LETTER

Impacts of Rodenticide and Insecticide Toxicants from Marijuana Cultivation Sites on Fisher Survival Rates in the Sierra National Forest, California

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Abstract

Secondary exposure of wildlife to pesticides has been well documented, yet exposure is typically associated with agricultural or wildland-urban interface areas. Wildlife in undeveloped areas is generally presumed free from risk. In 2009, a male fisher was found dead in the Sierra National Forest and subsequent necropsy revealed that the animal died of acute rodenticide poisoning. Follow-up testing revealed that 85% of fisher carcasses recovered by two research projects in the previous three years tested positive for rodenticides. Concern arose that exposure could predispose an animal to mortality from other causes, and that the underlying role of toxicants would escape notice. Further investigation indicated that the most likely source was the numerous illegal marijuana cultivation sites currently found on public lands throughout the western United States. To determine whether the presence of cultivation sites predisposed fishers to mortality from other sources, we related survival rates to the presence and number of cultivation sites found within that animal's home range over the past 10 years. Likelihood of exposure was related to the presence of cultivation sites, and female fisher survival was influenced by the number of cultivation sites within its home range. We discuss the conservation implications of this unexpected relationship.

Introduction

Secondary exposure of wildlife to anticoagulant rodenticides (AR) and other pesticides is widespread and has been well documented over the past 40 years. AR compounds have been found in numerous species including owls (Mendenhall & Pank 1980), bobcats (*Lynx rufus*; Riley *et al.* 2007), European mink (*Mustela lutreola*; Fournier-Chambrillon *et al.* 2004), polecat (*Mustela putorius*; Shore *et al.* 1999), stoats (*Mustela erminea*; Alterio & Brown 1997), badgers (*Taxidea taxus*; Proulx & Mackenzie 2012), mountain lions (*Puma concolor*; Litterel *et al.* 1988), and red-tailed hawks (*Buteo jamaicensis*; Stone *et al.* 1999). Testing is difficult, as it requires the recovery of liver tissue from an intact, nonscavenged carcass, yet when it is accomplished the occurrence of exposure is often found to be high. Dowding *et al.* (2010) found that 67% of European hedgehogs (*Erinaceus europaeus*) tested were positive for at least one AR compound. Hosea (2000) reported that 70% of animals sampled by the California Department of Fish and Wildlife, including bobcat, raccoon (*Procyon lotor*), red fox (*Vulpes vulpes)*, and coyote (*Canis latrans*), tested positive for AR exposure, and Riley *et al.* (2007) reported that 90% of Southern California bobcats tested were positive for exposure. And in a survey of 62 species in Spain, nocturnal raptors and carnivorous mammals showed the highest prevalence of AR exposure (62% and 38%: Sanchez-Barbudo *et al.* 2012).

Exposure of wildlife to other pesticides is likely to be equally widespread, yet can be more difficult to document. Unlike AR compounds, pesticides such as carbamate and organophosphate (OP) insecticides act rapidly and are less persistent in both the environment and within an animal's tissues (Grue *et al.* 1997). However, the direct and indirect implications of pesticide exposure to nontarget species have been well documented in relation to both responsible agricultural use and intentional misuse (Kendall & Smith 2003; Berny 2007; Richards 2011).

Impacts of exposure to AR and other pesticide compounds have been documented at local, regional, and global scales. Locally, toxicant exposure has been implicated in the wildlife declines due to both direct effects and interactions with other stressors such as parasites, pathogens, and predation (Berny *et al.* 1997; Winters *et al.* 2010; Lemus *et al.* 2011). Regionally, concern has been raised that widespread toxicant exposure may play a significant role in the population decline of species of conservation concern such as the European mink (*M. lutreola*) in France (Fournier-Chambrillon *et al.* 2004), sparrowhawks (*Accipiter nisus*) and kestrels (*Falco tinnunculus*) in Britain (Sibley *et al.* 2000), and the Eurasian otter (*Lutra lutra*: Lemarchand *et al.* 2011). Toxicant exposure has been linked with the worldwide decline of amphibians through interactions with parasites (Kiesecker 2002), pathogens (Rohr *et al.* 2008), environmental stressors (Relyea 2003), and trophic cascades (Relyea & Diecks 2008). Modeling efforts have also supported the concept that toxicant- related reductions in survival and reproduction may be sufficient to drive a population into negative growth (Roelofs *et al.* 2005).

