Investigating the extent, severity, and drivers of meso-carnivore exposure to anticoagulant rodenticides across the northeastern states, with an emphasis on fisher (*Pekania pennanti*)

This report summarizes the work of a regional collaboration that will be presented at NEAFWA in May 2023, with some pieces published separately by state, and with the regional and NY fisher work to be completed this August as part of a MS thesis and PhD dissertation.

Principle Investigators (fisher work):
Dr. Jacqueline L. Frair, **Stephanie Cunningham (PhD student), Georgianna Silveira (MS student)**
State University of New York, College of Environmental Science and Forestry

Principle Investigator (bobcat work):
Dr. David Needle
University of New Hampshire, New Hampshire Veterinary Diagnostic Laboratory

Academic collaborators (all species):
Dr. Krysten Schuler
Cornell Wildlife Health Lab, Cornell University, College of Vet. Med., Animal Health Diagnostic Center

Dr. Lisa Murphy, **Dr. Julie Ellis, and Erica Miller**
University of Pennsylvania, School of Veterinary Medicine

Dr. Chris Whittier
Tufts, Center for Conservation Medicine

State Agency collaborators (key contact persons)
Shevenell Webb, Maine Department of Inland Fisheries and Wildlife
Patrick Tate, New Hampshire Fish and Game Department
Kim Royar, Vermont Fish & Wildlife Department
Mandy Watson, New York Department of Environmental Conservation
Aaron Facka and **Tom Keller**, Pennsylvania Game Commission
Gretchen Fowles, New Jersey Department of Environmental Protection

Introduction

Rodents have been recognized as a threat to human health and livelihoods for millennia (van den Brink et al. 2018). Our defense arsenal includes physical methods (e.g., traps, barriers), chemical methods (e.g., toxic baits, fumigants, repellents), and biological/cultural remediation (e.g., sanitation, habitat manipulation, resistant plants, house cats). In both commercial and private applications, toxic compounds often prove most efficacious because they can remain potent for long deployment periods and a single bait station can dose numerous individuals (compared to a snap trap that can catch only one animal at a time before needing to be rebaited and reset). The most popular toxic compounds are **anticoagulant rodenticides** (ARs; Tosh et al. 2011), which interfere with the body's blood clotting ability (specifically the vitamin-K mediated synthesis of blood clotting factors in the liver) to render animals that have ingested ARs vulnerable over time to fatal hemorrhage precipitated by minor trauma, exertion, and other factors (Stone et al. 2003). As a result, the lethal effects of AR ingestion may be delayed by several days which, in practice, means now toxic rodents persist on the landscape as

potential prey. And importantly, AR toxicity acts the same whether ingested directly by consuming bait or indirectly by eating something else that consumed bait.

The persistence and toxicity of ARs render these compounds of particular concern for biomagnification in predators (Horak et al. 2018; Fernandez-de-Simon et al. 2018; López-Perea et al. 2019). Indeed, rodent control applications around the globe have led to widespread AR exposure in non-target species from insects to raptors and large carnivores (Hindmarch and Elliott 2017). Despite restrictions in the United States to protect non-target wildlife from AR poisoning[1], secondary AR exposure remains alarmingly widespread. For example, in California 83-96% of tested black bear (*Ursus americanus*), bobcat (*Lynx rufus*) and fisher (*Pekania pennanti*) had evidence of ≥1 AR in their system, most having been exposed to 2-5 different compounds (Riley et al. 2003, Riley et al. 2007, McMillin et al. 2008, Gabriel et al. 2012, Serieys et al. 2015). Riley et al. (2003) reported acute toxicity from AR exposure as the second leading cause of mortality in coyotes over a 9-year period in the Santa Monica Mountains National Recreation Area (bordering Los Angeles). Yet, lethal concentrations of ARs vary widely within and among species (Quinn 2019). For example, lethal concentrations may be as low as 0.17 µg/g for one wild felid (caracal; Serieys et al. 2019) while exceeding 5.81 µg/g in another (bobcat; Serieys et al. 2015). Less well understood are the **sublethal effects from chronic exposure to ARs**, which may suppress reproductive capacity or immune function, the latter increasing an individual's vulnerability to comorbidity factors such as parasites, disease, and predators (Riley et al. 2003, Riley et al. 2007, McMillin et al. 2008, Gabriel et al. 2012, Serieys et al. 2015, Wiens et al. 2019). In laboratory tests, sublethal exposure to ARs produced upwards of 70% mortality when combined with other stressors (Jaques 1959). In a long-term field study of bobcats, secondary AR exposure (at ≥0.05 ppm) was related to the severity of infection of notoedric mange (an ectoparasitic disease; Riley et al. 2007, Serieys et al. 2015). Likewise, a negative association between AR exposure and body condition has been observed in weasels and stoats (Elemeros et al. 2011). In studies of humans, dogs, and sheep the reproductive consequences of AR exposure have included increased miscarriage, fetal toxicosis, fetal congenital deformities, and decreased sperm counts (Ginsberg and Hirsh 1989, Munday and Thompson 2003, Robinson et al. 2005). Together with the other stressors acting on wildlife populations, AR exposure may thus pose an important challenge for population persistence in the long-term and maintaining sustainable harvests in the short-term. Moreover, widespread use of AR may suppress forest carnivore numbers and the ecosystem services they provide as natural agents of rodent control.

The predator species most at risk of AR exposure are those whose diets depend heavily on rodents (so called meso-predators), as well as those living in close proximity to landscapes heavily influenced by human activities (Hindmarch and Elliott 2017). The northeastern United States supports the highest rural human population densities in the nation (Figure 1; left panel), leading to a high degree of human-wildland interface (Figure 1; right panel). As a result, we anticipate forest carnivores in the northeastern US to be at particularly high levels of risk for chronic exposure to ARs.

Following the call from Hindmarch and Elliot (2017) and others, we focus not only on documenting the extent of AR exposure within northeastern meso-carnivores (an important first goal) but also attempt to illuminate spatio-temporal drivers of AR exposure and potential fitness-related implications to help guide effective conservation action.

