

# High Prevalence of Anticoagulant Rodenticide Exposure in New England Fishers (*Pekania pennanti*)

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## Research Article

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# Abstract

Anticoagulant rodenticides (ARs) are increasingly recognized as a threat to non-target species including native wildlife. Fishers (*Pekania pennanti*) are generally considered deep forest inhabitants that are not expected to have high exposure to ARs. To evaluate the distribution and levels of ARs in fishers, we analyzed liver samples from fisher carcasses (N = 45) opportunistically trapped across Vermont and New Hampshire between 2018 and 2019. Liquid chromatography-mass spectrometry was used to detect and quantify 11 different ARs in the liver tissue of each fisher at the time of trapping. All but one sample analyzed were positive for exposure to ARs, and 84% were positive for more than one type of AR. The most prevalent ARs detected were diphacinone (96%) and brodifacoum (80%). No samples had detectable levels of coumachlor, coumafuryl, difenacoum, pinodone, or warfarin. These results are mostly consistent with findings for fishers in the Pacific Northwest as well as with a variety of rodent specializing avifauna throughout the Northeastern USA, but show a higher prevalence of exposure and a different distribution of AR types than other studies. These results help establish current baseline exposure to ARs in fishers in the Northeast USA and suggest that ARs could pose a threat to wild mesocarnivore species in this region.

## Introduction

Anticoagulant rodenticides (ARs) are widely used to control rodent populations and reduce their damage in both urban and agricultural areas. In general, ARs are vitamin K1 antagonists that limit the synthesis of blood clotting factors (Hellemans et al., 1963) and herein will be categorized into first-, intermediate-, and second-generation ARs as outlined by Rattner and Harvey (2020): first-generation (FGARs) include dicoumarol, coumachlor, coumafuryl, and warfarin; intermediate generation (IGARs) include chlorophacinone, diphacinone, and pindone; second-generation (SGARs) include brodifacoum, bromadiolone, difenacoum, and difethialone. First generation ARs are less potent and have a shorter half-life, requiring rodents to repeatedly consume bait over several feedings in order to receive a lethal dose. In contrast, SGARs have a longer half-life and are more potent, usually making a single feeding lethal. Even if a target rodent has consumed a lethal dose of either compound, it will not succumb to the effects until a latent period of four to nine days later (Meehan 1985). This allows the rodent to repeatedly consume ARs beyond the lethal dose and become even more toxic to potential predators and scavengers. During this latent period, poisoned rodents uncharacteristically spend more time in open areas in a lethargic state- making them more susceptible to predation and enhancing the potential for non-target predator and scavenger exposure (Cox and Smith, 1992). This secondary toxicity and potential biomagnification could have damaging effects in ecosystems. Predators that exclusively or predominantly feed on rodents have proven to be at risk of secondary toxicity. Examples are well documented in avian species in the Northeastern United States including four different raptor species in Massachusetts (Murray and Tseng, 2008, Murray 2011, Murray 2017) and in seven different species across New York, New Jersey, and Connecticut (Stone et al. 1999, Stansley et al. 2014).

While ARs are detectable in a variety of biological samples, the liver is the optimal tissue for AR detection (Imran et al. 2015). Anticoagulant rodenticides will persist in the liver long after detectable levels in tissues like blood have dropped. Attempts to monitor for the presence of ARs in livers of wildlife are normally limited to post mortem sample analysis, hindering the ability to understand toxin level changes over time and only providing a snapshot at the time of death.

Currently, ARs are regulated under the Federal Insecticide, Fungicide, and Rodenticide Act by the Environmental Protection Agency (EPA). Where is states that ARs need to be strictly kept within 50 feet of a building for consumer use and 50–100 feet from man-made structures for professional and agricultural use, specifically to reduce the potential risk to non-target wildlife (Bradbury, 2008). While these regulations should help protect more rural wildlife that do not regularly approach buildings, AR toxicities in such mammals via secondary exposure have rarely been studied. This could be because wildlife that avoid human populated areas are presumed to have minimal risk to AR exposure. AR toxicities via secondary exposure have been noted in select rare and endangered predators, especially fishers in the Pacific Northwest (Riley et al., 2007).

Fishers (*Pekania pennanti*) typically avoid open and fragmented landscapes and preferentially reside in dense coniferous forests, a habitat that usually keeps them away from densely human-populated areas (Powell, 1993). Habitat loss, human encroachment, and possibly predator release have allowed fisher populations to expand in recent years especially in the Northeastern US where they may be increasingly found in peri-urban areas (LaPoint et al., 2015). Their diet is composed of mice, voles, shrews, rabbits, and a variety of other mostly small to medium sized mammals, reptiles, birds, insects, berries, (Powell, 1993), and even carrion (Zielinski et al., 1999). On the west coast, where some fisher populations are federally endangered, AR toxicity research has been ongoing and has shown fishers to be at high risk (Gabriel et al., 2012, Gabriel et al., 2015). In the Northeast where fishers are not threatened, AR exposure has not been well studied.