Most reports of AR and pesticide contamination in wildlife occur in or adjacent to agricultural, urban, or suburban settings where legal use of rodenticides and other pesticides is widespread (Erickson & Urban 2004; Riley *et al.* 2007; McMillian *et al.* 2008; Proulx 2011). Reports of misuse, such as the intentional poisoning of predators, are less common and generally associated with a single location or event (Allen *et al.* 1996; Wobeser *et al.* 2004). One well-documented exception to this was a population decline of red kites (*Milvus milvus*) in Spain following an outbreak of rabbit hemorrhagic disease and extensive predator poisoning intended to increase rabbit hunting yields (Villafuerte *et al.* 1998). However, little is known about the potential sources and risks of exposure for animals living in relatively undeveloped landscapes with little anthropogenic influences (Richards 2011; Gabriel *et al.* 2012).

Fishers (*Pekania pennanti*) are a species of significant conservation concern in the western United States. Populations are small and highly fragmented (Zielinski *et al.* 1995, 2005), and considered at high risk of extirpation from stochastic events such as disease or wildfire (Spencer *et al.* 2011). Considered old forest-obligate species, their conservation is often perceived to be at odds with fire and fuel reduction efforts (Scheller *et al.* 2011). They are currently deemed a candidate species, "warranted but precluded," under the United States Federal Endangered Species Act, are a candidate for listing under both the Oregon and California Endangered Species Acts, and are considered a sensitive species in the western United States by the U.S. Forest Service. In both Washington and California, reintroduction efforts have recently been undertaken in order to reinstate the species in parts of its historic range.

Despite over 40 years of protection, fisher populations have failed to expand and recolonize historically occupied habitat. Recent genetic work suggests that much of the fragmentation, previously attributed to human activities such as development and railroad logging, may be in fact date back to ice age events (Knaus *et al.* 2011; Tucker *et al.* 2012). Yet numerous ongoing research projects agree that across the western United States, fisher population growth rates hover near zero and population expansion is not occurring (C. Thompson, USDA Forest Service, unpublished data; R. Sweitzer, University of California at Berkeley, unpublished data, Zielinski *et al.* 2013). Significant research efforts have been underway for the past 5 years, intended to document fisher ecological requirements and limiting factors as well as help identify management options for integrating fisher conservation with effective fire and fuel management (Thompson *et al.* 2011).

In April 2009, a male fisher that appeared to be in excellent health was found dead by members of the UC Berkeley Sierra Nevada Adaptive Management Project (SNAMP) fisher research team (R. Sweitzer, unpublished data). Necropsy revealed that the animal had died of acute AR poisoning (Gabriel *et al.* 2012). Specifically, 250 ml of frank blood was observed in the thoracic and abdominal cavities and three AR compounds were detected in the liver: brodifacoum at 0.38 μg/g, bromodiolone at 0.11 μg/g, and chlorophacinone at <0.25 μg/g. Given this unexpected degree of exposure, archived liver samples from fishers previously submitted for necropsy from both the SNAMP and US Forest Service Kings River Fisher Project (KRFP) were tested for the presence of seven AR compounds. Over 83% of the samples submitted by these two research projects tested positive for the presence of at least one AR compound (Gabriel *et al.* 2012).

Following this surprising result, efforts were made to identify potential sources of exposure. As fishers in the

southern Sierra Nevadas inhabit mountainous terrain between 1000 and 2400 m, they do not come into contact with agricultural fields or suburban developments where AR use is most common. Although there are isolated cabins and other structures where AR compounds might be legally or illegally used, fishers are territorial and exposure from a single point source, such as an isolated cabin, would therefore be limited to the single resident animal and not widespread. Similarly, some fishers do exist on the fringe of rural communities and exploit anthropogenic food sources. However, the animals tested had been monitored via radio telemetry for most of their lives and most (>90%) had not ventured into these rural communities (C. Thompson, USDA Forest Service, unpublished data; R. Sweitzer, UC Berkeley, unpublished data). Instead, these animals inhabited public, wildland areas managed for recreation and forestry, areas considered free of many anthropogenic influences. Subsequent conversations with law enforcement officers identified illegal marijuana cultivation sites on public lands as a possible source of exposure. Beginning in 2000, hundreds of illegal cultivation sites associated with Drug Trafficking Organizations (DTOs) have been found and eradicated within the Sierra National Forest, and law enforcement agents report finding large quantities of rodenticides and other pesticides at these sites. These sites are often located far from developments and roads, and in remote parts of the forests where detection is unlikely (Gabriel *et al.* 2013). And while each cultivation site would be best described as a point-source for AR or pesticide contamination, the sheer number of sites identified makes it a landscape-level problem.