[1] https://www.epa.gov/rodenticides/restrictions-rodenticide-products

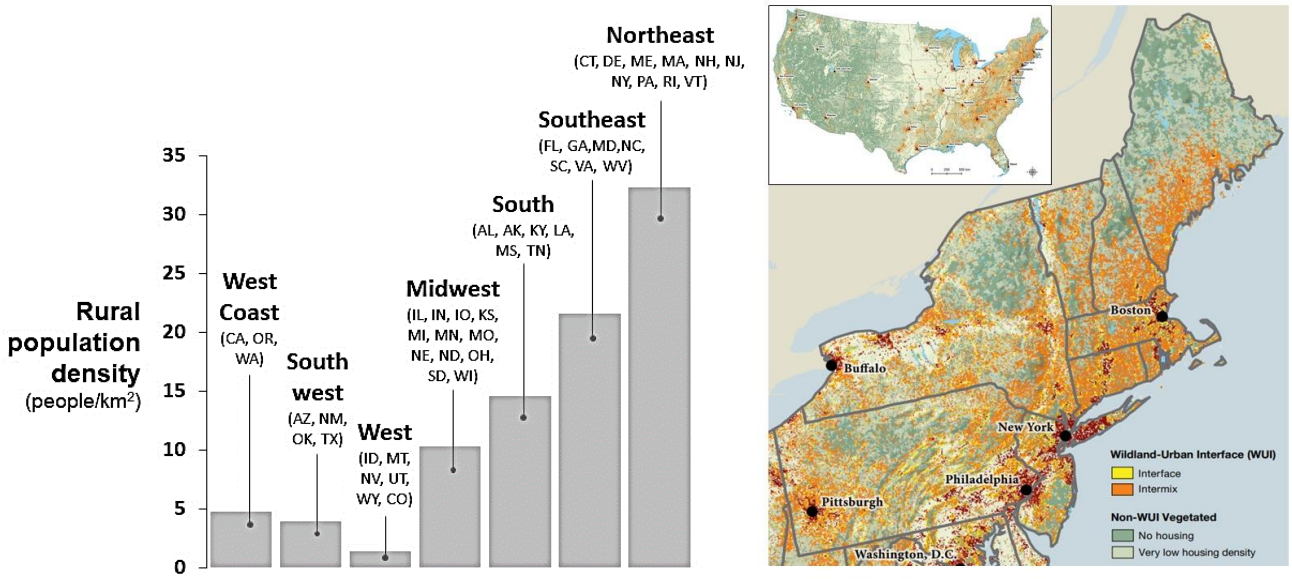


Figure 1. Differences in rural human population density across the US (data summarized from Demographia.com, 2000) and the corresponding map of wildland-urban interface (produced by the US Forest Service, 2010).

Project Goals and Supporting Objectives

The objectives of this are to:

1. Document the types, concentrations, and prevalence of ARs in northeastern meso-carnivores, with fisher (*Pekania pennanti*) as the focal species (Frair, ESF in the lead), bobcat (*Lynx rufus*) as a second focal species (Needle, UNH in the lead), and including other species of interest as available.

2. Map AR exposure in fisher across the Northeast and investigate potential landscape drivers (e.g., human developments, agricultural land use, and protected areas).

3. Relate AR exposure in fisher to measures of animal performance at the level of individuals (reproductive capacity, survival) and populations (e.g., trend).

Fisher, the focal study species.

Objective 1: Prevalence of ARs in meso-carnivores

General methods

Fisher became the focal species given a concurrent study tracking their productivity, recruitment, and survival within NY State (and the detection of 2 collared animals having died from AR poisoning), and alignment with general interest on fisher within the larger region. To acquire a "snapshot" of AR exposure within the standing live population of our target species we secured liver

samples as a byproduct of the regulated furbearer harvest. All field samples were collected by state agency personnel, and no outward indication of health concerns were expressed for any of the sampled individuals. To the extent possible we sought samples spanning the range of available habitat types and potential sources of AR exposure across each state. A second targeted sampling occurred for bobcat, with additional samples acquired from lynx (*Lynx canadensis*), river otter (*Lontra canadensis*), red fox (*Vulpes vulpes*) and gray fox (*Urocyon cinereoargenteus*) as desired by individual states. The great majority of these samples were collected between the Fall 2018 and Fall 2022 trapping seasons, however, some biobanked samples extend back to 2013. A sample of 23 incidental fisher mortalities from Pennsylvania (e.g., road killed animals) was also included after observing that rates of AR exposure were similar between the incidental and harvested sample of animals. NY further sampled fisher for 3 consecutive years, providing ≥100 samples/year, to investigate potential inter-annual variation in AR exposure rates.

We screened liver samples for 7 first-generation AR compounds (FGARs: chlorophacinone, coumachlor, coumafuryl, dicoumarol, diphacinone, pindone, valone, and warfarin) and 4 second-generation compounds (SGARs: brodifacoum, bromadiolone, difenacoum, difethialone) through the Pennsylvania Animal Diagnostic Laboratory System (PADLS; L. Murphy). Second generation ARs are more acutely toxic, requiring a single feeding only to deliver a lethal dose, and thus more highly regulated (Rattner & Mastrota 2017, Vandenbroucke et al. 2008). Quantification of AR concentrations had specification minimums of 0.010 ppm (brodifacoum, difenacoum), 0.025 ppm (bromadiolone), 0.050 ppm (chlorophacinone, difethialone, diphacinone), or 0.100 ppm (coumachlor, coumafuryl, dicoumarol, pindone, warfarin). Detections below these limits were marked as "trace" and considered positive detections (Gabriel et al. 2012).

