Great horned owl (*Bubo virginianus*) on an artificial perch at a cannabis cultivation in southern Humboldt County. Owls are an excellent natural rodent-control agent and providing perches and nest boxes for them can increase their presence on cultivation sites and reduce the need for rodenticides. Photo Credit: Ryan Mathis, CDFW

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Pesticides in California: their potential impacts on wildlife resources and their use in permitted cannabis cultivation

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The agricultural industry, including commercial cannabis cultivators, often relies on rodenticides and insecticides to help minimize damage from wildlife and insect pest species. Many of the most toxic pesticides are listed as California restricted materials, meaning they can only be purchased and used by certified applicators under a permit from a County Agricultural Commissioner. Despite the permit requirement and other restrictions, exposure of non-target wildlife to pesticides continues to occur throughout California. Non-target wildlife may be directly exposed through ingestion, inhalation, or dermal contact or secondarily exposed through ingestion of contaminated or poisoned prey. Exposure to pesticides can be lethal, or it can cause sublethal effects that impact species' immunology, reproduction, thermoregulation, morphology, and behavior. To date, information pertaining to pesticides is spread among disparate resources. Our review paper aims to synthesize a subset of this information. We provide an overview of insecticides and rodenticides and explore the potential effects that these pesticides may have on non-target wildlife species. We then outline current regulations regarding the use of these pesticides in cannabis cultivation, one of the fastest growing agricultural commodities in California.

Key words: acute poisoning, cannabis, insecticide, pesticide, regulations, rodenticide, sublethal effects, wildlife

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The agricultural industry often relies on pesticides to control wildlife and insect pests that damage plants by foraging on them or using them as nesting material (NDIC 2007). Following California Food and Agricultural Code section 12753(b), we define pesticides

as any substance, or mixture of substances, which is intended to be used for defoliating plants, regulating plant growth, or for preventing, destroying, repelling, or mitigating any pest species. Thus, the term pesticide is an overarching term that encompasses, for example, rodenticides, herbicides, insecticides, fungicides, acaricides, and nematicides. In this review, we focus on insecticides and rodenticides because they tend to be more acutely toxic to non-target wildlife species than other types of pesticides.

Pesticides are an issue of conservation concern because they can negatively impact species that are not the focus of pest control activities such as non-target wildlife (Table 1). Non-target wildlife may be directly exposed through ingestion, inhalation, or dermal contact or indirectly exposed through the ingestion of contaminated or poisoned prey (Berny 2007). An additional effect may be diminished prey resources for species that depend on insects or rodents as a food source (Hallmann et al. 2014). Exposure may happen in the area where pesticides were applied, or in surrounding soils, ground water, or surface waters given pesticides can move via spray drift, surface runoff, soil erosion, leaching, or irrigation return flows (Pimentel 2005; Baldwin et al. 2009). The off-target movement of pesticides has resulted in over 10% of the watersheds within California's North Central Coast, South Central Coast, Middle Sacramento, South Sacramento, and San Joaquin containing streams that are impaired by pesticides, where impairment is defined as surface waters that contain pollutants at levels that exceed protected water quality standards (SWRCB 2018). More than 50 pesticides and pesticide breakdown products were detected in the San Joaquin and Sacramento water basins alone (Baldwin et al. 2009; SWRCB 2018).

Currently, information pertaining to pesticides is spread among disparate scientific, management, and regulatory resources. The goal of our review is to synthesize a subset of this information, with the specific objectives of 1) providing an overview of insecticides and rodenticides, two groups of pesticides likely to impact wildlife, 2) describing the acute and sublethal effects of insecticide and rodenticide exposure on non-target wildlife, and 3) outlining current regulations regarding pesticide use on permitted cannabis cultivation sites. We focus on cannabis cultivation, specifically, because it is one of the fastest growing agricultural commodities in California. Further, we aim to distinguish permitted cannabis cultivation, which has stringent pesticide use regulations, from illegal cannabis cultivation, where the use of California and federally restricted pesticides and numerous ensuing environmental impacts have been documented (Gabriel et al. 2012, 2015, 2018).

PESTICIDE GROUPS

Insecticides

Among the most toxic pesticides are organophosphate and carbamate insecticides, known as the anticholinesterase pesticides (Fleischli et al. 2004). These insecticides function by inhibiting acetylcholinesterase (AChE), an enzyme essential to the functioning of the nervous system (Grue et al. 1997; Baldwin et al. 2009). When AChE accumulates in the nervous system due to inhibition by these chemical families, there is uninterrupted stimulation, loss of energy from signal receptors, and eventually, paralysis of respiratory muscles, asphyxiation, and death (Fleischli et al. 2004). Additionally, sublethal exposure to organophosphate and carbamate insecticides can cause short-term hypothermia, decreases in food consumption, weight loss, impaired vision, and altered sexual behavior, with effects tending to be especially acute in birds (Grue et al. 1997). Many insecticides made of

Table 1. Some of the non-target mammal and bird species documented to have pesticide residues in their systems or to have died from acute or secondary poisoning from pesticides. The table is based on information in Stone et al. (1999), Hosea (2000), Erickson and Urban (2004), Brakes and Smith (2005), Lima and Salmon (2010), Elliott et al. (2014), and CDPR (2018).

Mammals		Birds	
Common name	Species name	Common name	Species name
American badger	*Taxidea taxus*	American crow	*Corvus brachyrhynchos*
Bank voles	*Clethrionomys glareous*	American kestrel	*Falco sparverius*
Bobcat	*Lynx rufus*	Bald eagle	*Haliaeetus leucocephalus*
Coyote	*Canis latrans*	Barn owl	*Tyto alba*
Eastern chipmunk	*Tamias striatus*	Barred owl	*Strix varia*
Ermine	*Mustela erminea*	Burrowing owl	*Athene cooperii*
Field voles	*Microtus agrestis*	Common raven	*Corvus corax*
Fisher	*Pekania pennanti*	Cooper's hawk	*Accipiter cooperii*
Gray fox	*Urocyon cinereoargenteus*	Eastern screech owl	*Megascops asio*
Gray squirrel	*Sciurus carolinensis*	Golden eagle	*Aquila chrysaetos*
Heermann's kangaroo rat	*Dipodomys heermanni*	Great horned owl	*Bubo virginianus*
Long-tailed weasel	*Mustela frenata*	Long-eared owl	*Asio otus*
Mountain lion	*Puma concolor*	Northern harrier	*Circus cyaneus*
Opossum	*Didelphis virginiana*	Northern spotted owl	*Strix occidentalis caurina*
Racoon	*Procyon lotor*	Peregrine falcon	*Falco peregrinus*
Red fox	*Vulpes vulpes*	Red-shouldered hawk	*Buteo lineatus*
San Joaquin kit fox	*Vulpes macrotis mutica*	Red-tailed hawk	*Buteo jamaicensis*
Striped skunk	*Mephitis mephitis*	Red-winged blackbird	*Agelaius phoeniceus*
Vole	*Microtus* spp.	Sharp shinned hawk	*Athene striatus*
White-tailed deer	*Odocoileus virginianus*	Snowy owl	*Bubo scandiacus*
Woodmice	*Apodemus sylvaticus*	Song sparrow	*Melospiza melodia*
		Swainson's hawk	*Buteo swainsonii*
		Turkey	*Meleagris gallopavo*
		Turkey vulture	*Cathartes aura*

