



North Coast Resource Partnership Panel (NCRP)

Marijuana Cultivation: Impacts, Strategies, Upcoming Legislation & Economic Opportunities

October 16, 2014

12:15 Welcome/Context/Introductions:

Judy Morris, Trinity County Board of Supervisors, Vice-chair NCRP

12:30 Panel Presentation/Discussion

- Peggy O'Neill, Director & Nicole Sager, Senior Planner, Yurok Tribe Planning Department
- Matt St John, Executive Officer, Regional Water Quality Control Board, Region 1
- Cris Carrigan, Director, Office of Enforcement, State Water Resources Control Board
- Hezekiah Allen, Executive Director, Emerald Growers Association

1:10 Policy Review Panel (PRP) Round Table Discussion

3 minutes each PRP member – opportunity for panelists to comment and participate

Questions (PRP and Panel members may respond to any/all or address other topics)

1. What are the most important actions that state, local, federal and tribal governments and organizations can and should take **now** to address the social and environmental impacts of illegal cultivation?
2. If marijuana is legalized in California (potentially in 2016) what are proactive measures that should be taken to ensure healthy watersheds, communities and economy in the North Coast?
Or, What proactive strategies should the North Coast take now (legislation, advocacy) to ensure that legalization does not result in negative economic, social and environmental impacts to the region?
3. What are some potential funding mechanisms to replace the economic inputs from illegal cultivation that can benefit North Coast economies, communities and watersheds? To protect natural capital, working lands, healthy watersheds?

4. If marijuana is legalized and taxed, where should those revenues be allocated? Where do current illegal revenues go?
5. What is your vision for a post-legalization North Coast?
 - a. ongoing legal cultivation that is regulated and taxed like other agricultural commodities (or more, like in Colorado)?
 - b. small, environmentally sensitive “boutique” growers, much like a high end wine appellation?
 - c. all cultivation leaves the region and goes elsewhere – good riddance?
6. *Strategies for navigating the inconsistency among state and federal laws?*
7. *How do we account for the continuum from “provisioners” and “beneficiaries” – much like the source watershed relationship among Trinity and SoCal? Under various future legalization scenarios, how does the North Coast avoid losing natural resources and revenues from these agricultural products?*
8. *Should we direct staff to proactively incorporate data and analysis on this issue into our North Coast planning? If so, which impacts and issues? Short term (impact avoidance and regulatory strategies) and long term issues (policy changes under potential legalization)?*

PANELISTS: BACKGROUNDS/PERSPECTIVES

Peggy O’Neill, Director, Yurok Tribe Planning Department

I have worked in Indian country for the past 30 years and I view the marijuana grows as another wave of land grabbing and devastation to Indian lands. Instead of the “gold rush”, we are calling this the “green rush”. I have been responsible for the Public Utilities Department, Transportation Program, and Community Development for the Yurok Tribe for over a decade. Each of these programs have suffered the impacts of illegal marijuana grows; stealing water, destroying roads, and unsafe communities, etc.,. The grow areas are doubling every year on the reservation. This situation currently impacts every aspect of our Tribal government and the health and safety of our communities.

Nicole Sager, Senior Planner, Yurok Tribe Planning Department

As a land use planner for the past five years on the Yurok Tribe, I have witnessed devastating effects of large scale illegal marijuana cultivation on the reservation. The lands are illegally cleared of timber without regard to erosion controls, cultural sites are not a consideration, illegal water diversions of entire streams are impounded, and huge quantities of pesticides and fertilizers put into the ground water.

Matt St. John, Executive Officer, Regional Water Board

Matt St. John is the Executive Officer of the North Coast Regional Water Quality Control Board (Regional Water Board), a position he's held since May 2012. Mr. St. John has worked for the Regional Water Board since February 2001, previously serving as the Chief of the Timber Harvest and Non-Point Source Division and Supervisor of the Total Maximum Daily Load program. Before joining the Regional Water Board he worked as a consultant in the private sector for several years. St. John has a Master of Science degree in Environmental Engineering and Science from the University of Washington and a Bachelor of Science degree in Environmental Studies from the University of Vermont.

Cris Carrigan, Director, Office of Enforcement, State Water Resources Control Board

Cris Carrigan was appointed director of the Office of Enforcement replacing Reed Sato. Cris was a Senior Staff Counsel in the Office of Enforcement before being named director. He has been appointed as the Water Boards' statewide lead on developing a regulatory and enforcement program for cannabis cultivation. Cris was a partner at the law firm of Miller, Starr & Regalia, and a principal at the law firm of Morgan, Miller, Blair. Cris has administrative advocacy and trial experience under the Porter-Cologne Water Quality Control Act, the Clean Water Act, CERCLA, the California Environmental Quality Act and a variety of other state and federal environmental statutes. Cris is admitted to the California State Bar in 1998 and is admitted to practice law in all of the Courts of California, the United States Supreme Court, the Federal Court of Claims, the Ninth Circuit Court of Appeals, and all of the federal District Courts of California. Cris graduated from McGeorge School of Law and earned his Bachelor's degree from California State University, Sacramento.

Hezekiah Allen, Executive Director, Emerald Growers Association

Hezekiah Allen was born and raised in Humboldt County. He studied Politics and Government at Pacific University. After university he returned to the North Coast to work as a consultant, helping local residents increase water storage, implement conservation irrigation practices, and assisting local organizations with fundraising and strategic planning. In 2010 he was hired as the Executive Director of the Mattole Restoration Council. While with the MRC Allen was one of the first community leaders to call attention to the increasingly severe environmental impacts associated with illegal and unregulated marijuana cultivation. In 2013 he stepped down as ED of the MRC to focus all of his attention on advancing regulation for marijuana to help stem the tide of environmental and violent crimes associated with cultivation. He has presented at dozens of best management workshops, helped to author the widely distributed Best Management Practices guide, and has done on-site consultation with dozens of farmers throughout the region and the state. In June 2014 Hezekiah relocated to Sacramento after he was hired as the Executive Director of the Emerald Growers Association, a trade association focused on promoting the medicinal, environmental, economic, and social benefits of lawfully cultivated marijuana. For EGA he manages five programs: legislative affairs, market development, stewardship and environmental compliance, community development, and media relations.

Background Documents & Resources

[Anticoagulant Rodenticides on our Public and Community Lands: Spatial Distribution of Exposure and Poisoning of a Rare Forest Carnivore](#). Mourad W. Gabriel, Leslie W. Woods, Robert Poppenga, Rick A. Sweitzer, Craig Thompson, Sean M. Matthews, J. Mark Higley, Stefan M. Keller, Kathryn Purcell, Reginald H. Barrett, Greta M. Wengert, Benjamin N. Sacks, Deana L. Clifford. Published: July 13, 2012 DOI: 10.1371/journal.pone.0040163
(<http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0040163>)

[Impacts on Natural Resources from Toxicants Associated with Illegal Marijuana Cultivation on our Public and Tribal Lands](#), Powerpoint presentation, Mourad W. Gabriel, University of California Davis & Integral Ecology Research Center July 13, 2012
(<http://caforestpestcouncil.org/wp-content/uploads/2013/01/mourad-gabriel.pdf>)

M.W. Gabriel et al. 2013 "Silent Forests? Rodenticides on Illegal Marijuana Crops Harm Wildlife"
(http://www.iercecolology.org/wp-content/uploads/2013/03/Silent_Forests_by_Mourad_W._Gabriel_et_al.TWP_Spring_2013.pdf)

[Impacts of Surface Water Diversions for Marijuana Cultivation on Aquatic Habitats in Four Northwestern California Streams](#), Powerpoint presentation, Scott Bauer, Adam Cockrill & Jen Olson, California Department of Fish and Wildlife, Habitat Conservation Branch, Region 1, Coastal Conservation Planning
(http://www.waterboards.ca.gov/water_issues/programs/swamp/docs/cabw2013/twentytwo_mj_impacts.pdf)

Potential Economic Impact to Humboldt County If Marijuana Is Legalized - Jennifer Budwig. 2014.
<http://www.youtube.com/watch?v=ZOGxtOuPnBw>

Environmental impacts of outdoor marijuana growing: Scott Greacen at TEDxEureka.
https://www.youtube.com/watch?v=uM_AlzymIRE

The Fraser Institute, Public Policy Sources Paper, Marijuana Growth in British Columbia by Stephen T. Easton, 2004. <http://www.fraserinstitute.org/publicationdisplay.aspx?id=13187&terms=marijuana>

The Press Democrat, [Feds call for study of marijuana industry's effects on salmon](#), Glenda Anderson, October 2, 2014. <http://www.pressdemocrat.com/news/local/2914969-181/feds-call-for-study-of>

Politics Cheat Sheet, 5 States (And One City) Ready to Legalize Marijuana, Sam Becker, September 1, 2014. <http://wallstcheatsheet.com/politics/5-states-and-one-city-ready-to-legalize-marijuana.html/?a=viewall>

The New York Times, Marijuana Crops in California Threaten Forests and Wildlife, Felicity Barringer, June 2013. <http://www.nytimes.com/2013/06/21/us/marijuana-crops-in-california-threaten-forests-and-wildlife.html?pagewanted=all&r=0>

The New York Times, Rules for the Marijuana Market, Vikas Bajaj, Aug. 4, 2014.
<http://www.nytimes.com/2014/08/05/opinion/high-time-rules-for-the-marijuana-market.html?module=Search&mabReward=relbias%3Aw>

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Peer-Reviewed Publications

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

Published: July 2012 in *PLoS ONE*

Key Points of Interest

- 79% of CA fishers tested positive for rodenticides. Marijuana cultivation was implicated as the source.
- Four (4) fishers died directly due to anticoagulant rodenticides.
- One fisher kit had anticoagulant rodenticide: first case of transfer by mother's milk or in womb.
- Anticoagulant rodenticide exposure in fishers was common throughout their range in CA.

Impacts of rodenticide and insecticide toxicants from marijuana cultivation sites on fisher survival rates in the Sierra National Forest, California

Published: July 2013 in *Conservation Letters*

Key Points of Interest

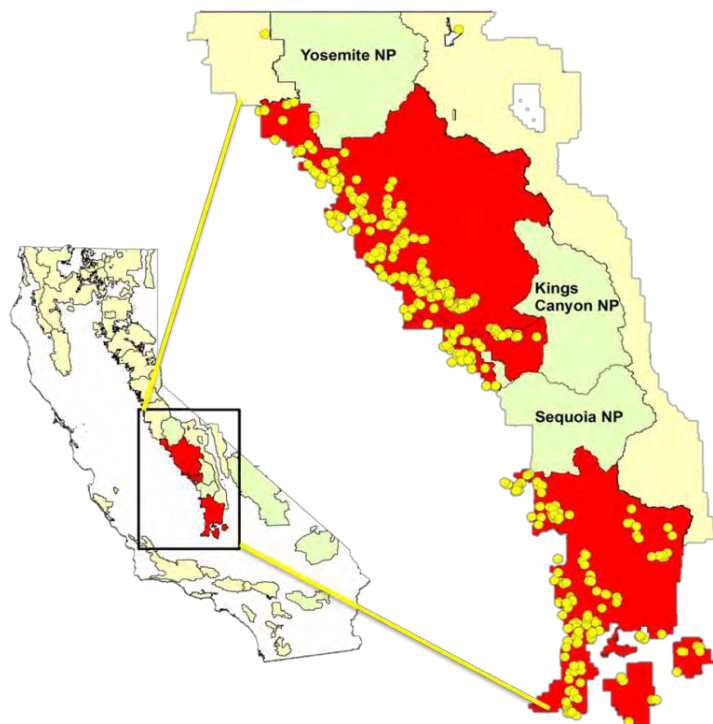
- Exposure of fishers to anticoagulant rodenticides rose from 79% to now 85%.
- Marijuana cultivation sites in a fisher's territory increased its chance of exposure.
- Marijuana cultivation sites in a female fisher's territory impacted its survival.
- Exposed fishers had 1-16 marijuana sites in their territories, non-exposed fishers had only 0-1 site.

Silent Forests? Illegal Marijuana Crops Harm Wildlife

Published: February 2013 in *Wildlife Professional*

Key Points of Interest

- Over 600 marijuana cultivation sites were remediated in the Central Sierras from 2005-2010.
- Incidents of biologists and forest ecologists being threatened or shot at by armed growers.
- Cost increases (25-40%) for projects due to safety concerns for biologists.
- Massive illegal use of fertilizer and banned and restricted-use chemicals within and near watersheds.



New Developing Research

- **Testing toxicant exposure in spotted owls and sympatric owl species where marijuana cultivation is rampant.**
 - Current data shows the 50% of owls tested are positive for rodenticides.
- **Testing watersheds for presence of banned, restricted-use and over the counter toxicants.**
 - Determine if trespass marijuana sites are contributing to fertilizer and toxicant/chemical loads in watersheds where endangered species and human communities live.
 - Water sampling throughout the Trinity River watershed
- **Creating sampling protocol for scientific monitoring impacts at grow sites.**
 - Generating average fertilizer loads per site.
 - Quantifying area and erosion impacts
- **Developing models of marijuana cultivation impacts on wildlife species of conservation concern.**
 - Investigating indirect effects of marijuana cultivation at sites (i.e. prey depletion, increased access through forest by large predators)
 - Example of impacts of trespass grow sites on public, tribal and private lands.
 - Sites within fisher habitat, in 2010-2011. 1,100+ trespass grow sites discovered.

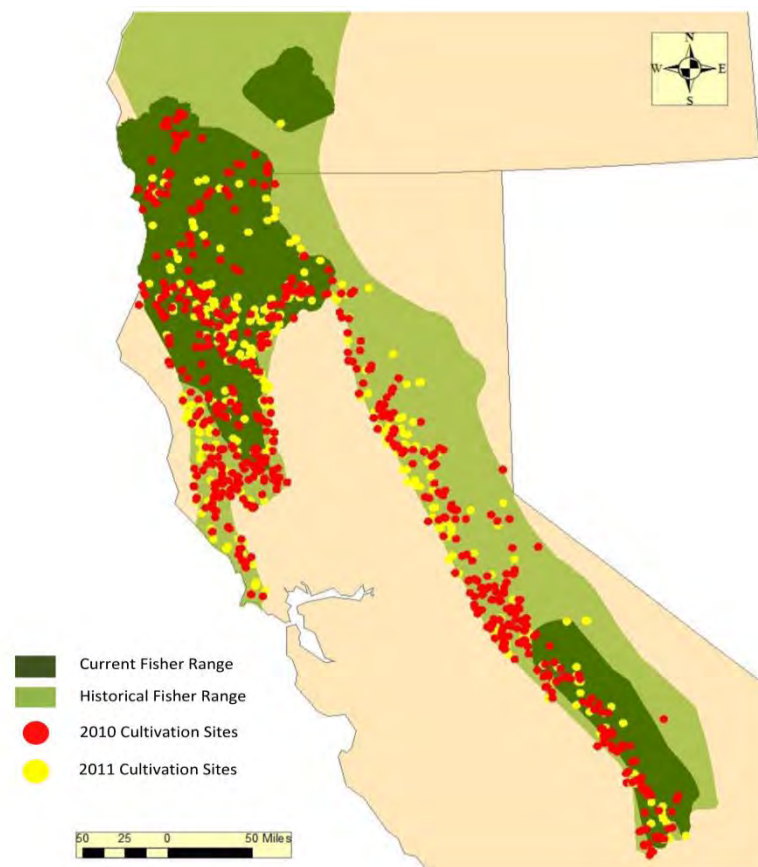


Figure 1: Map of California and the southern portion of Oregon denoting outdoor marijuana cultivation sites eradicated on public, tribal or private lands during 2010 and 2011 within both historic and current ranges of the Pacific fisher (*Martes pennanti*). Each site is buffered by 4000 meter radius which represents the home range of a male fisher.

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

Mourad W. Gabriel, Leslie W. Woods, Robert Poppenga, Rick A. Sweitzer, Craig Thompson, Sean M. Matthews, J. Mark Higley, Stefan M. Keller, Kathryn Purcell, Reginald H. Barrett, Greta M. Wengert, Benjamin N. Sacks, Deana L. Clifford

Published: July 13, 2012 • DOI: 10.1371/journal.pone.0040163

Abstract

Anticoagulant rodenticide (AR) poisoning has emerged as a significant concern for conservation and management of non-target wildlife. The purpose for these toxicants is to suppress pest populations in agricultural or urban settings. The potential of direct and indirect exposures and illicit use of ARs on public and community forest lands have recently raised concern for fishers (*Martes pennanti*), a candidate for listing under the federal Endangered Species Act in the Pacific states. In an investigation of threats to fisher population persistence in the two isolated California populations, we investigate the magnitude of this previously undocumented threat to fishers, we tested 58 carcasses for the presence and quantification of ARs, conducted spatial analysis of exposed fishers in an effort to identify potential point sources of AR, and identified fishers that died directly due to AR poisoning. We found 46 of 58 (79%) fishers exposed to an AR with 96% of those individuals having been exposed to one or more second-generation AR compounds. No spatial clustering of AR exposure was detected and the spatial distribution of exposure suggests that AR contamination is widespread within the fisher's range in California, which encompasses mostly public forest and park lands. Additionally, we diagnosed four fisher deaths, including a lactating female, that were directly attributed to AR toxicosis and documented the first neonatal or milk transfer of an AR to an altricial fisher kit. These ARs, which some are acutely toxic, pose both a direct mortality or fitness risk to fishers, and a significant indirect risk to these isolated populations. Future research should be directed towards investigating risks to prey populations fishers are dependent on, exposure in other rare forest carnivores, and potential AR point sources such as illegal marijuana cultivation in the range of fishers on California public lands.

Figures

Citation: Gabriel MW, Woods LW, Poppenga R, Sweitzer RA, Thompson C, et al. (2012) Anticoagulant Rodenticides on our Public and Community Lands: Spatial Distribution of Exposure and Poisoning of a Rare Forest Carnivore. PLoS ONE 7(7): e40163. doi:10.1371/journal.pone.0040163

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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 [1], [2], [3]. These toxicants are used to eradicate or suppress rodent pest populations in agricultural or urban settings to minimize economic losses [1], [4]. Generally, the mechanism of AR function is to bind and inhibit enzyme complexes responsible for the recycling of vitamin K₁, thus creating a series of deleterious clotting and coagulation impairments [4], [5]. The ARs are grouped into two classes: first-generation compounds, which require several doses to cause intoxication, and second-generation ARs, which are more acutely toxic often requiring only a single dose to cause intoxication and persist in tissues and in the environment [1], [4], [6], [7]. Rodents have started to develop resistance to both first-generation and second-generation ARs, prompting increasingly greater reliance on more acutely toxic compounds and increased distribution by AR users [1], [7], [8].

Primary exposure by ingestion of bait or secondary exposure through consumption of exposed prey has been documented in numerous species of endangered and common non-target wildlife [1], [3], [9], [10], [11], [12], [13]. Wildlife are thought to be at greatest risk of exposure to ARs in agricultural, urban or peri-urban settings, where large quantities of these compounds are often used [12], [14], [15]. However, little is known about the risks to wildlife in settings with little or no anthropogenic 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 fisher projects that generated data for exposure and mortality to anticoagulant rodenticides are outlined within the two isolated populations.

Fishers (*Martes pennanti*), a large mustelid and the largest member in the genus *Martes*, were once widely distributed throughout west coast of North America, but have experienced significant population declines, including extirpation from some regions and contractions of historic ranges [16], [17], [18]. Populations of fishers inhabiting California, Oregon and Washington have been designated as a Distinct Population Segment (DPS) and declared a candidate species for listing under the federal Endangered Species Act [17], [19]. The west coast DPS encompasses areas where fishers were extirpated from Washington and central and northern Oregon, a reintroduced population in the Cascade mountains of southern Oregon, and two extant and isolated populations, one spanning southern Oregon and northern California and another in the southern Sierra Nevada mountains of California [17], [19]. The population status of fishers in the southern Oregon/northern California is unknown; however population estimates for the isolated fisher population in the southern Sierra Nevada range from 150–300 fishers, with 120–250 in the adult age class [17], [20], [21]. Because fishers in the DPS occur in and are dependent on mid to late-seral stage coniferous and hardwood forests and are not associated with agricultural or urban settings, toxicants have not been previously considered a likely threat to fisher populations [17], [22], [23].



Figure 2. Enlarged map of fisher (*Martes pennanti*) project area for the northern California population at the Hoopa Valley Reservation Fisher project (HVRFP).

doi:10.1371/journal.pone.0040163.g002

We assessed the magnitude of AR exposure and poisoning among fisher carcasses submitted for necropsy from 2006 to 2011 as part of a collaborative effort studying threats to population persistence of fishers in California. Additionally, spatial analysis of telemetry data from sampled fishers was conducted in an effort to identify potential sources of AR in the environment. We hypothesized that due to fishers being a forest-dependent carnivore, exposure to ARs will be rare.

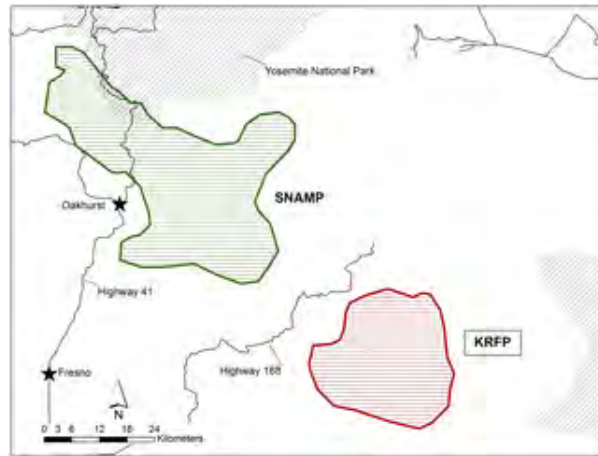


Figure 3. Enlarged map of fisher (*Martes pennanti*) project areas for the southern Sierra Nevada population: the Sierra Nevada Adaptive Management Project (SNAMP) and Kings River Fisher Project (KRFP).
doi:10.1371/journal.pone.0040163.g003

Fisher population	Number of fishers tested (P _{AR})	Number of AR exposed fishers (P _{AR})	Number of AR mortalities	% AR and range to all female (F) and male (M) exposed fishers	Chi-square	Probability Level	DF
Northern California	18 (11/7)	12 (7/5) 300	2	1.66 (50% - 8.33% range 1.66 - 1.66) 1.66 (50% - 8.33% range 1.66 - 1.66)	0.004	0.932	1
Southern Nevada	40 (18/22)	30 (15/15) 150	2	1.50 (50% - 6.66% range 1.50 - 1.50) 1.50 (50% - 6.66% range 1.50 - 1.50)	0.001	0.996	1
All California	58 (31/27)	42 (22/20) 450	4	1.61 (50% - 8.33% range 1.61 - 1.61) 1.61 (50% - 8.33% range 1.61 - 1.61)	0.004	0.932	1
Heterogeneity chi-square					0.001	0.97	1

Mean number (SD) of AR compounds detected per individual, range of numbers of AR per individual and standard deviation (SD) are given for all female (F) and male (M) fishers for each population. Chi-square and heterogeneity chi-square test analyzing variance between the sexes both within and between the populations.
doi:10.1371/journal.pone.0040163.t001

Table 1. Exposure and mortality due to anticoagulant rodenticides (AR) fishers (*Martes pennanti*) within the two isolated populations, northern California and southern Sierra Nevada.
doi:10.1371/journal.pone.0040163.t001

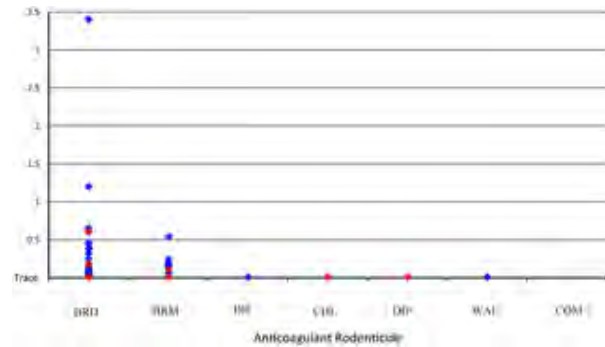


Figure 4. Quantification levels of anticoagulant rodenticides detected in California fishers.

Anticoagulant rodenticides (AR) brodifacoum (BRD), bromodiolone (BRM), difethialone (DIF), chlorophacinone (CHL), diphacinone (DIP), warfarin (WAF) and coumachlor (COM) parts per million (PPM) levels detected in positive fishers (*Martes pennanti*) in California. Blue diamonds represent AR quantification levels (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 Care and Use Committee (Protocol No. 16551).

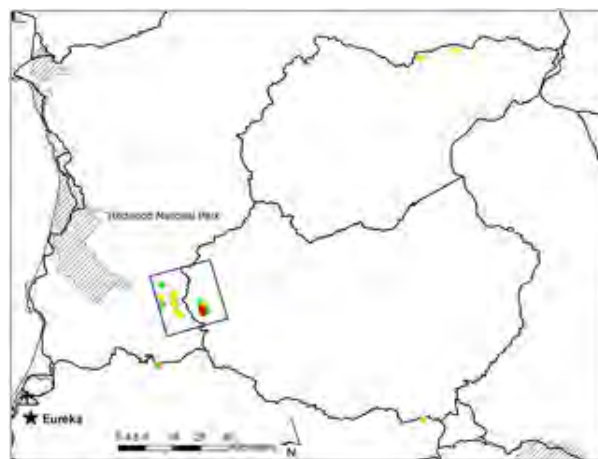


Figure 5. Exposure to and mortality from anticoagulant rodenticides (AR) in fishers (*Martes pennanti*) from the isolated northern California

population.