Results

We tested 1,065 livers from a total of 627 fisher (from ME, NH, VT, NY, PA), 271 bobcat (NH, VT, NY, PA, NJ), 101 river otter (PA), 46 lynx (ME), 11 red fox (NH), and 9 gray fox (NH). From these sampled we detected 8 of the 11 AR compounds, 4 FGAR and 4 SGAR (Table 1). The number of different compounds detected varied by state (Figure 2), with a low of 5 compounds detected in ME to a high of 8 compounds detected in both NY and PA. Three compounds (difacinone [FGAR], brodifacoum [SGAR], and bromadiolone [SGAR]) were detected in all 6 states.

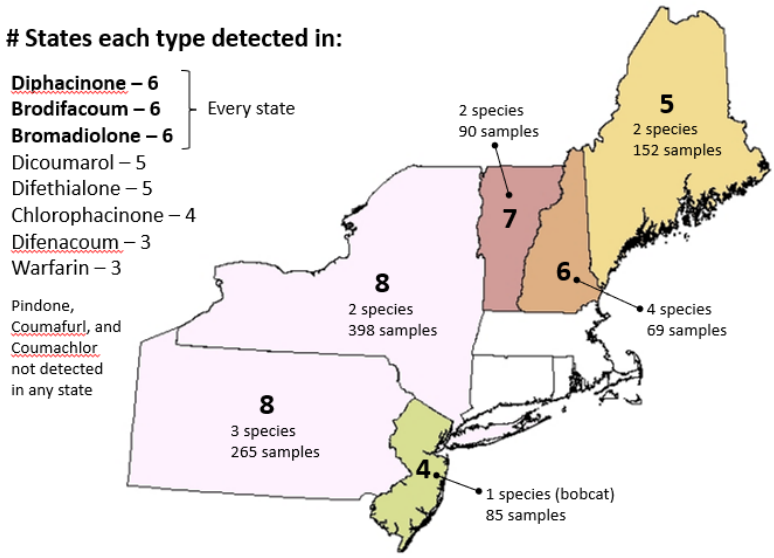


Figure 2. The number of AR compounds detected by state.

Table 1. Samples tested by species and state. Shown are the percent of samples testing positive for at least 1 AR compound (trace levels included) along with which AR compounds were detected within that species and state with First Generation (FG; gray) ARs indicated separately from the more potent Second Generation (SG; black) ARs.

Species	State	Sample years	# tests	% positive for ≥1 AR compound	Specific AR compounds detected										
					First-generation compounds							Second-generation compounds			
					Coumafuryl	Pindone	Coumachlor	Warfarin	Chlorophacinone	Dicoumarol	Diphacinone	Brodifacoum	Bromadiolone	Difethlalone	Difenacoum
Fisher	ME	2022	106	52.8%											
	NH	2020	15	93.3%											
	VT[a]	2018-2022	71	94.4%											
	NY	2018-2020	338	83.4%											
	PA[b]	2019-2022	97	70.1%											
Bobcat	NH		34	91.2%											
	VT	2021-2022	29	69.0%											
	NY	2016-2020	60	40.0%											
	PA[b]	2019-2022	63	49.2%											
	NJ	2013-2020	85	36.5%											
Lynx	ME	2021-2022	46	2.2%											
Red fox	NH	2020	11	100%											
Gray fox	NH	2020	9	100%											
River otter	PA[b]	2019-2022	105	17.1%											

[a] 45 samples from VT submitted for publication in Buckley et al. (2023, in revision)

[b] PA samples submitted for publication (Facka et al. submitted), lack specific data on AR compounds detected within bobcat and river otter, although Facka et al. (submitted) reports Warfarin also to have been detected within their study.

Fisher were exposed to the highest number of ARs overall (Table 1). For fisher, 970 tests detected AR residues, of which 51.4% yielded trace concentrations only and 48.6% yielded quantifiable concentrations. Of the 8 ARs detected, only 5 compounds returned quantifiable concentrations in fisher. For these 5 compounds, we observed similar mean levels among states (Figure 3), with the exception of Dicoumarol for which 1 animal in NY and 1 animal in PA registered concentrations of 2.14 and 1.78 ppm respectively. Importantly, AR concentrations are highly variable within a single individual over time, and as such quantification of AR impacts are better summarized by the *percent of the population exposed to one or more ARs* as well as the *number of different AR types* detected in an individual (van den Brink et al. 2018), both of which we consider next.

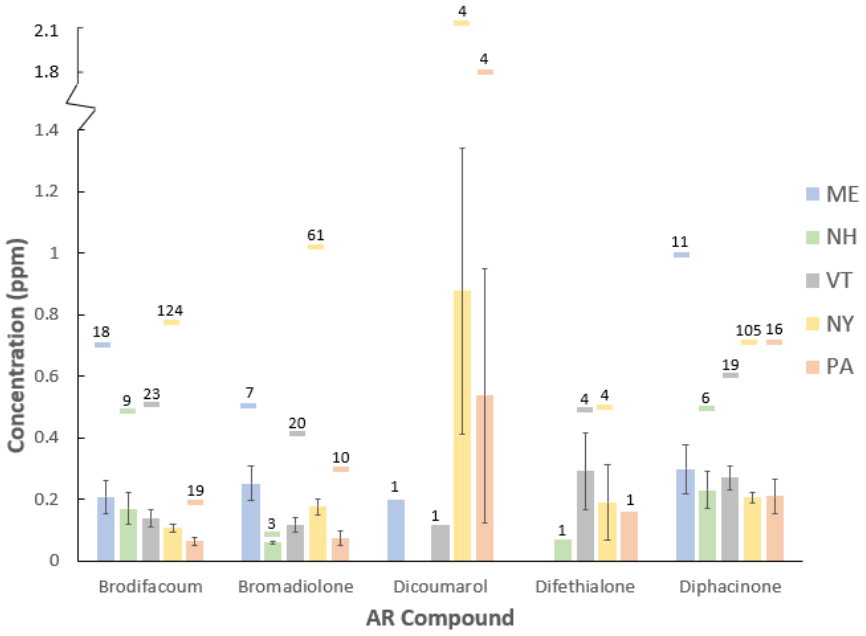


Figure 3. For ARs detected above trace levels in fisher, the bars indicate the mean ± SE concentration by compound and state, the corresponding lines indicate the maximum concentration observed for an individual sample, and the numbers indicate the sample size (i.e., the number of individuals for which concentration levels were quantifiable).