these chemical compounds are listed as California restricted materials (California Code of Regulations Title 3, Division 6, §6400) that can only be purchased and applied by certified applicators under permit from a county commissioner (Table 2). The use of organophosphates and carbamates has decreased substantially in the US in recent decades as newer insecticides have gained favor.

Table 2. California restricted materials (refer to California Code of Regulations Title 3, Division 6, §6400 for details) that can only be purchased and applied by certified applicators under permit from a county commissioner and their corresponding type and substance group.

Pesticide	Pesticide type	Substance group
Acrolein*	Herbicide	Aldehyde
Aldicarb	Acaricide / Insecticide / Nematicide	Carbamate
Aluminum phosphide	Insecticide / Rodenticide	Inorganic compound
4-aminopyridine	Acaricide / Avicide	unclassified
Azinphos-methyl	Acaricide / Insecticide	Organophosphate
Brodifacoum	Rodenticide	Hydrocoumarin
Bromadiolone	Rodenticide	Coumarin anticoagulant
Calcium cyanide	Fungicide / Herbicide	Fertilizer
Carbaryl*	Insecticide	Carbamate
Carbofuran	Acaricide / Insecticide / Nematicide	Carbamate
Chloropicrin	Insecticide / Nematicide	Unclassified
Chlorpyrifos*	Insecticide	Organophosphate
3-Chloro-p-toluidine hydrochloride	Avicide	
Dazomet*	Fungicide / Herbicide / Insecticide	Carbamate
Dicamba*	Herbicide	Benzoic acid
2,4-Dichlorophenoxyacetic acid*	Herbicide	Alkylchlorophenoxy
2,4-Dichlorophenoxybutyric acid	Herbicide	
2,4-Dichlorophenoxy propionic acid*	Herbicide	Aryloxyalkanoic acid
1,3-Dichloropropene	Bactericide / Nematicide	Halogenated hydrocarbon
Difenacoum	Rodenticide	Hydroxycoumarin
Difethialone	Rodenticide	Coumarin anticoagulant
Disulfoton*	Acaricide / Insecticide	Organophosphate
Endosulfan*	Acaricide / Insecticide	Organophosphate
Ethoprop*	Insecticide / Nematicide	Organophosphate

Table 2. continued.

Pesticide	Pesticide type	Substance group
Fenamiphos	Nematicide	Organophosphate
Lindane*	Acaricide / Insecticide	Organochlorine
Magnesium phosphide	Insecticide / Rodenticide	Inorganic compound
Metam sodium	Fungicide / Herbicide / Insecticide / Nematicide	Carbamate
Methamidophos	Acaricide / Insecticide	Organophosphate
Methidathion	Insecticide	Organophosphate
Methomyl*	Acaricide / Insecticide	Carbamate
Methyl bromide	Acaricide / Insecticide	Organophosphate
Methyl iodide	Insecticide / Nematicide	Alkyl iodide
2-Methyl-4-Chlorophenoxyacetic acid*	Herbicide	Auxin
Methyl isothiocyanate	Fungicide / Herbicide / Insecticide / Nematicide	Unclassified
Mevinphos	Acaricide / Insecticide	Organophosphate
Molinate	Herbicide	Thiocarbamate
Oxydemeton-methyl	Insecticide	Organophosphate
Paraquat	Herbicide	Bipyridylium
Parathion-methyl	Insecticide	Organophosphate
Phorate	Acaricide / Insecticide / Nematicide	Organophosphate
Phosphine gas	Insecticide	Unclassified
Potassium n-methyldithiocarbamate*		
Propanil	Herbicide	Anilide
Sodium cyanide		Inorganic compound
Sodium fluoroacetate	Rodenticide	Organohalide
Sodium tetrathiocarbonate		
Strychnine*	Avicide / Rodenticide	Plant derived
Sulfotep	Acaricide / Insecticide	Organophosphate
Sulfuryl fluoride	Fungicide / Insecticide / Rodenticide	Inorganic compound
Thiobencarb	Herbicide	Thiocarbamate
Tribufos	Herbicide	Organophosphate
Tributyltin	Fungicide	Organometal
Zinc phosphide*	Rodenticide	Unclassified*

* Includes exceptions from restriction

Synthetic pyrethroid insecticides were developed in the 1970s and began replacing organophosphates and carbamates in the 1990s due to their lower toxicity to mammals and birds (Bradbury and Coats 1989; Casida and Quistad 1998). Pyrethroids alter insect neural membranes, which disrupts electrical signaling in the nervous system and ultimately leads to paralysis and death (Soderlund 2010). While pyrethroids are less toxic to mammals and birds than anticholinesterase pesticides, they are still highly toxic to fish and invertebrates when introduced in aquatic habitats (Casida and Quistad 1998; Soderlund 2010). Further, they have a high potential to contaminate downstream habitats given their low solubility in water, high absorption coefficient, and stability in sediment (Bradbury and Coats 1989). Pyrethroids are widely used in agriculture and in structural pest control and can be applied by both pest control professionals and non-professionals such as homeowners.

Neonicotinoids were introduced in 1990, also to replace organophosphate and carbamate insecticides. They function by binding nicotinic acetylcholine receptors in the central nervous system of invertebrates and are now the most widely used insecticides in the world (Mineau and Palmer 2013; Hallman et al. 2014). The function of neonicotinoids, and their ability to persist in the environment, make them highly toxic to invertebrate pollinators and a contributor to the decline of grassland birds (Mineau and Palmer 2013). They are also water soluble, meaning they have a high propensity for runoff and ground water infiltration (Hallman et al. 2014). Consequently, several neonicotinoid-based insecticides (e.g., imidacloprid and thiamethoxam) are on California's Groundwater Protection List (California Code of Regulations Title 3, Division 6, §6800).