Green circles represent negative fishers, yellow circles represent exposed fishers, while red circles are fishers that died due to AR toxicosis.

doi:10.1371/journal.pone.0040163.g005

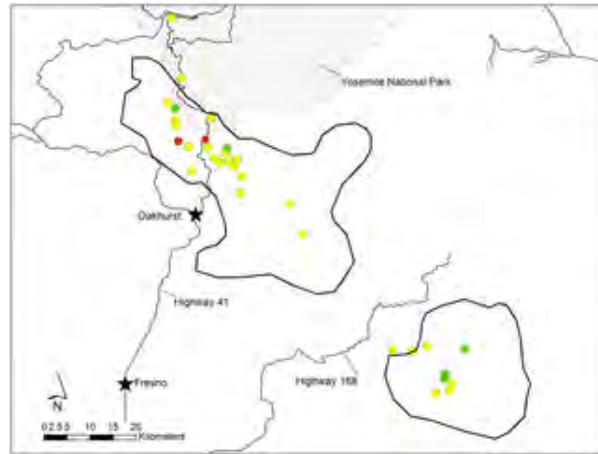


Figure 6. Exposure to and mortality from anticoagulant rodenticides (AR) in fishers (*Martes pennanti*) from the isolated southern Sierra Nevada population.

Green circles represent negative fishers, yellow circles represent exposed fishers, while red circles are fishers that died due to AR toxicosis.

doi:10.1371/journal.pone.0040163.g006

Study Area

Fishers were captured in box traps modified with a plywood cubby box (model 207, Tomahawk Live Trap Company, Tomahawk, Wisconsin, USA), sampled, and fitted with a VHF radio-collar and monitored via telemetry. Fisher carcasses were submitted from the two isolated California populations by three fisher monitoring projects (Figure 1). Carcasses from the northern California population were submitted by the Hoopa Valley Reservation Fisher Project (HVRFP), conducted in northwestern California within tribal, private and public lands, and non-monitored fishers on public and private lands throughout the northern Sierra Nevada/southern Cascade Mountain borderlands of north central California (Figure 2). Carcasses from the southern Sierra Nevada California population were submitted by the Sierra Nevada Adaptive Management Project (SNAMP) and the USDA Forest Service Kings River Fisher Project (KRFP); both projects were conducted on the Sierra National Forest 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, a mortality signal from the VHF collar was detected or when unmarked fisher carcasses were opportunistically observed at the project sites or adjacent areas. Fisher carcasses were stored in a -20 °C freezer 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. Liver samples were collected during necropsy and submitted for screening and quantification of seven ARs at CAHFS by liquid chromatography-tandem mass spectrometry for screening presence

of ARs and high-performance liquid chromatography to quantitate 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 ppm for BRD, 0.05 for WAF, BRM, and COM, and 0.25 ppm for DIP, CHL, and DIF. Detectable compound concentrations that were below quantitate limits were labeled as “trace” concentrations. All results were reported on a tissue wet weight basis and reviewed by a board-certified toxicologist [12], [24].



Figure 7. Condition of the undisturbed mortality site in which a fisher (*Martes pennanti*) mortality due to anticoagulant rodenticide from the southern Sierra Nevada population was found.

doi:10.1371/journal.pone.0040163.g007

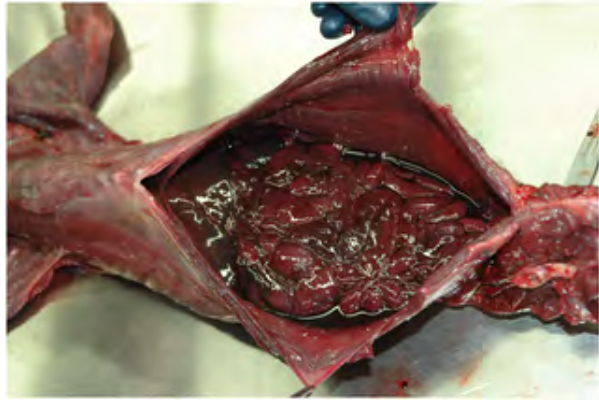


Figure 8. Thoracic cavity hemorrhaging containing 150 ml of frank blood due to coagulopathy after lethal exposure to anticoagulant rodenticides in a fisher (*Martes pennanti*) from the southern Sierra Nevada population.
doi:10.1371/journal.pone.0040163.g008

Age classification was determined by tooth wear, sagittal crest or testicular/teat development, field and laboratory observation, and monitoring of individual animals [17], [18], [25]. Fishers were classified as kits when fully or semi-altricial and dependent on milk for nourishment (roughly ≤ 10 weeks), juveniles if weaned and < 12 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. We compared the AR exposure prevalence between sexes within and between the two California populations using two-tailed heterogeneity chi-square tests of association [26]. The effects of sex and population on the number of anticoagulant rodenticides found per individual were analyzed with a two-way ANOVA [27]. All tests were conducted using the program NCSS (Number 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-range centroids to represent a centralized point within the core area of movement within each individual fisher home-range within each project area [28]. For each fisher, three centroids representing three sampling timeframes were calculated using ArcView 9.1 home range extensions (ESRI Inc., Redlands CA., USA) [29]. The first centroid incorporated all fisher locations from initial capture until death, irrespective of the monitoring time; the second centroid incorporated fisher locations collected six months prior to death; and the third centroid incorporated only the fisher locations collected three months prior to death. These two latter centroids containing locations collected over a shorter time period prior to death were calculated because some ARs have relatively short half-lives and any spatial clustering in these MCP centroids might suggest the locale of recent sources of AR exposure. Only fishers with ≥ 3 months of monitoring were used for spatial analysis, individuals that had less than or were opportunistically collected were excluded.



Figure 9. One of several nine-pound buckets of anticoagulant rodenticide removed from an illegal northern California marijuana operation within the 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 grow site within the southern Sierra Nevada fisher (*Martes pennanti*) project.
doi:10.1371/journal.pone.0040163.g010



Figure 11. Anticoagulant rodenticide bait pellets (bright green) with plant fertilizer freely dispersed around 2,000 plants from northern California marijuana grow site within the northwestern California fisher (*Martes pennanti*) project boundary.
doi:10.1371/journal.pone.0040163.g011

Centroids were analyzed by spatial scan statistics to determine whether exposure to ARs, exposure to different generation classes (1st and 2nd) of ARs, or exposure to individual compounds of ARs were distributed uniformly or spatially clustered in each of the two California populations [30]. SaTScan version 9.1.1 (M. Kulldorff, Harvard Medical School, Boston, MA, USA) was used to evaluate two separate models. First, a Bernoulli model utilizing count data was used to determine if spatial clustering occurred in exposed and non-exposed fishers, or in first or second-generation class AR exposure. The second model, a multinomial model using categorical data, was used to assign each fisher to a group based on the number of AR compounds detected and to examine possible clustering of individuals with high numbers of AR compounds [31]. SaTScan uses these models to scan the geographic area encompassing the MCP centroids to detect spatial clusters encompassing not more than 50% of the centroids [32]. The elliptical scanning window option was chosen for both models because it utilizes both circular and elliptical shapes to allow for a better fit to linear geographic features (i.e. drainages or ridgelines) that occur within the fisher's habitat [32], [33]. All statistical values from the 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 ($p > 0.05$) and the number of ARs per fisher ($p > 0.05$) were similar between populations and sexes (Table S1). The number of AR compounds detected per individual ranged from 1–4 (Table 1). Exposure to at least one AR among age classes ranged with one of 4 pre-weaned kits (25%), 4 of 4 (100%) juveniles, 12 of 17 (70%) sub-adults, and 29 of 33 (88%) adults. Both first and second generation ARs were detected, with BRD being most common and detected in 44 of the 46 (96%) exposed fishers, followed by BRM (16 of 46; 35%), DIP (8 of 46; 17%), CHL (four of 46; 9%), DIF (one of 46; 2%), and WAF (one of 46; 2%). Quantifiable levels of BRD (\bar{x} = 0.22 ppm; range trace –3.4 ppm) and BRM (\bar{x} = 0.12 ppm; range trace –0.54 ppm) were detected while only trace levels of other ARs were detected (Figure 4). No samples had detectable levels of COM and no 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). Brodifacoum was detected in 12 (92%), BRM in two (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). Brodifacoum was detected in 32 (97%), BRM in 14 (42%), 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 (all 12 from HVRFP) and 30 from the southern Sierra Nevada population (19 from SNAMP, 11 from KRFP). Of these fishers, 3-month MCP centroids were generated for 39 fishers, and 6-month centroids for 27 (Table S2). Spatial analysis for 6-month centroids from the KRFP could not be conducted because all fishers in the data set were AR exposed. Sixteen fishers were excluded from the analysis due to lack of monitoring data. No spatial clustering of AR exposure was detected for any of the temporal periods, specific AR compounds, generation class of AR, or distribution of numbers of ARs per fisher in any of the study areas (Table S2; Figure 5, Figure 6).

AR-Mortalities

Cause-specific mortality factors for all 58 fishers sampled ranged widely and included predation, infectious and non-infectious disease processes and vehicular strikes (M.W. Gabriel unpublished data). The cause of death for four of these fishers was attributed to lethal toxicosis, indicated by AR exposure with simultaneous coagulopathy and bleeding into tissues or cavities and ruling out any concurrent processes that might cause hemorrhaging [34]. Two of the four fishers killed by ARs were from the southern Sierra Nevada population, and two were from northern California (Table 1) and the case details are described below.

Southern Sierra Nevada

An adult male fisher was recovered on 15 April 2009, in the southern Sierra Nevada at the SNAMP project area. The fisher showed no signs of predation or scavenging (Figure 7). Gross necropsy determined that the fisher was in good nutritional (3.45 kg) and fair postmortem condition. Frank blood was observed in both the thoracic and abdominal cavities (150 ml and 100 ml respectively), and in the pericardial sac (7 ml) (Figure 8). The stomach and lower gastrointestinal tract contained some blood but no prey or formed feces, and no mucosal changes were noted. There were no other findings on gross examination. Histopathologically, no significant changes were observed in any tissues. Brodifacoum and BRM were detected and quantified in the liver sample at 0.38 ppm and 0.11 ppm, respectively, and 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 highway in the SNAMP project area approximately 3.7 km from Yosemite National Park. Vehicular strike was initially suspected as the cause of mortality due to the location of the carcass but lacerations, abrasions and visual evidence of trauma were not seen on gross examination of the intact carcass. The post-mortem state of the carcass was good and the nutritional state was poor (2.54 kg). Shallow subcutaneous hemorrhage was noted over the hindquarters and spinal column with no associated fractures, punctures or abrasions. There was approximately 20 ml of frank blood within the thoracic cavity. There was no evidence of pneumothorax, vessel ruptures, or visceral tearing. No blood or visceral damage was seen in the abdominal cavity. Stomach contents contained various rodent parts with formed feces in the descending colon. Histopathologically, no significant changes were observed in any tissues. Brodifacoum and BRM were detected and quantified at 0.60 ppm and 0.17 ppm, while one first generation AR, DIP was detected at a trace level within the liver tissue (Figure 4). No evidence was present to suggest that this fisher died due to vehicular trauma, despite its location on 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 in northwestern California at the HVRFP. Severe ectoparasitism on the carcass was noted in the field with ticks in both replete and non-replete stages. Predation was not suspected due to absence of external wounds. The gross necropsy determined that this fisher (2.65 kg) was in poor nutritional condition with no subcutaneous or visceral fat. Frank blood was present in the right external ear canal, nasal and oral cavities, within the lumen of the trachea and within the periorbital tissue with no associated skull fractures or punctures. The stomach was devoid of prey. The colon only contained semi-formed feces. Ectoparasitism was severe with approximately 48 female and 10 male American dog ticks (*Dermacentor variabilis*) and 8 female and 2 male western black-legged ticks (*Ixodes pacificus*) removed from various regions of the fisher. The liver sample from this 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. Field observations included no evidence of predation or scavenging. The nutritional state as well as the postmortem condition were poor. Gross necropsy determined that the fisher (2.89 kg) had no body fat present in any of the tissues. Frank blood was present in both thoracic and abdominal cavities. The stomach contained red and black fluid but no prey. Ectoparasitism was severe with 204 female and 27 male adult American dog ticks in both replete and non-replete stages on areas of the muzzle, chest, tops of fore-and hind-limbs as well as inguinal sections. Severe nematodiasis was seen in skeletal muscle throughout the body (trichinosis). Pulmonary nematodiasis (lungworm) was also noted in the marginal portions of the lungs. Histopathologically, no notable disease processes were seen but severe parasitism was noted. The liver sample for this fisher had quantifiable 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 died following maternal abandonment from their mothers death. One kit, a female fisher (0.32 kg) from KRFP tested positive for AR exposure. This kit was approximately six weeks of age and was recovered within a monitored maternal den tree shortly after maternal abandonment. Cause of death was determined to be acute starvation and dehydration. The liver tissue contained trace level of BRD but there was no associated hemorrhaging in any tissues, body cavities or lumina, suggesting that this finding was not clinically significant.

Discussion

Our findings demonstrate that anticoagulant rodenticides, which were not previously investigated in fishers or other remote forest carnivores, are a cause of mortality and may represent a conservation threat to these isolated California populations. This is the first documentation of exposure to ARs and of direct mortality from ARs in fishers anywhere in their geographic range. Earlier studies suggest ARs posed little or no additive mortality effects on non-target populations [7], [35], [36]. The shortfall of many of these studies was the utilization of common cosmopolitan species so they did not take in consideration that AR mortality may be additive in otherwise compromised populations. The spatially ubiquitous exposure observed within all post-weaning age classes and across the project areas in their contemporary range in California is of significant concern especially considering the recent work of Spencer et al. (2010), who demonstrated that even a small increase in human-caused mortality of 10–20% in the isolated Southern Sierra Nevada fisher population would be enough to prevent population expansion if other restrictive habitat elements were removed.

The high rate of exposure to second generation AR compounds (96% of exposed fishers) in these populations is surprising and cause for concern. This generation of ARs are not only more acutely toxic, but have long retention (>150 days half-life) through biphasic elimination in mammal tissues [1], [37]. Second-generation ARs are more toxic because death can occur from a single primary ingestion by a rodent [1], [5], [37], [38]. However, rodents can receive a lethal dose of second-generation ARs in one feeding bout and it can take up to 7 days before clinical signs manifest [1], [39]. Therefore, prey that have consumed a “super-lethal” dose of AR can pose a substantial risk to predators for several days prior to death [39]. In one study, a group of Norway rats (*Rattus norvegicus*) was given a choice between BRD bait and untreated food and another group had access only to the BRD bait [1]. Both groups consumed 10 and 20 median lethal doses (LD_{50}) on the first day and 40 to 80 LD_{50}

doses by day 6.5, respectively [1]. If sources for these toxicants are maintained for even short periods, exposed rodents, the main prey source for fishers in these 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], [39]. Emulsions used to increase palatability include sucrose, bacon, cheese, peanut butter, and apple flavors (Sure-Gro Inc., Brantford, Ontario, Canada and J.T. Eaton, Twinsburg, Ohio, USA), and thus could be palatable to generalist carnivores like fishers. Although we did not visually detect AR bait in the stomach or GI tracts of any fishers that died, primary poisoning cannot 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 in the function of normal clotting [5], [37], [40], [41]. The occurrence of AR -exposed wildlife dying from minor wounds that otherwise might have easily resolved themselves if ARs were not present suggests contributory lethal effects [1]. Several cases describe raptors receiving minor defensive lacerations or trauma from prey that lead to the raptor's death by exsanguination or hemorrhaging [1], [42]. Fishers actively pursue a wide array of terrestrial and arboreal prey [17], [18]. Hence, it is conceivable that a fisher could receive similar wounds or trauma from prey, or during the pursuit of prey. Consequently, if clotting mechanisms were compromised due to ARs, benign injuries could lead to serious complications [1], [42], [43], [44]. The leading causes of mortality within the USFWS DPS is intraguild predation (G.M.Wengert, unpublished data). It is possible that some of these cases, AR exposure could have compromised clotting mechanisms at the predation attempt and this deserves further study.

High levels of tick infestations were noted in two of the AR mortalities when compared to other sympatric species within the same project area [45]. In addition, locations of these replete ticks were in infrequent regions in other captures, most likely due to a lack of regular grooming. Whether ARs played a role by allowing 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 on rabbits and rats subjected to stressors such as severe decreases in ambient temperature (i.e. frostbite), approximately 10% of test animals died; however when animals were exposed to low non-lethal doses of anticoagulants and subjected to the same stressors, mortality rates increased to 40–70% [46]. It is unknown if stressors or injuries from environmental, physiological or 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 effects of AR exposure to a kit during fetal development or shortly after birth are unstudied. AR exposure in pregnant or whelping domestic canids varied, causing no clinical signs in some cases [47] but death due to coagulopathy immediately after delivery in other cases [48]. The female fisher who gave birth to this kit did not exhibit clinical signs at pre- or postpartum captures and monitoring of her maternal den site verified that one kit survived from that litter (Rebecca Green, United States Forest Service, personal communication). Nevertheless, clinical signs including hemorrhaging, inappetence and lethargy have been seen in domestic canid puppies of AR-exposed mothers [47], [48]. Mild to severe manifestations such as low birth weight, stillbirth or eventually neonatal death has been documented in several cases [47], [48], [49]. In one human study where pregnant women received low doses of warfarin due to severe risk of thromboembolic events, 33% of them had stillbirths, 28% had abortions, and 11% of the neonates died shortly after birth [50]. The range for congenital anomalies and miscarriages in pregnant females for prescribed doses of warfarin varied from 15 to 56% and long-term neurological symptoms have been reported in children that were exposed in-utero [51]. The fetotoxic effects of AR in pregnant fishers and their fetuses are unknown. In addition, because fishers exhibit delayed implantation of the blastocyst, whether ARs may cause pregnant females to abort or reabsorb the fetus merits further research [52], [53], [54]. The transfer of first generation ARs from mother to offspring in milk is not well-understood and there are no data on lactational transfer of second-generation ARs [49].

Quantification Levels

The quantity (ppm) of AR we observed in fisher liver tissues varied and overlapped extensively in both sublethal and lethal cases with no clear indication of a numeric threshold that might indicate an amount leading to morbidity or mortality. This lack of predictive ability has been shown in numerous wildlife cases [1], [12], [55]. For example, Brodifacoum, the most prominent AR compound detected in fishers in this study ranged considerably in lethal cases among individual mustelid species, with 0.32–1.72 ppm in stoats (*Mustela ermine*) [55], [56], [57], 0.7 ppm in least weasels (*Mustela nivalis*) [56], 1.47–1.97 in ferrets (*Mustela furo*) [57] and 9.2 ppm in American mink (*Mustela vison*) [3], [36]. In addition, there are stark differences for acute LD₅₀ doses among genera, where minute amounts of brodifacoum bait caused death in domestic canids but domestic felids required doses 5 to 40 times higher [38]. The same variability seen in both mustelids and other carnivores suggests that predicting clinical thresholds for fishers would be pre-mature [1], [58]. Furthermore, AR exposed fishers had an average of 1.6 AR types within their systems, and possible interaction effects from a combination of 2 or more AR compounds within a fisher and other species are entirely unknown [1], [37].

Potential Sources of AR

Spatial analyses did not reveal any obvious point sources of AR exposure. Instead, these analyses suggested that exposure is widespread across the landscape. Previous studies expected that exposure to AR compounds would be clustered near areas of human activity or inhabitations and that exposure would not be common outside of these areas [1], [12], [14], [24]. Incongruously, data from this study refuted this hypothesis thus making the finding even more significant. Furthermore, these exposures occurred within a species that is not closely affiliated with urban, peri-urban or agricultural settings in which second-generation ARs typically are [1], [12], [14], [24]. Federal and state regulations for anticoagulant rodenticide usage are specific for both generations. Before the June 2011 Environmental Protection Agency (EPA) regulations [39], second generation class ARs could be purchased at local retailers, with recommendations for placement in weather- and tamper-resistant bait containers no more than 50 feet from any building [39]. However, since June 2011, second generation ARs have not been available to consumers at retail, but only at agricultural stores (farm, tractor or feed stores) with additional form and weight restrictions [39]. These newly passed regulations are aimed at further restriction of irresponsible and illegal use of ARs [39]. However, we would have expected that with either pre- or post-June 2011 regulations, second generation AR exposed fishers would have overlapped with urban, peri-urban, or agricultural environments. This pattern is acknowledged in several studies, such as Riley et al. (2007) where bobcat (*Lynx rufus*) and mountain lion (*Felis concolor*) total quantification levels of AR exposure were associated with human-developed areas. Numerous studies have documented that secondary poisoning cases are closely associated with recent agricultural or urban pest eradication efforts [1], [13], [14], [24].

The majority of habitat that fishers in California and fishers throughout the DPS currently and historically occupied is not within or near agricultural or urban settings [17]. Several fishers that were exposed had been monitored their entire lives and inhabited public or community lands where human structures are rare or non-existent (M. Higley, R. Sweitzer, C. Thompson unpublished data). Therefore, exposure from first or second-generation AR use at or within 50 feet of residential or agricultural structures and settings were considered unlikely due to fisher habitat requirements and general lack of association with humans. This suggests that wide-spread non-regulated use of second generation second generation ARs is occurring within the range of fishers in California, especially on public lands.

A likely source of AR exposure to fishers is the emerging spread of illegal marijuana cultivation within California public and private lands [59], [60]. In 2008 in California alone, over 3.6 million outdoor marijuana plants were removed from federal and state public lands, including state and national parks, with thousands of pounds of both pesticides and insecticides found at grow sites [59], [60], [61]. In 2011, a three week eradication operation of marijuana cultivation removed over 630,000 plants and 23,316 kg of trash including 68 kg of pesticides within the Mendocino National Forest in the northern California fisher populations range [17], [62]. Anticoagulant rodenticides and pesticides are typically dispersed around young marijuana plants to deter herbivory, [60], [62], [63] but significant amounts of AR compounds are also placed along plastic irrigation lines used to draw water from in order to deter rodent chewing [60], [62], [63] (M.W. Gabriel, personal observation). A recent example in which over 2,000 marijuana plants were removed less than 12 km from one of the project areas revealed that plants on the peripheral edges as well as nearby irrigation had large amounts of second generation AR placed (Figure 9, Figure 10, Figure 11). Finally, just within a single eradication effort, multiple kilometers (>40 km) of irrigation line within National Parks and Forests in California were removed [60], [62]. Placement of ARs at the grow sites and along irrigation lines which jut out 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 but within a relatively short seasonal period between mid-April to mid-May. We cannot specify the exact explanation or source contributing to all AR mortalities that occurred within this short temporal period. This period is when females are providing for offspring as well as males searching for mates; however, preliminary spatial data for fishers in California document that females have

more confined home-ranges during this period, while males have slightly larger home-ranges (S. Matthews, R. Sweitzer, unpublished data).

Additionally, several books available to the general public identify the optimal time for planting marijuana outdoors is during mid to late spring, and seedlings are especially vulnerable to rodent pests [64], [65], [66]. Of additional concern is that April to May is the denning period for female fishers and a time when fisher kits are entirely dependent on their mothers [17], [18]. The documentation of a lactating female mortality attributed to AR toxicosis during this period suggests that most likely 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 and a candidate for protection under the Endangered Species Act, are not only being exposed to ARs, but ARs are a direct cause of mortality and indirect mortality (i.e. kit abandonment) in both of California's isolated populations. Consequently, these toxicants may not only pose a mortality risk to fishers but could also pose significant indirect risks by depleting rodent prey populations upon which fishers depend. The lack of spatial clustering of exposed individuals suggests that AR contamination is widespread within this species' range and illegal or irresponsible use of ARs continues despite recent regulatory changes regarding their use. Because we do not know the long-term ecological ramifications of these toxicants left on site long after marijuana grows are dismantled, heightened efforts should be focused on the removal of these toxicants at these and adjacent areas at the time of dismantling. Further regulation restricting the use of ARs to only pest management professionals as well as continued public outreach through state wide Integrated Pest Management programs may be warranted. In addition, promotion of compounds that do not possess the propensity for secondary poisoning (i.e. zinc phosphide) should be considered in non-professional use settings. Furthermore, ARs in these habitats may pose equally grave risks to other rare and isolated California carnivores such as the Sierra Nevada red fox (*Vulpes vulpes necator*), American marten (*Martes americana*), wolverine (*Gulo gulo*), gray wolf (*Canis lupus*) or raptors such as northern spotted owls (*Strix occidentalis caurina*), California spotted owls (*S.o. occidentalis*) and great gray owls (*Strix nebulosa*). Future research should be directed to investigating potential risks to prey populations as well as other sympatric species that may allow a better understanding of the potential AR 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.

Variable	DF	Sum of Squares	Mean Squares	F ratio	Probability Level
Fisher Population (A)	1	1.452	1.452	1.50	0.23
Sex (B)	1	1.335	1.335	1.38	0.245
Fisher Population*Sex (AB)	1	0.214	0.214	0.22	0.640

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s and sex on the number of anticoagulant rodenticides found per

used fishers within each California fisher project. Number of
ific AR types, generation class of AR and distribution of numbers of

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(DOCX)

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Author Contributions

Conceived and designed the experiments: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW DLC. Performed the experiments: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW DLC. Analyzed the data: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW BNS DLC. Contributed

reagents/materials/analysis tools: MWG LWW RHP RAS CT SMM JMH SMK KP RHB GMW DLC. Wrote the paper: MWG LWW RHP GMW BNS DLC. Acquisition of funding: MWG RAS CT SMM JMH KP RHB GMW.

