluating growth rate and energy
213 demands of altricial birds that can be translated to food intake (Sylvie et al. 2002; Sylvie et al.
214 2003; Masman et al. 1986; Weathers 1992). We estimate the food intake by estimating the
215 daily energy demands of kestrel nestlings using the equation (6) of peak daily metabolised
216 energy (DME) for altricial birds (Weathers 1992; Daan et al. 1989).

217 $$peakDME = 5.01W^{0.782} \quad (6)$$

Where: $peakDME$ is given in KJ/ds, is the daily metabolised energy of a growing kestrel nestling W is body weight in grams.

The $peakDME$ of 183.57 KJ/day was later used to obtain nestling food intake (Fi), which depends on the energy provided by prey (Ep), which was calculated using equation (7).

$$Ep = C_{prey} * p_{dry} * Aq * K \quad (7)$$

Where: Ep is the energy provided by a prey item given in KJ/g; C_{prey} is the percentage of the prey consumed by the kestrel discarding intestines set at 0.73; p_{dry} is the dry weight proportion of the prey; Aq is the assimilation quotient. The study performed by Masman (1986) provided the Aq (0.70) and p_{dry} (0.29) by obtaining the geometric mean of the values of the common vole (*Microtus arvalis*) in March, May, and July reported in the study. (KJ/g) is the energy provided by the dry weight of the prey, which was set at 21.4 KJ/g (Masman et al., 1986).

Ep of 3.19 KJ/g and $peakDME$ (KJ/day) were replaced in equation (8) to obtain the amount of food consumed by a kestrel nestling per day and food intake (Fi) in g wet weight/day (Masman et al., 1986).

$$Fi = \frac{BMR}{Ep} \quad (8)$$

The obtained values were replaced in equation (1) as FIR. Table 1 describes the values used and the amount of calculated food intake for an 8 day-old kestrel nestling.

Proportion of rodents in kestrel diet

The model was refined by running it with either a low or high proportion of rodents in the kestrel diet. We estimated the proportion of rodents in diet by plotting diet data from our field study (see Table SM5) and from published field studies (Table SM5). Based on the first and third quartile of the data distribution, we modelled scenarios with low (30%) and high proportion of rodents in diet (78%). The models assumed no variation between the diet of breeding and

non-breeding adults and nestling diet, but the predictive framework could be refined to model sex or age-specific diets if required.

2.2.2 SGAR concentrations in prey

SGAR uptake from prey depends both on the concentration of each SGAR in the prey and the percentage of available prey that contains the SGAR. Here, we assume a fixed concentration of each SGAR in prey, but the model could include a range of values and scenarios. The SGAR liver residue concentrations in prey were obtained from studies performed during and immediately after baiting campaigns to control rodent populations in Germany, France, England and Spain (Geduhn et al. 2014; Geduhn et al. 2016; Tosh et al. 2012; Hernández et al. 2013; Berny et al. 2014; Walther et al. 2021; Elmeros et al. 2019) (See Table SM1). The concentrations of each SGAR detected in the livers (and, where available, whole bodies) of wild rodent species were collated. For liver concentrations, a whole body factor was calculated by dividing liver residue concentrations by whole-body concentrations, yielding five individual factors from the three studies in the literature (Sage et al. 2008; Giraudoux et al. 2006; Newton et al. 1990). The geometric mean of these factors, 4.97, was used to convert liver residues to estimated whole-body concentrations, then the geometric mean of the reported whole body value ranges was calculated for each evaluated SGAR.

2.2.3 *SGAR usage*

Another variable included in our predictive framework was SGAR usage which we inferred from published field studies on the percentage of rodents in foraging ranges detected containing SGARs (Table SM 1, see pilot study in Supplementary Material 2). Not all captured rodents reported in the studies were positive for SGARs; of those that tested positive the range was 8-23% (Geduhn et al., 2016, 2014; Tosh et al., 2012). Here, we compare two scenarios, low frequency (8%) and high frequency (23%) of contaminated prey (PD) (i.e. low and high usage of SGARs in the kestrel foraging range), which were replaced in equation (1) (Table 1).

2.3 Risk assessment parameterisation

To determine the potential risk of an SGAR to exposed raptors, daily oral intake is then compared with a toxicity threshold value to derive a risk characterisation ratio.

2.3.1 Lethal threshold

First, we derived a *lethal threshold* based on the lethal effects (mortality) from avian acute oral toxicity studies performed on standard model species (See Table SM3). From the studies, the toxicological dose descriptor LD_{50} was taken.

The calculation of the *lethal threshold* was performed under two approaches, deterministic and probabilistic. The deterministic approach of evaluating risk consists of selecting, across all species tested, the endpoint with the lowest value and then applying an assessment factor (AF) of 300 to cover the inter- and intraspecies differences in sensitivity to SGARs (ECHA, 2018), as represented in equation 1.

$$Lethal\ \ deterministic\ threshold = \frac{LD_{50}}{AF} \quad (9)$$

The *lethal deterministic threshold* shown in Table 1 was calculated using the LD_{50} from a non-raptor model species and raptor species. The most sensitive species for Bromadiolone reported in the literature is an unnamed species from the family Partridge (Phasinidae). For Brodifacoum and Difenacoum the oral dose was obtained from a single dose exposure study in which rodents spiked with Brodifacoum and Difenacoum were fed to Barn owl (*Tyto alba*) (Newton et al. 1990), and Northern Bobwhite Quail *(Colinus virginianus*) for Difethialone (Table SM3).

The probabilistic approach consists of building a Species Sensitivity Distribution (SSD) using LD_{50} data from all the avian species tested and obtaining the hazardous concentration for 5% of the species (HC_5) (see Table SM6, Figure SM3). The HC_5 is the concentration that protects 95% of the species in a distribution. As the SSD approach is not suitable for datasets with fewer than 8 samples (Posthuma, Suter and Traas, 2001), this approach could only be used to derive the lethal threshold of Brodifacoum. There was not enough avian data for the other

293 SGARs. The Brodifacoum SSD was built using the package ssdtools in R using the following
294 distributions: Log-normal, Weibull and Gamma. The goodness-of-fit statistic was obtained
295 from the Kolmogorov-Smirnov test, and the most parsimonious model was selected by the
296 Akaike Information Criterion corrected for small samples (AICc) and AIC weight (AICω) (Todd
297 W, 2010; Thorley and Schwarz, 2018; Sorgog and Kamo, 2019). The lethal probabilistic
298 threshold was calculated according to equation 10.

299 $$Lethal\ probabilistic\ \ threshold = \frac{HC_5}{AF} \quad (10)$$

300 Where the HC_5 is divided by an assessment factor (AF) which varies according to the standard
301 deviation of the distribution. Because the best fit distribution was log-normal and the standard
302 deviation is 0.54 and to fulfil the assumptions of the SSD of protecting 95% of the species, the
303 AF was set at 5 (Sorgog and Kamo, 2019). The $Lethal\ probabilistic\ threshold$ for the
304 remaining SGARs were obtained by using the toxicity equivalency factors (TER) for
305 Difethialone > Brodifacoum > Bromadiolone = Difenacoum, 1, 0.8, and 0.5, respectively (Elliott
306 et al., 2024). We assumed Difenacoum and Bromadiolone have similar toxicity based on read-
307 across studies for Bromadiolone from Difenacoum studies by regulators (Biocidal Products
308 Committee, 2010a, 2009).

