Science of the Total Environment 643 (2018) 134–144

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

Anticoagulant rodenticide exposure in an Australian predatory bird increases with proximity to developed habitat

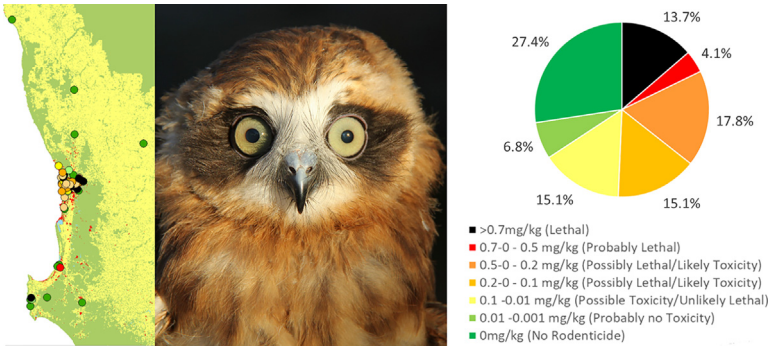
Michael T. Lohr

School of Science, Edith Cowan University, 100 Joondalup Drive, Joondalup, Western Australia 6027, Australia

HIGHLIGHTS

- Anticoagulant rodenticide (AR) exposure rates are poorly studied in Australian wildlife.
- ARs were detected in 72.6% of Southern Boobook owls found dead or moribund in Western Australia.
- Total AR exposure correlated with proximity to developed habitat.
- ARs used only by licensed pesticide applicators were detected in owls.
- Raptors with larger home ranges and more mammal-based diets may be at greater risk of AR exposure.

GRAPHICAL ABSTRACT



ARTICLE INFO

Article history:
Received 10 May 2018
Received in revised form 16 June 2018
Accepted 17 June 2018
Available online xxxx

Editor: D. Barcelo

Keywords:
Rodenticide
Southern Boobook
Ninox boobook
SGAR
Poisoning
Landscape

ABSTRACT

Anticoagulant rodenticides (ARs) are commonly used worldwide to control commensal rodents. Second generation anticoagulant rodenticides (SGARs) are highly persistent and have the potential to cause secondary poisoning in wildlife. To date no comprehensive assessment has been conducted on AR residues in Australian wildlife. My aim was to measure AR exposure in a common widespread owl species, the Southern Boobook (*Ninox boobook*) using boobooks found dead or moribund in order to assess the spatial distribution of this potential threat. A high percentage of boobooks were exposed (72.6%) and many showed potentially dangerous levels of AR residue (>0.1 mg/kg) in liver tissue (50.7%). Multiple rodenticides were detected in the livers of 38.4% of boobooks tested. Total liver concentration of ARs correlated positively with the proportions of developed areas around points where dead boobooks were recovered and negatively with proportions of agricultural and native land covers. Total AR concentration in livers correlated more closely with land use type at the spatial scale of a boobook's home range than at smaller or larger spatial scales. Two rodenticides not used by the public (difethialone and flocoumafen) were detected in boobooks indicating that professional use of ARs contributed to secondary exposure. Multiple ARs were also detected in recent fledglings, indicating probable exposure prior to fledging. Taken together, these results suggest that AR exposure poses a serious threat to native predators in Australia, particularly in species using urban and peri-urban areas and species with large home ranges.

1. Introduction

Anticoagulant rodenticides (ARs) are commonly used in residential, commercial, and agricultural settings for the control of rodent pests (Rattner et al., 2014b). They block the recycling of vitamin K in the

E-mail address: m.lohr@ecu.edu.au.

https://doi.org/10.1016/j.scitotenv.2018.06.207
0048-9697/

liver, which subsequently disrupts normal blood clotting in vertebrates (Park et al., 1984). ARs are often divided into first generation anticoagulant rodenticides (FGARs) and second generation anticoagulant rodenticides (SGARs) based on their chemical structure and when they were first synthesized. Unlike FGARS, SGARs are often lethal with a single feed and are substantially more persistent in liver tissue (Erickson and Urban, 2004).

AR exposure and subsequent mortality have been detected in non-target wildlife in all parts of the world where exposure has been tested (Laakso et al., 2010). Predatory bird species are particularly vulnerable to AR poisoning due to a greater susceptibility to most ARs than other bird species (Herring et al., 2017) and a prey base which frequently contains rodents targeted by the use of ARs. In some raptor species, mortality from AR exposure may have population-level impacts (Thomas et al., 2011). Unlike in Europe and North America, where the non-target impacts of ARs have been extensively studied, relatively little research has been conducted on AR exposure in Australian wildlife (Lohr and Davis, 2018; Olsen et al., 2013). This knowledge gap exists despite several lines of evidence suggesting that patterns of regulation and usage in combination with differences in faunal assemblages may increase the incidence and severity of non-target AR poisoning in Australia relative to better-studied areas of the world (Lohr and Davis, 2018).

Within Australia, patterns in the spatial distribution of AR exposure have not been studied in any wildlife species. A number of studies have addressed the spatial ecology of anticoagulant rodenticide exposure in non-target wildlife but have been primarily limited to North American mammals. Of these, some have focused on impacts within specific habitat types (Cypher et al., 2014; Gabriel et al., 2012). Studies examining patterns of AR exposure between urban and rural habitats have found correlations between the use of urban habitat and exposure rates in San Joaquin kit foxes (Mcmillin et al., 2008) and bobcats (Riley et al., 2007). A model developed to predict exposure patterns in San Joaquin kit foxes found that exposure was most likely in areas of low density housing on the urban/rural interface (Nogeire et al., 2015). Similar dynamics have been suggested but not tested in predatory bird species. Studies in North America and Europe have noted that predatory bird species which use more developed habitats tend to have greater rates of AR exposure than those which predominantly use more natural landscapes (Albert et al., 2010; Christensen et al., 2012). Additionally, a study in Spain noted a positive correlation between human population density and AR exposure in a sample of 11 species of predatory birds and mammals (López-Perea et al., 2015). The greater use of rodenticides and higher prevalence of targeted commensal rodents in human-dominated landscapes relative to natural areas is likely to drive these observed and suggested differences in non-target exposure. However, because AR usage patterns differ between urban and agricultural environments (Lohr and Davis, 2018) a need exists to evaluate the possibility of differences in non-target exposure patterns between different types of anthropogenic landscapes.

To address this knowledge gap, I sought to compare anticoagulant rodenticide (AR) exposure across intact native bushland and two different types of anthropogenic landscapes. Additionally, I undertook the first large-scale targeted testing of wildlife for AR exposure in the continent of Australia (Lohr and Davis, 2018). Testing was conducted on Southern Boobooks (*Ninox boobook*), which provide an excellent model to quantify the spatial distribution of threatening processes associated with fragmentation due to their presence across multiple habitat types and high abundance relative to other predatory bird species. To the best of my knowledge, no studies have directly addressed the relative impacts of different types of human land use on AR exposure in non-target wildlife. Understanding how different types of human land use impact the likelihood of AR exposure in non-target wildlife will be critical in evaluating risks to wildlife on a continental scale and will enable more effective targeting of measures to mitigate secondary toxicity.

2. Methods

Southern Boobooks are medium-sized hawk owls found across the majority of mainland Australia and adjacent parts of Indonesia and New Guinea (Olsen, 2011). They are assigned a conservation status of "Least Concern" by the IUCN ("*Ninox boobook*", 2018). Some taxonomies consider Southern Boobooks to be synonymous with the closely-related New Zealand Morepork (*Ninox novaseelandiae*) found in Tasmania and New Zealand but recent genetic and bioacoustic evidence suggests otherwise (Gwee et al., 2017). Boobooks are dietary generalists, consuming a wide variety of vertebrate and invertebrate prey (Higgins, 1999; Trost et al., 2008). These dietary habits make them an ideal model species for broad assessment of contamination of food webs by persistent pollutants like ARs. Their presence in most habitat types across Australia, with the exception of treeless deserts (Higgins, 1999), facilitates examination of differences in exposure across multiple habitat types and allows for future replication of this study at sites across the continent.

2.1. Specimen collection

Dead boobooks found in Western Australia were solicited from a network of volunteers, wildlife care centres, and government departments and were opportunistically collected when encountered. Boobooks euthanized by veterinarians and wildlife rehabilitators due to severe disease or injury were included. Dates and locations where each boobook was initially collected were recorded from the collector when possible. If liver tissue was identifiable and had a mass >3 g, it was removed and stored frozen at 20 °C until analysed for AR residues. A total of 73 usable boobook livers were stored for testing. While an effort was made to obtain boobooks from a diversity of geographical areas and habitat types throughout Western Australia, most samples originated in the more densely settled urban and peri-urban areas in the south-west of Western Australia in and around the city of Perth.

2.2. Rodenticide analysis

Liver samples were analysed by the National Measurement Institute (Melbourne, Australia) for residues of three FGARs (warfarin, coumatetralyl, and pindone) and five SGARs (difenacoum, bromadiolone, brodifacoum, difethialone, and flocoumafen) registered for use in Australia by the Australian Pesticides and Veterinary Medicines Authority. For each sample, 10 ml of reverse osmosis water and one gram of liver tissue were added to a 50 ml analytical tube and shaken for 15 min on a horizontal shaker. A 10 ml volume of 5% formic acid in acetonitrile solution was then added and the tube was shaken for an additional 30 min. QuEChERS extraction salt was added and the tube was shaken for an additional two minutes. The tube was then centrifuged for 10 min at 5100 rpm. After pipetting 3 ml of the supernatant into a 15 ml analytical tube, 5 ml of hexane was added and the tube was shaken for two minutes then centrifuged for 10 min at 5100 rpm. The hexane layer was removed using a vacuum pipette and discarded. A 1 ml aliquot of the supernatant was transferred to a 2 ml QuEChERS dispersive tube, shaken for one minute, and centrifuged at 13,000 rpm for three minutes. The QuEChERS supernatant was then filtered using a 0.45 μm filter. After filtration, 3 μl of coumachlor was added as an internal standard to 497 μl of the filtered extract and vortexed prior to LC-MS/MS analysis. A Waters TQS Tandem Quadrupole Detector Liquid Chromatograph-Mass Spectrometer (LC-MS/MS) and an Acquity UPLC CSH C18 100 × 2.1 mm column were used to quantify concentrations of each rodenticide. Recovery rates for each AR, were calculated using chicken liver samples spiked with analytical standards (Table 1).