Although direct mortality is obviously a concern, possibly more insidious is the potential for behavioral or physiological impacts associated with chronic or sublethal exposure (Grue *et al.* 1997; Fournier-Chambrillon *et al.* 2004; Berny 2007; Relyea & Diecks 2008). Chronic exposure to low doses of OP pesticides has been shown to significantly reduce the immune response of rats (Zabrodskii *et al.* 2012) and has been implicated in chronic neurological disorders in humans, including reduced memory and attention (Terry 2012). Sublethal doses of OP and carbamate pesticides have been shown to reduce thermoregulatory control in birds and mammals (Grue *et al.* 1997), induce pancreatitis in dogs and humans (Arnot *et al.* 2011), and cause partial paralysis associated with polyneuropathy (Paul & Mannathukkaran 2005; Lotti & Morretto 2006). Exposure to pesticides has also been shown to impair antipredator behavior: Cooke (1971) reported that tadpoles treated with DDT were more likely to be predated on by newts, and Farr (1977) found that exposure to an OP insecticide caused grass shrimp (*Palaemonetes pugio*) to be more easily captured by predatory fish. House sparrows exposed to a single, sublethal dose of the OP pesticide fenthion were 16 times more likely to be captured by a predator than controls within the same flock (Hunt *et al.* 1992).

Evaluating the impacts of pesticide exposures on free ranging wildlife can be difficult and is often limited to carcass counts in the field and detection of pesticides in postmortem samples, which primarily reflect acute intoxications. This is an opportunistic technique that can strongly underrepresent true mortality (Wengert *et al.* 2012). Many pesticides associated with acute mortalities can be detected from rather poor quality postmortem samples such as stomach contents and liver tissue, yet these samples are often unavailable in studies of free-living wildlife where animals are predated or scavenged (Morner *et al.* 2002). Assessing the sublethal impacts of pesticides exposures antemortem is often difficult as well, since the ability to detect specific pesticides is frequently impacted by low concentrations in only a few biological sample types. In addition, sample volumes can limit the sensitivity or breadth of analytical tests that can be performed and there are limited alternative biomarkers of adverse effect for many pesticides. Due to these challenges, studies linking pesticide exposure, particularly sublethal exposure, to morbidity or survival rates of free living animals are rare (Berny 2007; Richards 2011; Gabriel *et al.* 2012).

Analytical challenges notwithstanding, the ecological threat posed by contamination at these illegal marijuana cultivation sites is very real. In order to examine the potential impacts of AR and other pesticide use associated with illegal marijuana cultivation sites on fishers, we examined correlations between the number of known cultivation sites within an animal's home range and the presence of AR compounds in that animal's liver tissue. We also assessed whether the presence of illegal marijuana cultivation sites significantly impacted an individual's survival rate. We recognize that this is not necessarily a cause and effect relationship, nor was this a controlled and randomized study design. The illegal, clandestine nature of illegal marijuana cultivation, as well as all the challenges listed above, makes such a design impossible. Instead, we assumed that documented exposure to a limited suite of toxicants for which we could test (i.e., ARs) meant that the animal was at risk of exposure to all toxicants at the site, including those for which we did not test (OP and carbamate pesticides). This assumption is supported by the fact that fishers in the southern Sierra Nevada exploit a wide range of food resources including insects and carrion (Zielinski *et al.* 1999), and because baited pesticides, intended to kill mammals, are often found at these sites (M. Gabriel, UC Davis, personal observation). We also assumed that all illegal marijuana

cultivation sites are a potential source of exposure regardless of whether evidence of toxicants was recovered or not due to the fact that law enforcement agents often do not have the resources to carefully document and reclaim a site, and because stockpiles of these baited poisons are often cached or buried nearby in weatherproof, but not bearproof, containers (M. Gabriel, personal observation).

Methods

Study area

The study was conducted on the west slope of the southern Sierra Nevada, in the High Sierra and Bass Lake Ranger Districts of the Sierra National Forest, California. Field work was carried out between 1,000 and 2,400 m in elevation, corresponding to fisher occurrence in the region, and the study area included a mix of public and private land. The predominant forest cover types in the area are ponderosa pine (*Pinus ponderosa*), montane hardwood-conifer, and Sierran mixed conifer (Mayer & Laudenslayer 1989). Within the KRFP study area, the dominant private landowner is Southern California Edison (SCE) which maintains an active forestry program and does not utilize rodenticide or pesticide compounds (S. Byrd, SCE, personal communication). Other scattered, private inholdings do contain cabins or other seasonal structures where limited, legal use of rodenticide may occur. Within the SNAMP study area, additional development including the communities of Fish Camp, Sugar Pine, and Bass Lake exist that are occupied year-round.