309 2.3.2. Sublethal threshold

310 Chemical contaminants such as SGARs can elicit subtle but harmful and ecologically relevant
311 effects on individuals. We compared the daily intake of each SGAR to a threshold for sublethal
312 effects by using No Observable Effect Level (NOEL) values from reproductive studies because
313 of their ecological relevance (European Chemicals Agency, 2017; European Chemicals
314 Bureau, 2003; OECD, 2011) (See Table SM4). NOEL is the highest dose ingested where no
315 effects (e.g. mortality, lower egg hatch rate) were observed during the experimental
316 toxicological study. The lack of avian sublethal toxicity test studies for SGARs meant that the
317 sublethal threshold was first calculated with NOEL values obtained from reproductive tests
318 on Japanese quail (*Coturnix japonica*) evaluating Difenacoum toxicity. The competent

authorities approved the extrapolation of NOEL values for other rodenticides from the Difenacoum reproductive study as it was deemed unnecessary to perform more studies because of their similar mode of action (Biocidal Products Committee, 2007). The AF of 30 was used to address the interspecies variability in sensitivity (European Chemicals Agency, 2017, 2018; Aagaard et al., 2023). The sublethal threshold values of each SGAR are detailed in Table 1.

$$Sublethal\ threshold = \frac{NOEL}{AF} \quad (11)$$

2.3.3. Cumulative exposure

Chemical contamination of the environment, including from SGAR usage, can be prolonged and not limited to single exposures assumed by some regulatory approaches (Shore et al., 2018; Murray, 2020). Effects of longer-term, relatively low-level chronic exposure can be detrimental, though often less obvious (Murray, 2018). SGARs are known to bioaccumulate in the liver and can reach the thresholds for lethal and sublethal effects as exposure continues over time (Rattner et al., 2020). The rates of excretion and accumulation of SGARs in raptors is unknown and the few published pharmacokinetic studies were performed only in mammals (Horak, Fisher and Hopkins, 2018). Using the UK scenario, where SGAR baiting is advised to last up to 35 days to control a rodent infestation (CRRU, 2018), we estimated the cumulative exposure to the four rodenticides in the common kestrel using mammal excretion data (equation 12) (Aagaard et al., 2023; European Chemicals Agency, 2018). The cumulative exposure was then contrasted with our estimates of the lethal and sublethal thresholds to determine if prolonged exposure represents a risk.

$$Cumulative_{\mathrm{n}} = \sum_{n=1}^{35} E * (1 - EL)^{\mathrm{n}} + E \quad (12)$$

Where: $Cumulative_{\mathrm{n}}$ is the overall cumulative oral intake in ng/g of a kestrel over 35 days, E is the overall daily oral intake in ng/g of a kestrel of body weight/d, EL daily elimination/ excretion rate from black rats (*Rattus rattus*) for Bromadiolone (0.178) and Brodifacoum (0.530) (Horak, Fisher and Hopkins, 2018), from red fox (*Vulpes vulpes*) (Prat-Mairet et al.,

2017) for Difenacoum (0.250) and a standard value of 0.300 as the proportion of daily excretion rate in rodents for Difethialone (Larsen, 2003) because no avian data were available.

3 Results

3.1 Daily exposure of kestrels to individual SGARs

The exposure of kestrels to Brodifacoum, Bromadiolone, Difenacoum and Difethialone via diet was modelled for three life stages, with high and low SGAR usage and high and low proportions of rodents in diet (HS-HD, HS-LD, LS-HD, LS-LD). The models suggest that the concentration and type of SGAR in rodents determines the risk of exposure to kestrels. Figures 2a to 2d show that Brodifacoum has the highest dietary exposure. Kestrel exposure to Brodifacoum is 11 times more likely than to Bromadiolone, 63 times more than to Difethialone and 96 times more than to Difenacoum at all life stages and for all scenarios (Table SM7).

Different raptor life stages are predicted to be exposed to different amounts of SGARs, with the breeding male stage and nestling having higher exposure than the non-breeding stages and the breeding female stage, as shown in Figure 2. In this kestrel case study, this difference is due to the mass of food consumed relative to body size. Breeding male kestrels do the majority of hunting while females are incubating and brooding, have the highest food intake and thus SGAR exposure in all the evaluated scenarios. Nestlings (in our example an 8.3 day old 100g nestling) are more exposed to SGARs through diet than non-breeding adults, because of their high food intake compared to body size. Life stage specific diets could be incorporated into our model which could alter age-specific exposure and thus risk.

3.2. Risk assessment

Next, we assessed exposure compared to the lethal and sublethal thresholds. The lethal risk of SGAR exposure varied according to the approach used to calculate the threshold with the deterministic approach giving more conservative results than the probabilistic approach (Figure 3). The probabilistic lethal risk from exposure to SGARs via diet does exceed the risk characterisation ratio for any of the life stages in any of the scenarios. In contrast, under the

deterministic approach, Brodifacoum exposure represents a risk in all three life stages in three scenarios the high contamination and high rodent diet scenario (HS-HD), the high contamination and low rodent diet (HS-LD) and low contamination and high rodent diet (LS-HD) (RCR>1).

Breeding males and nestlings are estimated to be at risk of lethal effects under the probabilistic approach in all scenarios. Bromadiolone, Difenacoum, and Difethialone are not predicted to pose a lethal risk after a single exposure via diet.Breeding males and nestlings are predicted to be at risk of sublethal effects from exposure to Brodifacoum a in all scenarios and for difethialone in three scenarios (HS-HD, HS-LD, LS-HD) (Figure 3C). Non-breeding adults and breeding females are at risk of sublethal effects under all the scenarios for Brodifacoum exposure but not for Difethialone as LS-LD, HS-LD and LS-HD do not represent a risk. Bromadiolone exposure represents a sublethal risk to breeding males under the HS-HD scenario; the other three scenarios represent no risk. Non-breeding adults, breeding females and nestlings are not at risk of sublethal effects under any of the Bromadiolone exposure scenarios. Difenacoum exposure represents no risk of sublethal effects for any life stage or dietary scenario.

As raptors are likely to be exposed to mixtures of SGARs in diet, it is valuable to investigate risks posed by the two SGARs commonly reported in raptor carcasses. Difenacoum and Bromadiolone. Regardless of whether the lethal threshold is calculated using the probabilistic or deterministic approach, exposure to these SGARs together does not represent a risk for the kestrel in any life stages and dietary scenarios. For sublethal effects, this mixture represents a risk for breeding males, breeding females and nestlings under the HS-HD scenario (Figure 4).

3.3. Cumulative exposure

The cumulative exposure of kestrels to SGARs indicates that for all life stages and dietary scenarios it takes longer to reach the probabilistic threshold for lethal effects than the

deterministic threshold. Brodifacoum reaches the probabilistic threshold after 12days, with only the HS-HD scenario reaching the threshold for non-breeding male, and 3 days for breeding male and 8 days for nestlings(Figure 5A), for the other life stages and scenarios the cumulative exposure does not reach the probabilistic threshold after 35 days. Similarly, Bromadiolone, Difenacoum and Difethialone do not reach the probabilistic and deterministic lethal effects after 35 days of exposure (Figure 5B, 5C, 5D). Brodifacoum reaches the deterministic threshold in 2 days for the LS-LD scenario for the breeding female, non-breeding male and non-breeding female (Figure 5A). In the case of the sublethal exposure, Brodifacoum exceeded the threshold in all life stage and dietary scenarios (Figure 5A). Bromadiolone intake did not reach the threshold of 0.38 ng/g after 35 days for all the scenarios and life stages with the exception of breeding male and nestling in the HS-HD scenario exceeding the threshold of 0.38ng/g after 2 days of continuous exposure (Figure 5B). In contrast, Difenacoum does not exceed the threshold in the 35 days of treatment for any of the life stages e (Figure 5C). Difethialone sublethal exposure surpassed the threshold for non-breeding males, non-breeding females and breeding female after 4 to 7 days exposure in the scenarios of HS-LD and LS-HD. For Nestlings and breeding male under the HS-HD scenario, cumulative exposure reaches the threshold of 0.038ng/g within 1 day. For all life stages, the LS-LD it does not reach the threshold within 35 days (Figure 5D).

