Anticoagulant Rodenticides on our Public and Community Lands: Spatial Distribution of Exposure and Poisoning of a Rare Forest Carnivore

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Abstract

Anticoagulant rodenticide (AR) poisoning has emerged as a significant concern for conservation and management of non-target wildlife. The potential of direct and indirect exposure to toxicants is to suppress pest populations in agricultural or urban settings. The presence of ARs in forest lands has recently raised concern for fishers (Martes pennanti), a candidate for listing under the federal Endangered Species Act. To investigate the magnitude of this threat, we tested 58 carcasses for the presence and quantification of ARs, conducted spatial analysis of exposed fishers, and identified fishers that died directly due to AR poisoning. We found 46 of 58 (79%) fishers exposed to one or more second-generation AR compounds. No spatial clustering of AR exposure was detected and the spatial distribution of exposed fishers was widespread within the fisher’s range in California, which encompasses mostly public forest lands. These ARs, which some are acutely toxic, pose both a direct mortality or fitness risk to fishers, and a significant indirect risk to the species that fishers are dependent on, exposure sources such as illegal marijuana cultivation in the range of fishers on California public lands.


Editor: Wayne M. Getz, University of California, Berkeley, United States of America

Received: March 27, 2012; Accepted: June 1, 2012; Published: July 13, 2012

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Funding: These authors have no support or funding to report.

Competing interests: The authors have declared that no competing interests exist.

Introduction
Anticoagulant rodenticide (AR) exposure and poisoning has emerged as a conservation concern for non-target wildlife. Eradicate or suppress rodent pest populations in agricultural or urban settings to minimize economic losses [1], and inhibit enzyme complexes responsible for the recycling of vitamin K$_1$, thus creating a series of deleterious clotting and coagulation effects grouped into two classes: first-generation compounds, which require several doses to cause intoxication, or often requiring only a single dose to cause intoxication and persist in tissues and in the environment [1], [4], [6], first-generation and second-generation ARs, prompting increasingly greater reliance on more acutely toxic compounds [8].

Primary exposure by ingestion of bait or secondary exposure through consumption of exposed prey has been documented in numerous common non-target wildlife [1], [3], [9], [10], [11], [12], [13]. Wildlife are thought to be of greatest risk of exposure where large quantities of these compounds are often used [12], [14], [15]. However, little is known about the risk influences.

Figure 1. Fisher (*Martes pennanti*) current range in California and project areas.

Current range (shaded areas) of the two isolated California populations of fishers (*Martes pennanti*). Areas of mortality to anticoagulant rodenticides are outlined within the two isolated populations.

doi:10.1371/journal.pone.0040163.g001

Fishers (*Martes pennanti*), a large mustelid and the largest member in the genus Martes, were once widely distributed throughout western North America, but have experienced significant population declines, including extirpation from some regions and contractions of historic ranges inhabiting California, Oregon, and Washington have been designated as a Distinct Population Segment (DPS) under the federal Endangered Species Act [17], [19]. The west coast DPS encompasses areas where fishers were extirpated a reintroduced population in the Cascade mountains of southern Oregon, and two extant and isolated populations California and another in the southern Sierra Nevada mountains of California [17], [19]. The population status of the isolated fisher population in the southern Sierra Nevada range is unknown; however population estimates for the isolated fisher population in the southern Sierra Nevada range are unknown; however population estimates for the isolated fisher population in the southern Sierra Nevada range are 150–300 fishers, with the status of the isolated fisher population in the southern Sierra Nevada range unknown. Because fishers in the DPS occur in and are dependent on mid to late-seral stage coniferous forests, toxicants have not been previously considered a likely threat to fisher populations.
We assessed the magnitude of AR exposure and poisoning among fisher carcasses submitted for necropsy for threats to population persistence of fishers in California. Additionally, spatial analysis of telemetry data from sampled fishers was conducted to identify potential sources of AR in the environment. We hypothesized that due to fishers being a forest-dependent carnivore, exposure to ARs...
Table 1. Exposure and mortality due to anticoagulant rodenticides (AR) fishers (*Martes pennanti*) within the two isolated California and southern Sierra Nevada.
doi:10.1371/journal.pone.0040163.t001