Table 1
Limit of detection (LOD), limit of quantification (LOQ), average recovery, and relative standard deviation (RSD) for eight ARs in a spiked chicken liver matrix.

Compound	LOD (mg/kg)	LOQ (mg/kg)	Average recovery % (RSD)
Warfarin	0.001	0.002	94 (8.1)
Coumatetralyl	0.001	0.002	93 (7.6)
Bromadiolone	0.005	0.010	96 (9.5)
Difenacoum	0.005	0.010	96 (11.2)
Flocoumafen	0.005	0.010	103 (11.4)
Brodifacoum	0.005	0.010	92 (8.8)
Difethialone	0.005	0.010	91 (14.6)
Pindone	0.005	0.010	36 (13.5)

2.3. Statistical analysis

Total AR liver concentration is commonly used to compare toxicity risk when individuals are exposed to multiple rodenticides (Christensen et al., 2012) due to similarities in their modes of action and likely cumulative effects (Hughes et al., 2013). For this reason, the sum of all liver rodenticide concentrations above the limit of detection was calculated for each individual for the purposes of comparing differences in exposure by age, season, and land use. In order to compare seasonal trends in total AR concentration, boobooks were assigned to four groups based on their collection date: summer (December–February), autumn (March–May), winter (June–August), and spring (September–November). All boobooks with known collection months ($n = 71$) were included in the seasonal analysis. The Kruskal-Wallis test was used to assess whether significant differences existed in liver AR concentration by season.

Boobooks were assigned to age classes of less than one year ("hatch year") or greater than one year ("after hatch year") based on the presence of juvenile down and by examination of fluorescence patterns under ultraviolet light (Weidensaul et al., 2011). In one instance, it was not possible to determine age class due to degradation of porphyrins caused by prolonged exposure of ventral remiges to sunlight. A total of 72 boobooks of determined age class were available for analysis of the relationship between age and AR exposure. I used a Mann-Whitney-Wilcoxon test to determine whether total liver concentration of ARs varied between the two age classes. Results were considered significant if $p < 0.05$.

2.4. Exposure thresholds

The utility of rodenticide concentration in liver tissue as a means to diagnose lethal exposure has been questioned (Erickson and Urban, 2004; Thomas et al., 2011) as susceptibility to acute toxicity can vary among individuals and across species (Thomas et al., 2011). Exposure to multiple ARs adds additional complexity to the assessment of likely impacts from residual liver concentrations (Murray, 2017). However, a need exists to estimate likely impacts across exposed individuals and to compare the magnitude of exposure to previous studies. Accordingly, I identified relevant literature which established commonly used guidelines for outcomes of various exposure rates in related taxa to allow estimation of likely impacts on boobooks.

The Rodenticide Registrants Task Force suggested that a 0.7 mg/kg liver concentration of brodifacoum was likely to be toxic based largely on captive studies of Barn Owls (Kaukeinen et al., 2000), however this threshold estimate may be too high, as environmental conditions affecting wild birds may increase their susceptibility to ARs relative to captive birds (Mendenhall and Pank, 1980). Dowding et al. (1999) estimated a lethal liver concentration for brodifacoum of 0.5 mg/kg using 29 individuals from 10 species of birds. Numerous studies have reported thresholds of 0.2 mg/kg (Albert et al., 2010; Christensen et al., 2012; Hughes et al., 2013; Langford et al., 2013; López-Perea et al., 2015; Stansley et al., 2014; Walker et al., 2008) and 0.1 mg/kg (Albert et al., 2010; Christensen et al., 2012; Langford et al., 2013; Ruiz-Suárez et al., 2014; Shore et al., 2016; Stansley et al., 2014; Walker et al., 2008, 2011) as indices of lower limits at which lethal AR toxicity was likely to occur in predatory birds. These estimates were based on two studies examining wild barn owls: Newton et al. (1999, 1998) respectively. I also included a threshold of 0.01 mg/kg as this is the lowest published record of lethal SGAR toxicity in a predatory bird species (Stone et al., 1999). Boobook liver concentrations were compared against these thresholds (0.7 mg/kg, 0.5 mg/kg, 0.2 mg/kg, 0.1 mg/kg, and 0.01 mg/kg) to facilitate a comprehensive understanding of overall potential impacts of ARs across all sampled individuals.

2.5. Spatial analysis

Only boobooks with accurate location data were included in the spatial analysis. In one instance, two road-killed boobooks were recovered at the same location. One of these was randomly removed from the spatial analysis, leaving a total of 66 boobooks available for analysis. Land cover for the state of WA was classified into developed, agriculture, native vegetation or open water. The developed category included all areas with anthropogenic impervious surfaces (roads, buildings car parks, etc.) as well as intensive land uses that did not qualify as agriculture (mines, landfills, spots grounds, golf courses etc.). The agriculture category included a diversity of irrigated and dryland crops, orchards, and grazed areas. Intensive indoor animal agriculture was included in the developed category rather than agriculture because it consisted primarily of buildings and other impervious surfaces. Areas subjected to silvicultural practices were classified as part of the native vegetation category due to structural similarity. Additionally like native bushland, the only anticoagulant permitted for use in forestry is pindone which is used to control rabbits in areas too close to human habitation to allow the safe use of 1080. Percentages of each classification were calculated within circular buffer zones (areas of influence) of three different sizes around each location where a boobook was found. The two smaller buffer sizes were calculated to match the mean area of a boobook's core home range (7.3 ha) and total home range (145.1 ha) (Olsen et al., 2011). The largest buffer size was an arbitrarily large area with a 3 km radius. This larger buffer was included to account for the possibility of movement of contaminated prey into boobooks' home ranges from adjacent areas influencing the probability of boobook exposure to ARs. Because open water was not considered to be usable space, the percentages of the other three habitat types were calculated excluding any open water within the buffers.

I used general linear models with a negative binomial distribution, following methodology used by Christensen et al. (2012), to analyse differences in rodenticide exposure by habitat composition at the three different spatial scales. The Akaike Information Criterion AIC was used to rank models for habitat proportions at each spatial scale. Only single variable models were considered in the ranking due to nesting and correlation of habitat proportions and spatial scales. I calculated McFadden's pseudo-R^2 values for each habitat type and spatial scale combination. Statistical analysis was performed using RStudio 1.1.383 (RStudio, Inc., Boston, MA, USA).

3. Results

While I did not directly quantify physiological signs of rodenticide poisoning due to most carcasses being damaged as a result of vehicle collisions, during dissection I observed symptoms associated with acute lethal AR toxicity in at least nine boobooks exhibiting no sign of trauma. These symptoms included excessive bleeding from minor lacerations, pale or mottled livers, subdermal and muscular haemorrhage in the absence of trauma, blood in the thoracic cavity, and blood around the mouth and nares. Similar symptoms have been described in association with lethal AR toxicity in other raptor species (Murray, 2017).

Table 2
Percentage exposure, mean exposure and total detection of eight different anticoagulant rodenticides in livers of 73 Southern Boobooks in Western Australia.

	Coumatetralyl	Warfarin	Pindone	Difenacoum	Brodifacoum	Bromadiolone	Difethialone	Flocoumafen	Total
Percent exposed	0.000	2.740	0.000	15.068	72.603	31.507	8.219	2.740	72.603
Mean exposure (mg/kg)	0.000	0.000	0.000	0.004	0.260	0.019	0.015	0.011	0.310
Standard error	0.000	0.000	0.000	0.002	0.064	0.005	0.011	0.011	0.069
Maximum concentration (mg/kg)	0.000	0.002	0.000	0.097	4.002	0.214	0.775	0.818	4.002
Minimum concentration (mg/kg)	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Total detected (mg/kg)	0.000	0.003	0.000	0.287	18.994	1.421	1.063	0.834	22.606

ARs were detected in 72.6% of all boobook liver samples (Table 2) with a mean summed AR exposure of 0.310 mg/kg (SE 0.069246735) (Table 3). Approximately 17.8% of boobook livers contained greater than the suspected lethal threshold of 0.5 mg/kg total ARs (Fig. 1) with 13.7% above the more conservative limit of 0.7 mg/kg. Seven of the ten boobooks with AR liver concentrations above 0.7 mg/kg appear to have died directly of AR poisoning and the other three showed signs of poisoning described by Murray (2017) despite other apparent proximate causes of death. More than half of the boobooks tested had liver concentrations above 0.1 mg/kg (Fig. 1) and would likely have experienced at least some degree of coagulopathy (Rattner et al., 2014a). The majority of boobooks (65.8%) were exposed at a level above 0.01 mg/kg – the lowest observed lethal threshold in an owl (Fig. 1).

The three FGARs tested – coumatetralyl, warfarin, and pindone – were infrequently detected and accounted for only 0.01% of all ARs detected (Table 2). Coumatetralyl and pindone were not detected in any of the samples and warfarin was detected in two individuals at low levels (0.0024 mg/kg and 0.0014 mg/kg). The lower of these was below the limit of quantification. Detectable exposure to SGARs was substantially higher (Table 2). Brodifacoum – the most commonly detected SGAR – was found in 72.6% of samples and made up 84.0% of all rodenticides detected by mg/kg. It was detected in all liver samples containing AR residues (Table 2). Difethialone and flocoumafen, which were not known to be in use by the public were also detected in boobooks. Two or more ARs were detected in 38.4% of boobooks tested (Fig. 2). A maximum of five different ARs was detected in two individual boobooks.

Mean total liver concentration of ARs was not significantly different between age classes ($p = 0.34$). AR exposure was greatest in boobooks collected in winter and winter concentrations were significantly different from summer concentrations ($p = 0.026$) (Fig. 3). The livers of two recent fledglings still under parental care contained low but quantifiable amounts of brodifacoum (0.022 and 0.051 mg/kg) and difethialone (0.020 and 0.022 mg/kg).

Total AR exposure was positively correlated with the amount of developed area within buffers at all spatial scales (Table 4). Proportions of agriculture and bushland habitat within buffers were negatively correlated with total AR exposure at all spatial scales (Table 4). The three AIC top-ranked models quantified habitat composition at the scale of a full boobook home range and were all statistically significant (Table 4). The top-ranked model used developed habitat at the scale of a boobook's total home range and was highly significant ($p = 0.00182$). Correlations between the top three ranked models and total AR concentration were not particularly strong but are stronger than would be suggested by interpretation of traditional R^2 indices, as McFadden's pseudo-R^2 values falling in the range of 0.2 to 0.4 “represent an excellent fit” (McFadden, 1978).