References

1. Erickson W, Urban D (2004) Potential risks of nine rodenticides to birds and nontarget mammals: A comparative approach. United States Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substance, Washington DC.
2. Hoare JM, Hare KM (2006) The impact of brodifacoum on non-target wildlife: gaps in knowledge. *New Zeal J Ecol* 30: 157–167.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
3. Fournier-Chambrillon C, Berny PJ, Coiffier O, Barbedienne P, Dasse B, et al. (2004) Evidence of secondary poisoning of free-ranging riparian mustelids by anticoagulant rodenticides in France: implications for conservation of European mink (*Mustela lutreola*). *J Wildlife Dis* 40: 688–695.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
4. Berny P (2007) Pesticides and the intoxication of wild animals. *J Vet Pharmacol Ther* 30: 93–100.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
5. Valchev I, Binev R, Yordanova V, Nikolov Y, Pasha TN, et al. (2008) Anticoagulant rodenticide intoxication in animals—a review. *Turk J Vet Anim Sci* 32: 237–243.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
6. Ogilvie S, Pierce R, Wright G, Booth L, Eason C (1997) Brodifacoum residue analysis in water, soil, invertebrates, and birds after rat eradication on Lady Alice Island. *New Zeal J Ecol* 21: 195–197.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
7. Hadler MR, Buckle AP (1992) Forty five years of anticoagulant rodenticides-past, present and future trends. *P Fifteenth Vertebra Pest C* 15: 149–155.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
8. Lund M (1984) Resistance to the second-generation anticoagulant rodenticides. *P Eleventh Vertebra Pest C* 11: 89–94.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
9. Brakes C, Smith RH (2005) Exposure of non target small mammals to rodenticides: short term effects, recovery and implications for secondary poisoning. *J App Ecol* 42: 118–128.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
10. Hosea R (2000) Exposure of non-target wildlife to anticoagulant rodenticides in California. *P Nineteenth Vertebra Pest C* 19: 236–244.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
11. Stone WB, Okoniewski JC, Stedelin JR (1999) Poisoning of wildlife with anticoagulant rodenticides in New York. *J Wildlife Dis* 35: 187–193.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
12. Riley SPD, Bromley C, Poppenga RH, Uzal FA, Whited L, et al. (2007) Anticoagulant exposure and notoedric mange in bobcats and mountain lions in urban

southern California. *J Wildlife Manage* 71: 1874–1884.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

13. McMillin S, Hosea R, Finlayson B, Cypher B, Mekebri A, et al. (2008) Anticoagulant rodenticide exposure in an urban population of San Joaquin kit fox. *P Vertebra Pest C* 23: 163–165.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

14. Proulx G (2011) Field evidence of non-target and secondary poisoning by strychnine and chlorophacinone used to control Richardson's ground squirrels in southwest Saskatchewan. *P Ninth Prairie Conserv Endang Species C*: 128–134.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

15. Tosh DG, Shore RF, Jess S, Withers A, Bearhop S, et al. (2011) User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use. *J Environ Manage* 92: 1503–1508.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

16. Matthews SM, Mark Higley J, Scott Yaeger J, Fuller TK (2011) Density of fishers and the efficacy of relative abundance indices and small-scale occupancy estimation to detect a population decline on the Hoopa Valley Indian Reservation, California. *Wildl Soc Bull* 35: 69–75.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

17. Lofroth EC, Raley CM, Higley JM, Truex RL, Yaeger JS, et al. (2010) Conservation of fishers (*Martes pennanti*) in south-central British Columbia, Western Washington, Western Oregon, and California. Denver,: USDI Bureau of Land Management.

18. Powell RA (1982) The fisher: life history, ecology, and behavior: University of Minnesota Press Minneapolis, USA.

19. United States Fish and Wildlife Service (2004) 50-CFR Part 17 Endangered and threatened wildlife and plants; 12 month findings for a petition to list the west coast distinct population segment of the fisher (*Martes pennanti*); proposed rule. *Federal Register* 69: 18770–18792.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

20. Spencer W, Rustigian-Romsos H, Strittholt J, Scheller R, Zielinski W, et al. (2010) Using occupancy and population models to assess habitat conservation opportunities for an isolated carnivore population. *Biol Conserv* 144: 788–803.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

21. Spencer W, Rustigian H, Scheller R, Syphard A, Strittholt J, et al. (2008) Baseline evaluation of fisher habitat and population status, and effects of fires and fuels management on fishers in the southern Sierra Nevada. Final report prepared for USDA Forest Service, Pacific Southwest Region.

22. Zielinski WJ, Truex RL, Schlexer FV, Campbell LA, Carroll C (2005) Historical and contemporary distributions of carnivores in forests of the Sierra Nevada, California, USA. *J Biogeogr* 32: 1385–1407.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

23. Zielinski WJ, Truex RL, Schmidt GA, Schlexer FV, Schmidt KN, et al. (2004) Home range characteristics of fishers in California. *J Mammal* 85: 649–657.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

24. Ruder MG, Poppenga RH, Bryan JA, Bain M, Pitman J, et al. (2011) Intoxication of nontarget wildlife with rodenticides in northwestern Kansas. *J Wildl Dis* 47: 212–216.

[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

25. Coulter MW (1967) Ecology and management of fishers in Maine [Dissertation]: State University College of Forestry at Syracuse University.
26. Zar JH (1999) Biostatistical analysis. fourth edition. Upper Saddle River, NJ.: Prentice-Hall, Inc. 663 p.
27. Dowdy S, Wearden S (1991) Statistics for research. New York, NY: John Wiley & Sons. 629 p.
28. Powell RA (2000) Animal home ranges and territories and home range estimators. Research techniques in animal ecology: controversies and consequences. Columbia University Press, New York, New York, USA. pp. 65–110.
29. Rodgers AR, Carr A, Smith L, Kie J (2007) HRT: Home Range Tools for ArcGIS. Version 1.1. Ontario Ministry of Natural Resources, Centre for Northern Forest Ecosystem Research, Thunder Bay, Ontario, Canada.
30. Kulldorff M, Nagarwalla N (1995) Spatial disease clusters: detection and inference. Stat Med 14: 799–810.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
31. Jung I, Kulldorff M, Richard OJ (2010) A spatial scan statistic for multinomial data. Stat Med 29: 1910–1918.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
32. Kulldorff M (2001) Prospective time periodic geographical disease surveillance using a scan statistic. J R Stat Soc Ser A Stat Soc 164: 61–72.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
33. Kulldorff M, Huang L, Pickle L, Duczmal L (2006) An elliptic spatial scan statistic. Stat Med 25: 3929–3943.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
34. Maxie M, editor (2007) Jubb, Kennedy, and Palmer's pathology of domestic animals. 315–316 p.
35. North PM (1985) A computer modelling study of the population dynamics of the screech owl (*Otus asio*). Ecol Modell 30: 105–143.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
36. Kaukeinen D (1982) A review of the secondary poisoning hazard potential to wildlife from the use of anticoagulant rodenticides. P Tenth Vertebra Pest C 10: 151–158.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
37. Vandenbroucke V, Bousquet M, De Backer P, Croubels S (2008) Pharmacokinetics of eight anticoagulant rodenticides in mice after single oral administration. J Vet Pharmacol Ther 31: 437–445.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
38. Dubock A, Kaukeinen D (1978) Brodifacoum (Talon™ rodenticide), a novel concept. P Eighth Vertebra Pest C 8: 127–137.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
39. Bradbury S (2008) Risk Mitigation Decision for ten rodenticides. United States Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substance, Washington DC.

40. Petterino C, Paolo B (2001) Toxicology of various anticoagulant rodenticides in animals. *Vet Hum Toxicol* 43: 353–360.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
41. Shore RF, Birks JDS, Freestone P (1999) Exposure of non-target vertebrates to second-generation rodenticides in Britain, with particular reference to the polecat *Mustela putorius*. *New Zeal J Ecol* 23: 199–206.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
42. Papworth D (1958) A review of the dangers of warfarin poisoning to animals other than rodents. *The J R Soc Promot Health* 78: 52–60.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
43. Eason C, Murphy E (2000) Recognizing and reducing secondary and tertiary poisoning risks associated with brodifacoum; 2000. *ACS Publ* 771: 157–163.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
44. Newton I, Shore R, Wyllie I, Birks J, Dale L (1999) Empirical evidence of side-effects of rodenticides on some predatory birds and mammals. In: D. P Cowan, Feare CJ, editors. pp. 347–367. *Adv Vertebra Pest Manage*: Filander Verlag, Fürth.
45. Gabriel MW, Brown RN, Foley JE, Higley JM, Botzler RG (2009) Ecology of *Anaplasma phagocytophilum* infection in gray foxes (*Urocyon cinereoargenteus*) in northwestern California. *J Wildlife Dis* 45: 344–354.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
46. Jaques L (1959) Dicoumarol drugs and the problem of haemorrhage. *CMAJ* 81: 848–854.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
47. Munday J, Thompson L (2003) Brodifacoum toxicosis in two neonatal puppies. *Vet Pathol Online* 40: 216–219.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
48. Mackintosh C, Laas F, Godfrey M, Turner K (1988) Vitamin Kt treatment of brodifacoum poisoning in dogs. *P Thirteenth Vertebra Pest C* 13: 86–90.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
49. Greaves M (1993) Anticoagulants in pregnancy. *Pharmacol & Therapeu* 59: 311–327.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
50. Sareli P, England MJ, Berk MR, Marcus RH, Epstein M, et al. (1989) Maternal and fetal sequelae of anticoagulation during pregnancy in patients with mechanical heart valve prostheses. *Am J Cardiol* 63: 1462–1465.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
51. James AH, Abel DE, Brancazio LR (2006) Anticoagulants in pregnancy. *Obstet Gynecol Surv* 61: 59–69.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
52. Krackow S (1995) Potential mechanisms for sex ratio adjustment in mammals and birds. *Biol Rev* 70: 225–241.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
53. Rosenfeld CS, Roberts RM (2004) Maternal diet and other factors affecting offspring sex ratio: a review. *Biol Reprod* 71: 1063–1070.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)

54. Krüger O, Radford A, Anderson C, Liversidge R (2005) Successful sons or superior daughters: sex-ratio variation in springbok. *P Roy Soc Lond B Bio* 272: 375–381.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
55. Brown K, Alterio N, Moller H (1998) Secondary poisoning of stoats (*Mustela erminea*) at low mouse (*Mus musculus*) abundance in a New Zealand *Nothofagus* forest. *Wildl Res* 25: 419–426.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
56. Alterio N, Brown K, Moller H (1997) Secondary poisoning of mustelids in a New Zealand *Nothofagus* forest. *J Zool* 243: 863–869.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
57. Alterio N (1996) Secondary poisoning of stoats (*Mustela erminea*), feral ferrets (*Mustela furo*), and feral house cats (*Felis catus*) by the anticoagulant poison, brodifacoum. *New Zealand Journal of Zoology* 23: 331–338.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
58. Fisher P (2009) Residual concentrations and persistence of the anticoagulant rodenticides brodifacoum and diphacinone in fauna [Dissertation]: Lincoln University. 153 p.
59. Eth W (2008) Up in smoke: Wholesale marijuana cultivation within the national parks and forests, and the accompanying extensive environmental damage. *Pa State Environl Law Rev* 16: 451–451.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
60. Mallery M (2011) Marijuana national forest: encroachment on California public lands for cannabis cultivation. *Berkeley Undergrad J* 23: 1–50.
[View Article](#) • [PubMed/NCBI](#) • [Google Scholar](#)
61. Drug Enforcement Agency (2008) Record breaking seizures of marijuana throughout California for 2008. In: McEnry C, editor. Sacramento: United States Department of Justice.
62. United States Department of Agriculture (2011) Operation full court press concludes three week operation targeting illegal marijuana plants on public lands. In: United States Forest Service Pacific Southwest Region, editor: Press Release.
63. United States Department of Agriculture (2009) Marijuana on California's national forest: complete podcast. In: United States Forest Service Pacific Southwest Region, editor. Forest Focus.
64. Cervantes J (2000) Marijuana outdoors: guerilla growing. Vancouver, WA, USA: Van Patten Publishing. 144 p.
65. McCarthy T (2011) Growing marijuana: how to plant, cultivate, and harvest your own weed. New York, NY, USA: Skyhorse Pub Co Inc. 224 p.
66. Owner ST (2010) Marijuana outdoor growers guide. San Francisco, CA, USA: Green Candy Press. 142 p.



Silent Forests?

RODENTICIDES ON ILLEGAL MARIJUANA CROPS HARM WILDLIFE

By Mourad W. Gabriel, Greta M. Wengert, J. Mark Higley, Shane Krogan, Warren Sargent, and Deana L. Clifford



Credit: Timothy Archibald

Mourad W. Gabriel is a Doctoral Candidate at the Veterinary Genetics Laboratory at the University of California-Davis and President of the Integral Ecology Research Center.

Another mortality signal on the radio collar of a fisher (*Martes pennanti*) pulses on a wet spring morning, and fear of a repeat of the previous spring's mortalities looms in the backs of our minds. Hoopa tribal biologists scramble to recover the fisher quickly so that a necropsy can be performed to determine cause of death. The field crew reports back that the fisher is not dead but lethargic and lurching on the ground when it attempts to seek cover from approaching biologists. A conference call among researchers, a wildlife pathologist, and a veterinary toxicologist follows to determine the next course of action. Unfortunately, the consensus is humane euthanization. Though testing is ongoing, this is likely the sixth monitored fisher in California that has died from second-generation anticoagulant rodenticide (SGAR) toxicosis since 2009.

Linking SGARs to multiple deaths of a rare forest carnivore has been an alarming discovery. Even

more unsettling: We've learned that these deaths appear to be linked to illegal marijuana cultivation on community and public lands—a finding that raises serious concerns for the health of many species of wildlife including fishers, an Endangered Species Act candidate.

A Growing Concern

Beginning in 2008, full necropsies including toxicological screens—done at the University of California-Davis School of Veterinary Medicine and the California Animal Health and Food Safety Laboratory (CAHFS)—have been conducted to determine proximate and ultimate causes of mortality for fishers from the Hoopa Valley Reservation Fisher Project (HVRFP), Sierra Nevada Adaptive Management Project (SNAMP), and the U.S. Forest Service (USFS) Kings River Fisher Project (KRFP). These ongoing, long-term demographic projects encompass both tribal community forests within the HVRFP and public lands including Yosemite National Park and Sierra National Forest in the SNAMP and KRFP study areas.

Toxicology screening of 58 fishers from these community and public lands revealed that nearly 80 percent of the fishers had been exposed to anticoagulant rodenticide (AR) poisons, with 96 percent of those exposures being SGARs—results that we published recently in *PLoS ONE* (Gabriel et al. 2012). Concerned about this trend, we led an interdisciplinary collaboration including multiple stakeholders from the Hoopa Tribe, Integral Ecology Research Center, USFS, U.S. Fish and Wildlife Service, CAHFS, UC-Davis, SNAMP, and California Department of Fish and Wildlife, pooling together resources and expertise for a comprehensive approach to evaluate this emerging threat.

The fisher (*Martes pennanti*) is a cat-sized carnivore found in coniferous and mixed conifer and hardwood forests across Canada and in four regions of the United States, including New England, the Great Lakes, the northern Rockies, and the Pacific Northwest. Now a candidate species for listing under the Endangered Species Act, fishers in California are falling victim to rodenticides used on illegal marijuana crops scattered throughout the state's public and tribal lands.



Credit: John Jacobson/Washington Department of Fish and Wildlife

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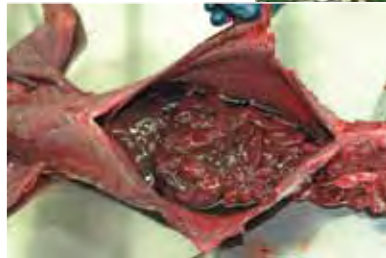


Spatial modeling suggested that fishers were exposed to SGARs ubiquitously throughout the study areas, contradicting current thought that wildlife are at greatest risk to these toxicants near agricultural, urban, or peri-urban settings, where the pesticides are legally used to eradicate or suppress rodent pest populations. However, lifetime monitoring of the California fishers showed that most of the exposed or poisoned individuals never overlapped any of those land-use types. In addition, the use of SGARs within the study areas, in adjacent timberlands, or within campgrounds would violate current state and federal regulations. As a result, our suspicions gravitated towards undiscovered illicit uses throughout the project areas. These suspicions were essentially confirmed after federal, state, and local law enforcement officers verified that the poisons were present at most marijuana cultivation sites found on public and tribal lands.

All of our documented SGAR fisher mortalities occurred from late April through early June, which is prime-time for marijuana seedling planting in California and likely the period of heaviest toxicant use to protect young plants from rodent damage. Regrettably, this is also a key time for female fishers to rear their kits. That unfortunate timing materialized when we discovered a lactating female fisher dead from SGAR poisoning in the Southern Sierra Nevadas. (California currently has two isolated native fisher populations, one within the north-western coastal mountains, where population estimates are unknown, and another within the Southern Sierra Nevadas, where estimates suggest fewer than 300 adults [Spencer et al. 2011]). Presumably, the dead mother's kits also died due to den abandonment.

In a separate instance, a rescue attempt on an abandoned fisher kit still dependent on its mother's milk was unsuccessful, and the kit was found dead of starvation. Most disconcerting was that SGARs were detected in the kit's tissues. This unexpected finding verified a transplacental or milk transfer of a SGAR from mother to kit, raising concern about fetotoxic or bioaccumulation effects of these pesticides, which are currently unknown.

These findings underscore the need to understand not only the direct impacts of these toxicants, but other possible indirect impacts that fishers and other wildlife may face at the population level. For example, we detected an average of 1.6 different types of ARs per fisher, with some fishers testing positive for four different toxic compounds. There



Credit: Mourad W. Gabriel



Courtesy of Mourad W. Gabriel

are no data on the possible interactions of two, three, or even four different ARs, or the effects they might have on animal health. Furthermore, we cannot yet determine whether a threshold level of exposure exists beyond which an animal cannot recover, since some fishers died with low levels of SGARs while others displayed no clinical signs even with much higher exposures. We wonder if these toxicants at sub-lethal doses lower resistance to environmental stressors, as seen in other studies, and whether the distribution of SGARs within the landscape will limit prey availability and create sink habitats near cultivation sites. This is just the beginning of a long list of potential cascading impacts now being discussed in California.

Problem Spreading Like Weeds

Illegal marijuana growing is not just a problem for wildlife. The High Sierra Volunteer Trail Crew is a nonprofit trail-maintenance crew that has spent the past seven years maintaining and cleaning trails throughout the Sierra Nevadas' national forests. In the mid-2000s, the group realized that risks associated with large-scale marijuana production

Pellets of anticoagulant rodenticide litter the ground beneath marijuana plants at an illegal grow site within occupied fisher habitat. Placed to kill rodents that might eat the valuable plants, these poisons—particularly second-generation anticoagulant rodenticides—have been linked to numerous deaths of fishers. The rare forest carnivores likely die of internal hemorrhaging (inset) after ingesting the anticoagulants or preying on rodents that have fed on the toxicants.



throughout most, if not all, California national forests threatened backcountry use of public lands. Since then, the trail crew's Environmental Reclamation Team (ERT) has remediated more than 600 large-scale marijuana cultivation sites on public lands. The numbers are daunting, especially when considering that these 600 sites were in only two of California's 17 national forests and may constitute only a fraction of the actual marijuana cultivation sites that exist in these forests. Tommy Lanier, Director of the National Marijuana Initiative, a White House supported program, states that "60 percent to 70 percent of the national marijuana seizures come from California annually, and of those totals, about 60 percent comes from public lands."

Based on data from ERT-remediated sites, at least 50 percent of them have SGARs. Beyond finding anticoagulant rodenticides, the team and other

uses are occurring in California, where marijuana cultivators place pourable carbamate pesticides in opened tuna or sardine cans in order to kill black bears, gray foxes, raccoons, and other carnivores that damage marijuana plants or raid food caches at grow-site encampments.

In many cases, law enforcement officers approaching grow sites observe wildlife exposed to what officers call "wildlife bombs" due to their high potential for mass wildlife killing. For example, as federal and state officers approached a grow site in Northern California, they discovered a black bear and her cubs seizing and convulsing as they slowly succumbed to the neurological effects of these pesticides. Because toxicants are usually dispersed throughout cultivation sites, it is remarkably difficult to detect and remove all pesticide threats.

Funding to document, quantify, and remediate the damage caused by illegal marijuana cultivation on public and tribal lands has been difficult to secure through state or federal agencies or even private foundations, possibly due to the common misperceptions that illegal marijuana cultivation is not an environmental but rather a social issue, and that it is not a significant threat to wildlife. Yet we propose that funding is strongly warranted to help researchers investigate toxicant exposure and implications throughout the forests' trophic levels, and to study impacts on all species of conservation concern, including fishers and the northern spotted owl.

Another common misperception is that it is the responsibility of law enforcement to not only protect our natural resources at illegal marijuana sites, but also to remove pesticides and remediate the sites. In truth, there is currently no standardized system for grow-site remediation. Recently, for example, we encountered more than 10 pounds of SGARs and 20 pounds of metaldehyde and carbamates from a single site that law enforcement officers had dismantled within fisher and northern spotted owl territories. Most of these toxicants were left untouched out of concern for the safety of the officers, who are not trained to handle and transport these highly toxic chemicals, especially in the frequent situation where these chemicals are unlabeled. Accordingly, without documentation of the environmental damage and threats from toxicants, and without funding for properly trained personnel, most poisons will continue to be left at grow sites, where they remain a catastrophic threat to wildlife.



Credit: Mourad W. Gabriel

Accompanied by armed escorts for security, Hoopa Tribe wildlife biologist J. Mark Higley (in green hard hat) documents corn stalks likely planted to provide food for growers of illegal marijuana (right foreground). Clearings for food crops, water diversions, fertilizers, and debris left by growers cause damage to natural wildlife habitat.

remediation groups frequently find and remove restricted and banned pesticides including organophosphates, organochlorines, and carbamates as well as thousands of pounds of nitrogen-rich fertilizers. Many of the discovered pesticides have been banned for use in the U.S., Canada, and the European Union, specifically certain carbamates, which gained notoriety worldwide after an explosion of public awareness about their use to kill African wildlife. Unfortunately, these same malicious



Effects Extend beyond Poison

Environmental threats from large-scale marijuana cultivation are certainly not limited to toxicant contamination. At most grow sites, it is standard practice to clear patches of forest within riparian corridors in order to provide enough sunlight for growing plants. The cumulative impact of these practices across the California landscape is unknown, but disheartening in its potential. Last year, at a site within the Hoopa Valley Indian Reservation in northern California, where 26,600 marijuana plants were removed, several acres of hardwood-conifer and alder forest had been cleared along one of the most productive Chinook and Coho salmon-bearing streams in the area. Under no circumstance would this clearing be allowed under the Tribe's management plans or current state or federal regulations established to protect habitat for the salmon.

Because growers prefer areas with a constant and abundant water supply, it is these sensitive habitats that suffer the greatest impacts from marijuana cultivation. Water diversions and pesticide-filled cisterns within streambeds feeding miles of plastic irrigation lines are all-too-familiar a sight. Human waste throughout these sites is also widespread, and because many of the sites on public and tribal lands are inhabited for several months of the year by drug-traffic organizations, extensive camp systems are set up with associated trash dumps and human latrine sites just meters away from water sources.

The camps and plantations are often guarded by armed drug traffickers, so concern for the safety of field crews, students, and biologists working on these lands is ever pressing. Wildlife professionals are fearful of unwittingly running into armed growers at active grow sites, with good reason. Recently, a federal biologist in the southern Sierra Nevada was chased by armed growers for 40 minutes through the national forest. "When we lost radio contact at one point for 10 minutes, we feared that the biologist was captured or possibly dead," says project supervisor Jodi Tucker of Sequoia National Forest. In another incident in the 2012 field season, biologists surveying for northern spotted owls on the Hoopa Reservation were

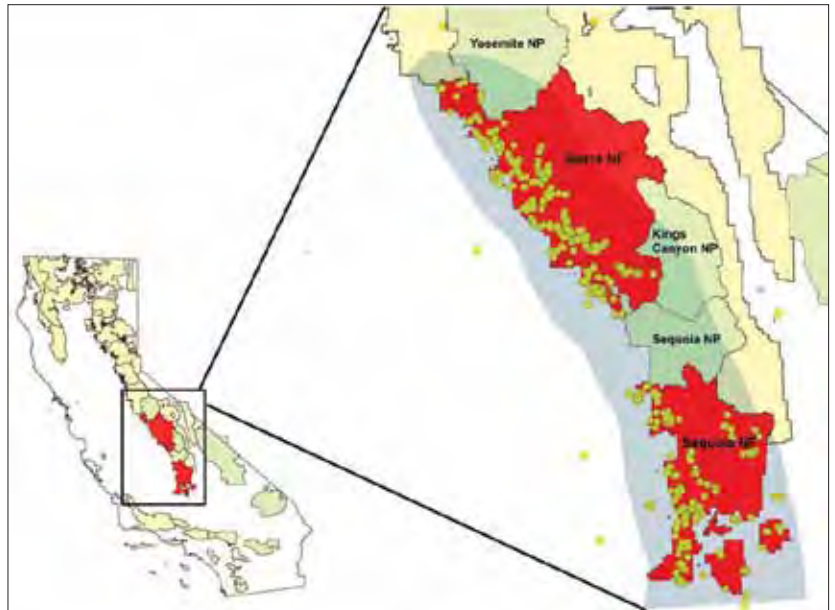


Credit: Environmental Reclamation Team



Credit: Environmental Reclamation Team

Volunteers with the Environmental Reclamation Team display thousands of pounds of garbage, chemicals, and other debris gathered at an illegal marijuana cultivation site in the Sierra Nevada Mountains. Plastic bottles refilled with unlabeled substances (left) sometimes contain carbamates, banned chemicals that growers use to kill bears, foxes, raccoons, and other animals that may harm pot plants or raid growers' food caches.



Credit: Greta M. Wengert

Dots scattered through California's Sierra and Sequoia National Forests represent some 600 illegal marijuana grow sites reclaimed by crews who removed trash, hazardous chemicals, water diversions, and rudimentary shelters left by growers. Blue shading represents current range of the fisher within the southern Sierra Nevada, where the population is estimated at fewer than 300 adults.



shot at by suspected illegal growers with high-caliber assault rifles. Luckily, no one was injured, but biologists avoided the survey area until the threat was addressed.

Due to heightened safety concerns and emerging patterns like these over the past several years, wildlife crews now are often composed of two individuals, whereas before, biologists worked independently in the field. The effects of these changes have not been fully ascertained, but it can be assumed that increased labor costs coupled with increased equipment and vehicle

expenditures are affecting the size, duration, and thoroughness of data for many studies on California's public and tribal lands.