Figure 4. Quantification levels of anticoagulant rodenticides detected in California fishers.
Anticoagulant rodenticides (AR) brodifacoum (BRD), bromadiolone (BRM), difethialone (DIF), chlorophacinone (CHL), diphacinone coumachlor (COM) parts per million (PPM) levels detected in positive fishers (*Martes pennanti*) in California. (ppm). Red diamonds represent levels in fishers that died due to AR ingestion.
doi:10.1371/journal.pone.0040163.g004

Methods

Ethics Statement

All procedures involving animals were reviewed and approved by the University of California, Davis, Animal Car
Figure 5. Exposure to and mortality from anticoagulant rodenticides (AR) in fishers (*Martes pennanti* population).
Green circles represent negative fishers, yellow circles represent exposed fishers, while red circles are fishers that died due to AR.

Figure 6. Exposure to and mortality from anticoagulant rodenticides (AR) in fishers (*Martes pennanti* population).
Green circles represent negative fishers, yellow circles represent exposed fishers, while red circles are fishers that died due to AR.

Study Area

Fishers were captured in box traps modified with a plywood cubby box (model 207, Tomahawk Live Trap Company) with a VHF radio-collar and monitored via telemetry. Fisher carcasses were submitted from the two isolated Cal populations of California within tribal, private and public lands, and non-monitored fishers on public and private lands throughout Mountain borderlands of north central California (Figure 2). Carcasses from the southern Sierra Nevada California Adaptive Management Project (SNAMP) and the USDA Forest Service Kings River Fisher Project (KRFP); both in the northern and central portions of this population’s extent (Figure 3).

Sample Collection

Deceased fishers were collected by project personnel whenever a fisher was determined to be inactive for >24 hours.
detected or when unmarked fisher carcasses were opportunistically observed at the project sites or adjacent areas, until a complete necropsy to determine causes of mortality was performed by a board-certified pathologist specializing in wildlife at the California Animal Health and Food Safety Laboratory System (CAHFS) or the University of California Davis Veterinary Medical Teaching Hospital in Davis, CA, USA. During necropsy, the carcasses were submitted for screening and quantification of seven ARs at CAHFS by liquid chromatography of ARs and high-performance liquid chromatography to quantitate positive samples. The AR compounds tested included first-generation diphacinone (DIP), chlorophacinone (CHL), and coumachlor (COM); and second-generation ARs, brodifacoum (BRD). The reporting limits were 0.01 ppm for BRD, 0.05 for WAF, BRM, and COM, and 0.25 ppm for DIP, CHL, and DI. Below quantitate limits were labeled as “trace” concentrations. All results were reported on a tissue wet weight basis and reviewed by a board-certified pathologist.

Figure 7. Condition of the undisturbed mortality site in which a fisher (Martes pennanti) mortality due to anticoagulant rodenticides in the Sierra Nevada population was found. doi:10.1371/journal.pone.0040163.g007
Figure 8. Thoracic cavity hemorrhaging containing 150 ml of frank blood due to coagulopathy after I. fisher (Martes pennanti) from the southern Sierra Nevada population.

doi:10.1371/journal.pone.0040163.g008

Age classification was determined by tooth wear, sagittal crest or testicular/teat development, field and laborato [17], [18], [25]. Fishers were classified as kits when fully or semi-altricial and dependent on milk for nourishment months of age, sub-adults when between 13–24 months of age, and adults ≥24 months of age [17], [18], [25].

Statistical Analysis

Prevalence of AR exposure among fishers was calculated for the total sample, each sex and each age class. W sexes within and between the two California populations using two-tailed heterogeneity chi-square tests of assc number of anticoagulant rodenticides found per individual were analyzed with a two-way ANOVA [27]. All tests \
Cruncher Statistical Software, Kaysville, UT, USA) with an alpha level $p = 0.05$.