Rodenticides

Rodenticides may also be used on agricultural sites to control known pests like mice (*Mus* spp.), roof rats (*Rattus rattus*), ground squirrels (*Spermophilus* spp.), and pocket gophers (*Thomomys* spp.; CDPR 2015). Anticoagulant rodenticides (ARs) function by inhibiting the synthesis of vitamin K in the liver, which delays coagulation and ultimately leads to hemorrhaging and death (Watt et al. 2005). There is a lag time between ingestion and death, meaning target species may ingest several toxic doses before they die (Herring et al. 2017). Effects of AR exposure can include acute poisoning, compromised immune systems, secondary poisoning through the consumption of exposed prey, and decreased ability to clot properly causing small injuries to bleed excessively (Gordon 1994). Anticoagulant rodenticides pose a threat to not only target and non-target wildlife, but also to children and pets—poison centers in the U.S. receive tens of thousands of reports of rodenticide exposure and ingestion annually (EPA 2011).

There are two types of ARs: first-generation and second-generation. Second-generation ARs were created after pest species began developing a resistance to first-generation ARs like warfarin (Hosea 2000). Second-generation ARs are more acutely toxic, more lipophilic (which increases their tissue accumulation and retention), and have longer half-lives (i.e., the time required for a concentration to decrease by half in a given organ like the liver; Hosea 2000; Erickson and Urban 2004). This means animals that ingest second-generation ARs can potentially carry the compound for years as compared to the shorter durations of first-generation ARs (CDPR 2018). Second-generation brodifacoum, bromadiolone, and difenacoum, for example, have hepatic half-lives of 113.5–350, 170–318, and 118 days, respectively, as compared to first-generation chlorophacinone, diphacinone, and warfarin

that have half-lives of < 2, 3, and 26.2 days, respectively (CDPR 2018). The longer half-lives also mean that target pest species have the propensity to consume multiple doses of second-generation ARs prior to death, leading to the bioaccumulation (i.e., higher concentrations) of second-generation ARs in their organs, in turn posing a greater risk to the predators and scavengers that may consume them (Stone et al. 2003; Riley et al. 2007; Lima and Salmon 2010). As a result of documented exposure to non-target wildlife, second-generation ARs (i.e., brodifacoum, bromadiolone, difenacoum, and difethialone) have been labeled as California restricted materials (Table 2).

Acute rodenticides like bromethalin, cholecalciferol, strychnine, and zinc phosphide act more rapidly than ARs and are available to both professionals and homeowners. Acute rodenticides have varying modes of action, all of which differ from anticoagulants. Bromethalin, for example, decreases adenosine triphosphate synthesis, which leads to a buildup of cerebral spinal fluid, damage to the central nervous system, and lastly, paralysis, convulsions, and death (Van Lier and Cherry 1988). Strychnine, alternatively, blocks the uptake of glycine, an inhibitory neurotransmitter, which leads to increased motor neuron impulses, respiratory muscle spasms, and ultimately respiratory failure (Lawrence et al. 2009). Secondary poisoning from acute rodenticides is uncommon, but they do pose a significant hazard to anything that may consume them including nontarget wildlife (e.g., striped skunks- *Mephitis mephitis* and raccoons- *Procyon lotor*), people, livestock, and pets (van den Brink et al. 2018). Strychnine, for example, is used to control pocket gophers (*Thomomys* spp.) and is only legally applied underground. Improper use and deployment of strychnine, however, has resulted in non-target losses of birds and mammals alike (Littrell 1990). Numerous cases of bromethalin intoxication have been reported in urban wildlife, also likely from improper bait placement (McMillin et al 2016). Lastly, cholecalciferol has been found at illegal cannabis cultivation sites in northern California and was assumed to be a contributing factor in the death of a fisher (*Pekania pennanti*) given it had signs of hypercalcemia (Gabriel et al. 2015).

EFFECTS OF PESTICIDE EXPOSURE

Acute effects

Acute poisoning can follow direct exposure through ingestion, inhalation, or dermal contact, or secondary exposure through ingestion of contaminated prey (Berny 2007, CDPR 2018). Acute effects from pesticides have been documented in target and non-target species alike, ranging from insect pollinators and other arthropods to birds and mammals, and can ultimately result in decreased species diversity (Tables 1, 3; Clarke et al. 1986; Warner 1994).

Secondary exposure to anticoagulant rodenticides.—In California, secondary exposure of predators and scavengers to ARs appears to be widespread. Sixty-nine percent of wildlife collected by California's Department of Fish and Wildlife (CDFW) in 1994–1999 and 92% of mountain lions (*Puma concolor*) tested by CDFW in 2015–2016 tested positive for one or more ARs (Hosea 2000; Rudd et al. 2018). Additionally, 89% of raptors collected by a public health surveillance program in 2007, 73.5% of endangered San Joaquin kit foxes (*Vulpes macrotis mutica*) collected in Bakersfield from 1985-2009, and 89% of bobcats (*Lynx rufus*) necropsied by the National Park Service in 1997–2012 tested positive for one or more ARs (Lima and Salmon 2010; Cypher et al. 2014; Serieys et al. 2015). While rodenticides have

Table 3. Examples of acute effects of pesticide exposure on non-target wildlife species.