4 Discussion

We evaluated the lethal and sublethal risk of SGAR exposure to raptors by implementing a predictive framework that integrated both field data and standard species toxicology test data. The risk was modelled by comparing the estimated daily oral intake of each SGAR by a raptor to a toxicity threshold obtained from lethal and sublethal toxicity studies on avian species. The common kestrel was the case study chosen to implement the framework because of the concern generated by population decrease and high residues of SGARs in liver samples from kestrels found dead in the wild. The modelling framework indicates that from the four evaluated SGARs, Brodifacoum exposure presents the greatest overall risk to kestrels. When the lethal

effects threshold is calculated using the deterministic approach, the risk is across ecological and SGAR usage scenarios for all life stages. In contrast, when the lethal threshold is determined using the probabilistic approach, SGARs do not represent a lethal risk .). In terms of risk of sublethal effects, Brodifacoum represents a risk under all scenarios to all life stages and Difethialone in all scenarios except for non-breeding adults and breeding females under the LS-LD scenario. Bromadiolone represents a risk of sublethal effects to breeding males under single exposure and in a mixture. Difenacoum represents minimal risk of lethal and sublethal effects under our modelling assumptions after a single exposure but can pose a risk as part of a mixture with Bromadiolone. Overall, the level of risk is associated with the type of SGAR, the concentration of SGAR residues in rodents, the proportion of rodents in diet, the proportion of contaminated rodent prey and the life stage of the raptor. If SGAR usage were to increase in duration or spatial scale, for example, or there was a shift to using more toxic SGARs or combinations of multiple products, then the estimated risk to raptors would increase.

4.1 Overall exposure

Our results can inform changes to SGAR management and raptor conservation by helping to predict when, how and which type of SGAR could be used to control rodents whilst minimising the likelihood of secondary poisoning. The SGAR with the highest dietary exposure level is Brodifacoum in all life stages and prey scenarios. Furthermore, the concentration of Brodifacoum in rodents is higher and accumulates faster than for Bromadiolone, Difenacoum, and Difethialone (Figure 5, Table SM1), probably because rodents accumulate Brodifacoum in the liver and most likely retain it until they die (liver half-life of up to 307.4 days and excretion rates in faeces of 14-21.7%) (Vandenbroucke et al., 2008; Horak, Fisher and Hopkins, 2018; Eason et al., 2002; Prat-Mairet et al., 2017). In contrast, the SGARs with predicted lower daily intake by raptors (Table SM7), Bromadiolone, Difenacoum and Difethialone, have a liver half-life in rodents of 28.1 days, 61.8 days and 28.5 days respectively (Vandenbroucke et al., 2008), with excretion rates of 25-53% (Horak, Fisher and Hopkins, 2018; Prat-Mairet et al., 2017; Larsen, 2003). Therefore, using SGARs with a high bioaccumulation factor, like

Brodifacoum, pose a particular risk of secondary poisoning because of their persistence in rodents. In addition, rodents do not immediately die after SGAR exposure; the latent period after consuming a lethal dose is 6 -10 days (Mathur and Prakash, 1981; Laakso, Suomalainen and Koivisto, 2010; Erickson and Urban, 2004; Bailey, Fisher and Eason, 2005). Rodents resistant to SGARs or exposed to non-lethal doses can become nomadic, expanding their homerange (McGee, McGilloway and Buckle, 2020). This has been reported in England, where 14% of rodents in farms with no active SGAR baiting were found to contain rodenticide residues in the liver (Tosh et al., 2012). Thus, our model suggests that to minimise sublethal and lethal effects on raptors, alternatives to Brodifacoum and Difethialone should be considered.

4.2 Risk and its drivers

Studies performed on the trophic transfer of SGARs to top predators and scavengers show that rodenticide residues in rodents modulate secondary exposure and its consequent effects (Geduhn et al., 2016; Coeurdassier et al., 2012; Tosh et al., 2011; Baudrot et al., 2020). During a SGAR baiting campaign, the barn owl's risk of exposure to rodenticides increased according to the proportion of pest species (e.g. *Mus musculus*, *Rattus rattus*) found in the owl pellets, i.e. in the diet. During winter, the risk increased as barn owls' prey more on rats, a target species for rodenticide usage, than non-target voles (Geduhn et al., 2016). Similarly, in France, the risk of exposure of red kites to Bromadiolone during a baiting campaign to control water voles (*Arvicola amphibius*) increased due to the higher availability of prey carcasses containing SGAR residues and the lack of removal of the rodent carcasses (Coeurdassier et al., 2012). In terms of SGAR management, we suggest that the quick removal of carcasses during baiting campaigns is crucial to minimise secondary exposure and its associated risk to raptors and other scavengers. Moreover, it would be interesting to consider how farm managers could encourage raptors to locate away from farm buildings especially during the breeding season and reduce reliance on synanthropic species and switch to non-pest prey by managing the wider landscape for wildlife including small mammals and birds (Broughton et al., 2014).

4.3 Model limitations and enhancements

Here, we assumed the proportion of rodents in the diets of raptors is the same for nestlings and for adults during the breeding and non-breeding seasons. However, the model could be refined to examine the effect of varying diets on different life stages and seasons of the year (Hernout et al., 2018). This could be informed by field data on raptor diets and/or ecological studies of seasonal population cycles in rodents (Village, 1982; Burthe et al., 2010; Carslake et al., 2011; Delattre et al., 1999). For example, (Geduhn et al., 2016) evaluated adult barn owl diet throughout the seasons and reported that in winter they rely more on rats and during spring and summer (breeding period of barn owls) the diet is mostly *Microtus* voles. Changes in raptor diet according to prey availability and energy demands modulate the risk of exposure and thus the effect of SGARs. The reliance of predators on SGAR target species during winter and the reported increase during autumn and winter in SGAR usage to protect grains in barns can increase the risk of exposure to raptors (Geduhn et al., 2016; Campbell et al., 2022). Therefore, in terms of management, our model outputs suggest that to control the accessibility of SGARs to the general public and pest control professionals during winter and refrain from using all SGARs during the breeding season of raptors.

The lack of studies on raptor toxicity has some effect on the RCR calculation because of differences in species sensitivity. The toxicological endpoints used were generally obtained from standard study species (eg. Japanese Quail (Coturnix japonica)), which are suspected to be more resilient to SGAR exposure than raptors (Rattner and Mastrota, 2018). The affinity of Vitamin K epoxide reductase (VKOR), the enzyme involved in making the coagulation factors, is 20 to 30 times less sensitive in granivorous species (eg. Japanese quail) than raptors whose VKOR is easily inhibited by SGARs (Khidkhan et al., 2024; Nakayama et al., 2020). These differences in species sensitivity to SGARs significantly influence both lethal and sublethal threshold calculations (Xu et al. 2015). This is evident in two key ways: first, through the large probabilistic thresholds where high LD50 values skew the HC5 (Wheeler et al. 2002) and subsequently affect the RCR (Figure 3); second, through toxicity variations between species that don't accurately reflect SGAR potency, as seen in the Brodifacoum-Difethialone

comparison where lethal effect thresholds are similar but Brodifacoum shows 3-fold greater toxicity for sublethal effects. This sublethal effect variability in birds (Rattner and Harvey 2021) was particularly apparent in our study, which incorporated two distinct endpoints - weight change and a read accros reproductive test results from two different species the Japanese quail (Coturnix japonica) and the American kestrel Falco sparverius.