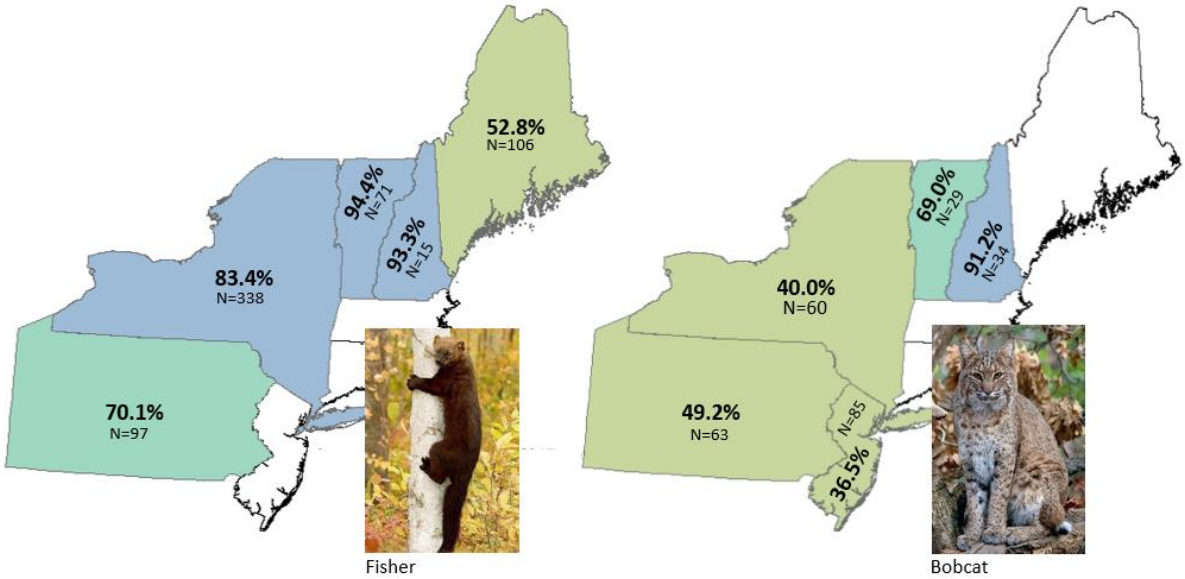


Figure 4. Percent of sampled fisher (left) and bobcat (right) testing positive for at least 1 AR compound at trace or higher levels of concentration.

The percent of the sampled fisher population exposed to at least 1 AR (at trace levels or higher) varied from a low of 52.8% in ME to a high of 93.3-94.4% in NH-VT (Figure 3 left panel), whereas bobcat exposure ranged 36.5% in NJ to 91.2% in NH (Figure 3 right panel). Lynx (in ME only) showed the lowest overall exposure rate at 2.2%. River otter (in PA only) tested positive at a rate of 17.1%, an unexpectedly high result given their primarily aquatic habit and dietary focus on aquatic sources of food rather than terrestrial small mammals. VT and NH, and to a slightly lesser degree NY, showed up as a "hot spot" for AR exposure. Although we observed a 26-52% decline in AR exposure in bobcats relative to fisher in VT, NY and PA, we detected only a 2.2% difference in their exposure level within NH. Likewise, red and gray foxes (in NH only) also exhibited high exposure levels.

As commercial products typically include a single active AR compound, residues from more than one compound in a given individual would result from unique exposure events and potentially arise from different landscape sources. We detected up to 6 different compounds within individual fisher,