Taxa	Species	Response	Source
Birds	Bald eagle (*Haliaeetus leucocephalus*), red-tailed hawk (*Buteo jamaicensis*), great horned owl (*Bubo virginianus*)	Birds were tested for poisoning by organophosphate insecticides from 1984-85. Insecticides were the cause of death for 8 eagles, 2 hawks, and 1 owl.	Henny et al. 1987
	103 bird species in 12 orders	8,877 avian carcasses, where death was attributed to organophosphates and carbamates, were recovered between 1980 and 2000 in the United States.	Fleischli et al. 2004
	10 common raptor species	Raptors, none of which were suspected of having rodenticide exposure, were collected as part of a public health surveillance program in California. Of the 96 birds tested, 87% (n = 83) tested positive for one or more anticoagulant rodenticides.	Lima and Salmon 2010
	Barred owls (*Strix varia*), eastern screech owls (*Megascops asio*), great horned owl	Among the 161 owls that were admitted to a wildlife clinic in Massachusetts from 2006 – 2010, 138 (89%) tested positive for anticoagulant residues, predominantly brodifacoum.	Murray 2011
	Barn owl (*Tyto alba*)	119 owls from British Columbia were tested for rodenticide exposure. 29% of owls had toxicosis symptoms, males had higher concentrations of SGARs than females, and juveniles were more likely to show signs of toxicosis than adults.	Huang et al. 2016
	Red-tailed hawk	Among the 97 hawks that were caught, 8 tested positive for diphacinone. Tests were based on blood samples; the half-lives of anticoagulant rodenticides in the blood are much shorter than in the liver.	Abernathy et al. 2018
	Northern spotted owls (*Strix occidentalis caurina*), barred owls	7 out of 10 northern spotted owls and 34 out of 84 barred owls tested positive for anticoagulant rodenticide exposure. Barred owls were lethally removed as part of a management action and northern spotted owls were opportunistically collected.	Gabriel et al. 2018
Mammals	Coyote (*Canis latrans*)	During a 9-year study in southern California, anticoagulant rodenticide exposure was the second leading cause of mortality; 83% of coyotes tested were exposed.	Riley et al. 2003

Table 3. continued.

Taxa	Species	Response	Source
	San Joaquin kit fox (*Vulpes macrotis mutica*)	In foxes (n=68) collected in Bakersfield from 1985-2009, 73.5% of livers tested positive for AR exposure.	Cypher et al. 2014
	Bobcat (*Lynx rufus*), mountain lion (*Puma concolor*)	90% of bobcat livers (n = 39) and 100% of mountain lion livers (n = 4) tested positive for AR exposure, including brodifacoum, bromadialone, diphacinone, and/or difethialone.	Riley et al. 2007
	Bobcat	Livers from 172 bobcats were sampled for anticoagulant rodenticide exposure from 1997-2012 in southern California. 89% of the samples came back positive, including both sexes and age classes. Fetal transfer of rodenticides was also documented.	Serieys et al. 2015
	Mountain lion	Out of 64 lions tested by California Fish and Wildlife, 92% had detectable levels of anticoagulant rodenticides; 67% were exposed to first-generation rodenticides and 92% to second-generation.	Rudd et al. 2018
	American badger (*Taxidea taxus*), red fox (*Vulpes vulpes*)	2.2x more badgers and 6.4x more foxes were observed per km of road in a study area with low poisoning as compared to a study area with high poisoning. Poisoning was from strychnine- and chlorophacinone-treated oat baits.	Proulx and MacKenzie 2012
	Fisher (*Pekania pennanti*)	101 fishers were collected for necropsy in California during 2007-2014, of whom 86 were exposed to one or more anticoagulant rodenticides (= 1.73 AR compounds/indiv).	Gabriel et al. 2015
	Wood mice (*Apodemus sylvaticus*), bank voles (*Clethrionomys glareolus*), field voles (*Microtus agrestis*)	Coumatetralyl anticoagulant rodenticide bait, pre-mixed with a marker dye, was deployed for routine rat control. 48.6% of individuals in local populations of non-target small mammals ate the bait as indicated by the presence of the marker dye in their feces.	Brakes and Smith 2015

Table 3. continued.

Taxa	Species	Response	Source
Herpetofauna	Wood frogs (*Rana sylvatica*), leopard frogs (*R. pipiens*), green frogs (*R. clamitans*), bullfrogs (*R. catesbeiana*), American toads (*Bufo americanus*), gray tree frogs (*Hyla versicolor*)	In an experimental setting, exposure to carbaryl (six concentrations- 6.5, 3.2, 1.6, 0.3, 0.03, and 0.0 mg/L) became up to 8x more deadly in green frogs and up to 46x more lethal in bullfrogs when combined with predatory stress (i.e., adult red-spotted newts- *Notophthalmus viridescens*). Synergistic interactions were also documented in leopard frogs and toads but not in wood frogs or gray tree frogs.	Relyea 2003
	European common frog (*Rana temporaria*)	Frogs were exposed to three application rates (recommended max, 0.1x, and 10x label rate) of six pesticides including three fungicides (pyraclostrobin, captan, and spiroxamine), two herbicides (bromoxyniloctanoate and fenoxaprop-P-ethyl), and one insecticide (dimethoate). Acute mortality ranged from 20-100% (= 60%) within seven days when pesticides were applied at the label rate.	Brühl et al. 2013
Multi-taxa	Mammals and birds	From 1971 – 1997 necropsies were done for wildlife submitted to a rehabilitation center in New York. Death from hemorrhage associated with anticoagulant rodenticides was documented in 51 cases; species ranged from owls to deer.	Stone et al. 1999
	Mammals and birds	Tissues from 74 dead animals were collected from 1994 – 1999 in California. 30 out of 43 mammals and 21 out of 31 birds examined (n = 21 species total) had rodenticide residues. Coyotes, bobcats, golden eagles (*Aquila chrysaetos*), and barn owls were among the species most frequently exposed.	Hosea 2000

been documented to negatively impact a wide array of non-target wildlife (Table 1), their impacts are particularly prevalent in the very species that help control rodent populations naturally like scavenging and predatory raptors (e.g., barn owl - *Tyto alba* and red-tailed hawk - *Buteo jamaicensis*) and mammals (e.g., bobcats and coyotes- *Canis latrans*; Gabriel et al. 2015; Elliott et al. 2016).

Secondary exposure to ARs has also been suggested as a contributing factor in the decline of threatened and endangered species (Gabriel et al. 2015, 2018). Northern spotted owls (*Strix occidentalis caurina*), for example, are a federal- and state-listed endangered species. One of their primary prey sources in northern California are dusky-footed woodrats (*Neotoma fuscipes*), which are also perceived as a threat to cannabis cultivation given that they forage on cannabis plants and use them as nesting material (Franklin et al. 2018). If ARs are used to control woodrats, particularly second-generation ARs at illegal cultivation sites, then the owls are at risk of secondary rodenticide exposure. In fact, Gabriel et al. (2018) found that 7 out of 10 northern spotted owls and 34 out of 84 barred owls (*S. varia*), an ecologically similar species, tested positive for AR exposure in northern California where thousands of illegal cannabis cultivation sites have been documented on private and public lands.