Because wildlife biologists are also avoiding some study areas due to safety concerns, study designs are now being altered to avoid known grow sites, thus further impacting quality and completeness of data. Research ecologist Craig Thompson from the USFS Pacific Southwest Research Station estimates that during each field season, 10 to 25 percent of the Kings River Fisher Project area becomes inaccessible due to safety concerns. In another telling example during the 2010-2011 field season, two radio-collared fishers in this study area pulsed mortality signals but could not be recovered due to their locations near known grow sites. Eventually, under escort by armed law enforcement officers, biologists recovered the collars, yet the carcasses—and any evidence of cause of death or rodenticide toxicosis—were long destroyed.

In his *Science* editorial “The Tragedy of the Commons,” Garret Hardin lamented the loss of our public resources due to the greed and inconsideration of some individuals (Hardin 1968). We believe the vast and ever-growing misuse of our public and tribal forests for the financial benefit of a few individuals is an enormous threat to these resources and a deplorable tragedy of the commons. Our public and tribal land and agencies are being hit on two fronts: first by having to endure the illegal use, take, and destruction of natural resources without our permission, then having to support the financial burden of renewing these lands from the disastrous ecosystem degradation that illicit cultivation produces. Regrettably, most of this is occurring without the knowledge of the public, whose land it is. Though this is a sad story that often brings surprise, disgust, and a feeling of helplessness in those hearing it for the first time, in the words of Rachel Carson, “The public must decide whether it wishes to continue on the present road, and it can do so only when in full possession of the facts.” ■

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Impacts of rodenticide and insecticide toxicants from marijuana cultivation sites on fisher survival rates in the Sierra National Forest, California

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Keywords

Anticoagulant rodenticide; fisher; marijuana; *Pekania pennanti*; pesticide; survival.

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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 recov-

ery of liver tissue from an intact, non-scavenged 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 $\mu\text{g/g}$, bromodiolone at 0.11 $\mu\text{g/g}$, and chlorophacinone at <0.25 $\mu\text{g/g}$. 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 & Mannathukkar 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 (*Palaeomonetes 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 (Mörner *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 Care 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 coumatolol (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 ± 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 ante-mortem 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 caliginosa*) 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 chlorophacinone 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 $\mu\text{g/g}$ 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 $\mu\text{g/g}$ liver concentration of BRD, and another necropsy of a great horned owl (*Bubo virginianus*) with 0.27 $\mu\text{g/g}$ BRM and 0.08 $\mu\text{g/g}$ 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 ex-

tremely 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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References

- Ahdaya, S.M., Shah, P.V. & Guthrie, F.E. (1976). Thermoregulation in mice treated with parathion, carbaryl, or DDT. *Toxicol. Appl. Pharm.*, **35**, 575–580.

- Allen, G. T., Veatch, J.K., Stroud, R.K. *et al.* (1996). Winter poisoning of coyotes and raptors with furadan laced carcass baits. *J. Wildlife Dis.*, **32**, 385–389.
- Alterio, N., Brown, K. & Moller, H. (1997). Secondary poisoning of mustelids in a New Zealand *Nothofagus* forest, London. *J. Zool.*, **243**, 863–869.
- Arnot, L.F., Veale, D.J.H., Steyl, J.C.A. & Myburgh, J.G. (2011). Treatment rationale for dogs poisoned with alicarb (carbamate pesticide). *J. S. Afr. Vet. Assoc.*, **82**, 232–238.
- Bailey, C., Fisher, P. & Eason, C.T. (2005). Assessing anticoagulation resistance in rats and coagulation effects in birds using small volume blood samples. *Sci. Conserv.*, **249**, 5–22.
- Berny, P. (2007). Pesticides and the intoxication of wild animals. *J. Vet. Pharm. Therapy*, **30**, 93–100.
- Berny, P.J., Buronfosse, T., Buronfosse, F., Lamarque, F. & Lorgue, G. (1997). Field evidence of secondary poisoning of foxes (*Vulpes vulpes*) and buzzards (*Buteo buteo*) by bromadiolone, a 4-year survey. *Chemosphere*, **35**, 1817–1829.
- Booth, I.H., Fisher, P., Heppelthwaite, V., & Eason, C.T., 2003. Toxicity and residues of brodifacoum in snails and earthworms. *DOC Science Internal Series*, **143**, New Zealand Department of Conservation, Wellington, NZ. 14 p.
- Burklow, T.R., Yu, C.E. & Madsen, J.M. (2003). Industrial chemicals: terrorist weapons of opportunity. *Pediatr. Ann.*, **32**, 230–234.
- Cooke, A.S. (1971). Selective predation by newts on frog tadpoles treated with DDT. *Nature*, **229**, 275–276.
- Cope, O.B. (1971). Interactions between pesticides and wildlife. *Ann. Rev. Entomol.*, **16**, 325–364.
- Davidson, R.S. & Armstrong, D.P. (2002). Estimating impacts of poison operations on non-target species using mark-recapture analysis and simulation modeling: an example with saddlebacks. *Biol. Conserv.*, **105**, 375–381.
- Dowding, C.V., Shore, R.F., Worgan, A., Baker, P.J. & Harris, S. (2010). Accumulation of anticoagulant rodenticides in a non-target insectivore, the European hedgehog (*Erinaceus europaeus*). *Environ. Pollut.*, **158**, 161–166.
- Environmental Protection Agency (EPA) (2009). Carbofuran; Final Tolerance Revocations; Final Rule. *Federal Register* **74** (93), 23045–23095.
- Erickson, W. & Urban, D. 2004. Potential risks of nine rodenticides to birds and nontarget mammals: a comparative approach. U.S. Environmental Protection Agency, Office of Pesticides Programs, Environmental Fate and Effects Division, Washington DC, USA.
- Farr, J.A. (1977). Impairment of antipredator behavior in *Palaemonetes pugio* by exposure to sublethal doses of parathion. *Trans. Am. Fish. Soc.*, **106**, 287–290.
- Fournier-Chambrillon, C., Berny, P.J., Coiffier, O. *et al.* (2004). Evidence of secondary poisoning of free-ranging riparian mustelids by anticoagulant rodenticides in France: implications for conservation of European mink (*Mustella lutreola*). *J. Wildlife Dis.*, **40**, 688–695.

- Gabriel, M.W., Wengert, G.M., Higley, J.M., Krogan, S., Sargent, W. & Clifford, D.L. (2013). Silent forests? *Wildlife Prof.*, **7**, 46-50.
- Gabriel, M.W., Woods, L.W., Poppenga, R. et al. (2012). Anticoagulant rodenticides on our public and community lands: spatial distribution of exposure and poisoning of a rare forest carnivore. *PLoS ONE* **7**: e40163. doi:10.1371/journal.pone.0040163.
- Golomb, B.A. (2008). Acetylcholinesterase inhibitors and Gulf War Illnesses. *Proc. Natl. Acad. Sci. U.S.A.*, **105**, 4295-4300.
- Gordon, C.J. (1994). Thermoregulation in laboratory mammals and humans exposed to anticholinesterase agents. *Neurotoxicol. Teratol.*, **16**, 427-453.
- Grue, C.E., Gilbert, P.L. & Seeley, M.E. (1997). Neurophysiological and behavioral changes in non-target wildlife exposed to organophosphate and carbamate pesticides: thermoregulation food consumption, and reproduction. *Am. Zool.*, **37**, 269-388.
- Grue, C.E., Hart, A.D.M. & Mineau, P. (1991). Biological consequences of depressed brain cholinesterase activity in wildlife. Pages 151-209 in P. Mineau, editor. Cholinesterase-inhibiting insecticides—their impact on wildlife and the environment. Elsevier Science Publishers B.V., Amsterdam, Netherlands.
- Hosea, R.C. (2000). Exposure of non-target wildlife to anticoagulant rodenticides in California. Proceedings of the 19th 507 Vertebrate Pest Conference, University of California, Davis, CA, USA.
- Hunt, K.A., Bird, D.M., Mineau, P. & Shutt, L. (1992). Selective predation of organophosphate-exposed prey by American kestrels. *Anim. Behav.*, **43**, 971-976.
- Janeway, C. A., Travers, P. & Walport, M. (2007). Immunobiology. 7th edition. Garland Science, New York, USA
- Jaques, L.B. (1959). Dicoumarol drugs and the problem of haemorrhage. *Can. Med. Assoc. J.*, **81**, 848-854.
- Keller, S.M., Gabriel, M., Terio, K.A. et al. (2012). Canine distemper in an isolated population of fishers (*Martes pennanti*) from California. *J. Wildlife Dis.*, **48**, 1035-1041.
- Kendall, R.J. & Smith, P.N. (2003). Wildlife toxicology revisited. *Environ. Sci. Technol.*, **37**, 179A-183A.
- Kiesecker, J.M. (2002). Synergism between trematode infection and pesticide exposure: a link to amphibian limb deformities in nature? *PNAS*, **99**, 9900-9904.
- Knaus, B.J., Cronn, R. Liston, A., Pilgrim, K. & Schwartz, M. K. (2011). Mitochondrial genome sequences illuminate maternal lineages of conservation concern in a rare carnivore. *BMC Ecol.*, **11**, 10. doi:10.1186/1472-6785-11-10
- Lemarchand, C., Rosoux, R. & Berny, P. (2011). Semi-aquatic top predators as sentinels of diversity and dynamics of pesticides in aquatic food webs: the case of Eurasian otter (*Lutra lutra*) and Osprey (*Pandion haliaetus*) in the Loire River catchment, France. Pages 298-310 in M. Stoytcheva, editor. *Pesticides in the modern world: risks and benefits*. InTech, Manhattan, NY, USA, ISBN 978-953-307-458-0.
- Lemus, J.A., Bravo, C., Garcia-Montijano, M., Palacin, C., Ponce, C., Magana, M.M & Alonso, J.C. (2011). Side effects of rodent control on non-target species: Rodenticides increase parasite and pathogen burden in great bustards. *Sci. Total Environ.* **409**, 4729-4734.
- Li, Q. & Kawada, T. (2006). The mechanism of OP pesticide-induced inhibition of cytolytic activity of killer cells. *Cell. Mole. Immunol.*, **3**, 171-178.
- Littrell, E. E. (1988). Wild carnivore deaths due to anticoagulant intoxication. *Calif. Fish Game*, **74**, 183.
- Lotti, M. & Moretto, A. (2006). Do carbamates cause polyneuropathy? *Muscle Nerve*, **34**, 499-502
- Macek, K.J. (1969). Screening of pesticides against fish, p. 92. In Progress in Sport Fishery Research, 1968. Bureau of Sport Fisheries and Wildlife, U.S. Res. Publ., **77**, 259 pp.
- Martin, P.A. & Solomon, K.R. (1991). Acute carbofuran exposure and cold stress: 537 Interactive effects in mallard ducklings. *Pestic. Biochem. Physiol.*, **40**, 117-127.
- Mayer, K.E. & Laudenslayer, W.F. (1989). A guide to wildlife habitats of California. California Department of Forestry, Sacramento, USA.
- McMillan, S.C., Hosea, R.C., Finlayson, B.F., Cypher, B.L. & Mekebri A. (2008). *Anticoagulant rodenticide exposure in an urban population of San Joaquin kit*, in R.M. Timm, M.B. Madon, editors. Proceedings of the 23rd Vertebrate Pest Conference **23**, 163-165.
- Mendenhall, V.M. & Pank, L.F. (1980). Secondary poisoning of owls by anticoagulant rodenticides. *Wildlife Soc. Bull.*, **8**, 311-315.
- Morner, T., Obendorff, D.L., Artois, M. & Woodford, M.H. (2002). Surveillance and monitoring of wildlife diseases. *Revue Scientifique et Technique de l'Office International des Epizooties*, **21**, 67-76.
- Olgun, S. (2004). *Immunotoxicity of pesticide mixtures and the role of oxidative stress*. PhD dissertation. Virginia Polytechnic Institute, Blacksburg VA.
- Paul, N. & Mannathukkaran, T.J. 2005. Intermediate syndrome following carbamate poisoning. *Clin. Toxicol.*, **43**, 867-868
- Proulx, G. (2011). Field evidence of non-target and secondary poisoning by strychnine and chlorophacinone used to control Richardson's ground squirrels in southwest Saskatchewan. Pages 128-134 in Danyluk, D., editor. *Proc. Ninth Prairie Conserv. Endang. Species Conf.* Winnepeg, MB, Canada.
- Proulx, G. & MacKenzie N. (2012). Relative abundance of American badger (*Taxidea taxus*) and red fox (*Vulpes vulpes*) in landscapes with high and low rodenticide poisoning levels. *Integr. Zool.*, **7**, 41-47.
- Rattner, B.A., Horak, K.E., Lazarus, R.S. et al. (2012). Assessment of toxicity and potential risk of the anticoagulant rodenticide diphacinone using Eastern screech-owls (*Megascops asio*). *Ecotoxicology*, **21**, 832-846.
- Relyea, R.A. (2003). Predator cues and pesticides: a double dose of danger for amphibians. *Ecol. Appl.*, **13**, 1515-1521.

- Releya, R.A. & Diecks, N. (2008). An unforeseen chain of events: lethal effects of pesticides on frogs at sublethal concentrations. *Ecol. Appl.*, **18**, 1728-1742.
- Richards, N. (2011). *Carbofuran and wildlife poisoning: global perspectives and forensic approaches*. John Wiley and Sons, West Sussex. UK. 304 pp.
- Riley, S.P., Bromley, C., Poppenga, R.H., Uzal, F.A., Whited, L. & Sauvajot, R.M. (2007). Anticoagulant exposure and notoedric mange in bobcats and mountain lions in urban Southern California. *J. Wildlife Manage.*, **71**, 1874-1884.
- Roelofs, W., Crocker, D.R., Shore, R.F. *et al.* (2005). Case Study Part 2: Probabilistic modelling of long-term effects of pesticides on individual breeding success in birds and mammals. *Ecotoxicology*, **14**, 895-923.
- Robertson, H.A & Colbourne, R.M. (2001). Survival of little spotted kiwi exposed to the rodenticide brodifacoum. *J. Wildlife Manage.*, **65**, 29-34.
- Rohr, J.R., Schotthoefer, A.M., Raffel, T.R. *et al.* (2008). Agrochemicals increase trematode infections in a declining amphibian species. *Nature*, **455**, 1235-1239.
- Sanchez-Barbudo, I.S., Camarero, P.R., & Mateo, R. (2012). Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. *Sci. Total Environ.*, **420**, 280-288.
- Scheller, R.M., Spencer, W.D., Rustigian-Romsos, H., Syphard, A.D., Ward, B.C. & Strittholt, J. (2011). Using stochastic simulation to evaluate competing risks of wildfires and fuels management on an isolated forest carnivore. *Landscape Ecol.*, **26**, 1491-1504.
- Shore, R.F., Birks, J.D.S. & Freestone, P. (1999). Exposure of non-target vertebrates to second-generation rodenticides in Britain, with particular reference to the polecat (*Mustela putorius*). *New Zeal. J. Ecol.*, **23**, 199-206.
- Sibley, R.M., Newton, I. & Walker, C.H. (2000). Effects of dieldrin on population growth rates of sparrowhawks 1963-1986. *J. Appl. Ecol.*, **37**, 540-546.
- Spencer, W.D., Rustigian-Romsos, H., Strittholt, J., Scheller, R., Zielinski, W. & Truex, R. (2011). Using occupancy and population models to assess habitat conservation opportunities for an isolated carnivore population. *Biol. Conserv.*, **144**, 788-803.
- Stone, W.B., Okoniewski, J.C. & Stedelin, J.R. (1999). Poisoning of wildlife with anticoagulant rodenticides in New York. *J. Wildlife Dis.*, **35**, 187-193.
- Susser, M. (1973). *Causal thinking in the health sciences: concepts and strategies of epidemiology*. Oxford University Press. Oxford, England.
- Terry, A.V. (2012). Functional consequences of repeated organophosphate exposure: potential non cholinergic mechanisms. *Pharmacol. Therap.*, **134**, 355-365.
- Thompson, H. (1996). Interactions between pesticides: a review of reported effects and their implications for wildlife risk assessment. *Ecotoxicology*, **5**, 59-81.
- Thompson, C.M., Purcell, K.L., Garner, J. & Green, R.E. (2010). Kings River Fisher Project progress report 2007-2010. Unpublished report to the USDA Forest Service, Pacific Southwest Research Center. Albany, CA. 37 pp.
- Thompson, C.M., Zielinski, W.J. & Purcell, K.L. (2011). Evaluating management risks using landscape trajectory analysis: a case study of California fisher. *J. Wildlife Manage.*, **75**, 1164-1176.
- Townsend, M.G., Bunyan, P.J., Odum, E.M., Stanley, P.I. & Wardall, H.P. (1984) Assessment of secondary poisoning hazard of warfarin to least weasels. *J. Wildlife Manage.*, **48**, 628-632.
- Tucker, J.M., Schwartz, M.K., Truex, R.L., Pilgrim, K.L. & Allendorf, F.W. (2012). Historical and contemporary DNA indicate fisher decline and isolation occurred prior to European settlement of California. *PLoS ONE*, **7**, e2803. doi:10.1371/journal.pone.0052803.
- Vidal, D., Alzaga, V., Luque-Larena, J.J., Mateo, R., Arroyo, L. & Vinuela, J. (2009). Possible interaction between a rodenticide treatment and a pathogen in common vole (*Microtus arvalis*) during a population peak. *Sci. Total Environ.*, **408**, 267-271.
- Villafuerte, R., Vinuela, J. & Blanco, J.C. (1998). Extensive predator persecution caused by population crash in a game species: the case of red kites and rabbits in Spain. *Biol. Conserv.*, **84**, 181-188.
- Webster, K.H. 2009. Validation of a prothrombin time (PT) 613 assay for assessment of brodifacoum exposure in Japanese quail and barn owls. Master's Thesis, Simon Fraser University
- Wengert, G.M., Gabriel, M.W. & Clifford, D.L. (2012). Investigating cause-specific mortality and diseases in carnivores: tools and techniques. Pages 294-313 in L. Boitani, R.A. Powell, editors. *Carnivore ecology and conservation: a handbook of techniques*. Oxford University Press, USA.
- Winters, A.M., Rumbelha, W.K., Winterstein, S.R., Fine, A.E., Munkhtsog, B. & Hickling, G.J. (2010). Residues in Brandt's voles (*Microtus brandti*) exposed to bromadiolone-impregnated baits in Mongolia. *Ecotoxicol. Environ.*, **73**, 1071-1077.
- Wobeser, G., Bollinger, T., Leighton, F.A., Blakley, B. & Mineau, P. (2004). Secondary poisoning of eagles following intentional poisoning of coyotes with anticholinesterase pesticides in western Canada. *J. Wildlife Dis.*, **40**, 163-172.
- Zabrodskii, P.F., Germanchuk, V.G., Kirichuk, V.F., Birdin, V.S. & Chuev, A.N. (2002). Combined effects of toxicants with various mechanisms of action and mechanical trauma on the immune system. *Bull. Experime. Biol. Med.*, **6**, 594-596.
- Zabrodskii, P.F., Lim, V.G. & Strel'tsova E.V. (2012). Disturbances of immune status and cytokine profile caused by chronic intoxication with OP compounds and their

- correction by administration of imunofan. *Eksp Klin Farmakol.* **75**, 35-37.
- Zielinski, W.J., Kucera, T.E. & Barrett, R.H. (1995). Current distribution of the fisher, *Martes pennanti*, in California. *Calif. Fish Game*, **81**, 104-112.
- Zielinski, W.J., Duncan, N.P., Farmer, E.C., Truex, R.L., Clevanger, A.P. & Barrett, R.H. (1999). Diet of fishers (*Martes pennanti*) at the southernmost extent of their range. *J. Mammal.*, **80**, 961-971.
- Zielinski, W.J., Truex, R.L., Schlexer, F.V., Campbell, L.A. & Carroll, C. (2005). Historical and contemporary distributions of carnivores in forests of the Sierra Nevada, California, USA. *J. Biogeogr.*, **32**, 1385-1407.
- Zielinski, W. J., Baldwin, J.A., Truex, R.L., Tucker, J.M. & Flebbe, P.A. (2013). Estimating trend in occupancy for the Southern Sierra Fisher (*Martes pennanti*) population. *J. Fish Wildlife Manage*, **4**, doi: <http://dx.doi.org/10.3996/012012-JFWM-002>.

5 States (And One City) Ready to Legalize Marijuana

SAM BECKER
SEPTEMBER 01, 2014



David McNew/Getty Images

It's an interesting time to stand on the sidelines and watch marijuana legalization efforts take over the country. Colorado and Washington both jumped the gun and passed initiatives to decriminalize and legalize cannabis by popular vote in 2012, and since then have both opened the first legal marijuana markets in the U.S. Legal retail sales began this year, and so far things have settled into place, and the novelty has started to wear off to some degree.

But many other states are following Washington and Colorado's path, getting closer and closer to legalization every election cycle. So far, legalization advocates have had to rely on voter-backed initiatives to get legislation passed, as the federal government seems as though it still won't budge on reclassifying cannabis out of its current schedule -1 status. Local governments across the country have taken baby steps towards ending prohibition, with many cities passing ordinances that either have decriminalized small amounts of marijuana or marked them as a lowest priority for law enforcement officials.

Many people are still struggling with the concept of legalized marijuana. For decades and generations, Americans grew accustomed to knowing marijuana as a powerful and dangerous drug — one that could lead to deaths and criminal behavior if it was allowed in their community. The past decade has really opened up a lot of people's eyes to the facts, which almost wholly dismiss those worries. The medical marijuana communities in several states have also shown the immense benefits cannabis can have for the sick, which is one of many factors that have led to a seismic shift in public opinion regarding marijuana legalization.

As time marches forward, more states are preparing for coming marijuana legalization initiatives, either derived from state legislators or from citizens themselves. A few states have gotten close in the past, but so far only Colorado and Washington have been able to pull through. That doesn't mean that several others aren't on the cusp, however.

Here are six states that are the closest to legalizing marijuana for recreational use in the near future, hot on the heels of Washington and Colorado.



Source: Thinkstock

1. Oregon

Perhaps the state that was the closest to becoming the third to end prohibition is Oregon, Washington's neighbor in the Pacific Northwest. Oregon has a reputation for being a hippy haven of sorts, although that stereotype really only holds true in a few select cities, like Portland, Eugene, and Corvallis. Despite the conservative-lean of most of the remainder of the state, Oregon still came very close to legalizing cannabis in 2012, but voters turned down a measure that would have probably done more harm than good.

This year, a new initiative is on the ballot for voters to consider, so Oregon could join its northerly neighbor in November. The *Huffington Post* [reports](#) 87,000 signatures had been collected to get the measure on this fall's ballot, and seeing that 57 percent of the state's residents support legalization, it's likely to pass. Legislators also like the possibility of up to [\\$40 million](#) in new tax

revenue.



Source: Thinkstock

2. California

Perhaps the biggest domino on the board that could drastically change the national landscape in terms of prohibition is California. If Oregon and California are both able to pass legalization measures, then the entirety of the U.S. west coast would be comprised of states that have ended prohibition, creating a Mecca of sorts for cannabis fans. Of course, California is the most populous — and probably most demographically complicated — state in the union.

California represents one of the world's largest economies all on its own, and if cannabis is legalized, it will have a dramatic effect

across the country. The state is already home to one of the most robust medical marijuana markets in the world, so the state's residents aren't exactly unfamiliar with the product either. Although it's not expected to reach the ballot until 2016, the wheels are in [motion](#) to make California one of the next states to end marijuana prohibition.

[See a Hawaiian Sweet & Sour Sausage Recipe](#)



Source: Thinkstock

3. Alaska

Sticking out west, and quite far north, Alaska has long been rumored to be on legalization's doorstep — although it hasn't happened just yet. Alaska has had some of the nation's most lax marijuana laws for a long time, likely due to its incredibly sparse

population, and vast landscape. Although law enforcement agencies have [maintained](#) that they don't plan on relaxing their duties when it comes to cannabis, even as other states have legalized, Alaskans are holding out hope.

Like Oregon, a ballot measure is in place for November, which will give Alaskans the opportunity to vote for legalization. There is a lot of support for passing the measure, but as some sources are [reporting](#), there is also a lot of resistance. If Alaska can stick with its west coast cousins and formally end marijuana's prohibition in the great white north, it should be a victory for Alaskans statewide and for entrepreneurs, legislators, and the state's budget as well.

Watch: How to Stick to a Trading Plan

schwab.com



Source: Thinkstock

4. Hawaii

Staying out west — way out west, that is — Hawaii should be one of a handful of states to opt for legalization. Hawaiians are famous for growing some of the most famous marijuana in the world, and it's a plant that is fairly heavily ingrained in the island culture. Although legalization efforts have been stopped short thus far, it's hard to believe that prohibition laws will remain intact very much longer, especially considering Hawaii's fiercely independent ideals regarding self-reliance and governance.

A [bill](#) to legalize was brought before legislators earlier this year, although it died shortly thereafter. Once again, it looks like the voters of the state will need to pass a voter-backed initiative in order for legalization to happen. Legislators will most likely need to take a close look at the revenue Colorado and Washington are bringing in to sway them back to the idea, and with the amount of

tourists the state sees annually, there's a lot of potential for heavy tax revenues that could be convincing.



Source: Thinkstock

5. Maine

Far from the western states that seem to dominate the legalization discussion, the northeastern bastion of Maine is also sitting pretty, getting ready to mount legalization efforts of its own. There was recently enough signatures [collected](#) to give the movement some momentum, and several cities across the state are looking at decriminalization efforts as well.

If Maine is able to pass legalization legislation, then some of its New England counterparts may follow suit as well. There are already groups working in states like Vermont to get initiative on state ballots, and if Maine is able to kick over the first domino in

the northeast, it should do nothing but help.

As David Boyer, Maine political director for the Marijuana Policy Project [told](#) local news affiliate *WCSH6* that, “We have bigger fish to fry. There’s violent crimes going on, there’s property crimes, and that is where our police resources should be spent.”



Bruce Bennett/Getty Images

6. Washington D.C.

The irony would be thick if D.C. was able to pass a legalization measure, wouldn't it? Well, voters living in the District of Columbia will get a chance to pull it off, just like Oregon and Alaska this November. There was an apparent [overwhelming](#) show of support in

order to get an initiative to the voters this fall, and although D.C. isn't actually a state, its residents look as ready to end prohibition within their jurisdiction as any other place in the country.