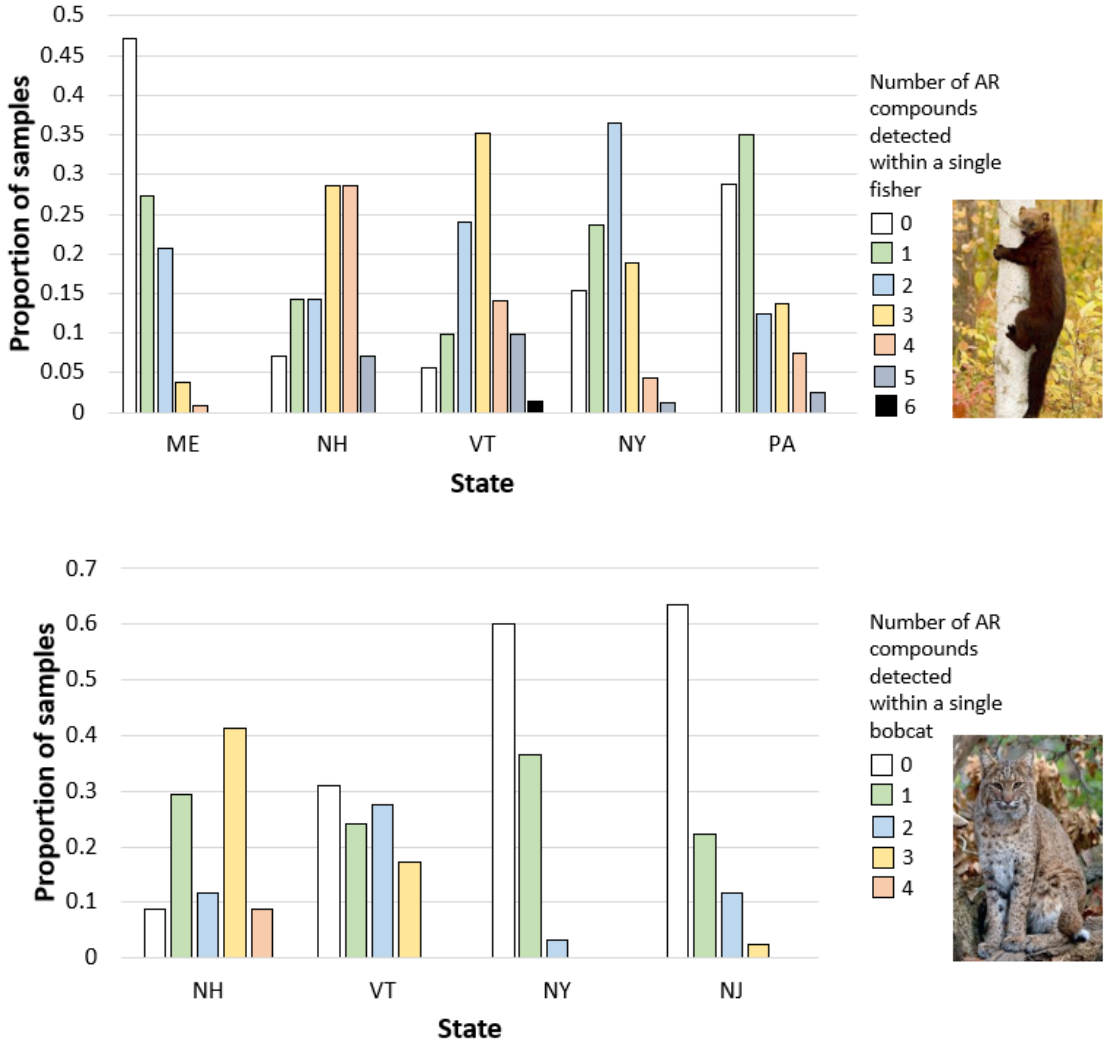


Figure 5. Proportion of sampled populations of fisher (top) and bobcat (bottom) exposed to each number of ARs as indicated.

and up to 4 compounds within individual bobcat (Figure 5). For fisher, more than half of the tested animals (61-79%) had residues of 2-6 compounds in NY, VT and NH (compared to 25-36% of the samples in ME and PA). For bobcat, 45-62% of the samples from VT and NH included residue from 2-4 compounds (compared to 3-14% in NY and NJ).

Objective 2: Mapping AR prevalence and isolating potential drivers

General methods

Using the multistate data from fisher, we:

1) Created a broad-scale map of the probability of fisher exposure to at least 1 AR and potential exposure to 1,2,3 or more compounds. This was accomplished using kriging, a process that relies solely on spatial autocorrelation among the data samples to fill in values for the spaces in-between samples.
2) Explored potential drivers of AR exposure by regressing landscape features (e.g., percent agriculture, wildland-urban intermix, and protected areas) against either a binary response (1 = at least 1 AR detected, 0 = no AR detected) or an ordinal response (0,1,2,3,…,6 types of ARs in the same sample).

Prior to fitting models, we had to deal with the uncertainty of the sample location, with "township" being the finest location resolution. Townships vary markedly in size and shape across this region, and taking the mid-point of a township as the location of the sample was an unsatisfactory solution. Instead, we embraced uncertainty in the sample location by randomly generating 10 possible x,y coordinates for each sample within its respective township, repeating each of our modeling efforts 10 times, and then averaging model results across the 10 sample iterations.

Kriging predicts values for each grid cell based on the pattern of spatial autocorrelation among samples (Krige 1966), and accounts for uncertainty in exact sampling locations better than other spatial interpolation

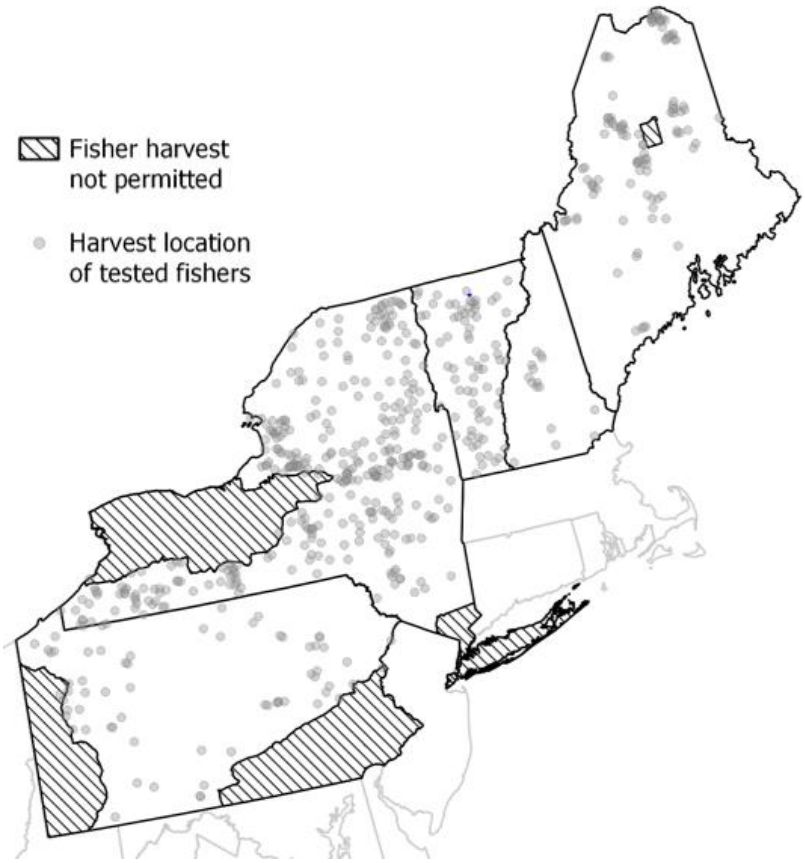


Figure 6. Approximate harvest location of the 597 fisher samples retained for spatial analyses (excluding those lacking sufficiently detailed location information).

methods. For this analysis we used a 30 km^2 grid cell to approximate the home size of male fishers (Arthur et al. 1989). The number of AR compounds was log-transformed prior to kriging and then back-transformed to its original scale. Kriging analysis and transformation were performed in R version 4.2.2 using the gstat package (Gräler et al. 2016, Pebesma 2004, R Core Team 2022).