4. Discussion

The overall proportion of boobooks with detectable AR exposure (72.6%) and the proportion of boobooks exposed to two or more rodenticides (38.4%) was high but within the range of estimates generated by studies in Europe and North America (Table 3). Mean total AR concentration in boobooks (0.310 mg/kg) was substantially higher than any other available published estimate with the exception of Red Kites (*Milvus milvus*) (0.413 mg/kg) in Denmark (Christensen et al., 2012). The extremely high mean exposure in boobooks may result from multiple causes. A large proportion of samples were obtained from urban and peri-urban areas where exposure is likely to be more prevalent. This was also the case in several other studies documenting high exposure rates and liver concentrations (López-Perea et al., 2015; Murray, 2017; Stansley et al., 2014). As a consequence, the sample of boobooks used in this study is probably not representative of Australia as a whole but may provide a useful estimate for other large human population centres elsewhere. Circadian activity patterns may also increase boobooks' risk of AR exposure relative to some other raptor species. Nocturnal species have been noted to have higher liver AR concentrations than diurnal species (Ruiz-Suárez et al., 2014; Sánchez-Barbudo et al., 2012). If owls using highly populated landscapes are at greater risk than other bird species, future evaluation of Powerful Owls which use urban and peri-urban areas and are listed as vulnerable in Victoria may be warranted. Southwest populations of Masked Owls (*Tyto novaehollandiae*) and Barking Owls (*Ninox connivens*), both of which are listed as P3 priority fauna (poorly known but thought to be possibly threatened) in Western Australia, may also be susceptible to AR poisoning in areas where developed habitats are encroaching on their remaining ranges.

As a consequence of the methodology used in sample collection, this study probably underestimates the proportion of lethal poisonings which actually occur. Anticoagulant rodenticides induce lethargy prior to mortality and lethally poisoned owls are more likely to die in nest hollows or roost sites in dense vegetation where their likelihood of detection by humans would be low (Newton et al., 1990). Similar underestimation of lethal toxicity has been suggested in studies of mammals exposed to ARs, as well (Mcdonald et al., 1998). Conversely, if haemorrhaging induced by sub-lethal exposure reduced a boobook's reaction time or ability to fly, it could increase the risk of other proximate sources of mortality (Newton et al., 1990) such as collisions with vehicles or windows. This could potentially increase its likelihood of being killed in a conspicuous location and subsequently collected for this study with the end result of inflating the number of sub-lethally exposed birds entering this study.

4.1. Individual rodenticides

A lack of detectable pindone residues in the livers of the boobooks sampled was unexpected because pindone is used within the Perth metropolitan area to control rabbits in urban bushlands and previous literature implicates similar control programs elsewhere in Australia in secondary poisonings of native raptors (Olsen et al., 2013) though this has recently been disputed (Olsen and Rae, 2017). Failure to detect pindone could be the result of a short retention time relative to more persistent SGARs (Fisher et al., 2003), its use in targeted and short-term control efforts, low overall usage relative to commercial and residential use of other anticoagulant rodenticides, or dietary patterns of boobooks precluding consumption of European rabbits (*Oryctolagus cuniculus*) – the species targeted by pindone applications. While it is possible that occasional localised exposure may occur, it appears that pindone, as currently applied in urban and peri-urban areas does not constitute a substantial threat to boobook populations relative to other rodenticides originating from commercial and residential sources. Future studies on impacts of pindone on native raptors should consider testing species which are more likely to prey on rabbits (Wedge-tailed

Table 3
Published rates of multiple second generation anticoagulant rodenticide exposure and percentages of individuals with exposure above two thresholds in predatory birds.

Species	Location	n individuals	% exposed	% multiple exposure	% >0.1 mg/kg	% >0.2 mg/kg	Mean exposure (mg/kg) (SE)	Source
Southern Boobook (*Ninox boobook*)	Western Australia	73	72.6	38.4	50.7	35.6	0.310 (0.069)	This study
Tawny Owl (*Strix aluco*)	United Kingdom	172	19.2	2.9	12.2	5.8	0.125	Walker et al., 2008
Barn Owl (*Tyto alba*)	United Kingdom	100	94	72	16			Shore et al., 2016
Red Kite (*Milvus milvus*)	Scotland	114	69.3	36		17.5	0.155 (0.017)	Hughes et al., 2013
Buzzard (*Buteo buteo*)	Scotland	479	44.3	14.2		2.1	0.047 (0.004)	Hughes et al., 2013
Kestrel (*Falco tinnunculus*)	Scotland	22	40.9	17.4		9.1	0.173 (0.082)	Hughes et al., 2013
Barn Owl (*Tyto alba*)	Scotland	63	34.9	17.5		17.5	0.076 (0.018)	Hughes et al., 2013
Tawny Owl (*Strix aluco*)	Scotland	34	38.2	5.9		2.9	0.047 (0.021)	Hughes et al., 2013
Sparrowhawk (*Accipiter nisus*)	Scotland	37	54.1	29.7		2.7	0.060 (0.016)	Hughes et al., 2013
Peregrine Falcon (*Falco peregrinus*)	Scotland	24	29.2	0		0	0.017 (0.007)	Hughes et al., 2013
Barn Owl (*Tyto alba*)	United Kingdom	58	84	52	17.2			Walker et al., 2011
Red Kite (*Milvus milvus*)	United Kingdom	18	94	89				Walker et al., 2011
Kestrel (*Falco tinnunculus*)	United Kingdom	20	100	95				Walker et al., 2011
Barn Owl (*Tyto alba*), Barred Owl (*Strix varia*), and Great Horned Owl (*Bubo virginianus*)	Canada	164	92		32	15	0.107	Albert et al., 2010
Great Horned Owl	Canada	123					0.016	Thomas et al., 2011
Red-tailed Hawk (*Buteo jamaicensis*)	Canada	58					0.005	Thomas et al., 2011
Golden eagle (*Aquila chrysaetos*)	Norway	16	73.3	31.3	25	6.3	0.051	Langford et al., 2013
Eagle owl (*Bubo bubo*)	Norway	8	62.5	25	37.5	12.5	0.087	Langford et al., 2013
Osprey (*Pandion haliaetus*)	Norway	3	0	0	0	0	0	Langford et al., 2013
Peregrine falcon (*Falco peregrinus*)	Norway	2	0	0	0	0	0	Langford et al., 2013
Gryfalcon (*Falco rusticolus*)	Norway	1	0	0	0	0	0	Langford et al., 2013
Red-tailed Hawk (*Buteo jamaicensis*)	USA	37	97	78				Murray, 2017
Barred Owl (*Strix varia*)	USA	24	88	42				Murray, 2017
Great Horned Owl (*Bubo virginianus*)	USA	17	100	71				Murray, 2017
Eastern Screech-Owl (*Megascops asio*)	USA	16	100	69				Murray, 2017
Red-tailed Hawk (*Buteo jamaicensis*)	USA	105	81	15	47	25	0.117	Stansley et al., 2014
Great Horned Owl (*Bubo virginianus*)	USA	22	82	18	36	9	0.07	Stansley et al., 2014
Eurasian Sparrowhawk (*Accipiter nisus*)	Spain (Canary Islands)	14	85.7				0.0577	Ruiz-Suárez et al., 2014
Long-eared Owl (*Asio otus*)	Spain (Canary Islands)	23	73.9				0.1322	Ruiz-Suárez et al., 2014
Common Buzzard (*Buteo buteo*)	Spain (Canary Islands)	9	26.3				0.0368	Ruiz-Suárez et al., 2014
Barbary Falcon (*Falco pelegrinoides*)	Spain (Canary Islands)	16	31.2				0.0915	Ruiz-Suárez et al., 2014
Kestrel (*Falco tinnunculus*)	Spain (Canary Islands)	21	66.6				0.219	Ruiz-Suárez et al., 2014
Barn Owl (*Tyto alba*)	Spain (Canary Islands)	21	76.2				0.1344	Ruiz-Suárez et al., 2014
All Species	Spain (Canary Islands)	104	63.5		34.8			Ruiz-Suárez et al., 2014
Scops Owl (*Otus scops*)	Spain (Majorca Island)	26	57.7			0	0.0134	López-Perea et al., 2015
Barn Owl (*Tyto alba*)	Spain (Majorca Island)	19	84.2			57.9	0.2337	López-Perea et al., 2015
Scops Owl (*Otus scops*)	Spain (Catalonia)	7	14.3			0	0.1584	López-Perea et al., 2015
Barn Owl (*Tyto alba*)	Spain (Catalonia)	22	54.5			13.6	0.1178	López-Perea et al., 2015
Tawny Owl (*Strix aluco*)	Spain (Catalonia)	27	77.8			29.6	0.0952	López-Perea et al., 2015
Eagle Owl (*Bubo bubo*)	Spain (Catalonia)	14	100			64.3	0.2896	López-Perea et al., 2015
Long-eared Owl (*Asio otus*)	Spain (Catalonia)	12	58.3			0	0.0111	López-Perea et al., 2015
Little Owl (*Athene noctua*)	Spain (Catalonia)	7	71.4			28.6	0.1972	López-Perea et al., 2015
Common buzzard (*Buteo buteo*)	Spain (Catalonia)	56	64.3			26.8	0.1253	López-Perea et al., 2015
Barn owl (*Tyto alba*)	Denmark	80	94		37.4	13.7	0.1141	Christensen et al., 2012
Buzzard (*Buteo buteo*)	Denmark	141	94		20.6	5.7	0.0745	Christensen et al., 2012
Eagle owl (*Bubo bubo*)	Denmark	10	100		70	70	0.1931	Christensen et al., 2012
Kestrel (*Falco tinnunculus*)	Denmark	66	89		27.2	13.6	0.099	Christensen et al., 2012
Little owl (*Athene noctua*)	Denmark	9	100		33.3	22.2	0.1186	Christensen et al., 2012
Long-eared owl (Asio otus)	Denmark	38	95		0	0	0.0194	Christensen et al., 2012
Marsh harrier (*Circus aeruginosus*)	Denmark	3	100		0	0	0.0123	Christensen et al., 2012
Red kite (*Milvus milvus*)	Denmark	3	100		0	66.7	0.413	Christensen et al., 2012
Rough-legged Buzzard (*Buteo lagopus*)	Denmark	31	84		12.9	0	0.0408	Christensen et al., 2012
Short-eared owl (*Asio flammeus*)	Denmark	5	100		0	0	0.015	Christensen et al., 2012
Tawny owl (*Strix aluco*)	Denmark	44	93		20.5	9.1	0.0784	Christensen et al., 2012
All Species	Denmark	430		73				Christensen et al., 2012

Eagles (*Aquila audax*) and Little Eagles (*Hieraaetus morphnoides*)) (Olsen et al., 2006) or scavenge rabbit carcasses (Whistling Kites (*Haliastur sphenurus*)) (Fuentes et al., 2005) and are at greater risk of secondary exposure.