Field data

Between February 2007 and December 2011, we captured and radio-collared fishers using protocols approved by the University of California at Davis and University of California at Berkeley Institutional Animal Case and Use committees. We captured fishers in Tomahawk box traps, baited with venison or chicken and equipped with a wooden cubby box attached to the back of the trap. Cubby boxes provide animals with a secure refuge where they are less likely to injure themselves biting at the wire cage. We transferred fishers from the trap into a metal handling cone, and anesthetized them for handling. We collared animals with either a Holohil or ATS VHR transmitter, weighing less than 40 g. After handling, we placed animals back into the cubby box and released them at the point of capture once they had fully recovered.

On the KRFP, we acquired location data using a combination of ground triangulation and walk-in techniques. Upon detecting an animal's signal, a technician immediately began collecting triangulation bearings. Given the rugged terrain fishers inhabit, technicians often collected 6–8 bearings before they felt comfortable about estimating the animal's position. If the animal appeared stationary, the technician attempted to follow the signal to the source and to identify the structure the animal was in. If successful, the technician used a handheld GPS unit to record the structure's location, and this information was used in place of the triangulation. If the walk-in was unsuccessful, meaning the animal moved before the technician identified the structure, the location was calculated using Locate II (Pacer, Nova Scotia, Canada). For home range analyses, we selected locations based on three or more bearings taken within 15 minutes and with an associated error polygon less than 10 ha. Mean location error was estimated at 97.1 m (SD = 89.4 m) based on the difference between triangulations and rest sites successfully located within 90 minutes of the triangulation bearings.

On the SNAMP, we relied primarily on aerial telemetry for location data. We conducted fixed wing flights 4–6 days per week weather permitting. The aircraft was equipped with one forward-mounted Yagi antennae for long-range detection and two side-mounted H-antennae for pinpointing animal location. When a signal was detected, the pilot oriented the flight path such that signal strength on the side mounted antennae was equal in order to pass directly over the collared animal. Once peak signal strength was achieved, the pilot circled back to mark the estimated location using either a mounted or handheld GPS unit. Mean location error, based on the use of test collars, was approximately 300 m.

On both projects, if a mortality signal was detected immediate attempts were made to recover a carcass. On KRFP, carcasses were generally recovered within 3–4 days of death. On SNAMP, due to the daily flights, carcasses were generally recovered within 24 hours. We submitted carcasses from both projects to the California Animal Health and Food Safety Laboratory at UC Davis for necropsy and cause-of-death identification. During necropsy, liver samples were collected and subsequently tested for the presence of AR using liquid chromatography-tandem mass spectrometry for screening presence of ARs and high-performance liquid chromatography to quantify positive samples. The AR compounds tested for included first-generation ARs, warfarin (WAF), diphacinone (DIP), chlorophacinone (CHL), and coumachlor (COM); and second-generation ARs, brodifacoum (BRD), bromodiolone (BRM), and difethialone (DIF). The reporting limits were 0.01 μg/g for BRD, 0.05 μg/g for WAF, BRM, and COM, and 0.25 μg/g for DIP, CHL, and DIF.

Locations of marijuana cultivation sites identified between 2002 and 2011 were provided by Sierra National Forest law enforcement officers. We included sites

identified between 2002 and 2007, before the start of the fisher monitoring program because (1) sites are often reused in subsequent years, (2) sites tend to be spatially clustered, and (3) the toxicants used at these sites may be cached and/or discarded after harvest, and contamination may continue for a number of years. Information on the toxicants found at each site was provided by both SNF law enforcement and the High Sierra Trail Crew (HSTC). HSTC is an all-volunteer organization dedicated to the maintenance of backcountry trails and facilities in the Sierra Nevada Mountains. In addition, they work extensively with law enforcement agents to assist with the reclamation of dismantled cultivation sites. An unknown and likely large percentage of cultivation sites remain undetected; however, the spatial clustering of these sites, associated with water availability and growing conditions, may limit the impact of undetected sites on our analyses. For example, it is likely that an animal whose home range overlapped three known sites actually overlapped five. Somewhat less likely due to the above-mentioned clustering, but possible, is the chance that an animal whose home range we thought to be clear of cultivation sites actually overlapped one or more. At the request of Sierra National Forest law enforcement, spatial data are not presented here.