To explore potential finer-scale drivers of AR exposure, for these same two response variables we fit regression models to compare the effects of agriculture, human development, and protected areas measured within 15-, 30-, and 60-km^2 buffers around each potential sample location. Within these buffers we calculated the percentage cover of agricultural land using the 2019 National Land Cover Database and considering either pasture or row crops or both classes together. We quantified human development using wildland-urban interface (WUI) data, which is defined by the Federal Register where human developments (namely buildings) occur adjacent to (interface) or intermixed within (intermix) vegetated wildland. Within each buffer we quantified the percentage of land in WUI interface or intermix. Lastly, using the Protected Areas Database of the United States (PAD-US) we calculated the percentage of area within each buffer covered by either GAP codes 1 and 2 (high degree of protection) or GAP code 3 (multiuse areas).

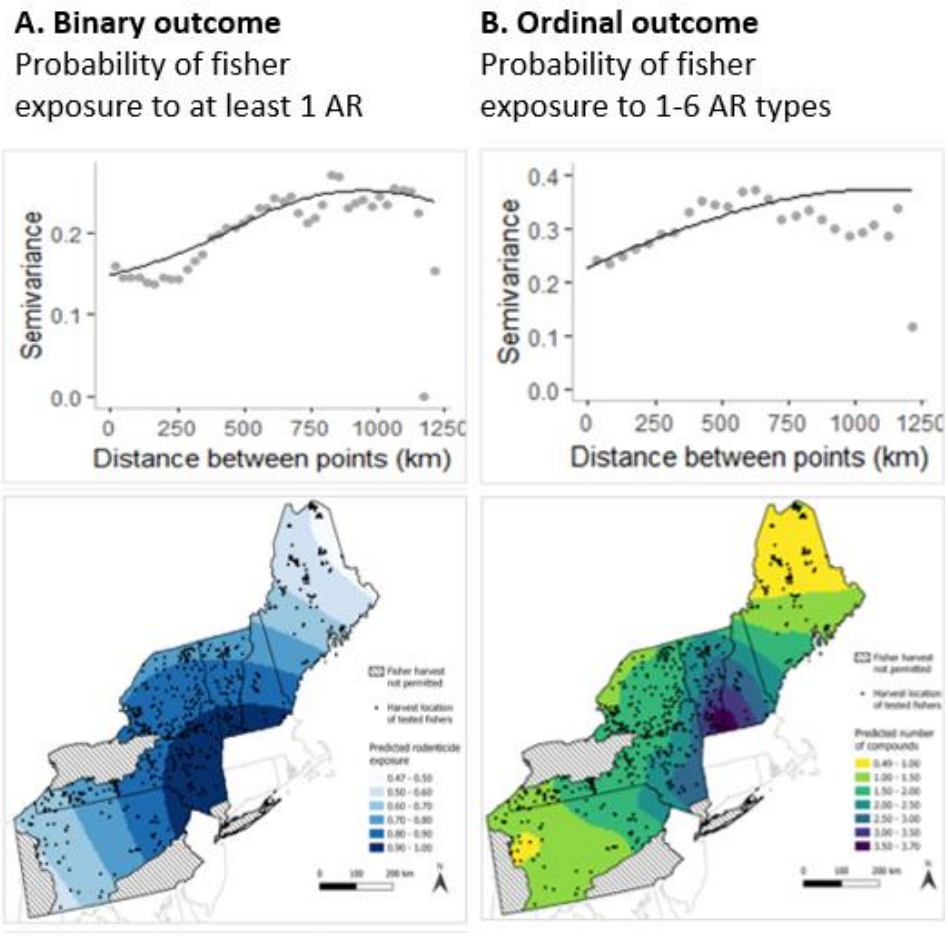


Figure 7. Kriging results for the binary (A) and ordinal (B) outcomes, with the semivariance (autocorrelation function) plotted at top and the mapped predictions based on those patterns of autocorrelation plotted at bottom.

Within each category of variables (agriculture, human development, protected areas), we used AIC model selection to determine the most informative variable(s), the most informative scale for selected variables, and whether the effect was linear (x) or non-linear (x+x^2). For the binary response variable, we fit logistic mixed-effects models using the R package lme4. For the ordinal response we fit under-dispersed Poisson mixed-effects models using the Conway-Maxwell-Poisson error distribution in R package glmmTMB. As this process involved fitting 10

different models (1 to each set of realized sample locations), we did not do model selection on the full model but rather fit a full model with variables representing each of three categories of landscape drivers. To evaluate variable importance we report the number of iterations (out of 10) in which a given parameter estimate was different from at α = 0.05.