Our framework can be implemented to predict differences in exposure to SGARs, focusing on sublethal endpoints (NOEL) including age- and sex-specific, and other relevant toxicological endpoints, where available. Martínez-Padilla et al., (2017) found that kestrel nestlings exposed to Bromadiolone had lower body mass and body condition associated with lower recruitment rate. In red kites, adults carry higher SGAR body burders because SGARs bioaccumulate overtime (Walker et al., 2016), barn owls exposed to SGARs increased their coagulation time (Hindmarch, Rattner and Elliott, 2019) and barn owls exposed to rodenticides have lower fledging success and brood size (Salim et al., 2014). Although previous studies indicated that SGARs can affect wild species in non-lethal ways that are hypothesized to link to population level effects (Rattner et al., 2014), there is a lack of studies with non-standard benchmarks that can be used to refine our framework. A key to addressing this is developing less-invasive (Biocidal Products Committee, 2007; Espín et al., 2016), inexpensive and quick biomarkers, such as the coagulation time of blood samples (Rached et al. 2020; Spadetto et al. 2025), that are correlated with fitness-related traits such as growth, survival and/or reproduction. This will allow enhancement of risk assessments and protection of wildlife (Rattner et al., 2020; Baudrot et al., 2020).

Across Europe, rat and mice populations are becoming resistant to Bromadiolone and Difenacoum (McGee, McGilloway and Buckle, 2020). Resistant rodents are potentially going to be carrying higher residue burdens and remain active in the environment for longer periods, so pose a greater risk to predators than non-resistant individuals. Also, unless tighter regulation is introduced, there is likely to be an increase in the use of more potent rodenticides in parts of the world where SGAR resistance is rising. SGAR sales and usage data are not freely available, but indirect metrics can be used to infer changes in spatial and temporal

patterns of usage. For example, in the UK (Ozaki et al., 2024, 2023; George et al., 2024) there are records of higher rates of detection of more potent SGARs, such as Brodifacoum, residues in buzzards (Ozaki et al., 2024), barn owls (Ozaki et al., 2023) and red foxes (*Vulpes vulpes*) (Campbell et al., 2024). In addition, Difethialone, a rodenticide not previously reported in non-target species in the UK, has started to be detected (Walker et al., 2021b; Shore et al., 2019; Walker et al., 2021a). Difethialone and Brodifacoum presence in livers of top predators is of concern because of their known toxicity to raptors (Elliott et al., 2024) and emphasises the need to implement or strengthen mitigation strategies.

5. Conclusions

Our modelling framework can be used to predict the risk to non-target wildlife associated with rodenticide usage and aims to be useful for regulators to explore the effects of changes to SGAR usage on specific raptor species. For example, our framework can model the potential impacts of baiting campaigns using different SGARs allowing the identification of the duration of active baiting that minimises effects on raptors within the focal area, optimal management strategy for removing rodent carcasses refined by the ecology of raptors, e.g. scavenging versus hunting, and availability of alternative prey species relative to target rodents. Raptor conservation managers can implement the model as it integrates the interaction between diet and life stages and the risk of lethal and sublethal effects on raptors which are often exposed to contaminants as well as multi-stressor variables such as competition and predation. Finally, future iterations of the framework could be used to model scenarios in countries where data on SGAR usage or raptor ecology are lacking and for species that are of conservation concern. Our model does emphasise the wide intra-species variation in sensitivity to SGARs, the paucity of toxicological endpoint data in non-model species and how this can affect the effectiveness of current methods to calculate lethal thresholds and thus protect wildlife. Overall, our framework provides the regulators, land managers and conservation organisations with a tool to identify areas and seasons where SGAR usage should be minimised in order to prevent lethal and sublethal impacts on non-target wildlife.

CRediT authorship contribution statement

Ciara Sanchez-Paredes: Writing – original draft, Visualization, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization, Project administration. **Malcolm Burgess:** Writing – review & editing, Supervision, Funding acquisition, Resources, Methodology, Investigation, Conceptualization. **Michael Fryer:** Writing – review & editing, Methodology, Conceptualization. **Gloria Dos Santos Pereira:** Writing – review & editing, Methodology. **Kathryn Arnold:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

All the used data is public available in the Supplementary Material, the link for the dietary data will be generated in dryad after submitting the manuscript.

Acknowledgments

We are thankful to the BTO licensed bird ringers that allowed us to monitor and collect data in the field- Colin Ackroyd, Ken Pearson, Colin Bradford, Colin Gibson, David Aitken, Eric Wood, Dave Hazard and Dave Tates. We appreciated the help of the field assistants- Briza Cristina Alves, Kimberly Holmes, Luke Andrews. We acknowledge the guidance of Dr. Georgia Jones and Jason Father during the rodent sampling and pilot study. We also thank farmers who allowed us access. We are thankful of Heather Carter and Darren Sleep for their guidance and knowledge during the laboratory work and for doing the Mass Spectrometer analysis. We acknowledge the Chemical Regulation Division from HSE for helping in the interpretation and implementation of the ECHA guidelines.

Funding

The project was funded by the ECORISC NERC CDT, reference number: NE/V012041/1, the RSPB and Natural England through the Action for Birds in England partnership.

Ethics

The animal monitoring in the Supplementary Material followed animal ethics permit approved by the University of York Animal Welfare and Ethical Review Body (AWERB).

References

Aagaard, A. et al. (2023). Risk assessment for Birds and Mammals. *EFSA Journal*, 21 (2). [Online]. Available at: doi:10.2903/j.efsa.2023.7790.

Backhaus, T. et al. (2004). Joint algal toxicity of phenylurea herbicides is equally predictable by concentration addition and independent action. *Environmental toxicology and chemistry / SETAC*, 23 (2), pp.258–264. [Online]. Available at: doi:10.1897/02-497.

Backhaus, T. and Faust, M. (2012). Predictive Environmental Risk Assessment of Chemical Mixtures: A Conceptual Framework. *Environmental science & technology*, 46 (5), pp.2564–2573. [Online]. Available at: doi:10.1021/es2034125.

Bailey, C., Fisher, P. and Eason, C. T. (2005). Assessing anticoagulant resistance in rats and coagulation effects in birds using small-volume blood samples. *Science for conservation* , (249), pp.5–21.

Baudrot, V. et al. (2020). Trophic transfer of pesticides: The fine line between predator–prey regulation and pesticide–pest regulation. *The Journal of applied ecology*, 57 (4), pp.806–818. [Online]. Available at: doi:10.1111/1365-2664.13578.

Berny, P. et al. (2015). VIGILANCE POISON: Illegal poisoning and lead intoxication are the main factors affecting avian scavenger survival in the Pyrenees (France). *Ecotoxicology and environmental safety*, 118, pp.71–82. [Online]. Available at: doi:10.1016/j.ecoenv.2015.04.003.

Berny, P. J., Fourel, I. and Lattard, V. (2014). Anticoagulant Rodenticides: Resistance and Residues in Norway Rats in France. *Proceedings of the Vertebrate Pest Conference*, 26. [Online]. Available at: doi:10.5070/v426110404.

Biocidal Products Comittee. (2016). *Assessment Report Difethialone Product-type 14 (Rodenticides)*. European Chemical Agency. [Online]. Available at: https://echa.europa.eu/documents/10162/d92237fb-4a5a-b1ef-c0e7-c8f009a12a9d.

Biocidal Products Committee. (2007). *Assessment Report Difethialone Product-type 14 (Rodenticides)*. European Chemicals Agency.