Toxicosis from anticoagulant rodenticides.—Cases of lethal poisoning from ARs in non-target wildlife are much rarer than secondary exposure. For a mortality to be attributed to AR exposure, the animal must have acute clinically significant signs of toxicant exposure including detectable levels of AR(s) in the liver, coagulopathy, and hemorrhaging that cannot be attributed to any other causes (Gabriel et al. 2015). One of the most notable documentations of a non-target wildlife species dying of toxicosis is fishers in California (Gabriel et al. 2012, 2015). Gabriel et al. (2015) found that 13 fishers had died of toxicosis, 11 from ARs specifically, and that the source of the rodenticides was likely illegal cannabis cultivation sites.

Acute effects of insecticides.—Acute effects from insecticides have also been documented in numerous non-target wildlife species (Relyea 2003; Fleischli et al. 2004). Insecticides, similar to ARs, tend to reduce the population sizes of predators and parasites that help control plant-feeding arthropods naturally like grassland birds (Pimentel 2005; Mineau and Palmer 2013). In addition, exposure to insecticides can contribute to the decline of threatened and endangered species. A study by Davidson and Knapp (2007) found that use of anticholinesterase insecticides upwind of sampled sites had a significant, negative influence on the probability that mountain yellow-legged frogs (*Rana muscosa*), a threatened species, would be present. They also found that the landscape-scale effect of anticholinesterase insecticides was stronger than that of fish, the primary variable (i.e., the introduction of nonnative fish to historically fishless areas) that has been attributed to the decline of yellow-legged frogs (Davidson and Knapp 2007). Amphibian species in general may be especially prone to pesticide exposure given their skin is highly permeable and the life cycle of some species encompasses aquatic and terrestrial phases, meaning they may be exposed to pesticides in two environments (Brühl et al. 2013).

Sublethal effects

Equally concerning to the acute poisoning of wildlife populations are the physiological, phenological, and behavioral impacts associated with sublethal exposure to pesticides (Table 4; Baldwin et al. 2009; Fraser et al. 2018). These impacts are cryptic in that they may

Table 4. Examples of sublethal effects of pesticide exposure on non-target wildlife species.

Taxa	Species	Response	Source
Birds	American kestrel (*Falco sparverius*)	Kestrels experienced hypothermia when they were given a single oral dose of the organophosphate methyl parathion (2.25 mg/kg wt) and were exposed to 10 hours of cold (temperature was normally within their thermoneutral zone).	Rattner and Franson 1983
	European starlings (*Sturnus vulgaris*)	Males decreased their singling and displaying by 50% following an oral dose of the organophosphate dicrotophos (2.5 mg/kg wt), which could reduce their reproductive success.	Grue and Shipley 1984
	European starlings	When given a single dose of the organophosphate dicrotophos, adults lost an average of 14% of their initial body weight within 24-hr of the dose.	Grue and Shipley 1984
	Northern bobwhites (*Colinus virginianus*)	After being fed a diet with varying concentrations of the organophosphate methamidophos, birds consumed less food and in turn, produced fewer eggs and had smaller clutch sizes.	Stromborg 1986
	Mallards (*Anas platyrhynchos*)	Ducklings exposed to low levels of carbofuran (0.22 mg/kg wt) experienced hypothermia and increased mortality at temperatures as high as 50° F.	Martin and Solomon 1991
	Mallards	Including methyl parathion (400 ppm) in the diet of mallards for 8 days resulted in a 50% reduction in egg production. The ducks also consumed only 16% of that of controls.	Bennett et al. 1991
	House sparrow (*Passer domesticus*)	After being exposed to a single sublethal dose of the organophosphate fenthion, sparrows were 16x more likely to be predated than controls within the same flock.	Hunt et al. 1992
	White-throated sparrow (*Zonotrichia albicollis*)	When given an oral dose of the organophosphate fenitrothion, birds fledged at lower body weights than controls.	Grue et al. 1997
	Insectivorous birds	The average intrinsic rate of increase in local bird populations was negatively associated with the concentration of imidacloprid, a neonicotinoid insecticide, likely because the birds (n = 15 species) feed their young almost exclusively with invertebrates.	Hallman et al. 2014
Mammals	Bobcats (*Lynx rufus*)	All bobcats with advanced mange (n = 19) had anticoagulant rodenticide compounds in their liver. Further, rodenticide levels were higher in bobcats that died of mange than those that died of other causes.	Riley et al. 2007
	Bobcat	Samples were collected from 124 bobcats presumed to be healthy from 2007 – 2012. Anticoagulant rodenticide exposure was positively associated with B-cell counts (i.e., the immune component that targets invading pathogens) and negatively associated with creatine (i.e., an indicator of kidney function).	Serieys et al. 2018

Table 4. continued.

Taxa	Species	Response	Source
	Common vole (*Microtus arvalis*)	Voles trapped in alfalfa fields treated with the anticoagulant rodenticide chlorophacinone had higher prevalence of the bacteria that causes tularemia, a zoonotic disease, than voles trapped in control fields.	Vidal et al. 2009
Herpetofauna	Wood frogs (*Rana sylvatica*)	Frogs were more susceptible to trematode infections in ponds with greater exposure to agricultural runoff, likely because of increased stress due to exposure to organochlorine pesticides and organophosphorus compounds (e.g., Altrazine and Malathion). This, in turn, led to a higher risk of limb deformities.	Kiesecker 2002
	African clawed frogs (*Xenopus laevis*)	Larvae were exposed to atrazine (0.01 – 200 ppb), a commonly used herbicide. Atrazine induced hermaphroditism, demasculinized the larynxes of males, and resulted in an up to 10-fold decrease in male's testosterone levels.	Hayes et al. 2002
	Leopard frogs (*R. pipiens*)	Frogs were exposed to a mixture of atrazine, metribuzin, aldicarb, endosulfane, lindane, and dieldrin for 21 days and then challenged with a parasitic nematode (*Rhabdias ranae*). Frogs exposed to pesticides had reduced T-cell proliferation (i.e., cells that defend against extracellular parasites) and more nematodes in their lungs than control animals.	Christin et al. 2003
	Green frogs (*R. clamitans*)	The net effect of exposure to glyphosate (3700 µg/L) and atrazine (201 µg/L), two commonly used herbicides, and to malathion (9.6 µg/L) and carbaryl (33.5 µg/L), two broad-based insecticides, was to increase frog susceptibility to trematode (*Echinostoma trivolvis*) infections.	Rohr et al. 2008
	Leopard frogs	Outdoor mesocosms that contained zooplankton, phytoplankton, periphyton, and larval amphibians were exposed to low concentrations of malathion, an insecticide. The malathion caused a decrease in zooplankton and subsequently an increase in phytoplankton, decrease in periphyton, and decrease in the growth and development of leopard frogs, which rely on periphyton as their food source.	Relyea and Diecks 2008
	Wood frogs, leopard frogs, American toads (*Bufo americanus*)	Outdoor mesocosms that included leaf litter, algae, zooplankton, and three species of tadpoles were exposed to Roundup herbicide and/or predator cues (i.e., newts or larval dragonflies). Roundup resulted in morphological changes (i.e., increase in tail depth) like the adaptive changes induced by dragonfly (i.e., predator) cues.	Relyea 2012

reduce individual fitness and population persistence without the obvious signs of pesticide exposure (Fraser et al. 2018).