Failure to detect coumatetralyl in any samples and the detection of warfarin at extremely low concentration in only two samples despite commercial availability to the public suggests that their relatively short half-life in liver tissue (Fisher et al., 2003) probably reduces the incidence and severity of secondary exposure and precludes bioaccumulation and biomagnification. This result is consistent with absence or low concentration and prevalence of FGARs relative to SGARs in other wildlife species since SGARs came into widespread use (Albert et al., 2010; Fourel et al., 2018; Murray, 2017; Ruiz-Suárez et al., 2014).

The detection of brodifacoum at rates an order of magnitude higher than all other ARs combined is probably attributable to a combination of its greater duration of persistence in liver tissue (Horak et al., 2018), more prevalent use, and incorporation into a greater number of commercially available rodenticide bait products. This is particularly concerning because captive studies suggest that brodifacoum is more likely to cause secondary toxicity in birds than any other tested ARs due to its high toxicity and long liver retention time (Erickson and Urban, 2004). Bromadiolone and difenacoum respectively, were the next most commonly detected in samples (Table 2). This is probably because, together with brodifacoum, they comprise the three SGARs commonly available in WA at retail stores. At present, brodifacoum, bromadiolone, and difenacoum probably pose the greatest threat of secondary poisoning to non-target wildlife of all ARs in use.

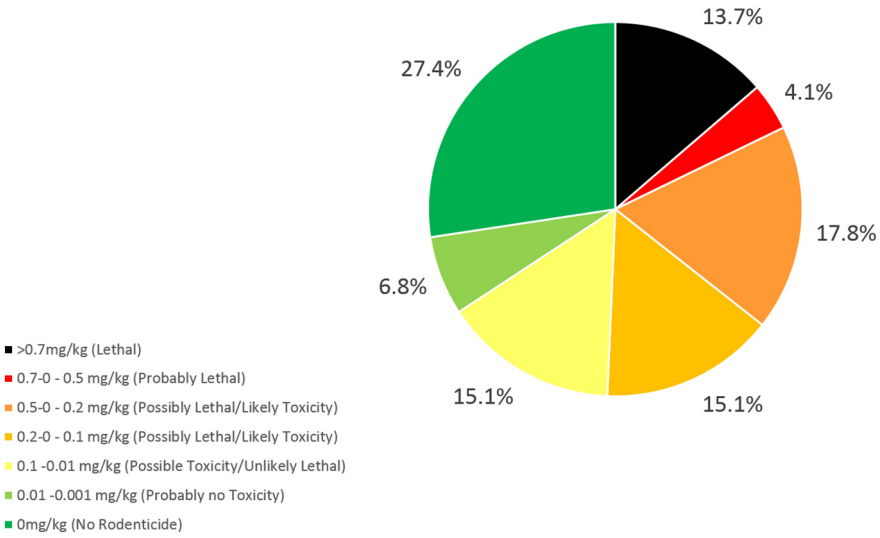


Fig. 1. Percentages of Southern Boobooks (n = 73) in Western Australia exposed to rodenticides stratified by total rodenticide liver concentration (mg/kg) thresholds indicating potential outcomes.

The detection of flocoumafen and difethialone – which are not readily available to the public due to sale in bulk quantities but are used by pest control professionals – indicates that at least some proportion of wildlife exposure is directly related to commercial pest control activities. Flocoumafen was the most prevalent rodenticide detected in liver tissue of one boobook, which died shortly after admission to a wildlife care centre and showed physiological signs of AR poisoning (pale mottled liver, subcutaneous haemorrhage, and large quantities of blood in the abdominal cavity). These findings have potentially serious implications for legislation attempting to curtail non-target exposure by limiting public access to SGARs. In the United States, legislation restricting the use of SGARs to licensed professionals went into effect in 2011 (Bradbury, 2008). However, a subsequent study found an increase in AR exposure in four predatory bird species in Massachusetts, USA following the ban (86% of 161 birds from 2006 to 2010 compared to 96% of 94 birds exposed from 2012 to 2106) perhaps due to an increased use of professional rodent control services (Murray, 2017). My findings provide additional evidence that use of ARs by professional pesticide applicators does contribute, at least to some degree, to poisoning of non-target raptors. However, the impacts of this source relative to private use are difficult to assess because other SGARs which are available to the public – particularly brodifacoum and bromadiolone – are in common use by professional pesticide applicators in WA. Taken together, these results cast doubt on whether regulations restricting sale of SGARs from private use will be sufficient to reduce widespread exposure and toxicity in predatory birds.

After the completion of this study, it was brought to my attention that diphacinone was also being used in Western Australia by commercial pesticide applicators. This FGAR has a relatively short half-life of three days in rat liver tissue and as a consequence is unlikely to bioaccumulate and cause secondary poisoning in predatory non-target wildlife (Fisher et al., 2003). The registration of diphacinone in Australia has expired. However, if diphacinone is re-registered, future monitoring projects should include diphacinone testing as it could potentially contribute to overall rodenticide exposure.

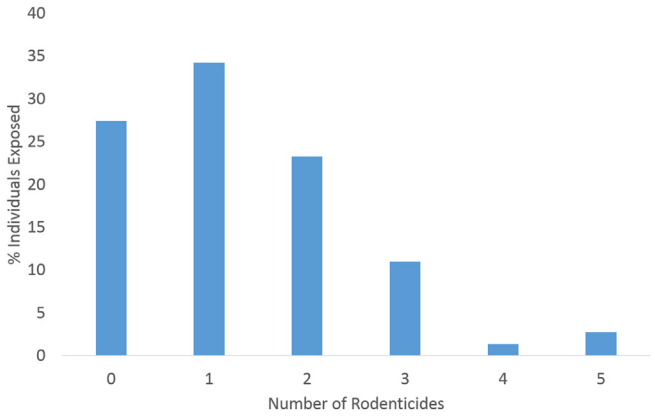


Fig. 2. Percentages of Southern Boobooks (n = 73) exposed to multiple anticoagulant rodenticides in Western Australia.

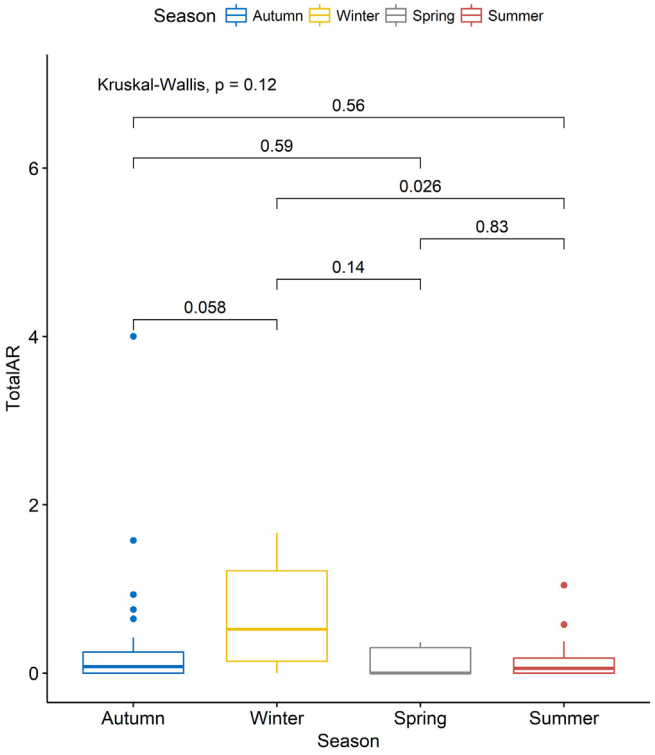


Fig. 3. Mean total anticoagulant rodenticide concentration (mg/kg) in liver tissue of Southern Boobooks (n = 71) in Western Australia by season.

Table 4
Akaike information criterion (AIC) ranking of models of the association between percentage of single land use types within buffers around collection points and total anticoagulant rodenticide liver concentration in Southern Boobooks (n = 66) in Western Australia at three different spatial scales (Big = 2827.4 ha buffer, Mid = 145.1 ha buffer, Small = 7.3 ha buffer.

Model	Estimate	Std. error	z value	Pr(>|z|)	AIC	McFadden's pseudo-R^2
Mid developed	2.1439	0.6876	3.118	0.00182	751.43	0.08675021
Mid agriculture	−2.4505	0.9844	−2.489	0.0128	754.28	0.05158204
Mid native vegetation	−2.5139	0.9584	−2.623	0.00871	754.35	0.05081192
Big agriculture	−3.0121	1.1147	−2.702	0.00689	754.51	0.04870524
Small developed	1.5092	0.6822	2.212	0.027	754.53	0.04854103
Big developed	1.7553	0.7547	2.326	0.02	754.83	0.04473145
Small agriculture	−1.6016	1.0237	−1.565	0.118	756.27	0.02641717
Small native vegetation	−1.364	0.9249	−1.475	0.14	756.59	0.02232542
Big native vegetation	−1.9017	1.066	−1.784	0.0744	756.8	0.01968855

Exposure to multiple rodenticides (38.4%) was relatively common in sampled boobooks but not as frequent as in some other predatory bird species (Christensen et al., 2012; Murray, 2017; Walker et al., 2011). The relatively high rate of multiple exposures and the presence of detectable levels of up to five different ARs in liver tissue suggests cumulative exposure from multiple prey items over an extended period of time. This hypothesis is supported by the finding that livers of adult raptors in Denmark contained multiple rodenticides more frequently than those of juveniles (Christensen et al., 2012). The prevalence of multiple exposures in boobooks is particularly concerning because laboratory studies on rats determined that warfarin sensitivity is increased after sub-lethal exposure to brodifacoum (Mosterd and Thijssen, 1991). If ARs have a synergistic effect rather than a purely additive effect, raptors may be negatively impacted at a lower threshold when exposed to more than one AR, leading to underestimates of negative impacts on non-target wildlife.