Biocidal Products Committee. (2009). *Assessment Report Difenacoum Product-type 14 (Rodenticides)*. European Chemicals Agency.

Biocidal Products Committee. (2010a). *Assessment Report Bromadiolone Product-type 14 (Rodenticide)*. European Chemicals Agency.

Biocidal Products Committee. (2010b). *Assessment Report Bromadiolone Product-type 14 (Rodenticides)*. European Chemicals Agency.

Biocidal Products Committee. (2016). *Assessment Report Difenacoum Product-type 14 (Rodenticides)*. European Chemicals Agency.

Broughton, R. K., Searle, K. R., Walker, L. A., Butler, A., Potter, E. D., Pereira, M. G., Carter, H., Sleep, D., Noble, D. G. and Johnson, A. C. (2022). Long-Term Trends of Second Generation Anticoagulant Rodenticides (Sgars) Show Widespread Contamination of a Bird-Eating Predator, the Eurasian Sparrowhawk (Accipiter Nisus) in Britain. SSRN Electronic Journal, pp.1–32. [Online]. Available at: doi:10.2139/ssrn.4144377.

Broughton, R. K. et al. (2014). Agri-environment scheme enhances small mammal diversity and abundance at the farm-scale. *Agriculture, ecosystems & environment*, 192, pp.122–129. [Online]. Available at: doi:10.1016/j.agee.2014.04.009.

Burthe, S. J. et al. (2010). Individual growth rates in natural field vole, Microtus agrestis, populations exhibiting cyclic population dynamics. *Oecologia*, 162 (3), pp.653–661. [Online]. Available at: doi:10.1007/s00442-009-1495-6.

Butet, A. et al. (2010). Responses of common buzzard (Buteo buteo) and Eurasian kestrel (Falco tinnunculus) to land use changes in agricultural landscapes of Western France. *Agriculture, ecosystems & environment*, 138 (3), pp.152–159. [Online]. Available at: doi:10.1016/j.agee.2010.04.011.

Butet, A., Rantier, Y. and Bergerot, B. (2022). Land use changes and raptor population trends: A twelve-year monitoring of two common species in agricultural landscapes of Western France. *Global Ecology and Conservation*, 34, p.e02027. [Online]. Available at: doi:10.1016/j.gecco.2022.e02027.

Campbell, S. et al. (2022). *Pesticide Usage in Scotland - Rodenticides on Arable Farms 2022*. Science & Advice for Scottish Agriculture (SASA).

Campbell, S. et al. (2024). Impact of changes in governance for anticoagulant rodenticide use on non-target exposure in red foxes (Vulpes vulpes). *Environmental Chemistry and Ecotoxicology*, 6, pp.65–70. [Online]. Available at: doi:10.1016/j.enceco.2024.01.001.

Carslake, D. et al. (2011). Spatio-temporal covariation in abundance between the cyclic common vole Microtus arvalis and other small mammal prey species. *Ecography*, 34 (2), pp.327–335. [Online]. Available at: doi:10.1111/j.1600-0587.2010.06334.x.

Christensen, T. K., Lassen, P. and Elmeros, M. (2012). High exposure rates of anticoagulant rodenticides in predatory bird species in intensively managed landscapes in Denmark. *Archives of environmental contamination and toxicology*, 63 (3), pp.437–444. [Online]. Available at: doi:10.1007/s00244-012-9771-6.

Coeurdassier, M. et al. (2012). The diet of migrant Red Kites Milvus milvus during a Water Vole Arvicola terrestris outbreak in eastern France and the associated risk of secondary poisoning by the rodenticide bromadiolone. *The Ibis*, 154 (1), pp.136–146. [Online]. Available at: doi:10.1111/j.1474-919X.2011.01193.x.

CRRU. (2018). *Guidance Permanent Baiting*. The Campaign for Responsible Rodenticide Use (CRRU) UK.

CRRU. (2021). *Best Practice and Guidance for Rodent Control and the Safe Use of Rodenticides*. Campaign for Responsible Rodenticide Use (CRRU).

Daan, S. et al. (1989). Intraspecific Allometry of Basal Metabolic Rate: Relations with Body Size, Temperature, Composition, and Circadian Phase in the Kestrel, Falco tinnunculus.

Journal of biological rhythms, 4 (2), pp.155–171. [Online]. Available at: doi:10.1177/074873048900400212.

Delattre, P. et al. (1999). Vole outbreaks in a landscape context: Evidence from a six year study of Microtus arvalis. *Landscape ecology*, 14 (4), pp.401–412. [Online]. Available at: doi:10.1023/A:1008022727025.

Eason, C. T. et al. (2002). Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. *Ecotoxicology* , 11 (1), pp.35–48. [Online]. Available at: doi:10.1023/A:1013793029831.

Elliott, J. E. et al. (2024). Anticoagulant Rodenticide Toxicity in Terrestrial Raptors: Tools to Estimate the Impact on Populations in North America and Globally. *Environmental toxicology and chemistry / SETAC*, 43 (5), pp.988–998. [Online]. Available at: doi:10.1002/etc.5829.

Elmeros, M., Bossi, R., Christensen, T. K., Kjær, L. J., Lassen, P. and Topping, C. J. (2019). Exposure of non-target small mammals to anticoagulant rodenticide during chemical rodent control operations. Environmental science and pollution research international, 26 (6), Springer Science and Business Media LLC., pp.6133–6140. [Online]. Available at: doi:10.1007/s11356-018-04064-3.

Erickson, W. and Urban, D. (2004). *Potential Risks of Nine Rodenticides to Birds and Nontarget Mammals: a Comparative Approach*. Environmental Protection Agency (US).

Espín, S. et al. (2016). Tracking pan-continental trends in environmental contamination using sentinel raptors—what types of samples should we use? *Ecotoxicology* , 25 (4), pp.777–801. [Online]. Available at: doi:10.1007/s10646-016-1636-8.

European Chemicals Agency. (2017). *Guidance on Biocidal Products Regulation: Volume IV Environment - Assessment and Evaluation (Parts B+C)*. European Chemicals Agency. [Online]. Available at: doi:10.2823/033935.

European Chemicals Agency. (2018). *Revised Emission Scenario Document for Product Type 14*. European Chemicals Agency. [Online]. Available at: doi:10.2823/660595.

European Chemicals Bureau. (2003). *Technical Guidance Document on Risk Assessment in support of Commission Directive 93/67/EEC on Risk Assessment for new notified substances - Commission Regulation (EC) No 1488/94 on Risk Assessment for existing substances - Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market*. European Commission Joint Research Centre., p.337. [Online]. Available at: doi:10.1002/mp.12308.

European Union. (2012). *Regulation (EU) no 528/2012 of the European Parliament and of the Council*. Official Journal of The European Union.

Geduhn, A. et al. (2014). Spatial and temporal exposure patterns in non-target small mammals during brodifacoum rat control. *The Science of the total environment*, 496, pp.328–338. [Online]. Available at: doi:10.1016/j.scitotenv.2014.07.049.

Geduhn, A. et al. (2016). Prey composition modulates exposure risk to anticoagulant rodenticides in a sentinel predator, the barn owl. *The Science of the total environment*, 544, pp.150–157. [Online]. Available at: doi:10.1016/j.scitotenv.2015.11.117.

George, S. et al. (2024). Anticoagulant rodenticide exposure in common buzzards: Impact of new rules for rodenticide use. *The Science of the total environment*, 944 (173832), p.173832. [Online]. Available at: doi:10.1016/j.scitotenv.2024.173832.