Spatial Analysis

For monitored fishers, telemetry locations were used to generate 95% minimum convex polygon (MCP) home-r the core area of movement within each individual fisher home-range within each project area [28]. For each fish timeframes were calculated using ArcView 9.1 home range extensions (ESRI Inc., Redlands CA., USA) [29]. Th initial capture until death, irrespective of the monitoring time; the second centroid incorporated fisher locations c centroid incorporated only the fisher locations collected three months prior to death. These two latter centroids prior to death were calculated because some ARs have relatively short half-lives and any spatial clustering in th sources of AR exposure. Only fishers with ≥3 months of monitoring were used for spatial analysis, individuals th were excluded.
Figure 9. One of several nine-pound buckets of anticoagulant rodenticide removed from an illegal northwestern California fisher (*Martes pennanti*) project boundary.
doi:10.1371/journal.pone.0040163.g009

Figure 10. Multiple packets of anticoagulant rodenticides found surrounding an illegal marijuana growth (*Martes pennanti*) project.
doi:10.1371/journal.pone.0040163.g010
Centroids were analyzed by spatial scan statistics to determine whether exposure to ARs, exposure to different individual compounds of ARs were distributed uniformly or spatially clustered in each of the two California populations. Harvard Medical School, Boston, MA, USA was used to evaluate two separate models. First, a Bernoulli model clustering occurred in exposed and non-exposed fishers, or in first or second-generation class AR exposure. The categorical data, was used to assign each fisher to a group based on the number of AR compounds detected at high numbers of AR compounds [31]. SatScan uses these models to scan the geographic area encompassing not more than 50% of the centroids [32]. The elliptical scanning window option was chosen for better fit to linear geographic features (i.e. drainages or ridgelines) that occur within the fisher's habitat models were generated by Monte Carlo simulations of 999 iterations and clusters evaluated for significance with alpha = 0.05.

Results

Population-level Exposure to AR

Forty-six of the 58 fisher carcasses tested (79%) were exposed to one or more compound of AR (Table 1). Frequency of exposure (p>0.05) were similar between populations and sexes (Table S1). The number of AR compounds detected at least one AR among age classes ranged with one of 4 pre-weaned kits (25%), 4 of 4 (100%) juveniles, 12 of first and second generation ARs were detected, with BRD being most common and detected in 44 of the 46 (96%) exposed fishers, DIP in two (15%), CHL in one (8%), and DIF in one (2%) of the 46 exposed individuals. Quantifiable levels of BRD (0.12 ppm; range trace 0.54 ppm) were detected while only trace levels of other AR compounds were detected (Figure 4). An indicator dye or AR bait was detected in either stomach or the GI contents of any fisher.

Northern California Fishers

Thirteen of 18 (72%) fishers from the northern California population were exposed to an AR compound (Table 1). BRD in one (15%), DIP in two (15%), CHL in one (8%), and WAF in one (8%) of the 13 exposed individuals.

Sierra Nevada Fishers

Thirty-three of 40 (83%) fishers from the southern Sierra Nevada were exposed to an AR compound (Table 1). BRD in one (2%), DIP in six (18%), CHL in three (9%), and DIF in one (3%) of the 33 exposed individuals.

Spatial Distribution of AR Exposure
Complete centroids were generated for 42 monitored fishers, 12 fishers from the northwestern California population (19 from SNAMP, 11 from KRFP). Of these fishers, 3-month MCP centroids were generated for 39 fishers, a (Table S2). Spatial analysis for 6-month centroids from the KRFP could not be conducted because all fishers in excluded from the analysis due to lack of monitoring data. No spatial clustering of AR exposure was detected for any of the temporal generation class of AR, or distribution of numbers of ARs per fisher in any of the study areas (Table S2; Figure 1).

AR-Mortalities

Cause-specific mortality factors for all 58 fishers sampled ranged widely and included predation, infectious and (M.W. Gabriel unpublished data). The cause of death for four of these fishers was attributed to lethal toxicosis, i coagulopathy and bleeding into tissues or cavities and ruling out any concurrent processes that might cause he were from the southern Sierra Nevada population, and two were from northern California (Table 1) and the case Southern Sierra Nevada

An adult male fisher was recovered on 15 April 2009, in the southern Sierra Nevada at the SNAMP project area scavenging (Figure 7). Gross necropsy determined that the fisher was in good nutritional (3.45 kg) and fair post the thoracic and abdominal cavities (150 ml and 100 ml respectively), and in the pericardial sac (7 ml) (Figure 8 contained some blood but no prey or formed feces, and no mucosal changes were noted. There were no other significant changes were observed in any tissues. Brodifacoum and BRM were detected and quantified in the livers.

CHL at trace levels (Figure 4).