Immunological effects.—One sublethal impact may be to an individual's immune system (Li and Kawada 2006; Zabroskii et al. 2012; Serieys et al. 2018). Insecticides like endosulfan and malathion have been found to compromise individuals' immune systems, leaving the animal more susceptible to parasitic infections and pathogens (Christin et al. 2003; Rohr et al. 2008). Leopard frogs (*Rana pipiens*) that were exposed to a mixture of four insecticides and two herbicides, for example, had fewer T-lymphocytes (i.e., one of the immune system cells that fight off extracellular parasites) and consequently, were at greater risk to parasitic nematodes (Christin et al. 2003). One study suggested that bobcats exposed to anticoagulant rodenticides, alternatively, experienced changes in their immune responses to allergens, tumors, viral infections, and novel pathogens (Serieys et al. 2018). Further, given immune responses are energetically costly, regular immune stimulation from anticoagulant rodenticide or insecticide exposure may lead to an overall decrease in fitness (Serieys et al. 2018).

Reproductive effects.—Pesticides may also adversely affect reproduction. Exposure to certain types of insecticides (e.g., organophosphates and neonicotinoids) has been documented to cause embryotoxicity (i.e., the transfer of residual pesticides from the parent to young), chemical castration, and a reduction in courtship displays, all of which can lead to reproductive failure (Grue and Shipley 1984; Grue et al. 1997; Pimentel 2005; Mineau and Palmer 2013). Additionally, mammals and birds have been shown to have reduced litter and clutch sizes, perhaps because insecticide and rodenticide exposure can decrease an animal's appetite (Bennett et al. 1991; Erickson and Urban 2004).

Thermoregulatory effects.—Sublethal exposure to pesticides like anticholinesterase insecticides can also impair thermoregulatory abilities, which can lead to death (Gordon 1994; Grue et al. 1997). Mallard ducklings (*Anas platyrhynchos*), for example, experienced hypothermia after being exposed to a low dose of carbofuran, with some ducklings dying at temperatures as high as 10° C, a temperature that would typically be within the species' thermoneutral zone (Martin and Solomon 1991). This suggests that pesticide exposure may pose an even greater risk to wildlife when weather conditions fall outside of the species' thermoneutral zone (Rattner and Franson 1983; Martin and Solomon 1991).

Morphological effects.—Altered morphology following exposure to certain types of insecticides has primarily been documented in amphibians. In a meta-analysis of experimental studies aimed at measuring the effects of chemical pollutants on amphibians, researchers found that the overall effect size of pollutants was a 535% increase in the frequency of abnormalities (e.g., limb deformities) as well as a 14.3% decrease in survival and 7.5% decrease in mass (Egea-Serrano et al. 2012). Frogs have also been documented to have inhibited growth of the larynx and to develop morphological phenotypes that are poorly suited for their environment (Kiesecker 2002; Relyea and Diecks 2008; Relyea 2012).

Behavioral effects.—Lastly, pesticides may alter the behavior, composition, and abundance of both predators and prey (e.g., insects and small mammals). Following pesticide exposure, arthropods exhibit altered search and attack behaviors, mammals have decreased coordination, motor skills, and response times, and fishes can develop swimming abnormalities, all of which make the respective individual more susceptible to predation (Pimentel 2005; Wolansky and Harrill 2008; Sanchez-Barbudo et al. 2012; Renick et al. 2015). Animals that are secondarily exposed to pesticides may also be at greater risk to predation if they too experience responses like reduced mobility and response times (Serieys et al. 2015). If

pesticides reduce the availability of food resources in a landscape, alternatively, then there can be cascading impacts throughout the food chain. In areas treated with insecticides, for example, insect prey populations like mosquitos and beetles have been shown to decline, which in turn has resulted in declines in the survival and abundance of insectivorous bird populations (Warner 1994; Hallmann et al. 2014).

REGULATIONS PERTAINING TO PESTICIDE USE ON PERMITTED CANNABIS CULTIVATION SITES IN CALIFORNIA

Commercial, adult-use cannabis cultivation was legalized in California in 2018. With legalization came a multitude of regulations pertaining to pesticide use. California state regulations restrict the use of pesticides in or around permitted cannabis cultivation if they are a) California restricted materials, b) on the ground water protection list, or c) not registered for a food use in California. California restricted materials (Table 2; California Code of Regulations Title 3, Division 6, §6400) are pesticides deemed to have a high potential to harm public health and the environment. They can only be purchased and used by, or under the supervision of, a certified applicator who has a permit issued by the County Agricultural Commissioner (CDPR 2014). The ground water protection list, alternatively, identifies pesticides that have the potential to pollute ground water (California Code of Regulations Title 3, Division 6, §6800), and restricts their use either statewide or in specified vulnerable areas (restrictions are pesticide-specific). Similar to other agricultural crops, cannabis cultivators must also: (1) comply with all pesticide label directions; (2) store chemicals in a secure building or shed to prevent access by wildlife; (3) contain any chemical leaks and immediately clean up any spills; (4) apply the minimum amount of product necessary to control the target pest; (5) prevent offsite drift; (6) not apply pesticides when pollinators are present; (7) not allow drift to flowering plants attractive to pollinators; (8) not spray directly onto surface water or allow pesticide product to drift to surface water and spray only when wind is blowing away from surface water bodies; (9) not apply pesticides when they may reach surface water or groundwater; and (10) only use properly labeled pesticides and consult with California Department of Pesticide Regulation (CDPR) if no label is available (CDFA 2019).