Being the heart of the federal government, a voter-backed legalization law could have some pretty resounding effects. It would be pretty hard for the federal government to continue justifying federal prohibition laws in say, Kentucky, while the city surrounding the nation's capital don't even enforce those laws themselves. One thing is for sure — it will be interesting to see what happens if D.C. is able to pull off a successful legalization effort.

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Forgotten Stars Of the 90's, What Do they Look Like Now!?

Feds call for study of marijuana industry's effects on salmon

BY GLENDA ANDERSON THE PRESS DEMOCRAT on October 2, 2014, 6:43PM10/02/2014

Following years of warnings from state Fish and Wildlife and forestry officials, the federal government this week called for further study of the effects of marijuana cultivation on threatened salmon populations in pot-rich areas like Northern California's Emerald Triangle, which includes Mendocino, Humboldt and Trinity counties.

The recommendations by federal fisheries officials were included in a document released Tuesday that lays out plans to rehabilitate 40 populations of threatened coho salmon in a wide geographic range that includes about 10,000 miles of streams and 13 million acres in southern Oregon and Northern California, including parts of Mendocino and Lake counties.

"We identified marijuana as one of the activities that contributes to the problems" fish face in some regions, said Julie Weeder, the recovery coordinator for the Northern California division of the National Oceanic and Atmospheric Administration's fisheries division, commonly called the National Marine Fisheries Service, which published the report.

The comprehensive, estimated 2,200-page report proposes some 3,000 recovery actions. There are about a half-dozen "highest priority recovery actions" for each of the 40 coho populations addressed in the plan. The top of the action list for rehabilitating fish populations in the Eel River system in Lake, Mendocino and Humboldt counties includes studying the effects of the marijuana industry on the fish and taking unspecified action to minimize its effects if necessary. There are no specific mitigation plans listed for pot because its effects need further study, Weeder said.

Some marijuana mitigations are already included in other recommended actions, such as stopping unauthorized water diversions from streams and rivers, Weeder said. Many illegal pot growers buy, rent or trespass and illegally divert water from streams that feed the threatened watersheds.

The priority lists also include well-established rehabilitation actions, such as restoring natural stream channels, reducing sediment buildup and increasing stream flows by reducing water diversions generally.

The plan — purely voluntary — is aimed at providing guidance to federal, state, local and tribal resource managers and private organizations and people who are pursuing rehabilitation projects or planning to do so.

"It provides the road map to recovery," Weeder said. The report describes historic and current conditions in the designated coho population areas, the threats facing the fish and how best to proceed toward increasing their numbers. It would cost an estimated \$5 billion to implement the entire plan, she said.

There currently is no funding pool identified for the project. It's likely the individual entities involved with coho restoration projects will pursue funding and that it will take decades to implement the plan.

The plan has been in the works since coho in southern Oregon and northern California were declared threatened in 1997. Several drafts were circulated before the final report was released Tuesday.

Marijuana cultivation was added to the list of fish dangers in about 2011, following recommendations by state Fish and Wildlife and forestry officials.

A state Fish and Wildlife study on four watersheds in Mendocino and Humboldt counties that was released earlier this year estimated that escalating marijuana production in Humboldt and Mendocino counties had the potential to suck streams dry, threatening decades of salmon restoration efforts.

The study used satellite images to determine that an average of 30,000 plants were growing in each of the four watersheds in 2012, an increase since 2009 of 75 percent, according to state Fish and Wildlife Senior Environmental Scientist Scott Bauer, who headed the study.

Researchers estimated each plant consumes 6 gallons of water a day, a rate that adds up to 180,000 gallons of water per day in each watershed — more than 160 Olympic-sized swimming pools — over the average 150-day growing cycle for outdoor plants.

Some marijuana advocates say that the plants use much less, closer to three gallons a day. But others have said that mature, tree-sized plants can use more, closer to 15 gallons a day.

Water isn't the only problem associated with marijuana cultivation.

Pot production also is polluting streams with pesticides, herbicides and sediment, a byproduct of clearing trees and building illegal roads to grow marijuana, wildlife, forestry and law enforcement officials say. It also is poisoning other wildlife.

State Fish and Wildlife spokesman Patrick Foy has called the habitat destruction from pot production "staggering."

Marijuana advocates say most people who grow pot for medicinal use are conscientious and are not causing environmental problems. It's the people who have moved to the area to grow large amounts for profit who are the problem, they contend.

Marijuana growing is just one factor among many purported to have affected Coho and other fish populations, according to the recovery plan. Some date back more than 100 years, such as the damming of the upper main stem of the Eel River above Potter Valley in 1908. Cape Horn dam and Scott Dam, which created Lake Pillsbury in 1922, blocked access to as much as 50 miles of spawning grounds and altered river flows, according to the report.

Similarly, the middle main stem of the Eel River was affected by the draining and diking of the Little Lake Valley outside of Willits in 1910 for grazing cattle and growing potatoes. The seasonal lake likely was productive habitat for Coho, according to the report.

The entire report may be found at: www.westcoast.fisheries.noaa.gov/stories/2014/29_09302014_soncc_plan_released.html

You can reach Staff Writer Glenda Anderson at 462-6473 or glenda.anderson@pressdemocrat.com. On Twitter @MendoReporter.

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The plan has been in the works since coho in southern Oregon and northern California were declared threatened in 1997. Several drafts were circulated before the final report was released Tuesday.

Marijuana cultivation was added to the list of fish dangers in about 2011, following recommendations by state Fish and Wildlife and forestry officials.

A state Fish and Wildlife study on four watersheds in Mendocino and Humboldt counties that was released earlier this year estimated that escalating marijuana production in Humboldt and Mendocino counties had the potential to suck streams dry, threatening decades of salmon restoration efforts.

The study used satellite images to determine that an average of 30,000 plants were growing in each of the four watersheds in 2012, an increase since 2009 of 75 percent, according to state Fish and Wildlife Senior Environmental Scientist Scott Bauer, who headed the study.

Researchers estimated each plant consumes 6 gallons of water a day, a rate that adds up to 180,000 gallons of water per day in each watershed — more than 160 Olympic-sized swimming pools — over the average 150-day growing cycle for outdoor plants.

Some marijuana advocates say that the plants use much less, closer to three gallons a day. But others have said that mature, tree-sized plants can use more, closer to 15 gallons a day.

Water isn't the only problem associated with marijuana cultivation.

Pot production also is polluting streams with pesticides, herbicides and sediment, a byproduct of clearing trees and building illegal roads to grow marijuana, wildlife, forestry and law enforcement officials say. It also is poisoning other wildlife.

State Fish and Wildlife spokesman Patrick Foy has called the habitat destruction from pot production “staggering.”

Marijuana advocates say most people who grow pot for medicinal use are conscientious and are not causing environmental problems. It's the people who have moved to the area to grow large amounts for profit who are the problem, they contend.

Marijuana growing is just one factor among many purported to have affected Coho and other fish populations, according to the recovery plan. Some date back more than 100 years, such as the damming of the upper main stem of the Eel River above Potter Valley in 1908. Cape Horn dam and Scott Dam, which created Lake Pillsbury in 1922, blocked access to as much as 50 miles of spawning grounds and altered river flows, according to the report.

Similarly, the middle main stem of the Eel River was affected by the draining and diking of the Little Lake Valley outside of Willits in 1910 for grazing cattle and growing potatoes. The seasonal lake likely was productive habitat for Coho, according to the report.

The entire report may be found at: www.westcoast.fisheries.noaa.gov/stories/2014/29_09302014_soncc_plan_released.html

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Marijuana Growth in British Columbia

by Stephen T. Easton

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Executive Summary

The cultivation and production of marijuana in British Columbia highlights the problems inherent in the enforcement of laws that are generally ignored by broad sectors of the populace. Some 7.5 percent of all Canadians report they use marijuana currently, and over their lifetimes, 23 percent report themselves as having used marijuana at least once.

This paper raises several issues that have the cumulative effect of suggesting that in the long term, the prohibition on marijuana cannot be sustained with the present technology of production and enforcement. To anyone with even a passing acquaintance with modern history, it is apparent that we are reliving the experience of alcohol prohibition of the early years of the last century.

In Canada, and more specifically British Columbia today, as with alcohol nearly a century ago, marijuana is too easily produced and exported to be controlled with the tools available to law enforcement in a free society. The return on investment is sufficiently great so that for each marijuana growing operation demolished, another takes its place.

For a modest marijuana growing operation of 100 plants, harvest revenue is from 13 kilograms of marijuana sold in pound blocks out the back door valued at \$2,600 per pound. This amounts to slightly less than \$20,000 per harvest. With four harvests per year, gross revenue is nearly \$80,000. A conservatively high estimate of production cost is about \$25,000. The return on invested money is potentially high: around 55 percent.

The underlying characterization of the marijuana grow operation is that it functions as a profit-maximizing activity in which the values of

output and costs yield a market equilibrium rate of return. Such an assumption permits an estimate of the total number of grow-ops. The range of estimates depends upon the value of the crop, the costs of production, the risk-adjusted rate of return to other small businesses, and the likelihood of discovery by the police. For the year 2000, the estimated number of “grow-ops” in British Columbia may be as high as 17,500. Combined with domestic consumption, numbers of this magnitude suggest that exports from British Columbia are worth nearly \$2 billion.

Why is it that indoor marijuana cultivation and consumption appear to take place more openly in BC than elsewhere in Canada? The most striking difference between BC and the rest of Canada lies in the rate at which offences are settled by charging the offender (or “cleared”). Only 13 percent of possession offences in BC are cleared by charge. Elsewhere in Canada over 60 percent of possession offences are cleared by charge. In addition, the penalties for conviction appear to be low.

In a sample of Vancouver marijuana growing operations “busted” by the police, most of those who were convicted received no jail time: 55 percent. Five more percent were sentenced to a single day or less and another 8 percent received sentences of between one day and 31 days, while still another 8 percent received 60 days. Some 11 percent were sentenced to 90 days. Of those who are repeat offenders, half are reconvicted within the year. Of the 35 percent who were fined, the average fine amounted to less than \$1,200: a small amount considering the size of most marijuana operations. While police resources are spent to destroy nearly 3,000 marijuana growing operations a year, the consequences are relatively small for those convicted.

Current public policy proposals emphasize decriminalization. Suppose, however, that marijuana were treated like any other product and were to be sold at retail cigarette value rather than in bulk. At current prices, a marijuana cigarette costs about \$1.50 to produce, and sells for around \$8.60. Since the consumer currently is willing to pay \$8.60, imagine a tax on marijuana cigarettes equal to the difference between the local production cost and the street price. This would transfer the revenue from the current producers and middlemen, many of whom are associated with organized crime, to the government. Crudely, government would have revenue of about \$7 per cigarette. Using conservative assumptions about Canadian consumption, this comes to revenue of over \$2 billion, and should marijuana be taxed on the same basis for export

(leaving aside obvious problems of international diplomacy with the United States), additional revenue could be generated. Further, policing assets currently involved in enforcing marijuana-related statutes could be deployed elsewhere.

What the analysis reveals is how widespread marijuana use is in Canada and how extensive production is in British Columbia. As a consequence, the broader social question becomes less about whether we approve or disapprove of local production, but rather who shall enjoy the spoils. As it stands now, growers and distributors pay some of the costs and reap all of the benefits of the multi-billion dollar marijuana industry, while the non-marijuana-smoking taxpayer sees only costs.

Marijuana Growth in British Columbia

The cultivation and production of marijuana in British Columbia highlights the problems inherent in the enforcement of laws that are generally ignored by broad sectors of the populace.¹ Some 7.5 percent of all Canadians report they use marijuana currently (or at least have done so during the past year). Of those aged 15 years and older, about 23 percent of the Canadian population report that they have used marijuana at least once in their life.² By province there are variations in recent marijuana use with British Columbia the highest at 11 percent, and Newfoundland and Ontario the lowest at 3.8 percent and 5.1 percent respectively. There is variation in use by age and sex, with younger people more likely to have used the drug than older people³ with males using at twice the rate of females.

This paper raises several issues that have the cumulative effect of suggesting that in the long term, the prohibition of marijuana cannot be sustained with the present technology of production and enforcement. To anyone with even a passing

acquaintance with modern history, it is apparent that we are reliving the experience of alcohol prohibition of the early years of the last century.⁴ In that sorry episode, on both sides of the Canada-US border the widespread demand for prohibited alcohol led to the rapid growth of criminal enterprises that expanded to produce the product that the general population desired.⁵ As a testament to the enduring significance of the period, recall that even today we cheer for Eliot Ness as he smashes the alcohol making stills of organized crime in endless television reruns of *The Untouchables*. Ironically, we may now sip a cocktail as we do so.

In Canada, and more specifically in British Columbia today, as with alcohol nearly a century ago, marijuana is too easily produced and exported to be controlled with the tools available to law enforcement in a free society. The return on investment is sufficiently great that for each marijuana growing operation demolished, another will take its place.

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- 1 I am indebted to several people who have read, commented, and offered insight about drafts this paper. Jason Clemens, Herbert Grubel, David Easton, Malcolm Easton, Kash Heed, Fred McMahon, Robert A. Jones, Niels Veldhuis, and Michael Walker each offered valuable insights but are not responsible for the content. Liv Fredrickson helped with data input as well as advice. Obviously I am responsible for errors.
 - 2 (Single *et al.*, 1999.) Contrast these figures with lifetime use of 8.1 percent for cocaine and 10.4 percent for LSD, speed, or heroin. On the legal side, 72 percent of the Canadian population has used alcohol in the past year, and 27 percent identify themselves currently as tobacco smokers.
 - 3 Among those 15 to 19 years old, about 25 percent have used in the past year (Single *et al.*, table 5.3). Although it is not in the survey data, it may very well be that the younger set—aged 9 and up, should actually be queried as well. Data from grade schools suggest that use of marijuana in the past year in grade 7 is typically around 10 percent or below. The percentage swells to around 30 percent or higher by grade 9 (*New Brunswick Student Drug Use Survey 2002 Highlights Report*; *Nova Scotia Student Drug Use 2002 Highlights Report*; *Prince Edward Island Student Drug Survey 2002 Highlights Report*). Data from other provinces are consistent with these figures.
 - 4 See, for example, Mark Thornton (1991), “Alcohol Prohibition was a Failure,” *Cato Policy Analysis* No. 157 (January).
 - 5 See, for example, Warburton (1932, chapter IX) or Thornton.

Although there are a host of important criminological, social, psychological, and economic issues associated with marijuana, this paper is primarily a framework that develops a series of “facts” and characterizations of the marijuana industry in British Columbia that can be revisited, revised, and challenged to make a sensible policy debate possible.⁶ The first two sections of the paper organize the discussion using the economist’s model of demand and supply with

an emphasis on the latter. Subsequent sections include a methodology and estimate of the number of marijuana growing operations (“grow-ops” as they are popularly known) in British Columbia, some discussion of why British Columbia appears to be a significant location for marijuana production, and some thoughts about the transformation of currently illegal returns into tax revenue were marijuana to be made legal.

Canadian Marijuana Consumption

Marijuana consumption is difficult to measure. Although there are plenty of data about marijuana *use* in Canada, very little is quantitatively oriented. To say that someone “uses” once or twice a week is not very specific about the *quantities* they are likely to use. Reuter suggests that a “very heavy user of marijuana consumes about 3 marijuana cigarettes per day” (1996, p. 7).⁷ In Australia, usage has been measured in the Australian Institute for Health and Welfare 1998 *National Drug Strategy Household Survey*.⁸ More Australians appeared to have tried marijuana (39 percent compared to 23 percent of Canadians), and more Australians have used marijuana “re-

cently” (18 percent compared to 7.5 percent in Canada).

The average marijuana cigarette is 0.4 to 1.0 grams in weight (Adams and Martin, 1997).⁹ For those who still think in Imperial units, there are about 28.35 grams in an ounce or about 453.6 grams in a pound. There are, of course, 1,000 grams in a kilogram. Consequently, even if marijuana use is measured in number of cigarettes, quantity is still difficult to assess. Loosely, 15 grams of marijuana generates between 15 and 30 cigarettes according to taste. I have found no correction for the strength of the active ingredients

6 I do not discuss the Canadian federal government initiatives to decriminalize small amounts of marijuana. Such a proposal deserves a separate and specific response.

7 On the other hand, asking around locally suggests that this is high for British Columbia leaf. Anecdotally, a heavy user is said to use one cigarette per day.

8 Digital document available at <http://www.aihw.gov.au/publications/health/ndshs98d/>. Although these data have more information about frequency of consumption, quantity must still be imputed.

9 Others find slightly lower values at roughly 0.39 grams per cigarette (W. Rhodes *et al.*, 1995, *What America’s Users Spend on Illegal Drugs*, 1988-93, Washington, D.C.: Office of National Drug Control Policy, p. 20, cited in Reuter, 1996.) In contrast, commercial cigarettes weigh-in at 0.77 grams, a weight that appears to have stabilized since 1988. Prior to 1988, the weight of a cigarette had fallen from over 1.6 grams in the early 1950s to about 0.77 today ([http://www.ncth.ca/NCTHweb.nsf/0/ac40b01bdef1ff99852569d60063e43b/\\$FILE/gdb6a-weight.pdf](http://www.ncth.ca/NCTHweb.nsf/0/ac40b01bdef1ff99852569d60063e43b/$FILE/gdb6a-weight.pdf)).

on the “weight” of the cigarette. Some people report that they consume as many as 60 cigarettes per day, but they are obviously exceptional.

Some limits on the size of the *internal* market for marijuana

If roughly 7.4 percent of the Canadian population currently uses marijuana, then with 25 million Canadians aged 15 or over this implies about 1.87 million users. Table 1 puts this consumption into some kind of numerical perspective.¹⁰ The first column identifies the number of users based on estimates of usage described in Single *et al.* (1999, Table 5.1) The second column gives an estimate in metric tons of internal Canadian marijuana consumption. The third column multiplies this by price to illustrate the size of the Canadian (con-

sumption) market. This of course does not include exports. The final column details the expenditure by Canadians on (legal) tobacco for the past few years to illustrate the scale of the internal market.

How large is the industry? Expenditures on illegal marijuana in Canada are roughly the same order of magnitude as those on legal tobacco products. Substantial though these numbers may be, however, they are not the central issue. Even as the Government of Canada apparently plans to reduce the penalty for consumption, most attention focuses on production for which the external market in the United States is simultaneously an economic goldmine and a political landmine. As the evidence will show, it is obvious that much of the British Columbia marijuana crop is grown for export.

Table 1: Estimates of the Internal Canadian Market for Marijuana, 1988-2000

Year	Current users (millions)	Total internal consumption* (thousands of kilograms)	Annual expenditure on marijuana* (billions of dollars)	Annual expenditure on tobacco (billions of dollars)
1988	1.38	111.0	1.4	
1990	1.10	92.1	1.5	
1991	1.11	87.9	1.5	
1992	1.13	92.2	1.6	
1993	0.96	81.1	1.2	
1994	1.71	152.1	2.0	
1995	1.73	154.1	1.7	
1996	1.75	156.1	1.7	
1997	1.78	158.2	1.7	2.5
1998	1.80	160.1	1.9	2.5
1999	1.82	162.0	1.7	2.4
2000	1.84	164.1	1.8	2.3

*Table 1A provides upper and lower estimates.
Sources: See Appendix Table 1A.

¹⁰ This table is derived from Appendix table 1A, which details the sources and methods of construction. Table 1 uses the “low” estimates from table 1A.

Producing Marijuana in British Columbia

There is very little hard information about the actual number of marijuana growing operations (“grow-ops”) in British Columbia. From the pattern of police enforcement we believe that the numbers have been increasing, but the actual scale of marijuana growing is difficult to know with assurance—for obvious reasons. From 1997 to 2000, Plecas *et al.* report that the number of grow-ops discovered and dismantled, or “busted” in the usual terminology, more than doubled: from 1,251 to 2,808. This issue is addressed below in the section titled “How Many Grow-ops are Out There?”

There are several ways to produce marijuana. I will discuss the outcomes of indoor supply, which is the most relevant to an urban setting and the current data set. Nearly 80 percent of all grow-ops discovered by police are indoor operations, although this reflects policing costs as well as the true distribution of grow-ops. Further, there are likely to be plenty of individual marijuana grow operations of a few plants that are not likely candidates to be busted and are conse-

quently are not included in the statistics. Before turning to the production side of the marijuana industry, however, there is the matter of price that permeates any discussion of the business. The next section develops a characterization of the relationship between price and quantity that is used throughout the rest of the analysis. This is important because evaluating marijuana quantities sold at per pound prices of production may lead to different interpretations of size and significance of the industry than by evaluating marijuana sales at the more expensive “per cigarette” level of consumption.

The price of the product

To give some idea of the value of marijuana (Appendix A discusses the estimates in detail), table 2 uses estimated values computed from cross-Canada data gathered by the RCMP from 1995 to 1999. Aggregating these data and estimating a relationship for British Columbia gives a sense of the values appropriate for different quantities of the drug.¹¹

Table 2: Retail Purchase Prices by Quantity of Purchase

Unit in which purchased	Year 2000 Canadian \$ unit price	Gram weight of purchase	Price per gram of the purchase
0.5 gram	8.6	0.50	17.16
1 gram	15.3	1.00	15.33
1 ounce	254.5	28.35	8.98
1 pound	2,613.0	453.60	5.76
1 kilogram	5,077.0	1000.00	5.08

The underlying estimation appears as equation 2 in Appendix A.

11 Not all units were actually purchased or reported in the raw data. For example, the kilogram price is an extrapolation of the estimated power function that relates price to quantity. All the other quantities were part of the data set.

The table's first column reports the unit of purchase. The second column reports the average price of the purchase of that unit. The third column indicates the number of grams in the purchase bundle in order to put the purchases into a common unit. The final column reports the implicit price per gram at the different quantities. As is expected, larger quantities are cheaper on a per gram basis.¹²

Growth cycle and "bud" size

Outdoor crops mature once a year. Each indoor crop takes between 6 weeks and 4 months to mature.¹³ To err on the side of caution, we will use a period that gives four harvests per year.

At harvest each plant produces one "bud" which is the structure that produces about 100 grams of usable marijuana. This, in turn, yields a dry weight of roughly 33 grams.¹⁴ Although they may not be a representative sample, data from Vancouver police drug busts suggest that in 1998 a bud weighed about 3.3 ounces (100 grams). In 1999 the average bud had increased to 4.3 ounces (122 grams). Most estimates (Plecas *et al.*, for example) take 100 grams as the relevant average. This assumption will also be made in what follows.

Potency

One frequently uttered sentiment is that British Columbia grown marijuana is on the stronger

end of the spectrum. This may be true, but it is tricky to document systematically. Data collected by the RCMP tend to suggest that the potency, the THC content, has remained roughly constant over the 1995 to 1999 period. Nationally, there was no obvious increase in the measured quality of marijuana acquired by the police from various activities: busts, buys, and the like. Within British Columbia, although the mean THC content has increased over the same period, that increase is not statistically significant.¹⁵ Consequently, although it is possible that there has been an increase in the THC content (if popular reports are to be believed), it remains to be observed systematically, though the raw numbers are not inconsistent with an increase in the late 1990s.

The house

The marijuana producer needs an establishment to house a grow-op. Typically, grow-ops have been found in rented houses. A house typically rents for about \$18,000 a year, though there is evidence that increasing the scale of production demands alternatives.¹⁶ Grow-ops arise (in part) because they have a very quick time to market compared to natural marijuana crops that have an annual cycle.¹⁷

The equipment necessary to run a grow-op includes supplies, lights, fans, seeds, and miscellaneous other materials. For a 100-plant operation,

12 For example, Caulkins (1994) finds a similar relationship for cocaine prices and quantities in the United States.

13 A relatively new phenomenon is that grow-ops are being found with "continuous cycle" harvesting. That is, there is a "circle" of plants with one at each stage in the productions process. Such a model takes more hands-on work, since one task or another has to be performed more frequently, but if the grow-op is busted by competitors, then there is much less market-ready product available. A clear trade-off is being made.

14 In addition, there are often several smaller buds, but I have not seen estimates of how many or how large they are.

15 Based on 2,089 BC observations, the THC (delta-9-tetrahydrocannabinol) content from 1995-1999 was 6.5, 6.9, 6.6, 7.1 and 7.4 percent (Ladds, 1999).

this amounts to about \$10,000.¹⁸ The electricity costs about \$2,500 per year. Many growers gladly pay for it. Others fear that the hydro company will notice the extensive residential use of electricity and might investigate.¹⁹ Still others simply steal the electricity.

Similarly, the grower cannot set up a generator in the back yard or on a balcony. It will make a conspicuous noise and will alert thieves who would help themselves to the maturing buds, an activity known as “grow-rips.” Obviously, there is no public recourse if you, as a grower, are burglarized. Nor can you carry theft insurance for the valuable crop. This may also help to explain the boom in “guard” dogs in some parts of British Columbia’s Lower Mainland as well as protection provided by organized crime for selected operations (Howell, 2002).

Ignoring electricity costs, table 3 reports that the total material cost of the operation is about \$28,000. Obviously what is missing is the labour cost. At a minimum wage of \$8 per hour over a 24-hour day to provide for constant security,

the cost of labour could add another \$70,000 to expenses. On the one hand, unlike the standard minimum wage paid and received, this is tax “free,” and even the most intensively farmed grow-op does not really need 24 hour care all the time. Consequently, this is a *very* high estimate of labour costs, and means that we will tend to understate the profitability of grow-ops. On the other hand, there is always the possibility of violence associated with grow-ops, which adds a premium to the usual market wage. For obvious reasons it is difficult to document labour usage and remuneration patterns systematically.²⁰

How much does such an operation produce?