Results

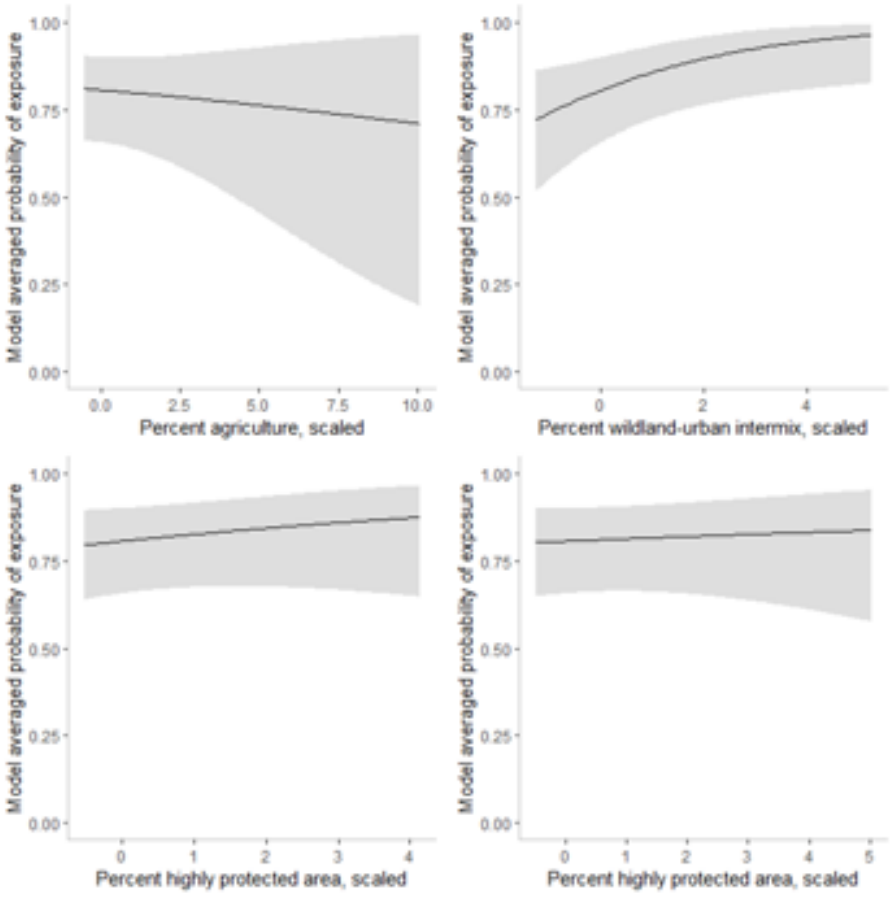
Semivariance plots indicate the range of autocorrelation in fisher samples to span ~750 km, which greatly exceeds the width of a given state in the region and indicates shared patterns of exposure across large swaths of space as exhibited in the resulting maps (Figure 7). Further, the semivariance plots indicate that ~40% of the variation in AR exposure can be explained by large-scale patterns of autocorrelation, leaving ~60% of the variance unexplained and likely due to finer-scale spatial patterns such as differential land use.

In our regression analyses, human development showed the most consistent relationship with both the binary (Table 2) and ordinal AR exposure outcomes. For the probability of exposure to at least 1 AR, human development was best modeled as the percentage of urban-wildland

Table 2. Summary of the global model predicting the probability of fisher exposure to at least 1 AR. The final variable type and scale used in the models is shown. The model was fit to 10 iterations of potential sample locations, with the average parameter estimates ($\overline{\beta}$) and average standard error ($\overline{SE}$) across all 10 iterations reported here. Also indicated is the number of iterations for which a variable was significant at α = 0.05 levels.

Variable Category	Variable type and scale used in models	$\overline{\beta} \pm \overline{SE}$	Number of iterations where P < 0.05
Intercept		1.43 ± 0.40	10
Agriculture	Crops, 60 km^2	-0.05 ± 0.10	0
Human development	Wildland-urban intermix, 60 km^2	0.34 ± 0.14	9
GAP 1 and 2 protected areas	60 km^2	0.13 ± 0.12	0
GAP 3 protected areas	60 km^2	0.04 ± 0.11	0

Figure 8. Partial slope plots showing the predicted effect of each covariate on the probability of fisher exposure to at least 1 AR.



intermix within a 60-km^2 buffer. For the probability of exposure to given number of AR compounds, percentage urban-wildland intermix was best measured within a 15-km^2 buffer. Besides these differences in scale the same patterns emerged, with urban-wildand intermix being positively associated with AR exposure probability. The percentage agriculture was the second most informative variable in each model, but whereas the coefficient for urban-wildland intermix was statistically significant in 80-90% of the iterations coefficients for the percentage agriculture were statistically significant in only 10-20% of the iterations. Moreover, the partial slope indicated a slightly negative effect of increasing agricultural cover on the probability of AR exposure in fisher. The extent of protected areas proved largely uninformative in all models.

Ongoing work

At present we are refining estimates of population trends for fisher across the region, which we will relate to changes in forest cover and condition, changes in climate, and, ultimately, potential AR exposure. We anticipate this final work to be completed by August 2023.

Objective 3: Exploring spatio-temporal drivers of AR exposure in NY and relating exposure to reproductive capacity

General methods

First, we explored spatio-temporal variation in fisher exposure to ARs across heterogenous New York State, an area spanning mixed agricultural and forested landscapes to wilderness areas within the Adirondack Park. We collected fisher data over a period of 3 years (2018-2020). The NY sample of fisher included 112 males and 226 females spanning 0.5-8.5 years of age (assuming birthdate of 1 April). Importantly, the NY fisher data indicated a 17% increase in the proportion of population exposed to at least 1 AR across the three years of sampling, given 76% in 2018 (N=100), 83% in 2019 (N=100), and 89% in 2020 (N=138). Therefore, some of the inter-state differences in AR exposure reported earlier may be due to among year differences in exposure rates.

As done previously for the regional analysis, we created 10 potential x,y coordinates for each observed sample location, fit full models to each set of sample locations, and report average coefficients across the 10 resulting models. Here we fit regression models to explain 1) the binary outcome (exposed to at least 1 AR), 2) the ordinal count outcome (# of different types of ARs), and 3) binary outcomes for specific AR compounds, namely the three most commonly detected ARs (brodifacoum, bromadiolone, difacinone). As described for the regional analysis, spatial variables were summarized within 15-, 30-, and 60-km^2 buffers. Here, spatial variables included percent agricultural land cover (cultivated crops and hay/pasture), percent forest, wildland-urban intermix or interface, and an index of beech mast availability (a combination of spatial variation in beech basal area x annual index of beech mast production). We anticipated a lagged effect between beech mast production and AR exposure in fisher given the intervening changes in small mammal communities in response to masting events. In addition, we included variables for fisher age and sex.

Second, we related AR exposure in fisher to differences in potential productivity. For this we retained 114 female fisher samples for which we had both tested for ARs and conducted histopathology

to count the number of corpora lutea. Corpora lutea indicate the maximum potential number of offspring that might be produced.