Regulations pertaining to pesticide use in cannabis cultivation are even more stringent when you incorporate federal regulations, or in this case the lack thereof. The United States Environmental Protection Agency (EPA) does not recognize cannabis as being a part of an existing crop group given it is illegal under federal law. This means there are no U.S. EPA-approved pesticide products for use on cannabis or U.S. EPA residue tolerance requirements (i.e., the amount of pesticide residue allowed to remain in or on each treated crop). Consequently, the only pesticides that can be legally applied to cannabis under California state law are pesticides with active ingredients that are exempt from residue tolerance requirements and either exempt from registration requirements or registered for a use broad enough to encompass cannabis (CDPR 2017). Most of these exempt pesticides are biorational like citronella or food-grade essential oils like cinnamon, garlic, and rosemary oils (CDPR 2017).

To monitor pesticide use on permitted cannabis cultivation sites, California's Bureau of Cannabis Control text of regulations (California Code of Regulations Title 16, Division 42) requires that cultivators have a pest management plan that includes "product names and active ingredient(s) of all pesticides to be applied to cannabis during any stage of plant growth" and "integrated pest management protocols including chemical, biological, and

cultural methods the applicant anticipates using to control or prevent the introduction of pests on the cultivation site." The Bureau of Cannabis Control also requires cannabis cultivators to submit 0.5 g of every cannabis batch to be tested for Category I (i.e., not registered for food use in California) and Category II Residual Pesticides (Tables 5, 6). If the sample exceeds any of the threshold values, then the batch from which the sample was taken will not be released for retail sale.

Table 5. Category I Residual Pesticides (California Code of Regulations Title 16, Division 42, §5719.1) that are not registered for food use in California (i.e., they cannot be used in or around cannabis cultivation sites), the pesticide type, and the substance group (IUPAC 2019). When a cannabis batch is tested for residual pesticides, the limit of quantitation is 0.10 μg/g or lower for all Category I Residual Pesticides.

Residual pesticide	Pesticide type	Substance group
Aldicarb[a,b]	Insecticide / Acaricide / Nematicide	Carbamate
Brodifacoum[a]	Rodenticide	Hydrocoumarin
Bromadiolone[a]	Rodenticide	Coumarin anticoagulant
Carbofuran[a]	Insecticide / Acaricide / Nematicide	Carbamate
Chlordane	Insecticide	Organophosphate
Chlorfenapyr	Insecticide / Acaricide / Miticide	Pyrrole
Chlorpyrifos[a*]	Insecticide	Organophosphate
Coumaphos	Ectoparasiticide	Organothiophosphate
Daminozide	Plant growth regulator	Unclassified
DDVP (Dichlorvos)	Insecticide / Acaricide	Organophosphate
Difenacoum[a]	Rodenticide	Hydroxycoumarin
Difethialone[a]	Rodenticide	Coumarin anticoagulant
Dimethoate[b]	Insecticide / Acaricide	Organophosphate
Ethoprop(hos)[a*,b]	Insecticide / Nematicide	Organophosphate
Etofenprox	Insecticide	Pyrethroid
Fenoxycarb	Insecticide	Carbamate
Fipronil	Insecticide	Phenylpzrazole
Imazalil	Fungicide	Imidazole
Methiocarb[b]	Insecticide / bird repellant	Carbamate
Methyl parathion	Insecticide / Acaricide	Organophosphate
Mevinphos[a]	Insecticide / Acaricide	Organophosphate
Paclobutrazol	Fungicide	Triazole
Propoxur	Insecticide / Acaricide	Carbamate
Spiroxamine	Fungicide	Morpholine
Thiacloprid	Insecticide	Neonicotinoid

[a] California restricted material
[a*] California restricted material with exceptions
[b] On the Groundwater Protection List

Table 6. Category II Residual Pesticides (California Code of Regulations Title 16, Division 42, §5719.1), the threshold value that provides the criterion for determining whether a cannabis sample passes or fails an analytical test by the Bureau of Cannabis Control (i.e., Action level), the pesticide type, and the substance group (IUPAC 2019).

Residual pesticide	Action level (µg/g)		Pesticide type	Substance group
	Inhalable cannabis goods	Other cannabis goods		
Abamectin	0.1	0.3	Insecticide	unclassified
Acephate[b]	0.1	5	Insecticide	Organophosphate
Acequinocyl	0.1	4	Acaricide	unclassified
Acetamiprid	0.1	5	Insecticide	Neonicotinoid
Azoxystrobin[b]	0.1	40	Fungicide	Strobilurin
Bifenazate	0.1	5	Insecticide/ Acaricide	Hydrazine carboxylate
Bifenthrin	3	0.5	Insecticide/ Acaricide	Pyrethroid
Boscalid[b]	0.1	10	Fungicide	Carboxamide
Captan	0.7	5	Fungicide / Bactericide	Phthalimide
Carbaryl[a*,b]	0.5	0.5	Insecticide	Carbamate
Chlorantraniliprole[b]	10	40	Insecticide	Anthranilic diamide
Clofentezine	0.1	0.5	Acaricide	Tetrazine
Cyfluthrin[a]	2	1	Insecticide	Pyrethroid
Cypermethrin	1	1	Insecticide	Pyrethroid
Diazinon[b]	0.1	0.2	Insecticide / Acaricide	Organophosphate
Dimethomorph[b]	2	20	Fungicide	Morpholine
Etoxazole	0.1	1.5	Acaricide	Diphenyl oxazoline
Fenhexamid	0.1	10	Fungicide	Hydroxyanilide
Fenpyroximate	0.1	2	Insecticide / Acaricide	Pyrazolium
Flonicamid	0.1	2	Insecticide / Aphicide	Pyridine
Fludioxonil[b]	0.1	30	Fungicide	Phenylpyrrole
Hexythiazox	0.1	2	Acaricide	Carboxamide
Imidacloprid[b]	5	3	Insecticide	Neoicotinoid
Kresoxim-methyl	0.1	1	Fungicide / Bactericide	Strobilurin
Malathion[b]	0.5	5	Insecticide / Acaricide	Organophosphate
Metalaxyl[b]	2	15	Fungicide	Phenylamide
Methomyl[a*,b]	1	0.1	Insecticide / Acaricide	Carbamate
Myclobutanil[b]	0.1	9	Fungicide	Triazole
Naled[a]	0.1	0.5	Insecticide / Acaricide	Organophosphate
Oxamyl	0.5	0.2	Insecticide / Acaricide	Carbamate
Permethrin	0.5	20	Insecticide	Pyrethroid
Phosmet	0.1	0.2	Insecticide / Acaricide	Organophosphate

Table 6. continued.