The land use in Vermont (VT) and New Hampshire (NH) are very similar with both states having a mosaic of farmland, human-developed land, and forested land interspersed across the state. Both VT and NH are highly forested 74% and 79% respectively, (Snyder and Sinclair, 2017, USDA, 2021) allowing remote habitat for fishers to be secluded from humans and our introduced toxins. The objective of this study was to determine if fishers in VT and NH are being exposed to ARs and if there are any patterns to such exposure.

## Methods

### Sample Collection

Carcasses were obtained by the Vermont Fish and Wildlife Department (VFWD) from animals legally trapped during the December 1st through December 31st 2018 season. Fishers from the New Hampshire Fish and Game (NHFG) legal trappings were between November 25th and December 31st, 2019. The 30

fisher samples from VT were selected from 23 different towns across the state with an effort to get a broad geographic distribution and limit repeated samples from the same towns. Samples from NH were representative of the distribution of harvested animals during the trapping season.

After collection, the VT carcasses were stored at -20°C until March 2019 when they were allowed to thaw for processing. Livers were dissected from thawed carcasses and placed into polyethylene storage bags (Whirl-pak, Nasco, Fort Atkinson, WI). These samples were re-frozen and shipped to the New Hampshire Veterinary Diagnostics Laboratory (NHVDL) and then transferred to Cummings School of Veterinary Medicine at Tufts where they remained frozen until being subsampled or directly submitted for analysis in May 2019. The NH carcasses were submitted to the NHVDL after harvest, and livers were dissected, placed in Whirl-pak bags, and refrozen. All samples from were sent for screening and quantification analysis of eleven ARs at the Pennsylvania Animal Diagnostic Laboratory System (PADLS) Toxicology Laboratory according to standard methods (Vudathala et al, 2010).

Fisher age was determined using lower premolar 4 tooth for cementum analysis at the Matson's Lab in Manhattan, Montana.

Quantitative AR results were based on the calibration curve prepared and run on the day of analysis. If the level of a rodenticide was below the established cutoff, but a peak was seen on the chromatogram at the correct retention time and with an appropriate ion ratio, it was reported as a trace amount detected. Screening and quantitative analysis was done for the following compounds at the specified detection limits: brodifacoum (0.010 ppm), bromadiolone (0.025 ppm), chlorophacinone (0.050 ppm), coumachlor (0.100 ppm), coumafuryl (0.100 ppm), dicoumarol (0.100 ppm), difenacoum (0.010 ppm), difethialone (0.050 ppm), diphacinone (0.050 ppm), pindone (0.100 ppm), warfarin (0.100 ppm).

## Results

The 45 fishers tested comprised 24 males, 20 females, and 1 unidentified individual and had an age class distribution of 28 juveniles, 9 subadults (~ 1 year old), 7 adults ( $\geq 2$  years old) and 1 that was unidentified. All but 1 fisher (98%) tested positive (trace or higher) for at least 1 of the 11 ARs (Table 1, Table 2). Most fishers (84%) were positive for more than 1 type of AR and more than half of fishers (58%) were exposed to 3 or more different AR compounds. Additionally, 1 individual from each state, both male, were exposed to 5 ARs. The most frequently detected AR compound was diphacinone, which is considered intermediate generation and was detected in 43 of the 45 samples (96%) at a range of 0.05 to 0.956 ppm. Brodifacoum was the most frequently detected SGAR with 36 samples (80%) testing positive at levels ranging from 0.01 to 0.468 ppm. The other ARs were detected with the following prevalence: bromadiolone (49%), chlorophacinone (24%), difethialone (20%), and dicoumarol (2%). Aside from 1 sample that had trace levels of dicoumarol, no samples had detectable levels of any FGARS (coumachlor, coumafuryl, difenacoum, or warfarin) nor the IGAR, pindone. Fishers from NH and VT had similar patterns for prevalence and detected levels of the different ARs. There were no obvious geographic patterns in either state. There were also no clear differences between age groups with the exception that juvenile

fishers (36%) had lower prevalence of exposure to bromadiolone compared to sub-adults (78%) or adults (71%).

## Discussion

The results of this study demonstrate that fishers in the northeast have a high prevalence of exposure to a variety of ARs. Non-target wildlife exposure to ARs in the northeast is well documented in birds of prey. In Massachusetts (MA), 95–99% of liver samples from birds of prey have been found positive for brodifacoum (Murray, 2011, Murray, 2017; Table 3). The high prevalence of brodifacoum exposure in our study (80%) is consistent with these regional findings as well as with a survey of fishers in CA where 92–97% of samples were positive for brodifacoum (Gabriel et al, 2012; Table 3). Our results are also consistent with CA fisher and MA raptor surveys in finding little or no exposure to FGARs, or the SGAR difenacoum and low to intermediate (3–49%) prevalence of exposure to bromadiolone and difethialone. However, our study found a higher prevalence of IGAR exposures with diphacinone prevalence at 96% being significantly higher than even the most exposed (15%) population of Central CA fishers ( $P < 0.0001$ , Fisher's exact test). While the exact amount and use of ARs by region is difficult to obtain, a 2017 survey of pest management companies in MA revealed that of the companies surveyed, the compounds predominantly used were; bromadiolone, difethialone, brodifacoum and diphacinone after 2011 (Memmott et al, 2017).