4.2. Rodenticide thresholds

The utility of detectable rodenticide concentration in liver tissue as a means to diagnose lethal exposure has been questioned (Erickson and Urban, 2004; Thomas et al., 2011) as susceptibility to acute toxicity can vary among individuals and across species (Erickson and Urban, 2004). However, it can be informative in comparing environmental exposure and as an index for potential impacts at the population level. Depending on the threshold used (0.7 mg/kg or 0.5 mg/kg), either 13.7% or 17.8% of boobooks tested had rates of exposure consistent with likely lethal outcomes. Confirmation of physical signs of rodenticide poisoning in all boobooks with AR liver concentrations above 0.7 mg/kg and the absence of other obvious causes of death in 70% of these individuals indicates that this threshold is a reasonable guideline for estimating likely lethal toxicity in boobooks. Regardless of the threshold used, the relatively high frequency of exposure at levels likely to be directly lethal is cause for concern. In combination with visible signs of AR poisoning, it indicates that exposure to ARs contributed substantially to mortality in boobooks found dead or brought to wildlife carers in the urban and peri-urban areas where most samples were collected.

Exposure at potentially dangerous but not necessarily lethal levels was also high relative to most published studies examining rodenticide exposure in wild raptors found dead or moribund. The proportion of boobooks exposed at levels above 0.2 mg/kg (35.6%) was higher than all other reported estimates except for in Barn Owls (*Tyto alba*) (57.9%) and Eagle Owls (*Bubo bubo*) (64.3%) in Spain (López-Perea et al., 2015) and Red Kites (*Milvus milvus*) (66.7%) in Denmark (Christensen et al., 2012). In all three species, the sample size was small (n < 20). The percentage of boobooks with total AR liver concentrations above 0.1 mg/kg (50.7%) was substantially greater than all previously reported species except for Red-tailed Hawks in New Jersey, USA (47%) (Stansley et al., 2014). At minimum, a threshold of 0.1 mg/kg should be considered potentially dangerous. In a laboratory study using Eastern Screech Owls (*Megascops asio*), diphacinone concentrations of ≥0.1 mg/kg in liver tissue were associated with coagulopathy (Rattner et al., 2014a). Coagulopathy is likely more dangerous to wild birds due to greater amounts of movement and injuries associated with capturing prey and may have synergistic interactions with environmental stressors which increase the chance of mortality (Erickson and Urban, 2004). SGARs are also more toxic than diphacinone and can logically be expected to have at least as great of an impact at the same threshold.

Sub-lethal exposure was common in boobooks regardless of the chosen threshold. The sub-lethal impacts of chronic AR exposure are poorly studied in wildlife. A number of lines of evidence suggest that even exposure below the threshold needed to cause lethal haemorrhage is not benign. While Thomas et al. (2011) take issue with the uncritical use of liver concentrations to assess likely toxicity, their probabilistic methodology examining AR toxicity in four raptor species predicted that 20% of individuals would experience quantifiable toxicity at levels as low as 0.08 mg/kg. Increased rates of parasitism and infectious disease have also been documented in association with AR exposure in bobcats (*Lynx rufus*) (Riley et al., 2007), Great Bustards (*Otis tarda*) (Lemus et al., 2011), and common voles (*Microtus arvalis*) (Vidal et al., 2009). In bobcats, immunosuppression and inflammatory response associated with chronic sub-lethal AR exposure and use of urban habitats may have led to an outbreak of notoedric mange (Serieys et al., 2018). Similar disruption of immune system function may occur in other chronically-exposed wildlife (Serieys et al., 2018). Several studies have also suggested the possibility of increased mortality rates via accidents, predation, vehicle collisions, nutritional stress, and blood loss following minor injury in wildlife exposed to sub-lethal doses of anticoagulant rodenticides (Albert et al., 2010; Mendenhall and Pank, 1980; Newton et al., 1990; Stone et al., 1999, 2003). If this dynamic is indeed consistent across wildlife species, the high rates of presumably sub-lethal exposure detected in boobooks are cause for concern. If sub-lethal exposure to ARs substantially increases the risk of parasitism and other sources of mortality, it is not appropriate to assess the overall impacts of anticoagulants on predatory bird populations based solely on documentation of direct lethal toxicity.

4.3. Spatial correlations

We observed weak but statistically significant correlations between AR exposure and habitat proportions in proximity to recovered boobook carcasses. The difference in the direction of correlations between AR exposure and proportions of agricultural and developed habitats, the consistency of the trends at different spatial scales, and the increasing strength of the trends at the most biologically meaningful spatial scale all suggest an actual difference in exposure risk between the two anthropogenic landscapes. Future studies on this topic should attempt to improve sample collection across different types of anthropogenic landscapes or focus on species for which samples are more readily available across study areas. A low sample size of boobook carcasses from landscapes predominantly comprised of native bushland or agriculture likely contributed to the low predictive value of top models.

The three top-ranked models for boobook AR exposure used habitat data at the scale of an average home range. Foraging behaviour likely explains the closer correlation of AR exposure and habitat type at the spatial scale of an average boobook home range relative to other spatial scales. The vast majority of foraging occurs within an animal's home range and its exposure to ARs can be expected to relate most closely to the proportions of habitat types likely to be sources of contamination of its prey base at this spatial scale. Boobooks have relatively small home ranges in comparison to other Australian owl species (Kavanagh and Murray, 1996; Soderquist and Gibbons, 2007). If risk of rodenticide exposure is related to developed area at the scale of an animal's home range, species with larger home ranges may be exposed over a broader portion of the landscape. This hypothesis is supported by the finding

that in bobcats – a species with a much larger home range than boobooks – the concentration but not the presence of ARs in liver tissue correlated with the proportion of developed habitat within their home range (Riley et al., 2007). Taken in combination, these results suggest that species with large home ranges are likely to be at risk of some degree of AR exposure if their home range encompasses even small areas of developed habitat. As a consequence, encroachment of human structures into large areas of natural habitat may have an impact on predatory species with large home ranges that is disproportionate to the area of habitat lost through development.

The positive correlation between total AR exposure and the proportion of developed area within buffers was expected due to the widespread use of rodenticides in commercial and residential settings. This pattern of exposure has been suggested following detection of high exposure rates in densely populated areas (López-Perea et al., 2015; Stansley et al., 2014) but, this appears to be the first instance where differences in exposure across habitat types has been directly quantified in a bird species. A number of other studies have examined the spatial patterns of AR exposure in wildlife. The trend in boobooks was similar to the correlation between developed areas and total AR exposure observed in a study of bobcats and mountain lions in California (Riley et al., 2007). Similarly, AR exposure was common (87%) in an urban population of San Joaquin kit foxes but no rodenticides were detected in individuals from a non-urban population (Mcmillin et al., 2008). Frequent AR exposure in wildlife inhabiting developed habitats is typically attributed to the "prevalent and wide-spread" use of ARs in urban areas (Cypher et al., 2014). Higher prevalence of commensal rodents which serve as vectors of ARs in urban areas may exacerbate this problem. A study in Canada demonstrated a higher proportion of rats in the diet of Barn Owls with territories containing more urban land use (Hindmarch and Elliott, 2014). Assuming that commensal rodents are an important vector of ARs, their higher relative proportion in the diets of urban owls may increase the incidence and severity of AR exposure. Boobooks are likely to be affected by this dynamic. In Canberra, Australia, boobook diets contained a higher percentage of mammal biomass in suburban areas (65.8%) than in woodland areas (26.0%) (Trost et al., 2008). Both the high prevalence of rodenticide use and the greater availability of potentially exposed commensal rodents likely contribute to the positive correlation between rodenticide exposure and developed habitat observed in boobooks.

A negative correlation between AR exposure and the proportion of bushland area within simulated home ranges was expected because rodenticides are seldom used in native habitats, aside from the use of pindone to control rabbits. Only one other study has tested spatial patterns of AR exposure in wildlife primarily using bushland habitats. Unlike patterns observed in boobooks, high exposure rates were unexpectedly detected in fishers (*Martes pennanti*) throughout areas of forested habitat, probably as a result of rodenticide use associated with illegal marijuana production (Gabriel et al., 2012). Similarly a threatened Spotted Owl (*Strix occidentalis*) with illegal marijuana cultivation within its home range was documented to have been exposed to brodifacoum despite being in a remote natural area (Franklin et al., 2018). Conservation and law enforcement professionals should be aware of this potential source of environmental contamination when attempting to mitigate damage caused by illegal marijuana cultivation in remote areas in Australia. Future work examining the distance rodenticides travel into bushland ecosystems from adjacent sources will be useful in gaining a better understanding of the relationship between fragmentation and rodenticide use. This could potentially lead to establishing appropriate sizes for SGAR exclusion zones around bushland areas containing sensitive fauna and reduce edge effects relating to SGARs.

The negative correlation between total AR exposure and the proportion of agricultural area within simulated home ranges was somewhat surprising, as rodenticides are known to be used in agricultural settings. AR exposure in wildlife has been attributed to agricultural application of ARs in the UK (Birks, 1998; Hughes et al., 2013), Spain (Lemus et al., 2011), France (Fourel et al., 2018), and Australia (Young and De Lai, 1997). Anecdotal accounts from farmers indicate that a variety of first and second generation products are used for asset protection around buildings and in grain storage areas in Western Australia (D. Thompson, personal communication, April 9, 2017). However, they are not licensed for use directly in crops or along crop perimeters. As a consequence, the total amount of bait deployed per unit area is likely to be substantially lower than in developed areas. However, in agricultural systems, total compliance with best practice application methods for SGARs may be rare and lack of compliance probably facilitates greater risk of secondary toxicity to native wildlife (Tosh et al., 2011). An anecdotal report of farmers in Western Australia requesting the FGAR pindone to control kangaroos (Twigg et al., 1999) – a use not allowed by the labelling – suggests that illegal use of ARs in agricultural contexts may be an issue in some areas. The widespread availability of SGARs to the public in Australia increases the risk that misuse could lead to localised impacts on non-target wildlife.