715 Giraudoux, P., Tremollières, C., Barbier, B., Defaut, R., Rieffel, D., Bernard, N., Lucot, É. and
716 Berny, P. (2006). Persistence of bromadiolone anticoagulant rodenticide in Arvicola terrestris
717 populations after field control. Environmental research, 102 (3), pp.291–298. [Online].
718 Available at: doi:10.1016/j.envres.2006.02.008.

719 Green, R. E. et al. (2004). Diclofenac poisoning as a cause of vulture population declines
720 across the Indian subcontinent. *The journal of applied ecology*, 41 (5), pp.793–800. [Online].
721 Available at: doi:10.1111/j.0021-8901.2004.00954.x.

722 Hernández, A. M. et al. (2013). Analysis of anticoagulant rodenticide residues in Microtus
723 arvalis tissues by liquid chromatography with diode array, fluorescence and mass
724 spectrometry detection. *Journal of chromatography. B, Analytical technologies in the*
725 *biomedical and life sciences*, 925, pp.76–85. [Online]. Available at:
726 doi:10.1016/j.jchromb.2013.02.032.

727 Hernout, B. V. et al. (2018). Interspecific variation in the spatially-explicit risks of trace metals
728 to songbirds. *The Science of the total environment*, 642, pp.679–689. [Online]. Available at:
729 doi:10.1016/j.scitotenv.2018.05.390.

730 Hindmarch, S., Rattner, B. A. and Elliott, J. E. (2019). Use of blood clotting assays to assess
731 potential anticoagulant rodenticide exposure and effects in free-ranging birds of prey. *The*
732 *Science of the total environment*, 657, pp.1205–1216. [Online]. Available at:
733 doi:10.1016/j.scitotenv.2018.11.485.

734 Horak, K. E., Fisher, P. M. and Hopkins, B. (2018). Pharmacokinetics of Anticoagulant
735 Rodenticides in Target and Non-target Organisms. In: Van den Brink, N. W. et al. (Eds).
736 *Anticoagulant Rodenticides and Wildlife*. Springer International Publishing. pp.87–108.
737 [Online]. Available at: doi:10.1007/978-3-319-64377-9_4.

738 Khidkhan, K., Yasuhira, F., Saengtienchai, A., Kasorndorkbua, C., Sitdhibutr, R., Ogasawara,
739 K., Adachi, H., Watanabe, Y., Saito, K., Sakai, H., et al. (2024). Evaluation of anticoagulant
740 rodenticide sensitivity by examining in vivo and in vitro responses in avian species, focusing
741 on raptors. Environmental pollution , 341, Elsevier., p.122837. [Online]. Available at:
742 doi:10.1016/j.envpol.2023.122837.

743 Laakso, S., Suomalainen, K. and Koivisto, S. (2010). *Literature Review on Residues of*
744 *Anticoagulant Rodenticides in Non-Target Animals*, TemaNord. Nordic Council of Ministers.
745 [Online]. Available at: doi:10.6027/tn2010-541.

746 Larsen, J. (2003). *Supplement to the methodology for risk evaluation of biocides Emission*
747 *scenario document for biocides used as rodenticides*. Danish Environment Protection Agency.

748 Lemus, J. A., Bravo, C., García-Montijano, M., Palacín, C., Ponce, C., Magaña, M. and Alonso,
749 J. C. (2011). Side effects of rodent control on non-target species: Rodenticides increase
750 parasite and pathogen burden in great bustards. The Science of the total environment, 409
751 (22), Elsevier B.V.., pp.4729–4734. [Online]. Available at:
752 doi:10.1016/j.scitotenv.2011.07.007.

753 Martínez-Padilla, J. et al. (2017). A negative association between bromadiolone exposure and
754 nestling body condition in common kestrels: management implications for vole outbreaks. *Pest*
755 *management science*, 73 (2), pp.364–370. [Online]. Available at: doi:10.1002/ps.4435.

756 Martínez-Ruiz, M. et al. (2023). Conservation letter: Effects of global climate change on
757 Raptors. *The Journal of raptor research*, 57 (1). [Online]. Available at: doi:10.3356/jrr-22-75.

758 Masman, D., Gordijn, M., Daan, S. and Dijkstra, C. (1986). Ecological Energetics of the

759 Kestrel. Ardea, 74, pp.24–39.

760 Massemin, S., Korpimaki, E., Poyri, V. and Zorn, T. (2002). Influence of hatching order on
761 growth rate and resting metabolism of kestrel. Journal of avian biology, 33, pp.235–244.
762 [Online]. Available at: doi:10.1034/j.1600-048X.2002.330305.x.

763 Mathur, R. P. and Prakash, I. (1981). Evaluation of brodifacoum against T. indica, M. hurrianae
764 and R. rattus. *The Journal of hygiene*, 87 (2), pp.179–184. [Online]. Available at:
765 doi:10.1017/S0022172400069382.

766 McGee, C. F., McGilloway, D. A. and Buckle, A. P. (2020). Anticoagulant rodenticides and
767 resistance development in rodent pest species – A comprehensive review. *Journal of Stored*
768 *Products Research*, 88, Elsevier Ltd. [Online]. Available at: doi:10.1016/j.jspr.2020.101688.

769 Méndez, D. et al. (2021). Large Andean condor aggregations at carcasses exacerbate the
770 threat of poisoning. *The Journal of raptor research*, 55 (3). [Online]. Available at:
771 doi:10.3356/jrr-20-26.

772 Molenaar, F. M., Jaffe, J. E., Carter, I., Barnett, E. A., Shore, R. F., Marcus Rowcliffe, J. and
773 Sainsbury, A. W. (2017). Poisoning of reintroduced red kites (Milvus Milvus) in England.
774 European journal of wildlife research, 63 (6), European Journal of Wildlife Research., p.94.
775 [Online]. Available at: doi:10.1007/s10344-017-1152-z.

776 Murray, M. (2018). Ante-mortem and Post-mortem Signs of Anticoagulant Rodenticide
777 Toxicosis in Birds of Prey. In: Van den Brink, N. W. et al. (Eds). *Richard F.* Springer Nature.
778 pp.109–134. [Online]. Available at: doi:10.1007/978-3-319-64377-9_5.

779 Murray, M. (2020). Continued anticoagulant rodenticide exposure of red-tailed hawks (Buteo
780 jamaicensis) in the northeastern United States with an evaluation of serum for biomonitoring.
781 *Environmental toxicology and chemistry*, 39 (11), pp.2325–2335. [Online]. Available at:
782 doi:10.1002/etc.4853 [Accessed 26 February 2025].

783 Naim, M. et al. (2011). Comparison of the Breeding Performance of the Barn Owl Tyto alba
784 javanica under Chemical and Bio-based Rodenticide Baiting in Immature Oil Palms in
785 Malaysia. *Dynamic biochemistry, process biotechnology & molecular biology*, 5 (Special2),
786 pp.5–11. [Online]. Available at:
787 http://www.globalsciencebooks.info/Online/GSBOnline/images/2011/DBPBMB_5(SI2)/DBPB
788 MB_5(SI2)5-11o.pdf.

789 Nakayama, S. M. M., Morita, A., Ikenaka, Y., Kawai, Y. K., Watanabe, K. P., Ishii, C.,
790 Mizukawa, H., Yohannes, Y. B., Saito, K., Watanabe, Y., et al. (2020). Avian interspecific
791 differences in VKOR activity and inhibition: Insights from amino acid sequence and mRNA
792 expression ratio of VKORC1 and VKORC1L1. Comparative biochemistry and physiology.
793 Toxicology & pharmacology: CBP, 228, Elsevier., p.108635. [Online]. Available at:
794 doi:10.1016/j.cbpc.2019.108635

795 Nakayama, S. M. M. et al. (2019). A review: Poisoning by anticoagulant rodenticides in non-
796 target animals globally. *The Journal of veterinary medical science / the Japanese Society of*
797 *Veterinary Science*, 81 (2), pp.298–313. [Online]. Available at: doi:10.1292/jvms.17-0717.