Although most estimates of production are speculative or designed to serve a particular purpose, Plecas *et al.* (p. 35) find that the average number of plants discovered in all marijuana grow-op busts around the province has been on the increase. Across British Columbia from 1997 to 2000 the average number of plants seized rose from 140 to

16 Recent busts reported in Vancouver newspapers suggest that new houses worth \$300,000 to \$400,000 are being purchased and used for a year or so for such purposes. Large-scale production at greenhouse operations in more rural settings has also been found recently. This suggests that the scale of grow-ops is increasing and is not inconsistent with observations by Plecas *et al.*

17 A quick introduction to marijuana grow operations is available to anyone who wishes to peruse the Internet. The detail and apparent sophistication of the technology is voluminous. The police have provided tips for spotting grow operations: http://www.city.richmond.bc.ca/emergency/police/grow_operations.htm. There is information on the types of lights and programs necessary to maximize indoor yield by following the links at sites such as: <http://www.cannabislink.ca>; or <http://www.cannabisnews.com>. Easier yet, try typing something like “marijuana growing” into a search engine.

18 This is typical in the sense that even though the average size is higher than 100 plants per grow-op, most operations still remain small, and the high average is due to some really large and spectacular busts of thousands of plants. There are relatively few of these in the data. As a result, although I call this typical, it is a statement about most likely to be observed rather than mean number of plants. The average number of plants found in grow-ops is rising.

19 Interestingly, there is irritation among some in law enforcement that the electricity supplier is not active in identifying likely grow-ops unless they fail to pay their bills. If they fail to pay, or are found bypassing the meter, then the electricity company expects prompt action by the police since it is a theft in progress.

20 Sharecropping (in which the financier and the grower split the crop) also is known. Some informal reports to the author suggest a 50-50 split is common.

Table 3: A Calculation of Vancouver Grow-ops

Revenue	Numbers	Comment
Number of plants	100	Near both mean and median in 161 busts VPD* busts from 1994-1999
Number of seasons	4	From 6 to 12 weeks
Total number of buds produced during one year	4 x 100 = 400	Each bud is roughly 100 grams
Total weight in kilograms	13.3	(400 x 100) x 1/1000 to account for dry weight
Price per pound (bulk)	\$2,600	See table 2 (2.2 pounds per kilo)
Annual value of sales	\$76,000	This is bulk (rounded)
Costs	Numbers	Comment
House rent	\$18,000	Assumes full year occupancy
Supplies	\$4,000	Fans, lights, containers, seeds, etc.
Wages (implicit or explicit)	\$2,000	Care and clipping of plants
Electricity**	\$2,500	Could be less if operator steals power
Operating Cost	\$24,500	(\$1,500 per pound)
Share to operator	\$38,000	50% of final product
Net revenue to investor*	\$13,600	50% of revenue less operating cost
Return on a dollar of cost	55%	(All figures rounded)

*Source: Wicksteed (2002) provides data about the size distribution of busts and the cost of supplies. House rents are a casual average from local newspapers. Plecas *et al.* provide estimates of the size of buds.

**Electricity at 0.57 cents per kWh implies an annual cost of \$2,500 for lighting this operation. More generally this amounts to roughly \$8.50 per plant.

180. There are apparently more operations, and an apparent increase in size of these operations.

A rough calculation of a marijuana grow operation

To get a sense of the numbers for a typical operation, assume a grow-op has 100 plants. This puts it in the “modest size for commercial use” category. Harvest revenue comes from 13.3 kilograms of marijuana sold in pound blocks out the back door at \$2,600 per pound.²¹ This amounts to slightly more than \$19,000 per harvest. Since there are four harvests per year (on the conservative side), gross revenue is about \$76,000. Even if

costs are about \$24,500, and the final sales are split equally with the operator, the net rate of return on invested money is potentially very high. The 100-plant grow-op makes around 55 percent return for a year’s worth of activity using the most conservative assumptions.

But the rate of return is not *really* 55 percent. There is the chance that you will be busted—either by your colleagues on the wrong side of the law, or by the police. If 10 percent of grow operations were busted by police, competitors, or thieves, then the expected annual rate of return is about 40 percent.²² This is still a fine rate of return if you can get it, but there are clearly risks in the busi-

21 This may be a little high currently, but see table 2. In discussing this figure with British Columbians who claim to know, they suggested that they were not able to get more than \$1,900 per pound. This is casual empiricism and serves to alert the reader to the gross uncertainties of any estimates. Consequently, in estimating the number of marijuana grow operations (below), it is appropriate to use a wide range of assumptions.

ness that are not about business. Interestingly, the observation that there are additional risks and our knowledge of the returns to the marijuana

grow-op business provide a mechanism for determining the number of marijuana grow-operations. This is discussed in the next section.

How Many Grow-Ops Are Out There?

One of the enduring problems facing anyone interested in the illegal, or “black,” or even gray economy, is to derive an estimate of the underlying level of total activity from the sample of those that are detected. There are problems in doing this. A few might be catalogued under some broad headings:

- sample selection—only the unlucky or the least capable are caught;
- varying intensity of effort on the part of the authorities—more police “fishing” means a higher catch, at least initially; and
- an uncertain feel for what the alternatives are facing the agents who are thinking of going into illegal production—can they find a remunerative line of work in the legal sector, or are their alternatives really all about illegal alternatives to, say, marijuana production?²³

This section proposes one calculation method to infer the number of grow-ops in British Columbia. More generally, it is a technique that could be used in a number of situations both current and historical. Although one may disagree in detail with *every* aspect of the analysis, it also provides a target to classify the underlying variables that may be important to any analysis of uncounted activities.

The approach

The underlying characterization is of the grow-op as a profit maximizing activity in which the value of output less costs, relative to the value of assets, yields the rate of return to assets. For each crop of a grow-op, all costs are fundamentally variable, so that we can write the rate of return as relative to costs.²⁴

If the industry is in equilibrium, then the return on capital (or costs) is equated to the rate of return

22 That is, with only a 90 percent chance of realizing your sales, the expected rate of return becomes:
 $((0.9 \times (\frac{1}{2} \times \$76,000) - 24,500) / 24,500)$.

23 There is still plenty of disagreement about the number of marijuana grow operations in British Columbia. Mark Hume of *The Globe and Mail* of January 12, 2004 reports: “Police estimate 2,000 to 3,000 grow-ops are producing BC bud in Greater Vancouver” (p. A2). On January 31, 2002, however, the *Vancouver Sun*’s Scott Simpson reports that the head of the Vancouver drug squad, Inspector Kash Heed, “could not estimate the number of growing operations in Vancouver, but said the number for the Lower Mainland has been pegged as high as 15,000” (<http://www.mapinc.org/mjcn.htm>). Interestingly, on a different page of the January 12, 2004 *Globe and Mail*, Peter Cheney reports police estimates that there are now 15,000 marijuana grow operations in Ontario (p. A6).

24 The alternative is to assume that the capital is used for a number of crop cycles. This would have the effect of increasing the value of output relative to the asset base. Consequently, this assumption biases the return to growing marijuana downward. The “true” returns on invested capital are likely to be higher.

in other industries or activities on the margin. This is the key observation underlying the estimation of the total number of illegal activities. It is what links the unobserved illegal activity to the known, legal world.

More formally, we write the value of output, PQ (price times quantity) less cost, C , relative to the value of capital, or in this case, cost. This gives a rate of return to investment (cost) in a particular year.

Thus R is a return over costs and looks like:

$$1. \quad R = [PQ - C] / C$$

The value of output less cost is net income, $PQ - C$, during the year, and the return over costs is akin to the usual calculation of the rate of return to capital. If we believe that the industry is in equilibrium, about which more will be said later, then the return on capital (or costs) is equated to the rate of return in other industries or activities on the margin. Thus $R = R^*$, where R^* is the market rate of return.

Unlike the market, however, a grow-op includes ingredients of extraordinary risk not captured by legal market entities. Let us add a probability of getting caught²⁵ in a grow-op and consequently the risk of losing the entire crop. If the probability of getting caught is π , then the harvester has a $(1 - \pi)$ probability of being able to sell quantity Q at price P . Compared to a riskless sale, this lowers the return to any given investment.²⁶

$$2. \quad [(1 - \pi)PQ - C] / C = R^*$$

The left-hand side tells us that the harvester has a $(1 - \pi)$ probability of being able to sell quantity Q at price P . Compared to a legal sale, this lowers the return to any given investment. The investor is assumed to lose the costs, C , whether the crop can be sold or not.

The expected return is equated to the return that the investor can get in any other sector of the economy, R^* . In effect, we assume that the potential investor in the marijuana business is faced with two options: Our potential producer can invest in those activities that are legal and receive a normal rate of return of R^* ; or our potential producer can invest in a grow-op that includes an extraordinary risk of crop loss.

A refinement

The market rate of return, R^* , constrains the amount of investment in marijuana grow operations. If more and more people get into the business, eventually it will drive the return below that which could be made in other business activities. This limits the size of the sector. Symmetrically, if the return to marijuana grow-operations is higher than the return in other activities, this leads to more investment going to the marijuana industry, eventually driving the return toward the market average. This basic framework may not fully capture the essential constraints on an illegal activity. Do potential growers of marijuana view the market return on funds as relevant in assessing their alternatives? If one were loaning funds to a grow-op producer, the lender may insist on a risk premium associated with the loan so that the constraint associated with an equilibrium in the

25 In this context, "getting caught" includes being shopped by unscrupulous competitors, as well as having your crops catch fire, or simply be stolen by thieves. A tip apparently led to the discovery of a "massive" hydroponic operation in Barrie, Ontario, in the old Molson brewery—a site in plain view of Highway 400 (*The Globe and Mail*, January 12, 2004, p. A1, A6.) In Vancouver, police speculate that a marijuana grow-operation is invaded each day by competitors.

26 The investor is assumed to lose the costs, C , whether the crop can be sold or not.

marijuana growing business is not the market return, R^* , but a return that is risk-adjusted above those associated with legal investments. As a result, the cost of funds that this group faces carries a risk premium relative to that of legal investments.²⁷

This suggests an expression like 3 is relevant to the basic equilibrium:

$$3. \quad [(1-\pi)PQ-C]/C=R^*+\pi$$

which equates the expected return on the left-hand-side to a higher-than-legal-market return by an amount of the risk, π . Although the risk may not simply be additive, Appendix B derives a form that is consistent with 3.

Calculating the number of grow-ops

How does all this help with a calculation of the number of grow-ops in British Columbia?

We need to assume something about π . We assume that it is the risk of being busted by the police.²⁸ If we assume that only the police bust grow-ops, then we can develop a measure of the total number of grow-ops in the province.

To see this, recall what we “know” in this context.²⁹

- We know the price of the product (see appendix B)
- We know the quantity of product for each operation—or at least we know the average output of those that are busted.
- We know the cost of the operation, although there are a few nagging issues that make this a more speculative calculation than the other data.
- We know the market return on legal enterprises—although this can be argued, the range of variation is likely not to matter much as will become apparent in the calculation.
- Finally, we also have a measure of the number of operations that have been busted around the province.³⁰

These data are sufficient to calculate the number of grow-ops. To see this, first consider the variable, π . Since π is the probability of being busted, we can think of π as being the ratio of busts relative to the total, T , the (unknown) number of grow-ops:

$$4. \quad \pi=B/T$$

27 Note that this is not the same as another experiment: should a person participate in the legal or illegal market? In this case, clearly the decision is based on R^* .

28 It also should include any other risk associated with being illegal rather than legal, e.g., lack of resources for redress of theft, extras security, and the like. Underestimating the risk will underestimate the number of grow-ops.

29 In this context, “know” is speculative under the best of circumstances.

30 This, of course, is police busts. It should also include “busts,” or thefts, or any other event that reduces the ability to sell the final product on the left-hand side of the equation. As discussed earlier, some reports have marijuana “rips” at one a day in the Vancouver area alone. Consequently, these calculations that use only police data to estimate the number of marijuana grow operations are very conservative.

Table 4: The Effect of Different Assumptions for Estimating the Number of Grow-Ops in BC

Actual Police Grow-op Busts	Assumed Return to Legal Activities	Assumed Ratio of Value to Cost	Market Return is R*		When the Return is risk Ad- justed, R*+ π	
			Implied Total Number of Grow-ops	Implied Probability of being Busted	Implied Total Number of Grow-ops	Implied Probability of being Busted
B	R*	PQ/C	T	π	T	π
2,800	10%	5.0	3,590	0.78	4,308	0.65
		4.5	3,706	0.76	4,529	0.62
		4.0	3,862	0.73	4,828	0.58
		3.5	4,083	0.69	5,250	0.53
		3.0	4,421	0.63	5,895	0.48
		2.5	5,000	0.56	7,000	0.40
		2.0	6,222	0.45	9,333	0.30
		1.9	6,650	0.42	10,150	0.28
		1.8	7,200	0.39	11,200	0.25
		1.7	7,933	0.35	12,600	0.22
		1.6	8,960	0.31	14,560	0.19
		1.5	10,500	0.27	17,500	0.16
		1.4	13,067	0.21	22,400	0.13
		1.3	18,200	0.15	32,200	0.09
		1.2	33,600	0.08	61,600	0.05

Since we know the number of operations that have been busted by the police, B, everything is “known” (however imperfectly) except for T, the total number of grow-ops at risk. That is, we know P, price, Q, quantity and R*, the rate of return on legal economic activity.

Some manipulation gives us the following expression:

$$5. \quad \pi = B/T = \{[(PQ/C)-(1+R^*)]/[1+(PQ/C)]\}$$

or, finally, an expression for the total number of grow-ops:

$$6. \quad T = B \cdot [1+(PQ/C)]/[(PQ/C)-(1+R^*)]$$

So what do the numbers look like? To illustrate: Let B = 2,800³¹; let R* = 10%; let (PQ/C) = 5

$$7. \quad T = 2,800 \cdot [(1+5)]/[5-(1.10)] = 2,800 \cdot [6/(3.9)] = 4,308$$

Table 4 reports what the theory implies for the number of grow-ops in British Columbia using various assumptions about the ratio of the value of output to costs. From the estimates in table 3, the number of grow-ops would be between 10,500 and 17,500 depending on the approach to risk. In later sections I use the 17,500 figure as I believe it best characterizes conditions in BC.

31 This is the number of “founded” cases in 2000 in all of British Columbia (Plecas *et al.*, 2002, p. 27.)

One point needs reinforcing. These are estimates for the numbers of “bustable” grow-ops. By that I mean that the small operations of a few plants that are for personal use generally are not “busted.” The Vancouver Police busted 30 grow-ops with fewer than 50 plants over a period of several years. The average was 117 plants, with a median of 95 plants. The fewest seized in a grow-op bust were 25 plants, and the most seized were over 1,100 plants (Wickstead, 2000a). A reasonable interpretation of the data in the table is that for grow-ops over 25 plants, these are the total number of “bustable” operations implied.³²

How reasonable are these estimates? If the reader wants a general rule for thinking about this, consider: what fraction of grow-ops is likely to be discovered and busted by the police? Suppose the police are able to bust one-half of all grow-ops. With 2,800 grow-op busts in the year 2000, it means that there were 5,600 grow-ops initially. If the police bust only 10 percent of grow-ops, then we can infer that initially there were 28,000 grow-ops. Although certainly not definitive nor a substitute for analysis, readers should use their “ingenuity guided by experience” to form their own tentative estimate.

Some of the limitations of this calculation

There are a number of limitations inherent in this calculation. First, the number of busts known is not the same as the number of actual busts as seen from the producers. We use known police busts.

Clearly, if there are grow-rips by competitors or “colleagues,” then the effect is to underestimate the riskiness of the enterprise.³³ Thus, the numbers in the table will underestimate the number of grow-ops. This is because the total number of grow-ops is, by formula, proportional to the number of busts as seen by the growers.

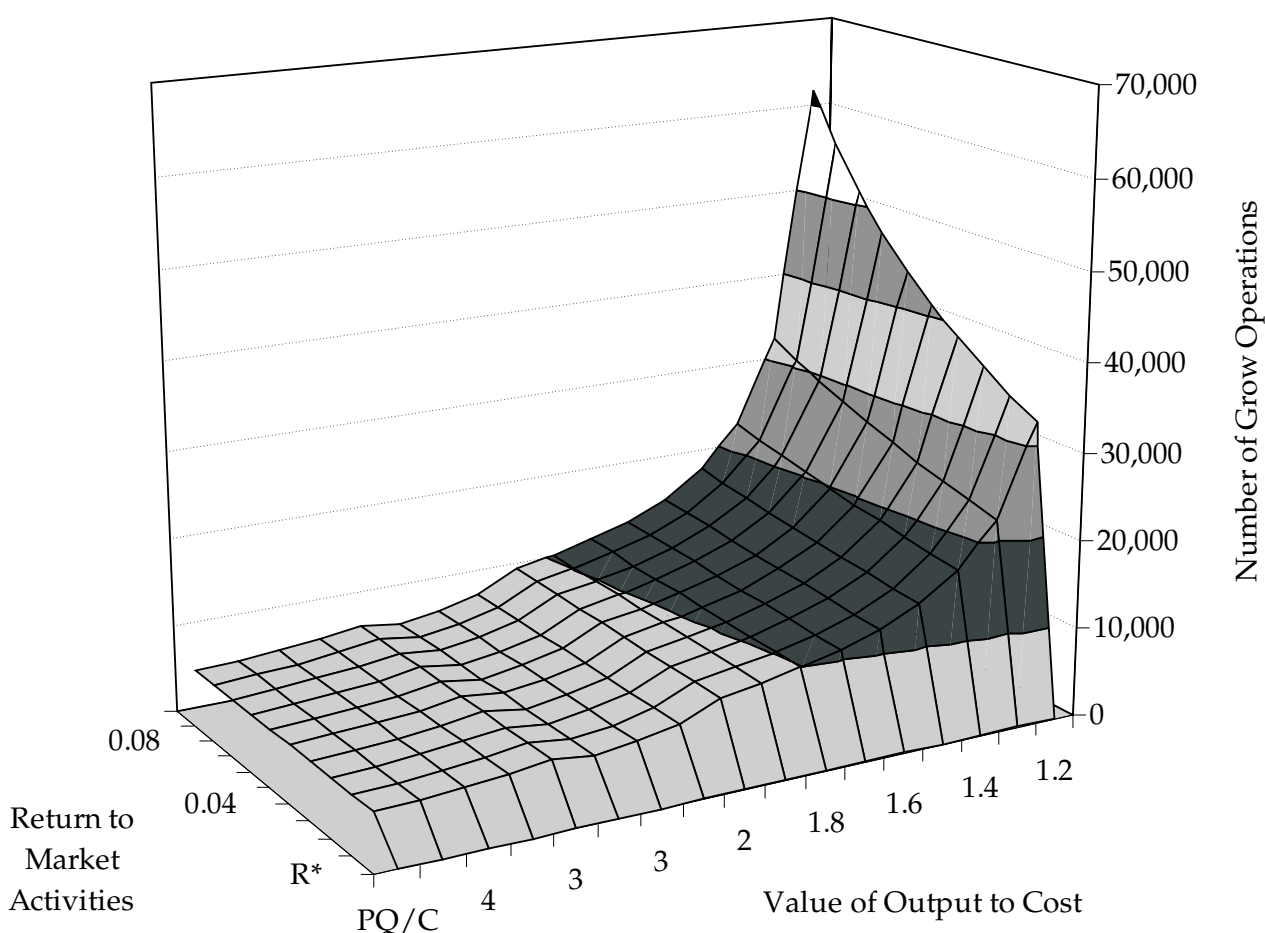
Second, increased enforcement implies increased numbers. Again, this is a consequence of the formula that requires the number of grow-ops to be proportional to the number of busts. The reason that the enforcement “doesn’t matter” in the calculation is that the only thing assumed to be important to the producer is the actual number of busts relative to the total that gives rise to the risk. Yet most of us would be concerned that the calculated number of grow-ops should not increase merely with increased enforcement. This is a limitation of the model in the text that must be addressed. The standard way to solve the problem (that is akin to simultaneity in enforcement and production) is discussed below in appendix C, “A Richer Model.”

Because of the many uncertainties associated with every ingredient of the formula, we want to look at a wide range of assumptions including different assumed rates of return available on outside investment. Figure 1 displays the patterns associated with a range of values relative to costs and rates of return. The ratio of value of sales to cost, PQ/C , is plotted on the “x”-axis; the measure of “ R^* ”, the market return on investment outside the industry (assuming additional risk at-

32 However, Plecas *et al.* report that in province wide data, there is at least one case in which a single plant was seized! For 1997-2000 they report the average number of plants seized increasing from 141 to 180 during the period.

33 According to Plecas *et al.*, 2002, table 2.6, about 57 percent of all files being opened for grow-ops comes from Crimestoppers or anonymous tips. These leave plenty of room for competitors as well as offended members of the general public to identify grow-ops. “Grow-rips” as they are known, appear to be increasing. The police are responding to more calls for break-ins that are for the purpose of stealing marijuana, but the thieves have, by mistake, targeted non-marijuana growing houses (O’Brian, 2004; *Vancouver Sun*, Jan. 20, 2004).

Figure 1: Number of Marijuana Grow Operations as a Function of the Value of Output and Rate of Market Return



tached) is plotted on the “y”-axis; and the “Number of Grow-ops” is along the vertical axis. Although not plotted, the value of π , the probability of being busted, like T , is a calculated value.

Estimates of the total number of grow-ops applied to the regions of British Columbia

The most recent characterization of the number of grow-ops in British Columbia is to be found in Plecas *et al.*, 2002. For the year 2000 they suggest a

figure of 2,808 incidents of busted grow-ops in British Columbia.

We can see the implications of the model by region if we are willing to go with a particular value of the rate of return and the value of output relative to costs. Table 5 takes model 2 in which the rate of return includes an explicit risk premium, and uses the value 1.5 for the ratio of the value of output relative to costs.

Although interesting, because they indicate the likely scope of the marijuana industry geographically, yearly variations in table 5 are

Table 5: Implied Number of Grow-ops by Region

District	1997	1998	1999	2000
Greater Vancouver	2,975	4,188	5,625	8,394
Fraser Valley	775	1,025	1,394	1,756
Squamish-Lillooet	81	106	106	206
Mainland/Southwest	3,831	5,319	7,125	10,356
Nanaimo	613	725	731	913
Comox-Strathcona	456	563	731	888
Capital	563	450	738	619
Cowichan Valley	275	519	581	406
Sunshine Coast	50	219	213	156
Alberni-Clayoquot	88	113	119	113
Powell River	—	100	94	119
Mount Waddington	38	63	75	56
Vancouver Island/ Coast	2,081	2,750	3,281	3,269
Thompson-Nicola	294	575	519	506
Central Okanagan	238	350	506	519
Northern Okanagan	169	313	294	500
Okanagan-Similkameen	175	231	269	344
Columbia-Shuswap	156	156	206	225
Thompson/Okanagan	1,031	1,625	1,794	2,094
Fraser-Fort George	144	175	269	406
Cariboo	144	181	163	381
Cariboo Overall	288	419	431	788
Central Kootenay	200	281	475	388
Kootenay Boundary	81	238	244	131
East Kootenay	88	125	138	181
Kootenay Overall	369	644	856	700
Kitimat-Stikine	63	75	75	156
Skeena-Queen Charlottes	44	38	31	13
Central Coast	6	—	—	6
North Coast Overall	113	113	106	175
Bulkley-Nechako	81	44	50	119
Stikine (region)	—	6	13	—
Nechako Overall	81	50	63	119
Peace River	25	31	69	44
Northern Rockies	—	6	13	6
Northeast Overall	25	38	81	50
Province Overall	7,819	10,956	13,738	17,550

Assumptions: Ratio of Sales to Costs (PQ/C) = 1.5

The Rate of Return to Enterprise: $R^* = 10\%$

π , the Probability of being Busted, is 16%

The Opportunity Cost for the grower is $(R^* + \pi)$

Table 6: The Export Consequences of Different Estimates of the Number of Grow-Ops

Value of Output to Cost Ratio* PQ/C	Number of Grow-Ops*	Marijuana Production in British Columbia (metric tons)**	Marijuana Exports*** from British Columbia (metric tons)	Retail Bulk Value of Exports**** (Billions of dollars)
5.0	4,308	102	72	0.36
4.5	4,529	108	77	0.39
4.0	4,828	115	84	0.42
3.5	5,250	125	94	0.47
3.0	5,895	140	109	0.55
2.5	7,000	166	136	0.68
2.0	9,333	222	191	0.96
1.9	10,150	241	211	1.05
1.8	11,200	266	236	1.18
1.7	12,600	299	269	1.34
1.6	14,560	346	315	1.58
1.5	17,500	416	385	1.93
1.4	22,400	532	502	2.51
1.3	32,200	765	735	3.67
1.2	61,600	1,464	1,433	7.17

*See table 4 for the basis of the estimates.

**Assume 33.3 grams per plant and 180 plants per grow-op (Plecas *et al.*), and 4 crops per year.

***British Columbia exports are BC production less BC consumption. National consumption from table 1. BC consumption is 13 percent of the national total, adjusted for consumption per user or 30,600 kg.

****Assumed price of \$5,000 per kg. (see table 2).

driven entirely by the number of busts in each region. Increased enforcement arising from local conditions are much more likely to have an impact in a region than they are in the overall scheme of things.

Potential British Columbia marijuana exports

Using the estimate of the number of grow-ops from table 4 will also allow an estimate of the total quantity of marijuana grown in British Columbia. Contrasted with the implicit demand of table 1, it gives a rough and ready sense of the level of exports by the industry. In table 6 the first column reports different possible output to cost ratios that are reasonable in assessing the British Columbia marijuana industry. Each of these num-

bers gives rise to an estimate of the number of grow-ops in the second column. The third column derives the implied quantity of production (measured in metric tons) associated with each of the estimates of the number of grow-ops. Since exports from British Columbia are the quantity of production less the amount absorbed domestically within the province, the estimate of the quantity of exports is generated by using the production figure of column four with the consumption from table 1 adjusted for the size of the province of British Columbia.

The value of exports is measured at an assumed price of \$5,000 (Canadian) per kilogram. This is a bulk value since it is purchased and shipped in quantity rather than cigarette by cigarette. Of course the value of the exports at final sale will

Table 7: The Value of Grow-op Marijuana Relative to GDP in British Columbia

	1997	1998	1999	2000
BC's Gross Domestic Product (GDP) (billions of dollars)	114.4	115.6	120.6	130.8
Grow-op Sales as a Percentage of BC GDP	1.1%	1.6%	2.4%	2.8%

depend upon the prices in the US and will be substantially greater.