The negative correlation between proximity to agricultural land and AR exposure may not be consistent throughout all Australian agricultural systems. In Queensland, declines in breeding owl abundance were attributed to broad-scale application of a brodifacoum-based rodenticide in canefields (Young and De Lai, 1997) but this product was subsequently removed from the market (Twigg et al., 1999). At present, brodifacoum is only registered for use in and around buildings in Australia (McLeod and Saunders, 2013) but can be freely purchased and applied without a license. While less toxic and persistent than brodifacoum, a coumatetralyl-based product is currently licensed for use in sugar cane, pineapple, and macadamia crops across Australia (Australian Pesticides and Veterinary Medicines Authority, 2017). More concerningly, during rodent plagues the SGAR bromadiolone has been used to bait field perimeters in New South Wales (New South Wales Department of Primary Industries, 2011; New South Wales Government: Department of Primary Industries, 2017).

4.4. Seasonal differences

The difference in AR exposure observed between boobook carcasses recovered in winter and those recovered in summer potentially reflects increased risk of exposure during winter when rodents make up a larger proportion of the diet. Boobooks are dietary generalists and one study indicates that boobook diet varies seasonally and includes higher proportions of vertebrates in winter than in autumn (Trost et al., 2008). This seasonal variation in diet may reduce the risk of accumulating lethal levels of ARs in boobooks relative to some other raptor species. Species preying predominantly on small mammals are likely to be at greater risk of exposure than species that prey predominantly on birds (Ruiz-Suárez et al., 2014). This hypothesis is supported by a lack of seasonal variation in AR exposure in Tawny Owls (*Strix aluco*) which feed consistently on bank voles (*Myodes glareolus*) and field mice (*Apodemus* spp.) (Walker et al., 2008). Similarly, in the United States, rodenticide exposure rates and concentrations did not vary significantly by season in Red-tailed Hawks (*Buteo jamaicensis*) (Stansley et al., 2014) which feed predominantly on mammals year-round. The only other study detecting seasonal variation in liver AR concentration found a significant difference in only one of five ARs tested (Christensen et al., 2012). This difference was attributed to an influx in autumn of migratory raptors from more sparsely populated regions with presumably less AR exposure risk (Christensen et al., 2012).

It is possible that consuming few rodents during a portion of the year allows boobooks to excrete sufficient levels of highly-persistent SGARs that total liver concentrations are less likely to accumulate to a lethal level. In this scenario, other raptor species which consistently consume rodents throughout the year – such as Masked Owls and Barking Owls – may be at elevated risk of lethal poisoning relative to boobooks. Alternately, seasonal variation in rodenticide exposure in boobooks could

be correlated with seasonal differences in rodenticide use patterns. Information on rodenticide sales is not publicly available, but anecdotal accounts from some Perth residents indicate greater use of rodenticides in winter in response to greater perceived abundance of commensal rodents. Improved knowledge of rodenticide application patterns and seasonal patterns of rodenticide exposure in species with a more consistent mammal-based diet would be useful in addressing these questions.

The high AR exposure rates observed in boobooks despite seasonal variation in the proportion of rodents in their diet highlights the need for additional study of exposure rates of other taxa which may potentially vector rodenticides. Documented exposure in raptors which prey primarily on birds indicates that non-rodent vectors may substantially contribute to AR exposure at higher trophic levels (Thomas et al., 2011). Invertebrates have been implicated in vectoring lethal levels of rodenticides to bird species including New Zealand Dotterels (*Charadrius obscurus aquilonius*) (Dowding et al., 2006) and nestling Stewart Island robins (*Petroica australis rakiura*) (Masuda et al., 2014) as well as an insectivorous mammal, the European hedgehog (*Erinaceus europaeus*) (Dowding et al., 2010). Reptiles could potentially also be effective vectors to higher trophic levels (Lohr and Davis, 2018). Further investigation of AR residues across more taxa is necessary to fully understand ecosystem-wide AR contamination and the vectors by which carnivorous species are exposed.

4.5. Rodenticide in fledglings

The detection of SGAR exposure in recent fledglings provides a possible indication as to why there was no significant difference in total AR exposure between hatch year boobooks and older adults. AR exposure prior to leaving the nest is particularly concerning from a conservation perspective. Suspected brodifacoum poisoning was previously reported as the likely cause of death of Norfolk Island Boobook chicks which were still in the nest (Debus, 2012) but there was no indication of physical examination or direct testing for AR exposure. Birds with growing feathers may be at additional risk of exsanguination (Newton et al., 1990). This may put chicks and recent fledglings at greater risk than adult birds which do not typically moult large proportions of their feathers simultaneously. Additional sub-lethal threats to chicks have also been reported. Stunted growth across several biometric measurements of nestling Barn Owls was observed in plots treated with anticoagulant rodenticides relative to control plots in Indonesia (Naim et al., 2010). While reduced prey availability due to rodent control likely had a negative influence on growth rates, nestlings in areas treated with the SGAR brodifacoum showed reduced growth when compared to areas where rodents were controlled with the FGAR warfarin or a biological rodent control agent (Naim et al., 2010), suggesting that AR exposure contributed to reduced nestling growth. Similarly, a dramatic reduction in breeding success occurred in a population of closely-related moreporks on Mokoia Island in New Zealand in the breeding season immediately following a broadscale distribution of brodifacoum as part of an attempted mouse eradication (Stephenson et al., 1999). While Stephenson et al. (1999) concede that the reduction in breeding success may have been related to a drop in prey availability rather than a direct effect of rodenticide toxicity, depression of breeding success by anticoagulant rodenticides is plausible. Laboratory testing also detected modest reductions in weight gain and wing growth in juvenile Japanese Quail (*Coturnix coturnix japonica*) exposed to sub-lethal doses of brodifacoum or difenacoum (Butler, 2010). Perhaps the most conclusive evidence of negative impacts of sub-lethal AR exposure on growing birds is the correlation observed between concentrations of bromadiolone in blood and reduced body condition observed in nestling Common Kestrels (*Falco tinnunculus*) (Martínez-Padilla et al., 2016).

Nest success may also be impacted in the early stages of nesting. Embryo toxicity has been observed in domestic chicken eggs injected with the anticoagulant rodenticide flocoumafen (Khalifa et al., 1992). It is also possible that exposure to anticoagulant rodenticides could impact egg viability via reductions in the integrity of eggshells. Exposure to therapeutic anticoagulants has resulted in bone density loss in humans by disruption of the vitamin K cycle and resultant suppression of calcification (Fiore et al., 1990; Resch et al., 1991; Monreal et al., 1991) though similar effects on bone density have not been observed in birds (Knopper et al., 2007). Residues of bromadiolone and chlorophacinone were detected in yolk and albumin of addled Barn Owl eggs in areas of palm plantations treated with rodenticides but no changes to eggshell thickness or morphology were detected (Salim et al., 2015). However, changes to barn owl egg morphology, reduced eggshell mass and decreased eggshell thickness have been observed when eggs contained higher concentrations of brodifacoum (Naim et al., 2012). While teratogenic effects of anticoagulant rodenticides are not widely reported in birds, one study suggested this possibility when the authors detected a single barn owl nestling in a plot treated with brodifacoum which failed to grow primary feathers and would have been unable to fly (Naim et al., 2010). Haemorrhage of oviducts in association with rodenticide poisoning has been observed in female raptors carrying eggs (Murray, 2017), suggesting that ARs may pose a particular risk to nesting females. Future assessments of population-level impacts of anticoagulant rodenticide exposure need to consider not only adult mortality, but also impacts on fecundity and recruitment.

5. Conclusion

My hypothesis that total AR exposure would vary between areas predominated by different types of anthropogenic landscape is to some degree supported by the finding of significant, though weak, relationships trending in opposite directions between total liver AR concentration and proportions of agriculture and developed land at the spatial scale of a boobook's home range. Understanding this dynamic is key to assessing landscape-level risk of AR poisoning across carnivores and scavengers in Australia. It will also facilitate future attempts to model exposure risk in endangered and priority taxa which may be susceptible and will enable more specific risk assessment prior to proposed future developments. The high rates and magnitude of AR exposure raise serious concerns about AR exposure in other Australian species. Future work should evaluate the impact of ARs on other Australian wildlife, particularly species utilizing urban and peri-urban areas, species with large home ranges, and species regularly consuming commensal rodents. The detection in boobooks of ARs presumed to be used only by professionals is concerning. Ongoing review of the registration of SGARs by the APVMA should take this into consideration when evaluating the efficacy of restricting SGARs to licensed pesticide applicators in reducing poisoning in non-target wildlife.

Acknowledgements

This project was supported financially by The Holsworth Wildlife Research Endowment via The Ecological Society of Australia, the BirdLife Australia Stuart Leslie Bird Research Award, the Eastern Metropolitan Regional Council's Healthy Wildlife Healthy Lives program, the Society for the Preservation of Raptors, the Edith Cowan University School of Science Postgraduate Student Support Award, and Sian Mawson.

I would like to extend my appreciation to the staff of National Measurement Institute's (NMI), Analytical Services Port Melbourne branch, in particular Hao Nguyen and her veterinary drug residue measurement team for their support in providing high quality measurements using NATA accredited LC-MSMS measurement techniques.

Dr. Cheryl Lohr provided valuable assistance in statistical analysis and Dr. Shaun Molloy graciously volunteered time to help develop necessary GIS layers. I would especially like to thank Kanyana Wildlife Rehabilitation, Native Animal Rescue, Native ARC, and Nature Conservation Margaret River Region and the many other individuals especially Simon Cherriman, Angela Febey, Amanda Payne, and Stuart Payne for

contributing samples. This manuscript was improved by comments from Dr. Robert Davis, Dr. Allan Burbidge and two anonymous reviewers. The photograph in the graphical abstract was provided by Simon Cherriman.

References

Albert, C.A., Wilson, L.K., Mineau, P., Trudeau, S., Elliott, J.E., 2010. Anticoagulant rodenticides in three owl species from Western Canada, 1988–2003. Arch. Environ. Contam. Toxicol. 58:451–459. https://doi.org/10.1007/s00244-009-9402-z.

Australian Pesticides and Veterinary Medicines Authority, 2017. Agricultural and Veterinary Permits Search [WWW Document]. URL https://portal.apvma.gov.au/permits?p_auth=MpDv1rrW&p_p_id=permitsportlet_WAR_permitsportlet&p_p_lifecycle=1&p_p_state=normal&p_p_mode=view&p_p_col_id=column-1&p_p_col_pos=1&p_p_col_count=3&_permitsportlet_WAR_permitsportlet_javax.portlet.action=search (accessed 8.4.17).