798 Nemček, V. (2013). Abundance ofraptors and habitat preferences ofthe common buzzard
799 Buteo buteo and the common kestrel Falco tinnunculus during the non-breeding season in an
800 agricultural landscape (western Slovakia). *Slovak Raptor Journal*, 7, pp.37–42. [Online].
801 Available at: doi:10.2478/srj-2013-0007.

802 Newton, I., Wyllie, I. and Freestone, P. (1990). Rodenticides in British barn owls.

Environmental pollution (Barking, Essex: 1987), 68 (1-2), pp.101–117. [Online]. Available at: doi:10.1016/0269-7491(90)90015-5.

OECD. (1984a). *OECD Guidelines for the Testing of Chemicals, Section 2 Test No. 205: Avian Dietary Toxicity Test*. OECD Publishing. [Online]. Available at: https://play.google.com/store/books/details?id=gu1Ns6XtFfIC.

OECD. (1984b). *OECD Guidelines for the Testing of Chemicals, Section 2 Test No. 206: Avian Reproduction Test*. OECD Publishing. [Online]. Available at: https://play.google.com/store/books/details?id=B4HCsIXf7OcC.

OECD. (2010). *Test No. 223: Avian Acute Oral Toxicity Test*. OECD Publishing. [Online]. Available at: https://play.google.com/store/books/details?id=Jutw2dHAzW0C.

OECD. (2011). *CHAPTER 4: Initial assessment of data*. The Organisation for Economic Co-operation and Development., pp.1–32. [Online]. Available at: http://www.oecd.org/chemicalsafety/risk-assessment/49188998.pdf.

Olker, J. H. et al. (2022). The ECOTOXicology Knowledgebase: A curated database of ecologically relevant toxicity tests to support environmental research and risk assessment. *Environmental toxicology and chemistry*, 41 (6), pp.1520–1539. [Online]. Available at: doi:10.1002/etc.5324.

Ozaki, S. et al. (2023). *Second generation anticoagulant rodenticide residues in barn owls 2022*. Lancaster: UK Centre for Ecology & Hydrology contract report to the Campaign for Responsible Rodenticide Use (CRRU)., p.25. [Online]. Available at: https://nora.nerc.ac.uk/id/eprint/536370/ [Accessed 9 July 2024].

Ozaki, S. et al. (2024). Significant Turning Point: Common Buzzard (Buteo buteo) Exposure to Second-Generation Anticoagulant Rodenticides in the United Kingdom. *Environmental science & technology*, 58 (14), pp.6093–6104. [Online]. Available at: doi:10.1021/acs.est.3c09052.

Pay, J. M. et al. (2021). Endangered Australian top predator is frequently exposed to anticoagulant rodenticides. *The Science of the total environment*, 788, p.147673. [Online]. Available at: doi:10.1016/j.scitotenv.2021.147673.

Posthuma, L., Suter, G. W., II and Traas, T. P. (2001). *Species Sensitivity Distributions in Ecotoxicology*. Lewis Publishers.

Prat-Mairet, Y. et al. (2017). Non-invasive monitoring of red fox exposure to rodenticides from scats. *Ecological indicators*, 72, pp.777–783. [Online]. Available at: doi:10.1016/j.ecolind.2016.08.058.

Rached, A. et al. (2020). Biomarkers Potency to Monitor Non-target Fauna Poisoning by Anticoagulant Rodenticides. *Frontiers in Veterinary Science*, 7 (December). [Online]. Available at: doi:10.3389/fvets.2020.616276.

Rattner, B. A. and Harvey, J. J. (2021). Challenges in the interpretation of anticoagulant rodenticide residues and toxicity in predatory and scavenging birds. Pest management science, 77 (2), pp.604–610. [Online]. Available at: doi:10.1002/ps.6137.

Rattner, B. A. et al. (2014). Adverse outcome pathway and risks of anticoagulant rodenticides to predatory wildlife. *Environmental science & technology*, 48 (15), pp.8433–8445. [Online]. Available at: doi:10.1021/es501740n.

Rattner, B. A. et al. (2020). Brodifacoum Toxicity in American Kestrels (Falco sparverius) with Evidence of Increased Hazard on Subsequent Anticoagulant Rodenticide Exposure. *Environmental toxicology and chemistry / SETAC*, 39 (2), pp.468–481. [Online]. Available at: doi:10.1002/etc.4629.

Rattner, B. A. and Mastrota, F. N. (2018). Anticoagulant Rodenticide Toxicity to Non-target Wildlife Under Controlled Exposure Conditions. In: *Anticoagulant Rodenticides and Wildlife*

Roos, S. et al. (2021). Annual abundance of common Kestrels (Falco tinnunculus) is negatively associated with second generation anticoagulant rodenticides. *Ecotoxicology* , 30 (4), pp.560–574. [Online]. Available at: doi:10.1007/s10646-021-02374-w.

RSPB. (2022). *Bird crime Report 2022 - Figthing raptor persecution*. The Royal Society for Protection of Birds.

Saad Soluiman Desoky, A. E.-A. (2016). Rodenticides (Anticoagulant). *International Journal of Research Studies in Zoology (IJRSZ)*, 2 (3), pp.1–6.

Sage, M., Courdassier, M., Defaut, R., Gimbert, F., Berny, P. and Giraudoux, P. (2008). Kinetics of bromadiolone in rodent populations and implications for predators after field control of the water vole, Arvicola terrestris. The Science of the total environment, 407 (1), pp.211–222. [Online]. Available at: doi:10.1016/j.scitotenv.2008.09.003.

Salim, H. et al. (2014). Sub-lethal effects of the anticoagulant rodenticides bromadiolone and chlorophacinone on breeding performances of the barn owl (Tyto alba) in oil palm plantations. *Slovak Raptor Journal*, 8 (2), pp.113–122. [Online]. Available at: doi:10.2478/srj-2014-0013.

Shore, R. F. et al. (2018). The Relative Importance of Different Trophic Pathways for Secondary Exposure to Anticoagulant Rodenticides. In: *Proceedings of 28th the Vertebrate Pest Conference*. 2018.

Shore, R. F. et al. (2019). *Second generation anticoagulant rodenticide residues in barn owls 2018*. UK Centre for Ecology and Hydrologycontract report to the Campaign for Responsible Rodenticide Use., pp.1–22. [Online]. Available at: http://pbms.ceh.ac.uk/sites/pbms.ceh.ac.uk/files/stewardship-2017-owls.pdf.

Smart, J., Amar, A., Sim, I. M. W., Etheridge, B., Cameron, D., Christie, G. and Wilson, J. D. (2010). Illegal killing slows population recovery of a re-introduced raptor of high conservation concern – The red kite Milvus milvus. Biological conservation, 143 (5), Elsevier BV., pp.1278–1286. [Online]. Available at: doi:10.1016/j.biocon.2010.03.002.

Sorgog, K. and Kamo, M. (2019). Quantifying the precision of ecological risk: Conventional assessment factor method vs. species sensitivity distribution method. *Ecotoxicology and environmental safety*, 183 (July). [Online]. Available at: doi:10.1016/j.ecoenv.2019.109494.