Analyses

To evaluate the relationship between potential and actual exposure, we estimated three separate home range metrics for each female fisher with at least 25 locations per home range. We excluded male fishers from the analyses despite the fact that AR exposure in males appears to be near universal (M. Gabriel, UC Davis, unpublished data). However, their large home ranges (2635 $\pm$ 1870 ha; Thompson *et al.* 2010) and extensive breeding season movements make both recovering carcasses and determining the source of exposure more difficult. During spring, when toxicant use associated with illegal cultivation sites is highest (M. Gabriel, personal observation), male fishers cover large areas in search of females, while females show more site fidelity associated with dens and are therefore more likely to reflect exposure within a bounded area. We calculated 95% and 50% adaptive kernel (ADK) home ranges using the Home Range Extension program for ArcGIS. We used 95% kernel ranges to represent the likelihood that an animal came into contact with toxicants at any point throughout its life. We used 50% kernel ranges to represent a more focused risk; the impact of cultivation sites located within key foraging or resting areas. We also calculated a 100% minimum convex polygon (MCP) using locations from either the last six months of an animal's life or July–December 2011 for animals still alive. This 6 month, 100% MCP was calculated to account for the half-life of many of these compounds in the environment, as well as the fact that limited evidence suggests that the sublethal effects of a single pesticide dose may last less than 30 days (Arnot *et al.* 2011). We used an MCP model to represent temporally limited exposure, instead of an ADK model, because ADK models estimate space use based on location clustering rather than absolute location, and therefore better represent habitat preference. However, in the 6-month model, we were more interested in the absolute probability of exposure given all movements during that time frame. We then calculated the number of identified cultivation sites within each home range.

For fishers that died and sufficient tissue was recovered for AR testing, we compared postmortem AR exposure with the number of cultivation sites found within that animal's home ranges using standard univariate statistics. For female fishers, we calculated survival using the known fate model in program MARK. We then compared this base model with three reduced models incorporating the number of cultivation sites in the 95%, 50%, and 6-month MCP home ranges as covariates. Similar approaches, relying on mark-recapture data, have been used to evaluate the impacts of management actions on nontarget species (Davidson & Armstrong 2002).

Results

Presence of toxicants at cultivation sites

Approximately 315 illegal marijuana cultivation sites have been located within the combined KRFP and SNAMP study areas since 2002. Numerous toxicants have been found at these sites including both over-the-counter rodent control products containing brodifacoum and bromadiolone, OP insecticides such as malathion, and carbamate pesticides such as carbofuran which is currently banned in the United States (EPA 2009, SNF Law Enforcement, personal communication). Prior to 2010, there was no detailed documentation of the majority of cultivation sites (High Sierra Trail Crew, personal communication). In 2010, volunteer reclamation crews began keeping detailed records of toxicants and empty product packaging found. Of the 36 sites reclaimed in 2010 and 2011, toxicants were found and removed from 80% including malathion, carbofuran, carbaryl, and deltamethrin insecticides, brodifacoum and zinc phosphate rodenticides, and at least two unidentified substances. Approximately 25 kg of unused toxicants were removed from these sites along with numerous empty packages (SNF law enforcement, personal communication).

Table 1 Survival estimates for female fishers in the southern Sierra National Forest, based on a known fate model in Program MARK. Base model includes no covariates; other models include the number of illegal marijuana cultivation sites within three different types of home range estimates as a spatial covariate (95% adaptive kernel, 50% adaptive kernel, 100% minimum convex polygon using locations collected 6 months prior to death)

Model	AICc	Delta AICc	AICc weight	Annual survival estimate	Comparison to base model	
					Chi-square	*P*
95%	210.198	0.000	0.529	0.752	4.906	0.027
Base	213.095	2.897	0.124	0.718	–	–
50%	213.621	3.423	0.095	0.735	1.483	0.223
6 mo	214.671	4.474	0.056	0.721	0.433	0.511

AR test results

Over the 5 year sampling period, 46 animals died and were subsequently necropsied and tested for the presence of AR compounds. Predation was the largest source of mortality (88%); other sources included starvation, infection, and one case of direct AR poisoning. Thirty-nine (85%) tested positive for the presence of one or more AR compound. The most common toxicant detected was brodifacoum, an acutely toxic second generation AR. The number of compounds detected per individual ranged from one to four. While more mortalities occurred during that period, predators typically consume the viscera of their prey leaving insufficient tissue to test. Of the 46 animals whose carcasses were recovered with sufficient tissue available for sampling, spatial data sufficient to estimate home ranges were available for 37. For a more detailed summary of AR results, see Gabriel *et al.* (2012).