Birks, J.D.S., 1998. Secondary rodenticide poisoning risk arising from winter farmyard use by the European polecat *Mustela putorius*. Biol. Conserv. 85 (3), 233–240.

Bradbury, S., 2008. Risk Mitigation Decision for Ten Rodenticides. Washington D. C., USA.

Butler, S., 2010. The Sub-lethal Effects of Second Generation Anticoagulant Rodenticides on Birds (Thesis Submitted for the Degree of Doctor of Philosophy at the University of Leicester by Sarah Elizabeth Butler Department of Biology University of Leicester September 2010 T).

Christensen, T.K., Lassen, P., Elmeros, M., 2012. High exposure rates of anticoagulant rodenticides in predatory bird species in intensively managed landscapes in Denmark. Arch. Environ. Contam. Toxicol. 63:437–444. https://doi.org/10.1007/s00244-012-9771-6.

Cypher, B.L., McMillin, S.C., Westall, T.L., Job, C.V.H., Hosea, R.C., Finlayson, B.J., 2014. Rodenticide exposure among endangered kit foxes relative to habitat use in an urban landscape. Cities Environ. 7, 8.

Debus, S.J.S., 2012. Norfolk Island Boobook chick deaths. Boobook 30, 6.

Dowding, J.E., Murphy, E.C., Veitch, C.R., 1999. Brodifacoum residues in target an non-target species following an aerial poisoning operation on Motuihe Island, Hauraki Gulf, New Zealand. N. Z. J. Ecol. 23, 207–214.

Dowding, J.E., Lovegrove, T. im G., Kast, S.N., Puckett, M., 2006. Mortality of northern New Zealand dotterels (*Charadrius obscurus aquilonius*) following an aerial poisoning operation. Notornis 53, 235–239.

Dowding, C.V., Shore, R.F., Worgan, A., Baker, P.J., Harris, S., 2010. Accumulation of anticoagulant rodenticides in a non-target insectivore, the European hedgehog (*Erinaceus europaeus*). Environ. Pollut. 158, 161–166.

Erickson, W., Urban, D., 2004. Potential Risk of Nine Rodenticides to Birds and Mammals: A Comparative Approach. Washington DC.

Fiore, C.E., Tamburino, C., Foti, R., Grimaldi, D., 1990. Reduced axial bone mineral content in patients taking an oral anticoagulant. South. Med. J. 83:538–542. https://doi.org/10.1097/00007611-199005000-00013.

Fisher, P., O'Connor, C., Wright, G., Eason, C.T., 2003. Persistence of four Anticoagulant Rodenticides in the Livers of Laboratory Rats. DOC Science Internal Series (Wellington, New Zealand).

Fourel, I., Sage, M., Benoit, E., Lattard, V., 2018. Liver and fecal samples suggest differential exposure of red fox (*Vulpes vulpes*) to trans- and cis-bromadiolone in areas from France treated with plant protection products. Sci. Total Environ. 622–623: 924–929. https://doi.org/10.1016/j.scitotenv.2017.12.053.

Franklin, A.B., Carlson, P.C., Rex, A., Rockweit, J.T., Garza, D., Culhane, E., Volker, S.F., Dusek, R.J., Shearn-Bochsler, V.I., Gabriel, M.W., Horak, K.E., 2018. Grass is not always greener: rodenticide exposure of a threatened species near marijuana growing operations. BMC Res. Notes 11:1–7. https://doi.org/10.1186/s13104-018-3206-z.

Fuentes, E., Olsen, J., Rose, A.B., 2005. Breeding diet at two whistling kite nests near Canberra. Aust. F. Ornithol. 22, 122–125.

Gabriel, M.W., Woods, L.W., Poppenga, R., Sweitzer, R.A., Thompson, C., Matthews, S.M., Higley, J.M., Keller, S.M., Purcell, K., Barrett, R.H., Wengert, G.M., Sacks, B.N., Clifford, D.A.L., 2012. Anticoagulant rodenticides on our public and community lands: spatial distribution of exposure and poisoning of a rare forest carnivore. PLoS One 7, e40163. https://doi.org/10.1371/journal.pone.0040163.

Gwee, C.Y., Christidis, L., Eaton, J.A., Norman, J.A., Trainor, C.R., Verbelen, P., Rheindt, F.E., 2017. Bioacoustic and multi-locus DNA data of Ninox owls support high incidence of extinction and recolonisation on small, low-lying islands across Wallacea. Mol. Phylogenet. Evol. 109:246–258. https://doi.org/10.1016/j.ympev.2016.12.024.

Herring, G., Eagles-Smith, C.A., Buck, J., 2017. Characterizing golden eagle risk to lead and anticoagulant rodenticide exposure: a review. J. Raptor Res. 51, 273–292.

Higgins, P.J., 1999. Southern boobook. Handbook of Australian, New Zealand and Antarctic Birds. Volume 4: Parrots to Dollarbird. Oxford University Press, Melbourne, pp. 864–875.

Hindmarch, S., Elliott, J.E., 2014. A specialist in the city: the diet of barn owls along a rural to urban gradient. Urban Ecosyst. 18:477–488. https://doi.org/10.1007/s11252-014-0411-y.

Horak, K., Fisher, P.M., Hopkins, B.M., 2018. Pharmacokinetics of anticoagulant rodenticides in target and non-target organisms. Emerg. Top. Ecotoxicol. 5:87–108. https://doi.org/10.1007/978-3-319-64377-9.

Hughes, J., Sharp, E., Taylor, M.J., Melton, L., Hartley, G., 2013. Monitoring agricultural rodenticide use and secondary exposure of raptors in Scotland. Ecotoxicology 22: 974–984. https://doi.org/10.1007/s10646-013-1074-9.

Kaukeinen, D.E., Spragins, C.W., Hobson, J.F., 2000. Risk-benefit considerations in evaluating commensal anticoagulant rodenticide impacts to wildlife. Proc. Vertebr. Pest Conf. 19, 245–256.

Kavanagh, R.P., Murray, M., 1996. Home range, habitat and behaviour of the masked owl Tyto novaehollandiae near Newcastle, New South Wales. Emu 96, 250–257.

Khalifa, B.A.A., Salem, F.M.S., Menha, M.A., 1992. Embryotoxic and teratogenic effects of flocoumafen in chick embryos and white rats. J. Appl. Anim. Res. 2:81–85. https://doi.org/10.1080/09712119.1992.9705936.

Knopper, L.D., Mineau, P., Walker, L.A., Shore, R.F., 2007. Bone density and breaking strength in UK raptors exposed to second generation anticoagulant rodenticides. Bull. Environ. Contam. Toxicol. 78:249–251. https://doi.org/10.1007/s00128-007-9122-x.

Laakso, S., Suomalainen, K., Koivisto, S., 2010. Literature Review on Residues of Anticoagulant Rodenticides in Nontarget Animals (Copenhagen).

Langford, K.H., Reid, M., Thomas, K.V., 2013. The occurrence of second generation anticoagulant rodenticides in non-target raptor species in Norway. Sci. Total Environ. 450–451:205–208. https://doi.org/10.1016/j.scitotenv.2013.01.100.

Lemus, J.A., Bravo, C., García-Montijano, M., Palacín, C., Ponce, C., Magaña, M., Alonso, J.C., 2011. Side effects of rodent control on non-target species: rodenticides increase parasite and pathogen burden in great bustards. Sci. Total Environ. 409:4729–4734. https://doi.org/10.1016/j.scitotenv.2011.07.007.

Lohr, M.T., Davis, R.A., 2018. Anticoagulant rodenticide use, non-target impacts and regulation: a case study from Australia. Sci. Total Environ. 634:1372–1384. https://doi.org/10.1016/j.scitotenv.2018.04.069.

López-Perea, J.J., Camarero, P.R., Molina-López, R.A., Parpal, L., Obón, E., Solá, J., Mateo, R., 2015. Interspecific and geographical differences in anticoagulant rodenticide residues of predatory wildlife from the Mediterranean region of Spain. Sci. Total Environ. 511: 259–267. https://doi.org/10.1016/j.scitotenv.2014.12.042.

Martínez-Padilla, J., López-Idiáquez, D., López-Perea, J.J., Mateo, R., Paz, A., Viñuela, J., 2016. A negative association between bromadiolone exposure and nestling body condition in common kestrels: management implications for vole outbreaks. Pest Manag. Sci. 73:364–370. https://doi.org/10.1002/ps.4435.

Masuda, B.M., Fisher, P., Jamieson, I.G., 2014. Anticoagulant rodenticide brodifacoum detected in dead nestlings of an insectivorous passerine. N. Z. J. Ecol. 38, 110–115.

McDonald, R.A., Harris, S., Turnbull, G., Brown, P., Fletcher, M., 1998. Anticoagulant rodenticides in stoats (*Mustela erminea*) and weasels (*Mustela nivalis*) in England. Environ. Pollut. 103, 17–23.

McFadden, D., 1978. Quantitative methods for analysing travel behaviour of individuals: some recent developments. In: Hensher, D.A., Stopher, P.R. (Eds.), Behavioural Travel Modelling. Croom Helm, London, p. 307.

McLeod, L., Saunders, G., 2013. Pesticides used in the management of vertebrate pests in Australia: a review. NSW Department of Primary industries, Orange, New South Wales.

Mcmillin, S.C., Hosea, R.C., Finlayson, B.F., Cypher, B.L., Mekebri, A., 2008. Anticoagulant rodenticide exposure in an urban population of the San Joaquin Kit Fox. In: Timm, R.M., Madon, M.B. (Eds.), Proceedings of the 23rd Vertebrate Pest Conference, pp. 163–165.

Mendenhall, V.M., Pank, L.F., 1980. Secondary poisoning of owls by anticoagulant rodenticides. Wildl. Soc. Bull. 8, 311–315.

Monreal, M., Olive, A., Lafoz, E., del Rio, L., 1991. Heparins, coumarin, and bone density. Lancet 338, 706.

Mosterd, J.J., Thijssen, H.H.W., 1991. The long-term effects of the rodenticide, brodifacoum, on blood coagulation and vitamin K metabolism in rats. Br. J. Pharmacol. 104, 531–535.