The second fisher mortality was a lactating adult female recovered on 2 May 2010 in the center of a paved rural km from Yosemite National Park. Vehicular strike was initially suspected as the cause of mortality due to the loc visual evidence of trauma were not seen on gross examination of the intact carcass. The post-mortem state of t (2.54 kg). Shallow subcutaneous hemorrhage was noted over the hindquarters and spinal column with no assor approximately 20 ml of frank blood within the thoracic cavity. There was no evidence of pneumothorax, vessel n damage was seen in the abdominal cavity. Stomach contents contained various rodent parts with formed feces significant changes were observed in any tissues. Brodifacoum and BRM were detected and quantified at 0.60 ppm was detected at a trace level within the liver tissue (Figure 4). No evidence was present to suggest that this fish the highway.

Northern California

A sub-adult male fisher was recovered on 4 May 2010 at the base of several riparian shrubs near a watercourse ectoparasitism on the carcass was noted in the field with ticks in both replete and non-replete stages. Predation wounds. The gross necropsy determined that this fisher (2.65 kg) was in poor nutritional condition with no subcutaneous fat present. The right external ear canal, nasal and oral cavities, within the lumen of the trachea and within the periorbital tissue stomach was devoid of prey. The colon only contained semi-formed feces. Ectoparasitism was severe with approx (Dermacentor variabilis) and 8 female and 2 male western black-legged ticks (Ixodes pacificus) removed from v fisher had quantifiable levels of BRD at 0.04 ppm as well as a trace level of CHL (Figure 4).

The second northern California fisher AR death, was an adult male recovered on 26 May 2010 at the HVRFP. F scavenging. The nutritional state as well as the postmortem condition were poor. Gross necropsy determined th the tissues. Frank blood was present in both thoracic and abdominal cavities. The stomach contained red and b 204 female and 27 male adult American dog ticks in both replete and non-replete stages on areas of the muzzle sections. Severe nematodiasis was seen in skeletal muscle throughout the body (trichinosis). Pulmonary nemat portions of the lungs. Histopathologically, no notable disease processes were seen but severe parasitism was n
levels of BRD at 0.61 ppm and trace levels of BRM (Figure 4).

Neonatal Transfer of AR

Necropsies and AR testing was performed on four kits who were all still dependent on mother’s milk when they mothers death. One kit, a female fisher (0.32 kg) from KRFP tested positive for AR exposure. This kit was approx monitored maternal den tree shortly after maternal abandonment. Cause of death was determined to be acute ≤ trace level of BRD but there was no associated hemorrhaging in any tissues, body cavities or lumina, suggestin

Discussion

Our findings demonstrate that anticoagulant rodenticides, which were not previously investigated in fishers or ol and may represent a conservation threat to these isolated California populations. This is the first documentation fishers anywhere in their geographic range. Earlier studies suggest ARs posed little or no additive mortality effe shortfall of many of these studies was the utilization of common cosmopolitan species so they did not take in co otherwise compromised populations. The spatially ubiquitous exposure observed within all post-weaning age cl: contemporary range in California is of significant concern especially considering the recent work of Spencer et z in human-caused mortality of 10–20% in the isolated Southern Sierra Nevada fisher population would be enoug habitat elements were removed.

The high rate of exposure to second generation AR compounds (96% of exposed fishers) in these populations i ARs are not only more acutely toxic, but have long retention (>150 days half-life) through biphasic elimination in more toxic because death can occur from a single primary ingestion by a rodent [1], [5], [37], [38]. However, rod ARs in one feeding bout and it can take up to 7 days before clinical signs manifest [1], [39]. Therefore, prey that a substantial risk to predators for several days prior to death [39]. In one study, a group of Norway rats (Rattus i untreated food and another group had access only to the BRD bait [1]. Both groups consumed 10 and 20 median lethal doses by day 6.5, respectively [1]. If sources for these toxicants are maintained for even short periods, exposed populations [17] can pose significant threats to their predators.

Many manufactures use “flavorizers” since the AR compound may be bitter and unpalatable to rodent pests [1], sucrose, bacon, cheese, peanut butter, and apple flavors (Sure-Gro Inc., Brantford, Ontario, Canada and J.T. E: palatable to generalist carnivores like fishers. Although we did not visually detect AR bait in the stomach or GI tr be completely ruled out.