Results

The number of AR compounds detected was greater in males than females and increased with increasing fisher age up to 4 years after which exposure levels declined (Figure 9a). This pattern was consistent when predicting the total number of compounds as well as when predicting individual compounds (all three ARs showed same response). As the proportion of area (within a 60 km^2 buffer) surrounding a sample location increased in wildland-urban intermix so too increased the number of AR compounds to which fisher were exposed (Figure 9c). We observed a slight trend for the lagged effect of our masting count on the number of compounds detected (Figure 9b), with increased beechnut production corresponding to an increased number of AR compounds in fisher. In contrast to our expectations, the amount of agricultural area did not inform the number of compounds detected in fisher.

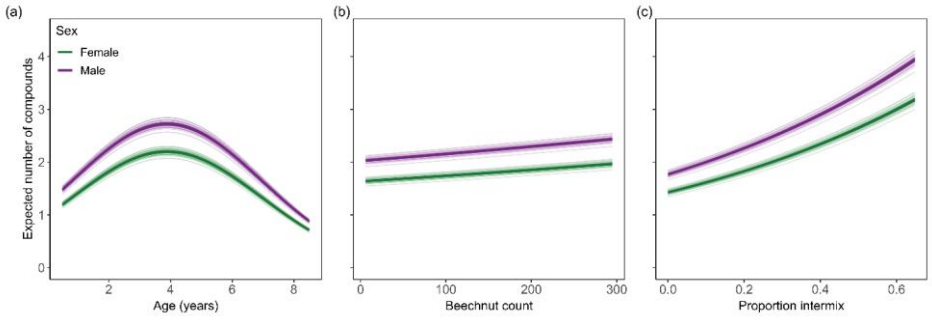


Figure 9. Partial effects of fisher age (a), beechnut count (b), and proportion of buffer classified as wildland-urban intermix (c) on the expected number of AR compounds detected within fisher.

With respect to specific AR compounds, the proportion of area classified as intermix remained a strong positive predictor, although with the magnitude of effect varying with AR type (Figure 10)

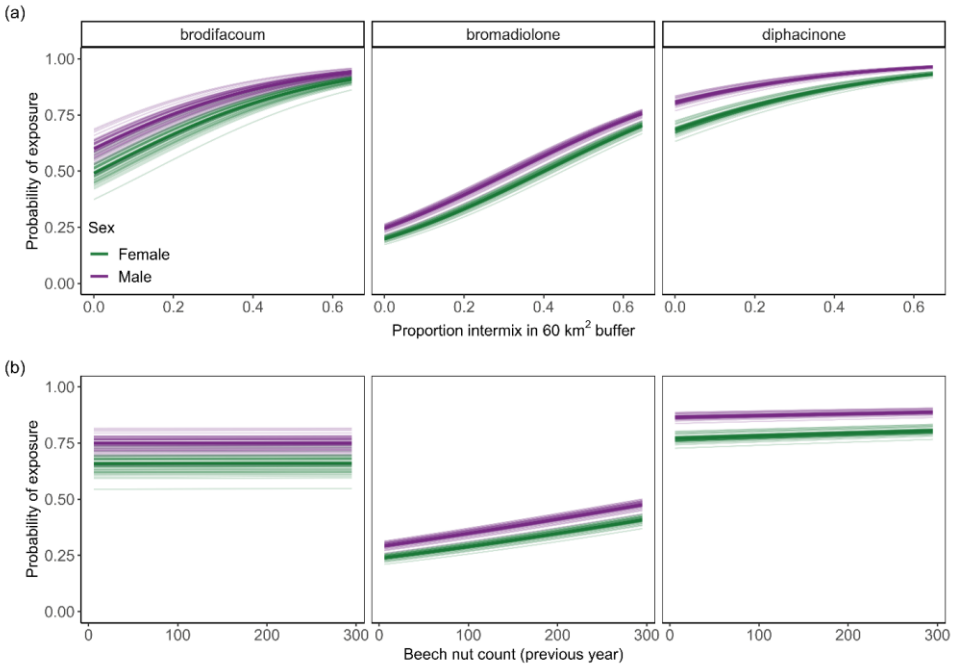


Figure 10. Partial effects of wildland-urban intermix (a) and lagged beechnut counts (b) on the probability of exposure to each of three specific AR compounds.

Ongoing work

We are currently relating AR exposure to potential fitness factors, such as productivity. Preliminary analyses indicate a slight negative trend (not statistically significant) between the number of AR compounds in a sample and total corpora lutea counts (Figure 11). Ongoing research will further attempt to relate collared fisher survival and recruitment rates to a measure of their blood-clotting ability in lieu of direct knowledge of AR exposure within live animals. We anticipate all analyses to be completed by August 2023.

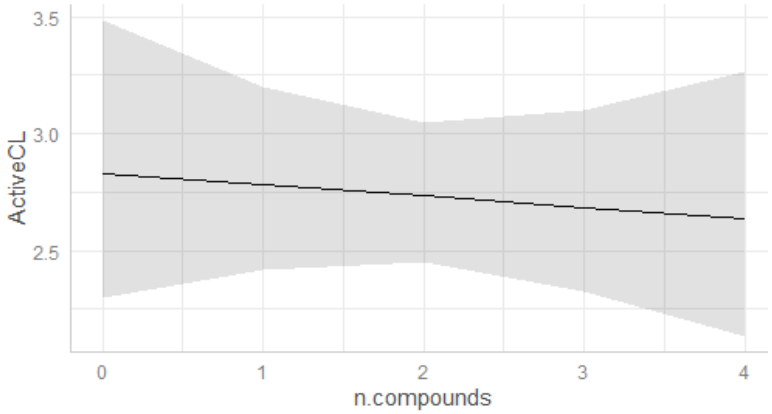


Figure 11. Relationship between the number of AR compounds detected in a fisher sample (x-axis) and the count of active corpora lutea (y-axis).

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