Murray, M., 2017. Anticoagulant rodenticide exposure and toxicosis in four species of birds of prey in Massachusetts, USA, 2012–2016, in relation to use of rodenticides by pest management professionals. Ecotoxicology 26:1041–1050. https://doi.org/10.1007/s10646-017-1832-1.

Naim, M., Noor, H.M., Kasim, A., Abu, J., 2010. Growth performance of nestling barn owls, Tyto alba javanica in rat baiting area in Malaysia. ARPN J. Agric. Biol. Sci. 5, 1–13.

Naim, M., Hafidzi, M.N., Sudharto, P.S., Caliman, J.P., 2012. Detection of rodenticide residues for raptor conservation. 3rd International Conference on Oil Palm and Environment (ICOPE). Bali, Indonesia, pp. 22–24.

New South Wales Department of Primary Industries, 2011. Mouse monitoring and baiting [WWW Document]. URL. http://www.dpi.nsw.gov.au/__data/assets/pdf_file/0008/387719/mouse-monitoring-and-baiting.pdf (accessed 8.21.17).

New South Wales Government: Department of Primary Industries, 2017. Mouse baiting FAQs [WWW Document]. URL. http://www.dpi.nsw.gov.au/biosecurity/vertebrate-pests/pest-animals-in-nsw/mouse-and-mice-plagues/mouse-baiting-faqs#Rodenticides (accessed 8.24.17).

Newton, I., Wyllie, I., Freestone, P., 1990. Rodenticides in British barn owls. Environ. Pollut. 68:101–117. https://doi.org/10.1016/0269-7491(90)90015-5.

Newton, I., Dale, L., Finnie, J., Freestone, P., Wright, J., Wyatt, C., Wyllie, I., 1998. Wildlife and Pollution: 1997/98 Annual Report.

Newton, I., Shore, R.F., Wyllie, I., Birks, J.D.S., Dale, L., 1999. Empirical evidence of side-effects of rodenticides on some predatory birds and mammals. In: Cowan, D.P., Feare, C.J. (Eds.), Advances in Vertebrate Pest Management. Filander Verlag, Fürth, pp. 347–367.

Ninox boobook, 2018. IUCN Red List Threat. Species. URL. http://www.iucnredlist.org/details/62023787/0 (accessed 6.7.18 [WWW Document]).

Nogeire, T.M., Lawler, J.J., Schumaker, N.H., Cypher, B.L., Phillips, S.E., 2015. Land use as a driver of patterns of rodenticide exposure in modeled kit fox populations. PLoS One 10, E0133351. https://doi.org/10.1371/journal.pone.0133351.

Olsen, J., 2011. What is a southern boobook? Australian High Country Owls. CSIRO Publishing, Collingwood, Victoria, pp. 15–19

Olsen, P., Rae, S., 2017. Invalid evidence for purported 'collapse' in the number of breeding little eagles in the Australian Capital Territory. Canberra Bird Notes. 42, pp. 245–249.

Olsen, J., Fuentes, E., Rose, A.B., Trost, S., 2006. Food and hunting of eight breeding raptors near Canberra, 1990–1994. Aust. F. Ornithol. 23, 77–95.

Olsen, J., Downs, J.a., Tucker, T., Trost, S., 2011. Home-range size and territorial calling of southern boobooks (*Ninox novaeseelandiae*) in adjacent territories. J. Raptor Res. 45: 136–142. https://doi.org/10.3356/JRR-10-92.1.

Olsen, J., Debus, S.J.S., Judge, D., 2013. Declining little eagles *Hieraaetus morphnoides* and increasing rabbit numbers near Canberra: is secondary poisoning by pindone the problem? Corella 37, 33–35.

Park, B., Scott, A., Wilson, A., Haynes, B., Breckenridge, A., 1984. Plasma disposition of vitamin K1 in relation to anticoagulant poisoning. Br. J. Clin. Pharmacol. 18:655–662. https://doi.org/10.1111/j.1365-2125.1984.tb02526.x.

Rattner, B.A., Horak, K.E., Lazarus, R.S., Goldade, D.a., Johnston, J.J., 2014a. Toxicokinetics and coagulopathy threshold of the rodenticide diphacinone in eastern screech-owls (*Megascops asio*). Environ. Toxicol. Chem. 33:74–81. https://doi.org/10.1002/etc.2390.

Rattner, B.A., Lazarus, R.S., Elliott, J.E., Shore, R.F., van den Brink, N., 2014b. Adverse outcome pathway and risks of anticoagulant rodenticides to predatory wildlife. Environ. Sci. Technol. 48:8433–8445. https://doi.org/10.1021/es501740n.

Resch, H., Pietschmann, P., Krexner, E., Willvonseder, R., 1991. Decreased peripheral bone mineral content in patients under anticoagulant therapy with phenprocoumon. Eur. Heart J. 12, 439–441.

Riley, S.P.D., Bromley, C., Poppenga, R.H., Uzal, F.A., Whited, L., Sauvajot, R.M., 2007. Anticoagulant exposure and notoedric mange in bobcats and mountain lions in urban Southern California. J. Wildl. Manag. 71:1874–1884. https://doi.org/10.2193/2005-615.

Ruiz-Suárez, N., Henríquez-Hernández, L.A., Valerón, P.F., Boada, L.D.B., Zumbado, M., Camacho, M., Almeida-González, M., Luzardo, O.P., 2014. Assessment of anticoagulant rodenticide exposure in six raptor species from the Canary Islands (Spain). Sci. Total Environ. 485:371–376. https://doi.org/10.1016/j.scitotenv.2014.03.094.

Salim, H., Hafidz, M.N., Noor, H.H., Omar, D., Kasim, A., Abidin, C.M.R.Z., 2015. The effects of rodenticide residues deposited in eggs of Tyto alba to eggshell thickness the effects of rodenticide residues deposited in eggs. Sains Malaysiana 44:559–564. https://doi.org/10.17576/jsm-2015-4404-10.

Sánchez-Barbudo, I.S., Camarero, P.R., Mateo, R., 2012. Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. Sci. Total Environ. 420:280–288. https://doi.org/10.1016/j.scitotenv.2012.01.028.

Serieys, L.E.K., Lea, A.J., Epeldegui, M., Armenta, T.C., Moriarty, J., VandeWoude, S., Carver, S., Foley, J., Wayne, R.K., Riley, S.P.D., Uittenbogaart, C.H., 2018. Urbanization and anticoagulant poisons promote immune dysfunction in bobcats. Proc. R. Soc. B Biol. Sci. 285, 20172533.

Shore, R.F., Walker, L.A., Potter, E.D., Pereira, G., 2016. Second Generation Anticoagulant Rodenticide Residues in Barn Owls 2015. Lancaster, UK.

Soderquist, T., Gibbons, D., 2007. Home-range of the powerful owl (*Ninox strenua*) in dry sclerophyll forest. Emu 107, 177–184.

Stansley, W., Cummings, M., Vudathala, D., Murphy, L.A., 2014. Anticoagulant rodenticides in red-tailed hawks, *Buteo jamaicensis*, and great horned owls, *Bubo virginianus*, from New Jersey, USA, 2008 – 2010. Bull. Environ. Contam. Toxicol. 92:6–9. https://doi.org/10.1007/s00128-013-1135-z.

Stephenson, B.M., Minot, E.O., Armstrong, D.P., 1999. Fate of moreporks (*Ninox novaeseelandiae*) during a pest control operation on Mokoia Island, Lake Rotorua, North Island, New Zealand. N. Z. J. Ecol. 23, 233–240.

Stone, W.B., Okoniewski, J.C., Stedelin, J.R., 1999. Poisoning of wildlife with anticoagulant rodenticides in New York. J. Wildl. Dis. 35:187–193. https://doi.org/10.7589/0090-3558-35.2.187.

Stone, W.B., Okoniewski, J.C., Stedelin, J.R., 2003. Anticoagulant rodenticides and raptors: recent findings from New York, 1998–2001. Bull. Environ. Contam. Toxicol. 70:34–40. https://doi.org/10.1007/s00128-002-0152-0.

Thomas, P.J., Mineau, P., Shore, R.F., Champoux, L., Martin, P.a., Wilson, L.K., Fitzgerald, G., Elliott, J.E., 2011. Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada. Environ. Int. 37:914–920. https://doi.org/10.1016/j.envint.2011.03.010.

Tosh, D.G., Shore, R.F., Jess, S., Withers, A., Bearhop, S., Ian Montgomery, W., McDonald, R.A., 2011. User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use. J. Environ. Manag. 92:1503–1508. https://doi.org/10.1016/j.jenvman.2010.12.014.

Trost, S., Olsen, J., Rose, A.B., Debus, S.J.S., 2008. Winter diet of southern boobooks *Ninox novaeseelandiae* in Canberra 1997–2005. Corella 32, 66–70.

Twigg, L.E., Lowe, T.J., Martin, G.R., Gray, G.S., 1999. A Review of the Anticoagulant Pesticide Pindone.

Vidal, D., Alzaga, V., Luque-Larena, J.J., Mateo, R., Arroyo, L., Viñuela, J., 2009. Possible interaction between a rodenticide treatment and a pathogen in common vole (*Microtus arvalis*) during a population peak. Sci. Total Environ. 408:267–271. https://doi.org/10.1016/j.scitotenv.2009.10.001.

Walker, L.A., Turk, A., Long, S.M., Wienburg, C.L., Best, J., Shore, R.F., 2008. Second generation anticoagulant rodenticides in tawny owls (*Strix aluco*) from Great Britain. Sci. Total Environ. 392:93–98. https://doi.org/10.1016/j.scitotenv.2007.10.061.

Walker, L.A., Chaplow, J.S., Llewellyn, N.R., Pereira, M.G., Potter, E.D., Sainsbury, A.W., Shore, R.F., 2011. Anticoagulant Rodenticides in Predatory Birds 2011: A Predatory Bird Monitoring Scheme (PBMS) Report. Lancaster, UK.

Weidensaul, C.S., Colvin, B.A., Brinker, D.F., Huy, J.S., 2011. Use of ultraviolet light as an aid in age classification of owls. Wilson J. Ornithol. 123:373–377. https://doi.org/10.1676/09-125.1.

Young, J., De Lai, L., 1997. Population declines of predatory birds coincident with the introduction of Klerat rodenticide in north Queensland. Aust. Bird Watch. 17, 160–167.