The specific levels of individual AR concentrations are difficult to interpret because these values are labile with expected decline after initial exposure and deposition in the liver. Moreover, the half-life of these compounds in fishers, and indeed any carnivores, is largely unknown (Kopanke et al., 2018). Likewise, lethal doses have not been determined for fishers for any of these compounds and studies of other mustelids have found large disparities even for the most common compound, brodifacoum (Fournier-Chambrillon et al., 2004, Alterio, 1996, Alterio et al., 1997). Clinical disease and presumed death in fishers has been observed in individual fishers in CA with AR concentrations in the liver of 0.38–0.66 ppm brodifacoum, 0.11–0.17 ppm bromadiolone, and trace amounts of chlorophacinone or diphacinone. While we did not detect brodifacoum levels that high in any of the VT fishers, we did find 2 NH fishers within that range (0.39 and 0.47 ppm). Our study also found individuals, which were notably live trapped, with much higher levels of bromadiolone (up to 0.29 ppm) and diphacinone (up to 0.96 ppm).

The widespread and often improper use of ARs may be a contributing factor to the bioaccumulation of ARs in rodent populations and could contribute to biomagnification of ARs within non-target species through secondary toxicity. This has important implications for the regulation of ARs and how they might negatively impact broader ecosystems. Larger predators including some common in the northeast (e.g., coyote, bobcat, domestic dog) have been known to kill or scavenge fishers (Gabriel et al., 2015), which could compound their own secondary AR exposure from rodents. As past research has shown, the loss of a major predator or predators could severely impact the balance of a sensitive or well-established ecosystem (Sergio et al., 2008 and Zielinski, 2014). In addition to these effects in the trophic cascade,

ARs may also persist in our ecosystem posing more environmental concerns (Hernández et al., 2013, Liu et al, 2015).

Although the cause of death for the fishers in our study was exclusively from being trapped for their pelts, sub-lethal AR levels may still be a risk for ineffective blood clotting. Fishers often suffer lacerations from prey while hunting, or through intraspecific trauma while defending territory. Minor wounds that would have otherwise healed, could prove much more severe as has been seen in raptors (Erickson et al. 2004). Persistent, sub-lethal AR levels may also suppress normal immune functions making predators more susceptible to diseases. Furthermore, neonatal transfer of ARs from a lactating female fisher to altricial kits has been documented (Gabriel et al., 2012) and may affect species fitness.

While this study starts to fill an information gap, there were limitations. Sample collection was opportunistic from available trapped fishers and location data was limited to the town where fishers were trapped. More intentional sampling and specific geolocations from a larger dataset would further our understanding of AR exposure risk in New England fishers. The carcass storage conditions between trapping and initial liver collection presumably varied widely within the maximum of 84 hours by which carcasses needed to be tagged by state authorities. The December time frame would predictably ensure a level of natural cold storage though some tissue degradation might occur. However, this should not be a significant factor as AR compounds persist in tissue samples and would only be expected to be higher in fresh tissues; the liver values found in this study are therefore a minimum.

This study shows trapped fishers are highly exposed to a wide spectrum of ARs across Vermont and New Hampshire. Whether this tends to be true of fishers across the northeast and whether this exposure poses a significant health risk, are open but important questions. Further understanding the source of ARs and their influence up the food chain from rodents to fishers is equally important and could help identify possible interventions including policies that may minimize fisher and similar mesocarnivore exposure to ARs. For example, it would be helpful to know whether fishers are directly consuming rat poison, rodents poisoned by legal human AR use, or are consuming forest rodents that might be poisoned from illegal use of ARs similar to the situation in CA related to remote marijuana cultivation.

Importantly, regardless of the source and whether the AR use is legal or not, the near universal exposure of the fishers sampled suggests that AR exposure is widespread and represents an underestimated health risk to wild fishers. It follows that the same risk exists for other mammalian rodent predators such as bobcats, foxes, and marten, all species essential to their ecosystems in the northeast. Wider use of AR alternatives, policy intervention, more public outreach and education about the propensity for secondary toxicity, and wider surveillance for wildlife health effects are warranted.

## Declarations

**Author Contribution Statement:**

Jacqueline Y Buckley and Christopher Whittier were the main drivers for the concept, design, data analysis, and interpretation, and prepared tables for the project. Jacqueline Y Buckley wrote the main manuscript text and acquired data through tissue subsample collection. Walter Cottrell, David Needle, Patrick Tate, and Kimberly Royar acquired data through sample collection and engaged in discussions for the methodology portion of the manuscript. All Authors provided critical revisions and approved the final version of this manuscript.

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This study made opportunistic use of carcasses from fishers legally harvested and in the possession of the New Hampshire and Vermont Fish and Wildlife Department and was therefore exempt from the Tufts University and University of New Hampshire Animal Care and Use Committees (IACUC) protocol approval.

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The authors have no competing financial interest in the research presented the authors report no competing financial or professional interests.

## Data Availability Statement

All data generated or analyzed during this study are included in this published article.

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## Tables

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