Spadetto, L., Gómez-Ramírez, P., León-Ortega, M., Zamora-López, A., Díaz-García, S., Zamora-Marín, J.M., Tecles-Vicente, F., Pardo-Marín, L., Fenoll, J., Calvo, J. F., et al. (2025). Exploring anticoagulant rodenticide exposure and effects in eagle owl (Bubo bubo) nestlings from a Mediterranean semiarid region. Environmental research, 264 (Pt 2), Elsevier BV., p.120382. [Online]. Available at:doi:10.1016/j.envres.2024.120382.

Spadetto, L., Gómez-Ramírez, P., Zamora-Marín, J. M., León-Ortega, M., Díaz-García, S., Tecles, F., Fenoll, J., Cava, J., Calvo, J. F. and García-Fernández, A. J. (2024). Active monitoring of long-eared owl (Asio otus) nestlings reveals widespread exposure to anticoagulant rodenticides across different agriculturallandscapes. The Science of the total environment, 918, p.170492. [Online]. Available at:doi:10.1016/j.scitotenv.2024.170492.

Sumasgutner, P. et al. (2014a). Conservation related conflicts in nest-site selection of the Eurasian Kestrel (Falco tinnunculus) and the distribution of its avian prey. *Landscape and urban planning*, 127, pp.94–103. [Online]. Available at: doi:10.1016/j.landurbplan.2014.03.009.

Sumasgutner, P. et al. (2014b). Hard times in the city-attractive nest sites but insufficient food supply lead to low reproduction rates in a bird of prey. *Journal of Wildlife Rehabilitation*, 34 (3), pp.17–31. [Online]. Available at: doi:10.1186/1742-9994-11-48.

Sylvie, M. et al. (2002). Influence of hatching order on growth rate and resting metabolism of kestrel nestlings. *Journal of avian biology*, 33 (3), pp.235–244. [Online]. Available at: doi:10.1034/j.1600-048X.2002.330305.x.

Sylvie, M. et al. (2003). Nestling energy expenditure of Eurasian kestrels Falco tinnunculus in relation to food intake and hatching order. *Avian science*, 3 (1), pp.1–12. [Online]. Available at: https://www.researchgate.net/publication/258359973_Nestling_energy_expenditure_of_Eurasian_kestrels_Falco_tinnunculus_in_relation_to_food_intake_and_hatching_order/download.

Thorley, J. and Schwarz, C. (2018). ssdtools: An R package to fit Species Sensitivity Distributions. *Journal of Open Source Software*, 3 (31), p.1082. [Online]. Available at: doi:10.21105/joss.01082.

Todd W, A. (2010). Uninformative Parameters and Model Selection Using Akaike's Information Criterion. *The Journal of wildlife management*, 74 (6), pp.1175–1178. [Online]. Available at: doi:10.2193/2009-367.

Tosh, D. G. et al. (2011). Does small mammal prey guild affect the exposure of predators to anticoagulant rodenticides? *Environmental pollution* , 159 (10), pp.3106–3112. [Online]. Available at: doi:10.1016/j.envpol.2011.03.028.

Tosh, D. G. et al. (2012). Rodenticide exposure in wood mouse and house mouse populations on farms and potential secondary risk to predators. *Ecotoxicology* , 21 (5), pp.1325–1332. [Online]. Available at: doi:10.1007/s10646-012-0886-3.

U.S. Environmental Protection Agency. *ECOTOX Knowledgebase*. [Online]. Available at: https://cfpub.epa.gov/ecotox/ [Accessed 12 March 2024].

Van Den Brink, N. W. et al. (2018). *Anticoagulant rodenticides and wildlife*, Emerging Topics in Ecotoxicology. van den Brink, N. W. et al. (Eds). Cham: Springer International Publishing., pp.195–227. [Online]. Available at: doi:10.1007/978-3-319-64377-9.

Vandenbroucke, V. et al. (2008). Pharmacokinetics of eight anticoagulant rodenticides in mice after single oral administration. *Journal of veterinary pharmacology and therapeutics*, 31 (5), pp.437–445. [Online]. Available at: doi:10.1111/j.1365-2885.2008.00979.x.

Village, A. (1982). The Home Range and Density of Kestrels in Relation to Vole Abundance. *The Journal of animal ecology*, 51 (2), pp.413–428. [Online]. Available at: doi:10.2307/3974.

Village, A. (1990a). Diet and Prey Selection. In: *The Kestrel*. Bloomsbury Publishing Plc,.

Village, A. (1990b). The Breeding Cycle : The Nestling Stage. In: *The Kestrel*. Bloomsbury Publishing Plc. [Online]. Available at: doi:10.5040/9781472597434.ch-013.

Walker, L. A. et al. (2016). *Anticoagulant rodenticides in red kites (Milvus milvus) in Britain 2010 to 2015: a Predatory Bird Monitoring Scheme (PBMS)*. UK Centre for Ecology and

Hydrlogy., pp.1–14. [Online]. Available at: http://www.ceh.ac.uk/http://nora.nerc.ac.uk/orcanbedownloadeddirectlyfromthePBMSwebsite http://pbms.ceh.ac.uk/.

Walker, L. A. et al. (2017). *Anticoagulant rodenticides in red kites (Milvus milvus) in Britain 2015*. 18, UK Centre for Ecology and Hydrology., pp.1–18. [Online]. Available at: http://nora.nerc.ac.uk/%0Ahttp://pbms.ceh.ac.uk/.

Walker, L. A. et al. (2021a). *Second generation anticoagulant rodenticide residues in red kites 2019*. UK Centre for Ecology and Hydrology contract report to Natural England.

Walker, L. A. et al. (2021b). *Second generation anticoagulant rodenticide residues in red kites 2020*. UK Centre for Ecology and Hydrology report for Natural England.

Walther, B., Geduhn, A., Schenke, D., Schlötelburg, A. and Jacob, J. (2021). Baiting location affects anticoagulant rodenticide exposure of non-target small mammals on farms. Pest management science, 77 (2), Wiley., pp.611–619. [Online]. Available at: doi:10.1002/ps.5987.

Weathers, W. W. (1992). Scaling nestling energy requirements. The Ibis, (134), pp.142–153. [Online]. Available at: doi:10.1111/j.1474-919X.1992.tb08391.x.

Wheeler, J. R., Grist, E. P. M., Leung, K. M. Y., Morritt, D. and Crane, M. (2002). Species sensitivity distributions: Data and model choice. Marine pollution bulletin, 45 (1-12), pp.192–202. [Online]. Available at: doi:10.1016/S0025-326X(01)00327-7.

Wemer, L. et al. (2021). Reduced ectoparasite load, body mass and blood haemolysis in Eurasian kestrels (Falco tinnunculus) along an urban–rural gradient. *Science of Nature*, 108 (5). [Online]. Available at: doi:10.1007/s00114-021-01745-x.

Weathers, W. W. (1992). Scaling nestling energy requirements. The Ibis, (134), pp.142–153. [Online]. Available at: doi:10.1111/j.1474-919X.1992.tb08391.x.

Wheeler, J. R., Grist, E. P. M., Leung, K. M. Y., Morritt, D. and Crane, M. (2002). Species sensitivity distributions: Data and model choice. Marine pollution bulletin, 45 (1-12), pp.192–202. [Online]. Available at: doi:10.1016/S0025-326X(01)00327-7.

Xu, F.-L., Li, Y.-L., Wang, Y., He, W., Kong, X.-Z., Qin, N., Liu, W.-X., Wu, W.-J. and Jorgensen, S. E. (2015). Key issues for the development and application of the species sensitivity distribution (SSD) model for ecological risk assessment. Ecological indicators, 54, Elsevier BV., pp.227–237. [Online]. Available at: doi:10.1016/j.ecolind.2015.02.001.