Relationship between home range, survival, and exposure rate

Female fisher home range averaged 1096 ± 637 ha ($N = 46$). The average number of cultivation sites within fisher home ranges was 5.3 for 95% ADK, 1.1 for 50% ADK, and 3.7 for 6-month 100% MCP. The relationship between the number of cultivations sites within the animals' home range and the presence of AR compounds detected at necropsy did not differ significantly between exposed and unexposed animals for the 95% and 50% ADK home ranges ($P = 0.235$ and 0.837) based on a 2-sample *t*-test. However, females with AR exposure had more cultivation sites within their 6-month 100% MCP home ranges than those without exposure (mean = 4.0 and 0.67, range = 0–16 and 0–1, $P < 0.001$). The base survival model estimated annual female survival at 0.718. The best performing model included the number of cultivation sites in the 95% ADK home range as a spatial covariate (Table 1).

Discussion

We found evidence that female fisher survival was related to the number of marijuana cultivation sites the animal was likely to encounter. Due to the difficulties outlined earlier, it is challenging to relate antemortem pesticide exposure with likelihood of mortality from sources such as predation or vehicular strike. However, the fact that fishers more likely to encounter cultivation sites suffered significantly higher rates of mortality indicates that exposure may predispose an animal to dying from other causes. It also opens the door for a wide range of conservation concerns based on research conducted on other species and in other venues.

The relationship we observed between the 6-month MCP and the probability of exposure likely reflects the persistence of these toxicants in an animals' tissue and our ability to detect contamination. It may also indicate a decline in toxicant availability at older sites due to remediation, environmental degradation, or consumption. Less clear is why the overall survival data were best explained by a model incorporating the number of cultivation sites in the 95% ADK home range but not the 50% ADK or 6-month MCP. The fact that both smaller ranges are embedded within the 95% ADK range may indicate that more cultivation sites within the 95% ADK range produces a greater overall risk of long-term repeated exposure, and that this may be a significant factor in survival. It may also indicate that current postmortem tests for AR compounds may not best represent the hazards of long-term exposure to multiple toxicants. Additional research is necessary to better understand how exposure risk may vary across the landscape, or what behavioral characteristics may predispose a fisher to exposure.

On both projects, the vast majority of location data were collected during daylight hours due to safety concerns. This could lead to an underrepresentation of time spent in developed areas, as has been observed for bobcats and coyotes (S. Riley, National Park Service, personal

communication). However, fishers are active throughout all hours of the day and territory mapping has indicated that diurnal locations give an accurate representation of habitat use (Thompson *et al.* 2010). Similarly, while the difference in location accuracy and sample size between the two research projects may introduce fine-scale differences in interpretation, it is unlikely to impact home-range scale analyses.

Exposure of wildlife to pesticides is widespread; however, the use of rodenticides and insecticides around illegal marijuana cultivation sites is a fundamentally different scenario than has been previously addressed by wildlife researchers. Typically, wildlife is exposed to these compounds through either legal application such as agricultural spraying, use within 50 ft of a building, or exotic pest removal programs. At cultivation sites, an inherently illegal activity where regulations are disregarded, multiple toxicants are used in large quantities with the intent of poisoning anything that might harm the crop.

These pesticides are used in conjunction with large quantities of fertilizer, raising the possibility of uptake into surrounding vegetation. In addition, cultivation sites are often near stream channels. Thus, not only terrestrial but aquatic wildlife are potentially exposed. Given the facts that the primary compounds in OP and carbamate pesticides were initially developed as nerve agents in World War II (Grue *et al.* 1997), that the use of pesticide-based weapons is an ongoing concern (Burklow *et al.* 2003; Terry 2012), and that exposure to multiple neurological agents is one plausible scenario for the elusive Gulf War Illness (Golomb 2008), the contamination occurring at illegal marijuana cultivation sites is more akin to leaking chemical weapon stockpiles than typical use or misuse of agricultural products (Zabrodskii *et al.* 2012). It should also be noted that even though marijuana is a high-profile crop, cultivation of any crop on national forest lands is illegal and it is the method of cultivation and the extensive use of toxicants, not the particular crop, which results in environmental contamination.