A reasonable supposition, given that British Columbia absorbs slightly more than its 13 percent of Canada's population, is that British Columbia's consumption is roughly between 21 and 54 metric tons (from table 1). The quantity of output is vastly greater: between 100 and 1,460 metric tons.³⁴ It is reasonable to conclude that most of the British Columbia crop is exported to the United States or in some measure to the rest of Canada. The estimate that appears to me to be the most reasonable (albeit tentative) generates exports of nearly \$2 billion in year 2000.

The size of the British Columbia marijuana industry

To put this into some kind of perspective, table 7 measures the value of production of marijuana from grow-ops at between 1 percent and 2.8 percent of British Columbia's Gross Domestic Product (GDP) that was roughly \$130 billion in 2000.³⁵

However useful this is insofar as it scales the cost of domestic production by comparing the wholesale value of BC's marijuana crop to GDP, the ratio is inflated since we are using final sales and not the value-added of the marijuana grow industry.³⁶

To measure the value of the marijuana crop at final sale prices properly, we need to use the prices associated with the quantities that are sold on the retail market: the gram, ounce, pound, kilo etc., amounts since prices per unit vary by quantity. Similarly, prices vary by region and by type of product. Using a statistical analysis of price per gram as a function of quantity sold, region, urban-rural, and other variables, we can construct a retail price model for sales. If we were to assume that marijuana were sold by the pound, then in British Columbia in the year 2000, the retail price is about \$2,600 in urban British Columbia. If we were to assume that marijuana was sold by the ounce, then it would be worth about \$4,100 per pound on average. By the cigarette, a pound would sell for \$7,800.

34 That is, with 7,000 to 17,500 grow-ops each producing about 13.3 kilograms annually, the total harvest is between 168 and 420 metric tons. Specifically, 33.3 grams per plant x 180 plants x 4 crops per year = 24 kilograms per year per grow-op.

35 Sales to the general public are assumed to be in the ounce range. In any case, table 2 permits the reader to calculate his or her own valuation.

36 Since GDP measures value added rather than final sales, the size of the marijuana industry appears too large relative to other industries. Rather than try to "guild the bud" by further refinements of the value added of the marijuana grow operations, the comparisons should be taken for what they are: an effort to get some sense of the overall scale of economic activity in the marijuana industry in BC. Obviously we can construct a value-added measure consistent with our representative grow-op of table 2, but this is placing a great deal of weight on a rather speculative calculation.

Table 8: The Value* of the BC Marijuana Harvest by Region Measured at “per Cigarette” Values (in millions of dollars)

District	1997	1998	1999	2000
Greater Vancouver	950	1,328	2,319	3,422
Mainland/Southwest	1,224	1,687	2,937	4,222
Vancouver Island/Coast	665	872	1,353	1,333
Thompson/Okanagan	329	515	740	854
Provincial Total	2,497	3,474	5,664	7,156

*The assumptions underlying quantities for this table are the same as those for table 5.

So what are the bounds to a measure of retail value of sales? To answer this we need a measure of the price of what is sold. Significantly, the unit in which the marijuana is sold is an important consideration. From our estimates in table 2 and the supporting discussion in appendix A, we know the relationship between price per gram and quantities sold—be it a fraction of a gram, or by the kilo, and various quantities in between.

To carry this to the extreme, suppose that the British Columbia producers’ crop was to be valued at the per cigarette street cost: the smallest and most expensive retail unit. Table 8 gives a sense of the values.

Table 8 reflects the retail value of the product from each of British Columbia’s regions. The producers do not, of course, receive these amounts. Like many agricultural products, the “middle-man” receives much of the difference between the final sale price and the original producer. Transportation, packaging, marketing, and risk of confiscation by various compet-

itors and law enforcement are all part of the difference.

Although the values do not reflect the actual receipts by the growers in each region, the numbers do reflect an estimate of the contribution to ultimate street sales made by each region should the final product be sold at British Columbia retail prices in British Columbia. Estimating the “true” street value of the actual product would necessitate knowing exactly where final consumption took place: both at home and in the United States.³⁷

Although many underground activities have consequences for society ranging from alcohol prohibition of the 1920s to drug prohibitions today, economists have had a difficult time in describing the extent of production. The British Columbia marijuana industry is a good place to begin to study this problem. While decentralized, the characteristics of the grow-ops are relatively well known, and there is a considerable volume of product, much of which heads to the US.

37 There is a substantial marijuana trade with the US.

Why Does it Happen in British Columbia?

Although current federal initiatives to decriminalize the possession of small quantities of marijuana may change the traditional location of marijuana production, one of the enduring, frequently-asked questions is why it is that marijuana cultivation and consumption have traditionally taken place more openly in BC than elsewhere in Canada. Is it British Columbia's *in-door* climate? What is different on the Coast?³⁸

Although there is no simple answer to such a question, several statistical observations may bear on the issue. One outstanding statistic is that possession incidents are not "cleared by charge" as frequently in British Columbia as they are in Canada's other provinces.³⁹ Although there are differences between BC and the rest of Canada for charges with respect to other drugs, the difference is greatest with respect to marijuana. Second, a look at the pattern of arrests and penalties facing marijuana growers in Vancouver also gives a sense of the consequences for (some) marijuana growers.

Table 9 reports drug incidents and charges for 2001. Only 13 percent of possession offences in BC are cleared by charge. Elsewhere in Canada over 60 percent of possession offences are cleared by charge. Even though BC has nearly twice as

many offences relative to population as the rest of Canada, clearing by charge is one-fifth of that elsewhere in Canada. The reasons for such a pattern may depend upon the courts, the prosecutors, or the police, but it is surely indicative of a difference in perspective at some level in the enforcement of the law.⁴⁰

Is clearing by charge the relevant data for explaining the size of the British Columbia marijuana industry? Are fines lower here than elsewhere? Probably not, but why this industry has been so successful in British Columbia and less so elsewhere remains a topic of serious interest. In that spirit, the next section considers the effect of being caught ("busted") in a marijuana grow-operation. Although I do not have comparative data on those caught for growing marijuana elsewhere in Canada, the kinds of punishments in British Columbia are consistent with a marginal level of deterrence.

What happens to marijuana growers?

Local conditions in British Columbia obviously play a role in the production of marijuana. If British Columbians really are producing the massive quantities of the drug that I have suggested, is-

38 Recent high-profile police busts in Ontario and Quebec make it clear that marijuana growing is no longer unique to British Columbia.

39 Actually, BC is far less likely to clear offences by charge than the rest of Canada for almost any drug possession offence. "Clearing by charge" means that a file is sent to Crown prosecutors for action on a criminal charge. Files can be closed in other ways if, for example, the person the police believe committed the crime has died or is being charged with a more serious offence on another charge.

40 The observation that BC does not often charge for marijuana possession (nor, for that matter, other drug possession), and yet the province has a particularly potent marijuana crop is a puzzle. Theory would suggest that if enforcement is very enthusiastic, then the crops would be small and of high potency. A less strict criminal enforcement environment would be expected to produce crops that are less strong and less intensively cultivated. BC appears to be the opposite.

Table 9: Drug Crimes and Drug Charges in Canada and British Columbia, 2001

Incidents Known to the Police	Actual Number in Canada	Actual Number in BC	BC as a Share of Canada	Incidents Cleared by Charge in BC	Incidents Cleared by Charge in Canada Net of BC
Heroin—Possession	504	367	73%	37%	80%
Trafficking	403	258	64%	74%	86%
Importation	58	13	22%	23%	22%
<i>Heroin—Total</i>	965	638	66%	51%	75%
Cocaine—Possession	5,478	1,744	32%	38%	82%
Trafficking	6,265	1,876	30%	70%	81%
Importation	490	53	11%	28%	36%
<i>Cocaine—Total</i>	12,233	3,673	30%	54%	79%
Other Drugs—Possession	3,982	675	17%	25%	59%
Trafficking	2,472	329	13%	43%	76%
Importation	1,302	231	18%	17%	14%
<i>Other Drugs—Total</i>	7,756	1,235	16%	28%	57%
Cannabis— Possession	49,639	11,757	24%	13%	62%
Trafficking	11,124	2,098	19%	62%	73%
Importation	739	203	27%	4%	21%
Cultivation	9,122	3,477	38%	27%	37%
<i>Cannabis—Total</i>	70,624	17,535	25%	22%	61%

Note: 2001 population: CANADA: 31,081,887; BC: 4,095,934. BC's population is 13% of Canada's.

Sources: Statistics Canada, *Canadian Crime Statistics 2001*, cat. no. 85-205 XIE, pp. 17 and 37.

sues of local law enforcement are clearly part of the cost of doing business. This section explores some of the consequences from fragmentary data arising from charges and convictions when grow-operation busts take place. Although the discussion is entirely in the context of Vancouver data, since Vancouver is an important source of British Columbia marijuana it is clearly a significant environment. The first subsection looks at the consequences for being caught by the Vancouver police in a marijuana grow-operation over the 1996-1999 period.⁴¹ A second subsection characterizes those who are caught to see whether the punishments meted out give any hint about their

effectiveness in deterring illegal marijuana grow operations. There are obviously many other important questions to be answered, such as connections with organized crime, and the financing and money laundering and trading for other illegal drugs, but the data are not able to inform us on these issues.

Sentencing those found guilty

Table 10 details the outcomes for those who were sentenced after being convicted of offences associated with the busting of marijuana grow-ops in Vancouver. The first column indicates the num-

41 The raw data for this section relies on Wickstead, "Who Wants to be a Millionaire?" It relates to Vancouver between 1996 and 1999.

ber of days of the sentence. The second column gives the percentage of all those convicted (for whom we have relevant data, as some were still awaiting sentencing), and the third column reports the cumulative percentage of those sentenced, up to and including the number of days indicated.

Most who were charged and convicted received no jail time. In table 10, the first row indicates that 55 percent of convictions received zero days' jail time. Five percent of those convicted received a single day in jail. Another 8 percent received sentences between 1 day and 31 days, and still another 8 percent received 60 days. Some 11 percent were sentenced to 90 days. Sentences for the remaining 11 percent were spread out from 120 days to 540 days.

A number of ingredients go into sentencing. For the data available, the number of prior convictions (of any type) and the size of the operation in which the convicted person was caught appear to be positively associated with the length of the sentence, although it is clear that much more than those factors must influence sentencing.

Statistical analysis reveals that an additional prior conviction will increase the length of the sentence by on average, a little over three and one-half days.⁴² Similarly, the value of the grow-operation affects sentencing. A \$100,000 increase in the imputed value of the grow-op tends to add over 16 days to sentencing. However, what is equally interesting is that these two variables—prior convictions and the value of the operation—account for only about 16 percent of the explanation of the length of sentence. “Other factors” explain the length of sentences associated with marijuana grow-op busts. Whether this has to do with the

**Table 10: Sentenced Jail Time
for Those Convicted in
Marijuana Grow-Operations**

Days	Percent Sentenced	Cumulative Percent
0	55.3	55.3
1	4.4	59.6
30-46	7.9	67.5
60-61	7.9	75.4
90	11.4	86.8
120	1.8	88.6
150	0.9	89.5
180	6.1	95.6
240	0.9	96.5
270	0.9	97.4
540	2.6	100.0
Total	100.0	100.0

Note: 114 observations.

Source: Wickstead, 2000a.

judge in whose court the case is heard, the prosecutor who works the case, the defense counsel who defends, or specific details of the case not captured by our data, clearly more research has to be done to reach an understanding of the reasons for the observed durations of sentences.

As might be expected, cultivation and drug trafficking were the majority of offences for which there were convictions. Table 11 indicates the range of days for those convicted of cultivation. One half, 50 percent, received no jail time. Two received 540 days. All but a handful received 90 days or fewer as a sentence. Of course not all these days are actually spent in jail since after one-sixth of a sentence, roughly, a convicted person is eligible for parole, and days in jail before conviction count for two days served after conviction.

⁴² See appendix E for the statistical details of the analysis.

Table 11: Days Sentenced for Cultivation Offence

Days of Sentence	Percent	Cumulative Percent
0	50.0	50.0
1	6.0	56.0
30-59	8.4	64.3
60-61	9.5	73.8
90	13.1	86.9
120	2.4	89.3
150	1.2	90.5
180	6.0	96.4
240	1.2	97.6
540	2.4	100.0
Total	100.0	100.0

Note: 84 observations.

Source: Wickstead, 2000a.

Outside of the loss of your equipment and product, how important are the personal costs for having been convicted in a marijuana grow operation dismantled by the Vancouver Police Department? Who are some of the people who are growing marijuana and are they deterred from returning to the business? To explore this issue we can look at some of the current producers' past run-ins with the law. What do their criminal records reveal?

Time between convictions

Although charges are not the same as convictions, past convictions and current charges provide their own feel for the drumbeat of suspect economic activity in the marijuana trade. Figure 2 plots the histogram of the days between charges for those apprehended in current grow-ops. Prior

charges were varied, although many relate to marijuana.

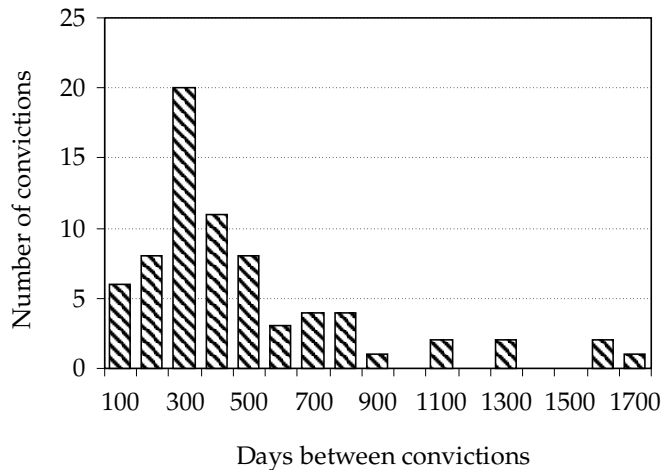
The distribution in figure 2 (reported in the legend) shows that the average time between convicted offences is about 14 months. In the figure, the horizontal axis shows the number of days between convictions. The vertical axis shows the frequency with which each number of days between charges is observed. The median is 11 months (328 days). This means that as many are charged in under 11 months as after 11 months. So among those with more than a single arrest, if charges are leveled this frequently, it is reasonable to suggest that whatever it is that many of these people are doing, they are continuing to do it!⁴³ From the point of view of an ongoing business, court time, or a charge, are simply part of the costs of doing business.

This sense is heightened by the data in table 12 that reports the outcome of all the charges for which data are available about those who were charged in the Vancouver police busts, many who have had multiple incidents in the past.

The first column of table 12 reports the number and proportion of all those who are currently charged with running a grow-op (or who face other charges arising from the arrest) and who have been convicted in the past. Of those now charged, about 70 percent were convicted and only 3 percent acquitted. Twenty-two percent had charges stayed with four percent discharged or dismissed.

Among the 670 convictions, there were 237 fines imposed (a little over a third of those convicted.) These fines averaged \$1,167. To put this into per-

43 Two observations were excluded as the time between charges was 4,500 and 5,000 days. These were well above any other observations. The data in the text use a cutoff of 2,000 days. The mean for the whole sample, including the two very high observations, was 551 days.

Figure 2: Days Between Convictions

spective, with only 100 plants, we saw about \$170,000 per operation in sales. The effective fine is far less important than having to set up all over again in another house. Recall that the equipment costs over \$10,000 and that with the bust, the producer lost the last crop, seed, and house lease.⁴⁴

Restitution is theoretically a tool that can be used to undo the damage of the grow-op. Destruction of a house, damage to power connections, and miscellaneous damage to other facilities are all the types of things eligible for restitution. What is

the record? Of the 167 cultivation cases, 11 involved restitution. These had a mean of about \$3,500. Of the 167 cases, 45 paid fines for which the average \$2,550. Only two fines were over \$6,500. Compared to the rewards of growing marijuana, these are not substantial amounts.

In summary

Marijuana production in British Columbia is substantial. Based on Vancouver data, a third of those who are caught are repeat offenders while two-thirds are first-time offenders. The penalties for being caught growing marijuana do not appear to be particularly stringent, and repeat offenders appear to average being caught marginally less than once a year. Fines appear to be modest and not sufficient to deter the behaviour. It is difficult to evaluate a policy that induces police to assign resources to catch nearly 3,000 grow operations a year, yet treats offenders to what must be seen as relatively minor punishment. These punishments do not seem to prevent recidivism. As argued in earlier sections, it is too profitable to prevent new people moving into production and to prevent old producers from rebuilding.

Legalization in Canada: Suppose We Tax it Like Other Sins?

What kind of money are we talking about if we try to reduce the crime and punishment associated with marijuana? Although there are many issues associated with the full or even partial legalization of marijuana, one of the most important is how much the demand for marijuana

changes when the price changes. Measuring the demand for legal products is hard task, but it is doable, and forms core employment for legions of economists. For marijuana, an illegal product, it is a more difficult job and impossible to do directly.⁴⁵ Fortunately, some issues can be ad-

⁴⁴ In a case I recently observed, the convicted grower asked the judge in all innocence, "Do you want that in cash?" causing all in the courtroom to shake their heads.

Table 12: The Result of Past Charges of those Currently Charged in Busts of Grow-ops

	Convicted	Stayed	Acquitted	Discharged	Dismissed	Fines
Number	670	212	26	21	23	237
Percent of charged	0.70	0.22	0.03	0.02	0.02	
Percent of convicted						35
Average fine of those fined						\$1,167

Source: Wickstead, 2000a.

dressed without detailed knowledge of the elasticity of demand.

Crude estimates in a revenue “switching” regime

Based on the grow-op data, for an investor we have assumed relatively high costs of around \$62,600 to produce, conservatively, 400 plants per year. That works out to \$156 per plant, and a plant produces 33.3 grams for a production cost of \$4.70 per gram.⁴⁶ A gram makes anywhere from one to three cigarettes. So today, *with the substance illegal*, we are looking at a per-cigarette wholesale price of \$1.60 to \$4.70 as opposed to the current “retail” price of \$8.60 per half gram.⁴⁷ This is still more expensive than tobacco, but then the tobacco industry has had a head start on mass production techniques, and by including very expensive labour costs, these are extreme

assumptions about the production costs of marijuana.⁴⁸

What about tax revenue? If we substitute a tax on marijuana cigarettes equal to the difference between the local production cost and the street price that people currently pay—that is, transfer the revenue from the current producers and marketers (many of whom work with organized crime) to the government, leaving all other marketing and transportation issues aside we would have revenue of (say) \$7 per cigarette. If you could collect on every cigarette and ignore transportation, marketing, and advertising costs, this comes to over \$2 billion on Canadian sales⁴⁹ and substantially more from an export tax, and you forego the costs of enforcement and deploy your policing assets elsewhere.⁵⁰

Notice that we have merely substituted government taxation for the premium on illegality. We

45 Appendix F reviews some approaches to an estimate of the demand for marijuana.

46 To make the point that these “estimates” are fraught with uncertainty, I will round the numbers ruthlessly.

47 Contrast this with the current price of tobacco cigarettes that sell for about 24 cents of which 9 cents is production and distribution. Tax makes up the difference.

48 In the long run, the cost of producing both tobacco and field marijuana is likely to be similar since both are weeds amenable to cultivation. A pound of tobacco wholesales for about \$3 Canadian a pound (between \$1.75 and 2.00 per pound US depending on the grade. See <http://www.ers.usda.gov/publications/agoutlook/Jan1999/ao258b.pdf>).

49 That is, from appendix table 1A, year 2000 low weight is 160,000 kg, or 160,000,000 grams. Assume .5 grams per cigarette or 320 million cigarettes. At a cost of approximately \$1.60 per cigarette, available revenue (plus transport and marketing that are assumed to be negligible) is 320 million cigarettes x (\$8.60 - \$1.60) = \$2.24 billion.

have not changed anything else. We have kept the price the consumer pays the same, and we have not altered the structure of production. We would still grow marijuana in “flower pots” except now it would be in the open and taxed like any other commodity at the retail level.

Importantly, this approach has the effect of transferring to the government revenue currently received by illegal producers as reward for their cost of production and risk.⁵¹ Unless we wish to continue to transfer these billions from this lucrative endeavor to organized crime, this policy should be considered. Not only would we deprive some very unsavory groups of a profound source of easy money, but also resources currently spent on marijuana enforcement would be available for other activities.

Advanced production techniques

If we were to assume that the wholesale price of marijuana would fall if it were legalized, since it would become cheaper to produce with proper mass production techniques—remember the difference between gin produced in hidden stills during Prohibition and modern distilleries—then both the cost and retail prices would most certainly fall. If we assume that the elasticity of demand is 0.6—a common estimate for tobacco and alcohol demand (see appendix F)—at

the current price, then dropping the price from \$8.60 to \$0.10 per cigarette would increase the quantity consumed by nearly 60 percent, but less than in proportion to the fall in price. However, by increasing taxes, the \$8.60 per cigarette retail price can be maintained with an increase in government revenue of another few billion dollars. The simplest taxation arithmetic is basic. The government can transfer revenue from organized crime and other small producers to itself by taxing a legal product to the level consumers have already revealed they are willing to pay. There are questions about how we collect taxes on exports, and what would happen should the US retaliate against our legalization, but the basic argument would be the same: we affect no change in price, we only transfer the revenue from current producers.

As for those current producers who argue for legalization, recall the old proverb, “Be careful what you wish for; your wish may be granted.” Many of those who advocate legalization for pecuniary reasons are perhaps thinking primarily of the increase in demand associated with legalization.⁵² However, as with the transition from prohibition to legalization of liquor early in the last century, we may note that very few of the “ma and pa” stills are currently in operation. Although there is always room for home and boutique production, large, sophisticated industries would quickly supplant local suppliers of marijuana with a corresponding decrease in costs.

50 Of course marijuana enforcement is only one aspect of drug enforcement and only one aspect of overall enforcement. There are economies of scope and scale that may well make this issue more complicated. Further, since we believe a lot of the product is sold in the US, it is unlikely that Canada would be able to collect much of this revenue.

51 In a wild flight of fancy, the government could even choose not to tax, but current policy obviously emphasizes taxes on “sin,” and in this, marijuana is no different than tobacco, alcohol, and gambling, and no doubt would be taxed accordingly.

52 The current Canadian proposal to decriminalize up to 15 grams of marijuana possession is an interesting exercise. It has the potential to increase demand without legalizing supply. If prices rise at all, it is likely that they will rise in the short run. In all probability, the supply response will be sufficiently great to keep the price stable in the medium and long term. Higher prices in the short run will only reward current producers—including organized crime. I hope these are merely unintended consequences of an inadequately thought out policy shift.

Conclusion

Marijuana is grown all over the world. In British Columbia (as in other provinces, notably Quebec and Ontario), it is a significant crop that fuels organized crime. Marijuana production appears to have been growing robustly during the past decade. Like many illegal products and services, it is difficult to measure the level of marijuana production. This is particularly the case when it is cheap to set up a grow operation and the market is substantial. In this paper I have reported a methodology for estimating the output of illegal production. Using estimates of marijuana growing in British Columbia based on this methodology, I have developed an estimate about the overall size of the local market and the implied level of exports.

The analysis reveals how widespread is the use of marijuana in Canada and how extensively it is produced in British Columbia. Consequently, the broader social question becomes less whether or not we approve or disapprove of local production, but rather who shall enjoy the spoils. As it stands now, growers and distributors pay some of the costs and reap all of the benefits of the multi-billion dollar marijuana industry while the non-marijuana-smoking taxpayer sees only costs. Alcohol prohibition in the US expanded organized crime in North America. Removing alcohol prohibition generated many problems, but none like those afflicting society in the days of Al Capone and his ilk. Removing the prohibition on marijuana production would permit society to replace today's gift of revenue to organized crime with (at the very least) an additional source of revenue for government coffers.