Sub-lethal AR Exposure

In addition to the risk from lethal toxicosis, sub-lethal AR exposure may compromise fishers through a reduction The occurrence of AR -exposed wildlife dying from minor wounds that otherwise might have easily resolved the lethal effects [1]. Several cases describe raptors receiving minor defensive lacerations or trauma from prey that hemorrhaging [1], [42]. Fishers actively pursue a wide array of terrestrial and arboreal prey [17], [18]. Hence, it i or trauma from prey, or during the pursuit of prey. Consequently, if clotting mechanisms were compromised due complications [1], [42], [43], [44]. The leading causes of mortality within the USFWS DPS is intraguild predation some of these cases, AR exposure could have compromised clotting mechanisms at the predation attempt and

High levels of tick infestations were noted in two of the AR mortalities when compared to other sympatric specie locations of of these replete ticks were in infrequent regions in other captures, most likely due to a lack of regul more ticks to obtain a blood meal due to immobilization due to compromised clotting factors is unknown.
Furthermore, sublethal AR exposure may decrease an animal’s resilience to environmental stressors. In a study, severe decreases in ambient temperature (i.e. frostbite), approximately 10% of test animals died; however when anticoagulants and subjected to the same stressors, mortality rates increased to 40–70% [46]. It is unknown if even pathogenic factors could predispose fishers to elevated mortality rates when coupled with AR exposure.

**Neonatal Transfer of AR**

The documentation of neonatal or lactational transfer of AR to a dependent fisher kit was unexpected, and the or shortly after birth are unstudied. AR exposure in pregnant or whelping domestic canids varied, causing no clinical coagulopathy immediately after delivery in other cases [48]. The female fisher who gave birth to this kit did not monitoring of her maternal den site verified that one kit survived from that litter (Rebecca Green, United States Forest Service, personal communication). Several fishers that were exposed had been monitored their entire lives and inhabited public or community lands where human stencilled or peri-urban or agricultural settings in which second-generation ARs could be purchased at local retailers, with recommendations for placement in wea- (EPA) regulations [39], second generation class ARs could be purchased at local retailers, with recommendations for placement in wea-

**Quantification Levels**

The quantity (ppm) of AR we observed in fisher liver tissues varied and overlapped extensively in both sublethai threshold that might indicate an amount leading to morbidity or mortality. This lack of predictive ability has been example, Brodifacoum, the most prominent AR compound detected in fishers in this study ranged considerably 0.32–1.72 ppm in stoats (Mustela erminea) [55], [56], [57], 0.7 ppm in least weasels (Mustela nivalis) [56], 1.47–1.67 American mink (Mustela vision) [3], [36]. In addition, there are stark differences for acute LD50 doses among genera, where minute amos caused death in domestic canids but domestic felids required doses 5 to 40 times higher [38]. The same variability suggests that predicting clinical thresholds for fishers would be pre-mature [1], [58]. Furthermore, AR exposed f systems, and possible interaction effects from a combination of 2 or more AR compounds within a fisher and otf

**Potential Sources of AR**

Spatial analyses did not reveal any obvious point sources of AR exposure. Instead, these analyses suggested that previous studies expected that exposure to AR compounds would be clustered near areas of human activity or outside of these areas [1], [12], [14], [24]. Incongruously, data from this study refuted this hypothesis and makes it clear that exposures occurred within a species that is not closely affiliated with urban, peri-urban or agricultural settings in [14], [24]. Federal and state regulations for anticoagulant rodentcide usage are specific for both generations. Bi (EPA) regulations [39], second generation class ARs could be purchased at local retailers, with recommendations for placement in wea-

The majority of habitat that fishers in California and fishers throughout the DPS currently and historically occupied is not within or near urban or agricultural stores (farm, tractor or feed stores) with additional form and weight restrictions [39]. These newly passed regulations are a testament to irresponsible and illegal use of ARs [39]. However, we would have expected that with either pre- or post-June 2011 would have overlapped with urban, peri-urban, or agricultural environments. This pattern is acknowledged in se (Lynx rufus) and mountain lion (Felis concolor) total quantification levels of AR exposure were associated with documented that secondary poisoning cases are closely associated with recent agricultural or urban pest erad
Therefore, exposure from first or second-generation A structures and settings were considered unlikely due to fisher habitat requirements and general lack of associat non-regulated use of second generation second generation ARs is occurring within the range of fishers in Califc A likely source of AR exposure to fishers is the emerging spread of illegal marijuana cultivation within California alone, over 3.6 million outdoor marijuana plants were removed from federal and state public lands, including sta both pesticides and insecticides found at grow sites [59], [60], [61]. In 2011, a three week eradication operation and 23,316 kg of trash including 68 kg of pesticides within the Mendocino National Forest in the northern Califor rodenticides and pesticides are typically dispersed around young marijuana plants to deter herbivory, [60], [62], placed along plastic irrigation lines used to draw water from in order to deter rodent chewing [60], [62], [63] (M.V.) which over 2,000 marijuana plants were removed less than 12 km from one of the project areas revealed that pl irrigation had large amounts of second generation AR placed (Figure 9, Figure 10, Figure 11). Finally, just withi km) of irrigation line within National Parks and Forests in California were removed [60], [62]. Placement of ARs great distances from the grow site itself may explain why there are no defined clusters of AR exposure.