Based upon work conducted to date, fishers in the southern Sierra Nevada appear highly susceptible to all pesticide exposure (Gabriel *et al.* 2012). Unlike fishers in other parts of the country, which are larger bodied and tend to consume fewer, larger prey items, fishers in the southern Sierra Nevada exploit a wide range of resources including small mammals, birds, carrion, insects, fungi, and other plant material (Zielinski *et al.* 1999). Both AR and carbamate pesticide compounds have been found in invertebrates sampled at cultivation sites (M. Gabriel, unpublished data), and bioaccumulation of AR has been documented in both earthworms (*Aporrectodea calignosa*) and snails (*Cantareus asperses*) (Booth *et al.* 2003). Therefore, fishers are potentially directly exposed through the consumption of toxicants mixed with bait, and secondarily exposed through scavenging and predating upon contaminated small mammals and insects.

Often, marijuana growers return to productive sites in subsequent years even if the site was found and eradicated by law enforcement (Sierra National Forest law enforcement, personal communication; M. Gabriel personal observations.). They also cache pesticides near sites for future use, so even if a site is found and eradicated the cache may remain undetected and can continue to contaminate a site for several years (M. Gabriel, unpublished data). Therefore the potential for chronic exposure by second and third-order predators is plausible.

Exposure to rodenticide and insecticide compounds has been implicated in a number of behavioral and physiological conditions. Chronic exposure to low doses of OP pesticides has been shown to significantly reduce the immune response through reduced activity of the Th1 and NK cells, which are essential components in combating both intra and extracellular pathogens (Li & Kawada 2006; Janeway *et al.* 2007; Zabrodskii *et al.* 2012), and Riley *et al.* (2007) speculated that AR exposure predisposed both bobcats and mountain lions to notoedric mange. Vidal *et al.* (2009) found that voles exposed to the anticoagulant chlorophacine had a higher incidence of infection by the zoonotic pathogen *F. tularensis*. In 2009, four fishers on the combined SNAMP and KRFP study areas died as a result of infection with canine distemper. The timing and spacing of the mortalities suggested an epizootic event moving through the region (Keller *et al.* 2012). It is possible that the widespread pesticide contamination observed at marijuana cultivation sites might compromise the immune response of numerous individuals within the population, thus making a population more susceptible to a variety of pathogens and parasites. However, much additional work needs to be undertaken to answer this question.

Another concern is the number of different toxic compounds located at illegal cultivation sites and the potential for additive or synergistic effects (Thompson 1996). In laboratory tests with bluegill (*Lepomis macrochirus*) exposed to 37 combinations of various pesticides, effects were additive in 59% of combinations and synergistic in 35% (Macek 1969). In another experiment, the OP pesticides malathion and EPN dosed at one-fortieth and one-fiftieth of the LD50 doses, respectively, resulted in 100% mortality in domestic dogs (Cope 1971), indicating the potential for strong synergistic interactions between these compounds. Malathion in particular, a compound often found at illegal cultivation sites, has been shown to act synergistically with other pesticides (Olgun 2004). Given the variety of toxicants found at illegal cultivation sites and the fact that as many as four AR compounds were

detected in an individual fisher (Gabriel *et al.* 2012), the risk of interactive effects should be seriously considered.

The ability of an animal to recover from physical injury has also been shown to be negatively impacted by exposure to OP pesticides and ARs. OP exposure at sublethal doses, combined with physical injury, increased the likelihood of mortality in injured rats due to reduced immune system activity (Zabrodskii *et al.* 2002). Similarly, secondary sublethal exposure to ARs has been shown to reduce the blood-clotting activity in numerous animals including screech owls (*Otus asio*: Rattner *et al.* 2012), weasels (*Mustela nivalis*: Townsend *et al.* 1984), barn owls (*Tyto alba*: Webster 2009), and rats (*Rattus norvegicus*: Bailey *et al.* 2005). Erickson & Urban (2004) reported multiple instances where predators with liver concentrations of ARs as low as 0.03 μg/g died as a result of excessive bleeding from minor wounds inflicted by prey. For example, the authors reported a necropsy of a red-tailed hawk that "seemed to have exsanguinated through a minor toe wound," and was found to have a 0.46 μg/g liver concentration of BRD, and another necropsy of a great horned owl (*Bubo virginianus*) with 0.27 μg/g BRM and 0.08 μg/g BRD that "died from hemorrhaging of minor wounds inflicted by prey."