References

- Australian Institute for Health and Welfare. 1998 *National Drug Strategy Household Survey*. Available digitally at <http://www.aihw.gov.au/publications/health/ndshs98d/>.
- Bergerman, Roy (2000). RCMP Memorandum 2000-03-01. "Re: Marihuana Potency—Project SERRE II."
- Caulkins, Jonathan P. (1994). "What is the Average Price of an Illicit Drug?" *Addiction* 89: 815-819.
- Cheney, Peter (2004). "The Massive Secret Inside Barrie's Former Brewery." *Globe and Mail* (January 12).
- Clements, K.W. and M. Daryal (1999). "The Economics of Marijuana Consumption." Paper presented at the 28th Conference of Economists, Economic Society of Australia. Melbourne: La Trobe University (September).
- Daryal, M. (1999). *The Economics of Marijuana*. Unpublished BEc Honours thesis. University of Western Australia.
- (2002). "Prices, Legalisation, and Marijuana Consumption." Available digitally at <http://www.econ.ilstu.edu/UAUJE/PDF's/issue2002/mertdaryal.pdf> (Accessed May 2003).
- Centre for Addiction and Mental Health (1999). *Ontario Student Drug Use Survey*. Toronto: Centre for Addiction and Mental Health.
- El Sohly, Mahmoud (2000). "Potency Trends of Delta9-THC and Other Cannabinoids in Confiscated Marijuana from 1980-1997." *Journal of Forensic Science* (Jan.) 45(1): 24-30.
- Grossman, Michael, Frank J. Chaloupka, and Ismail Sirtalan (1998). "Empirical Analysis of Alcohol Addiction." *Economic Inquiry* (January): 39-48.
- Howell, Mike (2000). "Pot Growers Mistreating Guard Dogs" *Vancouver Courier* (January 14) Available digitally at <http://www.vancourier.com/013102/news/013102nn4.html>.
- Hume, Mark (2004). "Tangled Police Investigation Rattles BC's Liberal Party." *Globe and Mail* (January 12).
- Ladds, John (1996). RCMP Memorandum 1999-06-28. "Marihuana Quantitation Report."
- New Brunswick Student Drug Use Survey 2002 *Highlights Report*. Available digitally at <http://www.gnb.ca/0378/pdf/StudentDrugUseSurvey2002ENG.pdf>.
- Nisbet, Charles T. and Firouz Vakil (1972). "Some Estimates of Price and Expenditure Elasticities of Demand for Marijuana Among UCLA Students." *Review of Economics and Statistics* 54, pp. 473-475.
- Nova Scotia Student Drug Use 2002 *Highlights Report*. Available digitally at http://www.gov.ns.ca/heal/downloads/2002_NSDrugHighlights.pdf.
- O'Brian, Amy (2004). "Violent 'Grow Rips' on the Rise. Police Alarmed at Number of Break-ins by Suspects Looking for Pot." *Vancouver Sun* (January 10).
- Plecas, Darryl, Yvon Dandurand, Vivienne Chin, and Tim Segar (2002). *Marijuana Grow Operations in British Columbia: An Empirical Survey 1997-2000*. Vancouver: International Centre for Criminal Law Reform and Criminal Justice Policy.
- Prince Edward Island Student Drug Survey 2002 *Highlights Report*. Available digitally at <http://www.gov.pe.ca/hss/drugsurvey/index.php3>.
- Reuter, Peter (1996). "The Mismeasurement of Illegal Drug Markets: The Implications of Its Irrelevance." In S. Pozo, *Exploring the Underground Economy*. Kalamazoo, MI; Upjohn Institute for Employment Policies.
- Rhodes, William, Mary Layne, Patrick Johnson, and Lynn Hodik (2000). *What America's Users Spend on Illegal Drugs: 1988-1998*. US Government: Office of National Drug Control Policy (December).
- Saffer, H. and Frank Chaloupka (1999). "The Demand for Illicit Drugs." *Economic Inquiry* 37 (3): 401-411.
- Silverman, Lester P. and Nancy L. Spruill (1977). "Urban Crime and the Price of Heroin." *Journal of Urban Economics*. Vol. 4, pp. 80-103.
- Simpson, Scott (2002). "Sweep Nets 20 Marijuana Grow Operations." *Vancouver Sun* (January 31). Available digitally at <http://www.mapinc.org/mjcn.htm>.
- Single, Eric, Bob Williams, and Diane McKenzie (1999). *Canadian Profile: Alcohol, Tobacco & Other Drugs*. Ottawa: Canadian Centre on Substance Abuse and the Addiction Research Foundation of Ontario.

- Statistics Canada (2002). *Canadian Crime Statistics 2001*. Catalogue no. 85-205 XIE (December).
- Thies, C. and F. Register (1993). "Decriminalisation of Marijuana and the Demand for Alcohol, Marijuana and Cocaine." *The Social Science Journal* 30: 385-99.
- Thornton, Mark (1991). "Alcohol Prohibition was a Failure." *Cato Policy Analysis* No. 157 (January).
- Vancouver Sun* (2004). "Eight Suspected in Being Involved in Marijuana-ripoff Crime Ring." (Jan 20).
- van Ours, Jan C. (1995), "The Price Elasticity of Hard Drugs: The Case of Opium in the Dutch East Indies, 1923-1938." *Journal of Political Economy*. Vol. 103, no. 2, pp. 261-279.
- Warburton, Clark (1932). *The Economics of Prohibition*. Reprinted AMS Press: New York: 1968; original printing Columbia University Press, 1932.)
- White, Michael D. and William A. Luksetich (1983). "Heroin: Price Elasticity and Enforcement Strategies." *Economic Inquiry*. Vol. XXI (October): 557-564.
- Wickstead, P.D.G. (2000a). "Who Wants to be a Millionaire?" Mimeo. 217 pp.
- ____ (2002b). "Waivers—Pleading Guilty in Vancouver." Mimeo. 57 pp.
- Yuan, Yuehong and Jonathan P. Caulkins (1998). "The Effect of Variation in High-Level Domestic Drug Enforcement on Variation in Drug Prices." *Socio-Economic Planning Sciences* 32 (4): 265-276.

Appendices

Appendix Table 1A

Table 1A puts Canadian marijuana consumption into some kind of numerical perspective that is commensurate with the degree of uncertainty associated with it. Row 1 identifies the number of users based on estimates of usage described in Single *et al.* (1999, table 5.1). User numbers are im-

puted (using rates of change from Rhodes *et al.*) for years not sampled. Row 2 gives the actual surveyed percentage of Canadians over the age of 15 who are users. Row 3 assumes per-user consumption of marijuana cigarettes (based on US data.) Rows 4 and 5 use two estimates for the size of

Table 1A: Estimates of the Internal Canadian Market for Marijuana, 1988-2000

	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
1. Millions of Current Users in Canada ^a	1.38	1.41	1.10	1.11	1.13	0.96	1.71	1.73	1.75	1.78	1.80	1.82	1.84
2. Actual surveyed users as a % of the population 15 or older*		6.5	5.0			4.2	7.4						
3. Number of cigarettes used per month**	16.9	17.3	17.6	16.6	17.2	17.8	18.7	18.7	18.7	18.7	18.7	18.7	18.7
Weight of one cigarette													
4. Low (grams)***	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
5. High (grams)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Implied Average Annual Marijuana Consumption per user (grams):													
6. Low weight estimate	77.0	79.5	82.0	76.2	78.4	82.4	86.5	86.5	86.5	86.5	86.5	86.5	86.5
7. High weight estimate	202.8	207.6	211.2	199.2	206.4	213.6	224.4	224.4	224.4	224.4	224.4	224.4	224.4
8. Price per ounce (in year 2000 \$C)****	370.3	377.6	476.1	474.0	482.3	418.0	382.7	321.5	303.9	308.1	331.9	303.9	303.9
9. Price per gram \$C	13.0	13.3	16.8	16.7	17.0	14.7	13.5	11.3	10.7	10.8	11.7	10.7	10.7
Total Canadian Internal Consumption (in thousands of kgs—metric tons)													
10. Low weight average	106.3	111.7	90.1	84.8	88.2	78.8	147.7	149.7	151.6	153.7	155.5	157.4	159.4
11. High weight average	279.8	291.9	232.0	221.5	232.3	204.3	383.2	388.2	393.2	398.6	403.3	408.2	413.4
Total Canadian Internal Consumption Annual Expenditure (in billions of dollars)													
12. Low weight average	1.4	1.5	1.5	1.5	1.6	1.2	2.0	1.7	1.7	1.7	1.9	1.7	1.8
13. High weight average	3.6	3.9	3.9	3.7	3.9	3.0	5.2	4.4	4.2	4.3	4.7	4.4	4.4
14. Amount Canadians Spend on Tobacco										2.5	2.5	2.4	2.3

Notes: All figures are in 2000 Canadian dollars.

^aData from surveys reported by Single (1999) interpolated with rates of growth of US use reported in Rhodes *et al.* (2000)

*Single (1999).

** US data (Rhodes *et al.*)

***US data (Rhodes *et al.*) converted from ounces to grams.

****Author's calculation using Canada-wide data for 1998-2001 and US data to track relative price movement. See the section below on pricing marijuana in Canada. Rhodes *et al.* use 1/2 ounce as a purchase unit. This accounts for the difference between the prices in rows 8 and 9 and those of table 2 in the text. All are derived from the pricing formula of appendix A.

each marijuana cigarette. These are reasonable low and high values. The price estimates are developed (Appendix A) and are adjusted by an available US price series for marijuana to account for relative price movements.⁵³ The next two rows refer to the high and low estimates of metric tons of internal Canadian marijuana consumption. The final rows multiply this by price to illustrate the size of the Canadian (consumption) market. Of course this does not include exports.

The final rows of table 1A indicate that the bounds on Canadian domestic consumption of

marijuana bracket substantial differences. Appropriate interpretation of such uncertainty is that we need to know more about the true quantitative measures of consumption to understand how much of the crop is used locally and how much is exported. How large is the industry? To illustrate the internal market, the final row of table 1 lists Canadian expenditures on legal tobacco. Notice that the value of legal tobacco expenditures lies roughly in the middle of the two estimates of the value of Canadian consumed marijuana.

Appendix A: Pricing Marijuana in British Columbia and Canada

What prices are used to evaluate the quantities of marijuana sold? This is an interesting question that has been explored in the context of gram quantities of heroin and cocaine as distinct from pound or kilogram quantities. Using gram prices leads to a higher evaluation of the amount of a drug than using the bulk quantity value. If there is a systematic relationship between them, then it is less important since one or the other form of pricing may be relevant to a particular problem, but one can go either forward or backward to generate the price relevant to the question being asked, and with knowledge about quantities sold, an average price can be generated.

Locally, Plecas *et al.* suggest:

Current estimates of the average wholesale market value of a kilogram of dry local marijuana in British Columbia, sold in large quantities of a kilogram or more, vary from \$3,500 to \$7,500 per kilogram. Estimates of the retail value of a kilogram of dry local marijuana in British Columbia, sold by the pound or by the ounce, vary

between \$3,500 and \$9,000 per kilogram. One can reasonably assume that the average market price in British Columbia during the period [1997-2000] considered was probably somewhere between \$5,000 and \$7,000 per kilogram. (p. 37)

Caulkins (1994) considers the problem of quantity discounts in the following way. Let $P(x)$ be the market price of x grams (note this is *not the price per gram of x grams sold* but the price of x grams sold). If $f(x)$ is the distribution of retail sales – the frequency with which each gram quantity x is sold, then the total amount paid is $\int P(x)f(x)dx$ and the total quantity purchased is $\int xf(x)dx$. The average price paid for the total consumption of marijuana is then

$$1. \quad \bar{P} = \frac{\int P(x)f(x)dx}{\int xf(x)dx}.$$

To know the value of final sales of the total amount sold, multiply \bar{P} by total quantity sold.

⁵³ All prices, however, are in 2002 Canadian dollars.

While this formula is undoubtedly correct, we do not have good information about the true distribution of quantities sold, $f(x)$. Further, we need to assume something about the relationship between price and quantity sold. What is assumed is that $P(x) = ax^\beta$ in which the power reflects the quantity discount. If $\beta = 1$, then price is proportional to quantity. If $\beta < 1$, then there are quantity discounts and the price per gram is falling with increasing quantities. How fast it falls depends on β .

In general, if $P(1)$ is the price of one gram, then $P(1) = \alpha$, and $P(x) = P(1)x^\beta$ so that increases in price are relative to the gram price.⁵⁴

To understand marijuana pricing in British Columbia we have the RCMP data from 1995-1999.

The relevant approach is to estimate the relationship $\ln(P) = \alpha + \beta \ln(Q)$ where price is the price per unit for the chosen quantity and the term “LN” refers to the natural logarithm. For example, based on the data available we find the equation for table 2 in the text:

$$\begin{aligned} 2. \quad \ln(P) &= 2.73 + 0.84 \ln(Q) \\ &\quad (31.31) \quad (39.3) \\ R^2 &= 0.95 \\ N &= 86 \end{aligned}$$

In comparison, Caulkins (1994) finds that $\beta = 0.80$ for heroin based on the US Drug Enforcement Administration’s STRIDE data with some 301 observations. I find the similarity between the two estimates striking in light of the different product and location. Taken at face value, it suggests that

the cost of the cutting, repackaging, and retailing are adding to cost in a similar way in both disparate data sets.

But there is clearly more to the price than simply a power function of the observed relationship between quantity and price. There are other dimensions to the pricing function for which this literature does not usually control.

Fortunately, the price data come with some additional information attached as to the location of purchases and the type of marijuana purchased. In British Columbia, for example, I find that equation 3 in the table below best characterizes the relationship between price per gram and independent attributes such as weight in which the marijuana is sold, urban or rural, home grown or commercial, and whether or not the crop was grown hydroponically. Also included in this national data set are provincial dummies and whether the purchase was of imported marijuana or not.

In Equation 3, where PPG is the price per gram, WEIGHT is the actual weight sold, CITY is a dummy variable for urban or rural; HG refers to home grown (as distinct from “commercial”); HYDRO refers to hydroponically grown.⁵⁵ There are also a series of dummy variables for provinces. The regression suggests that there is, for example, a 1.7 percent increase in the price per gram for a 10 percent increase in the quantity unit sold. The data also suggest that there is a discount on home-grown marijuana and a premium for hydroponic marijuana. Similarly, marijuana sold in the city is cheaper than that sold in rural areas.

54 That is $d\ln[p(x)/p(1)] = \beta \cdot d\ln(x)$ so that β is the percentage increase in price with respect to a percentage increase in quantity. A value of $\beta < 1$ means that when quantity purchased increases by 10 percent, the price increases by less than 10 percent.

55 The form of this equation is similar to that of 2 except that we are looking at price per gram on the left hand side. The coefficient on the natural logarithm of weight is consequently $\beta - 1$ which implies that a point estimate of $\beta = 0.83$.

Equation 3—Full

Dependent Variable: LOG(PPG)

Price per gram of marijuana

Included observations: 86

Variable	Coefficient	Std. Error	t-Statistic	Prob.
LN(WEIGHT)	-0.2	0.0	-9.3	6.9E-14
CITY	-0.33	0.14	-2.38	0.02
HG	-0.59	0.25	-2.40	0.02
HYDRO	0.36	0.14	2.59	0.01
IMPTD	0.10	0.18	0.52	0.60
ALTA	0.06	0.20	0.31	0.76
SAS	0.16	0.16	0.98	0.33
MAN	0.26	0.20	1.30	0.20
ONT	0.12	0.16	0.76	0.45
QUE	0.21	0.25	0.82	0.41
NUN	1.1	0.2	5.8	1.E-07
NWT	0.53	0.25	2.12	0.04
NS	0.49	0.18	2.67	0.01
C	2.6	0.1	29.	3.3E-41
R-squared	0.66	Mean dependent var		2.25
Adjusted R-squared	0.60	S.D. dependent var		0.64
S.E. of regression	0.41	Akaike info criterion		1.18
Sum squared resid	11.9	Schwarz criterion		1.58
Log likelihood	-36.9	F-statistic		10.9
Durbin-Watson stat	1.33	Prob(F-statistic)		2.4E-12

The variable IMPTD refers to whether the product was imported or local. Among the provincial dummies, British Columbia is the home province and consequently does not appear on the list. The provincial dummies are self-explanatory. Other than British Columbia, those that do not appear were excluded because of problems with a small number of observations.

The points of interest in the provincial dummies is that there is a substantial increase in price associated, not surprisingly, with Nunavut and the Northwest Territories, and a premium for Nova Scotia. The rest of the provinces have prices not distinguishable from those in British Columbia. Overall, about 60 percent of the price variance is explained, and of that, about 50 percent is explained without provincial dummies.

Appendix B: Risk and the Alternatives

Suppose that an investor has a bond that pays \$1 per year in perpetuity. The formula relating the price of the \$1 per year and the rate at which the future is discounted to the present at the interest rate, r , is:

$$4. \quad P_b = (1/r).$$

If we have an investment that is likely to be destroyed in any period at a rate of $(1-\pi)$, then the price of the \$1 per year is now:⁵⁶

$$5. \quad P_b = (1-\pi)/(r+\pi).$$

Since P_b and the rate of discount are inverses, the discount of the future is:

$$6. \quad (1/P_b) = (r+\pi)/(1-\pi)$$

The text assumes for analytic simplicity that this is approximated⁵⁷ by $(r+\pi)$ and that in turn, this is represented by, $R^*+\pi$: the alternative return available to our grow-op operator. It is an alternative at the same risk as would be found in the grow-op business, which is what puts all legal investments at risk.

Appendix C: A Richer Model Police Enforcement Enthusiasm

The primary problem with the model thus far is that it does not take into account different conditions that affect the number of busts carried out by the police (or for that matter by others who want to rip off grow-ops.)

Grow-op busts as a function of resources spent

To see how this affects the framework developed above, assume that the number of busts, B , is a product of the number of grow-ops, T ; the number of police assigned to the "grow-busters," N ; the amount of security installed by the grow-ops themselves, S ; and other stuff, x . This leads to an expression:

$$7. \quad B = \exp(b_0) \cdot T^{b_1} N^{b_2} S^{b_3} x^{b_4}$$

that can be rewritten in log-linear form as:

$$8. \quad \ln(B) = b_0 + b_1 \ln(T) + b_2 \ln(N) + b_3 \ln(S) + b_4 \ln(x).$$

Since we know that the number of busts is related to the total number of grow-ops as:

$$9. \quad T = B \cdot \left(\frac{1}{1 - \left(\frac{C \cdot (1 + R^*)}{P} \right)} \right)$$

or, for simplicity write as:

$$10. \quad T = B \cdot v$$

where the expression in equation 9 in large brackets is v .⁵⁸

Now take the natural log of both sides of 10 and substitute from 8 so that we have:

56 That is, $P_b = \sum_{t=0}^{\infty} \left(\frac{1-\pi}{1+r} \right)^t - 1$

57 Clearly this is a better approximation, the smaller is π .

$$\ln(T) = b_0 + b_1 \ln(T) + b_2 \ln(N) + b_3 \ln(S) + b_4 \ln(x) + \ln(v).$$

This leads to a reduced form for the total number of grow-ops, T^* , as:

$$\ln(T^*) = \left(\frac{1}{1-b_1} \right) [(b_0 + b_2 \ln(N) + b_3 \ln(S) + b_4 \ln(x)) + \ln(v)]$$

Without further identification of the coefficients, little can be said. However, if we assume that all except b_3 are positive, and that only a fraction of grow-ops are busted so that $0 < b_1 < 1$, then the number of grow-ops will be greater than those developed by our formula by an amount, proportional to v raised to the power $[1/(1-b_1)]$ for given values of the other variables.

Since b_1 is such an important number, we may want to know something about it. It is the scale effect of grow-ops on the number of busts. It is not obvious that it is a large number. Suppose that there was plenty of “space” and an additional grow-op faced no constraints that were different than those that had gone before. Holding everything else constant, the coefficient is the change in the number of busts because of a change in the number of grow-ops. This is likely to be a small number. Unless there is crowding or congestion—as has been alleged in some locales—the change in the number of busts because of an additional grow-op is likely to be small.

Suppose, for example, that $b_1 = 0.01$. That is, an increase of 100 grow-ops increased the likelihood that 1 additional bust would take place. In this case, the estimates in the table would have to be increased as a function of v raised to the power

$[1/(1-b_1)]$. If v is 5, then the estimate is increased by 1.6 percent. If $b_1 = 0.1$, then the estimates would increase substantially. If the value of b_1 is not too large, it is not likely to impart much of a downward bias to the estimates.

Notice that we can, in fact, estimate a relationship that calculates b_1 in principle. Writing the equation for the number of busts, B , which is at least partially observable, as a reduced form, that is as a function of T^* , the equilibrium number of grow-ops, we have an estimating equation:

$$\ln B = \ln T^* - \ln(v)$$

that reduces to the measurable:

$$\ln B = \left(\frac{1}{1-b_1} \right) (b_0 + b_2 \ln(N) + b_3 \ln(S) + b_4 \ln(x) + \ln(v)) - \ln(v)$$

or,

$$\ln B = \left(\frac{b_0}{1-b_1} \right) + \left(\frac{b_2}{1-b_1} \right) \ln(N) + \left(\frac{b_3}{1-b_1} \right) \ln(S) + \left(\frac{b_4}{1-b_1} \right) \ln(x) + \left(\frac{b_1}{1-b_1} \right) \ln(v)$$

that permits identification of the coefficients and a reduced form estimate of the impact of the different variables on the number of busts.

Since we can know at least the number of police, N , tasked to finding grow-ops, and we have our estimates for v , subject to the vagaries of S and x , we can estimate b_1 . A first step in this analysis is in Appendix D below.

58 Note that the value of v is likely to lie somewhere between 1.2 and 3 and depends entirely on the cost of production, revenue, and yield on alternative opportunities.

Appendix D: Delay Times and the Number of Grow-Ops

To get an estimate of the delay times we use data from Plecas *et al.* for 32 regions. In the regression we have the log of the time to bust, D, regressed against the log of the number of busts, B. The panel data are based on eight regions and four years of data using a fixed effect model since the regions do not change and may have individual characteristics. The coefficient on D tells us the effect of delay on the number of busts. In this case, a

10 percent increase in the time of delay results in a 1.4 percent decrease in the number of busts. In terms of the model, it suggests that the effect of the number of grow ops measured is affected by the number of grow ops. With more delay, fewer grow-ops are discovered. Although there may be many reasons for this, the subtleties of the model in appendix C are clearly an issue that should be investigated.

Dependent Variable: LOG(B?)

Method: GLS (Cross Section Weights)

Sample: 1997 2000

Included observations: 4

Number of cross-sections used: 8

Total panel (unbalanced) observations: 31

One-step weighting matrix

White Heteroskedasticity-Consistent Standard Errors & Covariance

Variable	Coefficient	Std. Error	t-Statistic	Prob.
LOG(D?)	-0.14	0.017	-8.48	0.0000
YEAR	0.22	0.013	16.7	0.0000
Fixed Effects				
C—C				4.14
K—C				4.44
M—C				6.80
NC—C				2.70
T—C				5.40
V—C				5.95
NE—C				1.86
NK—C				2.28
Weighted Statistics				
R-squared	0.998	Mean dependent var		6.73
Adjusted R-squared	0.997	S.D. dependent var		4.33
S.E. of regression	0.216	Sum squared resid		0.98
F-statistic	12060	Durbin-Watson stat		2.49
Prob(F-statistic)				0.00
Unweighted Statistics				
R-squared	0.988	Mean dependent var.		4.45
Adjusted R-squared	0.98	S.D. dependent var.		1.66
S.E. of regression	0.218	Sum squared resid.		0.996
Durbin-Watson stat.	2.81			

Appendix E

The regression underlies the remarks in the text. It is a regression of sentenced days in jail on prior offences and the value of the grow-op as estimated by the police. The coefficient on PRIORS tells us the effect of a change in the number of prior offences on the length of sentence. On average, an additional prior offence adds about 3.58 days to the sentence. The number of priors runs from 0 to 25 so in the extreme, priors may add 90 days to a sentence. Looking at the coefficient on the value of grow-ops (measured in units of \$100,000 as reported by police), an increase of \$100,000 implies an increase of about 16 days in

sentenced jail time. Since the estimated value of the marijuana grow operations runs between \$75,000 and \$3.6 million, the effect on sentencing can be substantial. At the extreme, the value can add 540 days to the jail sentence.

Also of interest is the adjusted R^2 that indicates that about 16 percent of the variance of days sentenced can be explained by the two variables in the regression. This is the basis for the remarks in the text suggesting that there is much left to explain: 84 percent, to be precise.

Dependent Variable: SENDAYS

Included observations: 111

Variable	Coefficient	Std. Error	t-Statistic	Prob.
C	-8.85	15.8	-0.56	0.58
PRIORS	3.58	1.79	1.99	0.05
VALUE/100000	16.2	4.09	3.97	0.00
R-squared	0.17	Mean dependent var		52.1
Adjusted R-squared	0.16	S.D. dependent var		101.
S.E. of regression	93.1	F-statistic		11.2
Log likelihood	-659.	Prob(F-statistic)		0.00

Appendix F: The Demand for Marijuana

Although not used in this analysis, a critical value for many problems with respect to marijuana is the elasticity of demand. The elasticity of demand measures the percentage change in the quantity consumed associated with some percentage change in price. Although conventionally expressed as numbers like 0.5 or 1 or 1.5, elasticities are negative since an increase in price reduces the quantity demanded. An elasticity of 1 implies that a 10 percent fall in price is associated with a 10 percent increase in quantity. An elasticity of less than one means that a fall in price of say, 10 percent, engenders an increase in the quantity consumed of less than 10 percent.

One approach to finding a value for the elasticity of demand for the consumption of marijuana is to use an analogy. We can measure the demand for other addictive substances that are legal and com-

monly used, such as tobacco, for which the elasticity of demand is about 0.5; and for alcohol, another addictive substance, for which the measured elasticity is between 0.18 and 0.86 in the short run.

Estimates for marijuana use span values between 1.4 and 0.1. However, it is important to recall that these estimates are not of the usual kind. They estimate some form of usage rather than quantity. The fact that you smoke once a month is recorded rather than the quantity of marijuana that you purchase. Survey data suggest a very inelastic demand for marijuana (0.2), while purchase-related data tend to find elasticities around 1.0 (Nisbet and Vakil, 1972) although Clements and Daryal (1998) and Daryal (2002) find elasticities between 0.5 and 0.1. Saffer and Chaloupka (1999) estimate an elasticity for marijuana use of 0.28 and 0.44.

About the Author

Stephen T. Easton is a professor of Economics at Simon Fraser University and a Senior Scholar at The Fraser Institute. He received his A.B. from Oberlin College and his Ph.D. from the University of Chicago. Recent works published by The Fraser Institute include *Privatizing Prisons* (editor, 1998), *The Costs of Crime: Who Pays and How Much? 1998 Update* (with Paul Brantingham, 1998), and *Rating Global Economic Freedom* (editor, 1992). He was also co-author of *A Secondary Schools Report Card for British Columbia* (1998), *The 1999 Report Card on British Columbia's Secondary Schools, Boys, Girls, and Grades: Academic Gender Balance in British Columbia's Secondary Schools* (1999), and *The 1999 Report Card on Alberta's High Schools*. Other publications about education include "Do We Have a Problem Yet? Women and Men in Higher Education," in David Laidler (ed.), *Renovating the Ivory Tower: Canadian Universities and the Knowledge Economy* (Toronto: C.D. Howe Institute 2002), pages 60–79; "Plus ça change, plus c'est la même chose" in Stephen B. Lawton, Rodney Reed, and Fons van Wieringen, *Restructuring Public Schooling* (Berlin: Springer-Verlag, 1997) and *Education in Canada: An Analysis of Elementary, Secondary and Vocational Schooling* (Vancouver: The Fraser Institute, 1988). His editorials have been carried by the *Vancouver Sun*, the *Globe and Mail*, the *Financial Post*, the *Ottawa Citizen*, the *Stirling chain* and many other newspapers around the country. Professor Easton continues his work as co-author of the Institute's *Report Cards* on schools in Alberta and British Columbia.