It is noteworthy that the AR fisher mortalities we documented occurred in different areas of their California range mid-April to mid-May. We cannot specify the exact explanation or source contributing to all AR mortalities that o when females are providing for offspring as well as males searching for mates; however, preliminary spatial dat more confined home-ranges during this period, while males have slightly larger home-ranges (S. Matthews, R. d

Additionally, several books available to the general public identify the optimal time for planting marijuana outdoor especially vulnerable to rodent pests [64], [65], [66]. Of additional concern is that April to May is the denning pei entirely dependent on their mothers [17], [18]. The documentation of a lactating female mortality attributed to AR kits would be abandoned and die from female mortalities during this time.

In conclusion, this study has demonstrated that fishers in the western DPS, which are of conservation concern i Species Act, are not only being exposed to ARs, but ARs are a direct cause of mortality and indirect mortality (i. populations. Consequently, these toxicants may not only pose a mortality risk to fishers but could also pose sigir populations upon which fishers depend. The lack of spatial clustering of exposed individuals suggests that AR c and illegal or irresponsible use of ARs continues despite recent regulatory changes regarding their use. Becaus of these toxicants left on site long after marijuana grows are dismantled, heightened efforts should be focused c areas at the time of dismantling. Further regulation restricting the use of ARs to only pest management professi state wide Integrated Pest Management programs may be warranted. In addition, promotion of compounds that (i.e. zinc phosphide) should be considered in non-professional use settings. Furthermore, ARs in these habitats California carnivores such as the Sierra Nevada red fox (Vulpes vulpes necator), American marten (Martes ame or raptors such as northern spotted owls (Strix occidentalis caurina), California spotted owls (S.o. occidentalis) i should be directed to investigating potential risks to prey populations as well as other sympatric species that ma sources contributing to these exposure and mortality rates from anticoagulant rodenticides.

Supporting Information

Table_S1.docx
Table S1: A two-way ANOVA analyzing the effects of California fisher (Martes pennanti) populations and sex on the number of anticoagulant rodenticides found per individual.

<table>
<thead>
<tr>
<th>Variable</th>
<th>DF</th>
<th>Sum of Squares</th>
<th>Mean Squares</th>
<th>F ratio</th>
<th>Probability Level</th>
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<td>0.214</td>
<td>0.22</td>
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</table>

Table S2.

Results of spatial scan statistics to detect clusters of anticoagulant rodenticide (AR) exposed fishers within each California fisher minimum convex polygon (MCP) centroids used for each temporal period, specific AR types, ge ARs per fisher (number of AR positive fishers per test in parentheses) are shown.

(DOCX)

Acknowledgments

We would like to acknowledge the contributions of two anonymous reviewers as well as the following people an Veterinary Medical Teaching Hospital and pathologists Megan Jones and Patricia Gaffney and the (UCD) graduates and Richard Brown, the biologists at all the projects sites including Kerry Rennie, Rebecca Green, Tessa Smith, was provided by Integral Ecology Research Center, California Animal Health and Food Safety Laboratory Syste
States Forest Service, National Park Service, United States Fish and Wildlife Service and the Bureau of Indian. Linda Munson for initially taking the fisher health project under her wing. Her mentorship and contributions to will be appreciated.

Author Contributions

Conceived and designed the experiments: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW DLC. Performed the experiments: SMM JMH SMK KP RHB GMW. Analyzed the data: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW. Contributed reagents/materials/analysis tools: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW BNS DLC. Wrote the paper: MWG LWW RHP GMW BNS DLC.

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