Finally, sublethal exposure to pesticides has been shown to cause short-term hypothermia in both birds and mammals (Grue *et al.* 1991; Gordon 1994). Martin & Solomon (1991) reported that mallard ducklings (*Anas platyrhynchos*) exposed to a sublethal dose of carbofuran suffered hypothermia and enhanced mortality at 10 °C. Ahdaya *et al.* (1976) reported that the LD50 dose of either OP or carbamate pesticides was reduced by as much as a factor of 5 at both higher and lower temperatures in mice, indicating that exposed animals were unable to adequately thermoregulate, and Jaques (1959) documented similar interactions between temperature and AR compounds. Given that fisher exposure to these contaminants peaks in the spring (Gabriel *et al.* 2012) when females are providing for dependent kits and temperatures are highly variable, reduced thermoregulatory ability could result in female mortality, a reduction in her ability to forage, and kit abandonment. Furthermore, it has been documented that AR compounds can be transferred from a female fisher to dependent kits through lactation (Gabriel *et al.* 2012), and female fishers frequently provision weaned kits with small mammals (C. Thompson, personal observation). Therefore, the possibility that kit survival could be reduced must be considered as well.

The association between illegal marijuana cultivation sites, AR and other pesticide exposure, and fisher mortality is strong yet speculative. Determining a cause and effect relationship would require novel testing procedures and either an experimental framework or an extremely challenging, logistically difficult collaboration between the scientific and law enforcement communities, given the inherent dangers of visiting and monitoring these sites. In order to evaluate the strength of the association between AR exposure and mange in native felid predators, Riley *et al.* (2007) modified a framework for inferring causal relationships in wildlife disease (Susser 1973), and applied it to the contamination of free ranging wildlife: strength of the association, specificity of the association, coherence with current knowledge about the effects of exposure, time sequence, and consistency. We have established a statistically significant association between AR exposure and female fisher survival. Specificity of the association and coherence with current knowledge is difficult to address due to the numerous ways in which pesticide exposures may manifest and influence survival rates. While more information is needed, the relationship between a fisher's movements over the last 6 months of its life and access to AR contaminated cultivation sites suggests a relevant time sequence. To the best of our knowledge consistency of the relationship cannot yet be addressed, as this is the first reported analysis of the potential impacts of illegal marijuana cultivation sites on the survival of free ranging carnivores. Increasing the amount and breadth of testing, as well as the development of accurate ante-mortem testing procedures, will dramatically enhance our ability to interpret the population-level impacts and represents the quickest route to establishing cause and effect relationships.

The potential existence of an underlying, anthropogenic-based, previously unrecognized factor increasing mortality rates for a USFWS candidate species previously thought to be free of such influences raises significant conservation concerns. Under current research protocols such a factor could easily go unnoticed; cause of death is often determined in wildlife research yet once the mortality has been categorized based on field or genetic evidence, underlying causes are rarely investigated. Yet this emerging stochastic risk has the potential to shift a population from a positive to a negative growth rate, putting a sensitive population further in peril. Based on long-term carnivore monitoring data, Zielinski *et al.* (2013) concluded that fishers in the southern Sierras showed stable occupancy rates over the past 8 years. Yet Spencer *et al.* (2011) suggested that the population was not expanding despite the existence of suitable, unoccupied habitat, potentially due to high mortality rates. The authors state that a 10–20% reduction in survival would be sufficient to interfere with population expansion, and conclude that increased mortality is likely limiting the natural recolonization of unoccupied habitat. While data quantifying the impacts of secondary poisoning on nontarget wildlife survival

rates are rare, Robertson & Colbourne (2001) estimated that secondary exposure to brodifacoum increased the natural mortality rate of little spotted kiwis (*Apteryx owenii*) by 3–19%, and Davidson & Armstrong (2002) estimated that the survival rate of a rare New Zealand bird, the saddleback (*Philesturnus carunculatus rufusater*), was reduced by 45% following a brodifacoum-based rodent control operation. Given the breadth of potential direct and indirect impacts described above, the possibility that widespread AR exposure is reducing fisher survival rates sufficiently enough to limit population expansion must be considered.

Future work is needed to (1) improve the antemortem biomarkers used to indicate exposure to pesticides; (2) document the spatial and temporal scales of environmental contamination and wildlife exposure; (3) more fully evaluate the risk of exposure to diverse species; and (4) determine the potential population-level impacts for species of conservation concern. Although we do not yet have the data to interpret the long-term ecological consequences of this unprecedented level of site-specific contamination on public lands, the negative impacts are clear and priority must be given to the identification, documentation, and reclamation of these sites, and educating the public about these illegal actions on their communal lands.

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