Residual pesticide	Action level (µg/g)		Pesticide type	Substance group
	Inhalable cannabis goods	Other cannabis goods		
Piperonyl butoxide	3	8	Other	Cyclic aromatic
Prallethrin	0.1	0.4	Insecticide	Pyrethroid
Propiconazole[b]	0.1	20	Fungicide	Triazole
Pyrethrins	0.5	1	Insecticide	unclassified
Pyridaben	0.1	3	Insecticide / Acaricide	Pyridazinone
Spinetoram	0.1	3	Insecticide	Spinosym
Spinosad	0.1	3	Insecticide	Natural substance
Spiromesifen	0.1	12	Insecticide	Tetronic acid
Spirotetramat	0.1	13	Insecticide	Tetronic acid
Tebuconazole[b]	0.1	2	Fungicide	Triazole
Thiamethoxam[b]	5	4.5	Insecticide	Neonicotinoid
Trifloxystrobin	0.1	30	Fungicide	Strobilurin

[a] California restricted material
[a*] California restricted material with exceptions
[b] On California's Groundwater Protection List

The legalization of commercial, adult-use cannabis cultivation came with a bountiful number of pesticide regulations. These regulations aim to help minimize potential environmental impacts of permitted cannabis cultivation and are one of the many ways in which legal cultivation is delineated from illegal cultivation. At legal cultivation sites, for example, the toxic pesticide products that tend to result in acute and sublethal effects in non-target wildlife species cannot be legally applied. At illegal cultivation sites, alternatively, the use of these toxic pesticides and numerous ensuing environmental impacts have been well documented (Gabriel et al. 2012, 2015, 2018). Given legal cannabis cultivation is still in its infancy in California, however, there are many knowledge gaps. We encourage studies focused on the types and quantities of pesticides being used at permitted cannabis cultivation sites and assessments of whether cultivators are fully abiding to regulations. We also encourage studies aimed at improving our understanding of how pesticide use in cannabis cultivation relates to other agricultural industries, and if there are any pesticide-related impacts unique to cannabis given it tends to be grown in rural and forested areas (Butsic et al. 2018). Lastly, we urge studies comparing pesticide use at legal vs. illegal cannabis cultivation sites and documentation of any subsequent environmental impacts as this information would likely highlight some of the benefits of legalization.

Author contributions

Conceived and designed the study: LNR, ADB
Collected the data: LNR, ADB
Performed the analysis of the data: LNR, SM, ADB
Authored the manuscript: LNR, SM, ADB, EC
Provided critical revision of the manuscript: SM, ADB, EC

LITERATURE CITED

Abernathy, E. V., J. M. Hull, A. M. Fish, and C. W. Briggs. 2018. Secondary anticoagulant rodenticide exposure in migrating juvenile red-tailed hawks (*Buteo jamaicensis*) in relationship to body condition. Journal of Raptor Research 52:225–231.

Baldwin, D. H., J. A. Spromberg, T. K. Collier, and N. L. Scholz. 2009. A fish of many scales: extrapolating sublethal pesticide exposures to the productivity of wild salmon populations. Ecological Application 19:2004–2015.

Bennett, R. S., B. A. Williams, D. W. Schmedding, and J. K. Bennett. 1991. Effects of dietary exposure to methyl parathion on egg laying and incubation in mallards. Environmental Toxicology and Chemistry 10:501–507.

Berny, P. J. 2007. Pesticides and the intoxication of wild animals. Journal of Veterinary Pharmacology and Therapeutics 30–93-100.

Bradbury, S. P., and J. R. Coats. 1989. Comparative toxicology of the pyrethroid insecticides. Pages 133-177 in P. de Voogt, editor. Reviews of Environmental Contamination and Toxicology. Springer, New York, NY, USA.

Brakes, C. R., and R. H. Smith. 2005. Exposure of non-target small mammals to rodenticides: short-term effects, recovery and implications for secondary poisoning. Journal of Applied Ecology 42:118–128.

Brühl, C. A., T. Schmidt, S. Pieper, and A. Alscher. 2013. Terrestrial pesticide exposure of amphibians: an underestimated cause of global decline? Scientific Reports 3:1135.

Butsic, V., J. K. Carah, M. Baumann, C. Stephens, and J. C. Brenner. 2018. The emergence of cannabis agriculture frontiers as environmental threats. Environmental Research Letters 13:124017.

California Department of Food and Agriculture (CDFA). 2019. Final program environmental impact report. Available from: https://www.cdfa.ca.gov/calcannabis/PEIR.html (July 2019).

California Department of Pesticide Regulation (CDPR). 2014. Notice of final decision concerning brodifacoum (second generation anticoagulant rodenticide), July 18, 2014. Available from: https://www.cdpr.ca.gov/docs/registration/canot/2014/ca2014-09.pdf (May 2019).

California Department of Pesticide Regulation (CDPR). 2015. Legal pest management practices for marijuana growers in California. Available from: https://www.cdpr.ca.gov/docs/cannabis/questions.htm (May 2019).

California Department of Pesticide Regulation (CDPR). 2017. Cannabis- pesticides that are legal to use. Available from: https://www.cdpr.ca.gov/docs/cannabis/can_use_pesticide.pdf (February 2019).

California Department of Pesticide Regulation (CDPR). 2018. An investigation of anticoagulant rodenticide data submitted to the Department of Pesticide Regulation. Pesticide Registration Branch, Sacramento, CA, USA.

Casida, J. E., and G. B. Quistad. 1998. Golden age of insecticide research: past, present, or future? Annual Review of Entomology 43:1–16.

Christin, M. S., A. D. Gendron, P. Brousseau, L. Ménard, D. J. Marcogliese, D. Cyr, S. Ruby, and M. Fournier. 2003. Effects of agricultural pesticides on the immune system of *Rana Pipiens* and on its resistance to parasitic infection. Environmental Toxicology and Chemistry 22:1127–1133.

Clarke, R. G., P. J. Weatherhead, H. Greenwood, and R. D. Titman. 1986. Numerical responses of red-winged blackbird populations to changes in regional land-use patterns. Canadian Journal of Zoology 64:1944–1950.

Cypher, B. L., S.C. McMillin, T.L. Westall, C. Van Horn Job, R.C. Hosea, B.J. Finlayson, and E.C. Kelly. 2014. Rodenticide exposure among endangered kit foxes relative to habitat use in an urban landscape. Cities and the Environment 7:8.

Davidson, C., and R. A. Knapp. 2007. Multiple stressors and amphibian declines: dual impacts of pesticides and fish on yellow-legged frogs. Ecological Applications 17:587–597.

Egea-Serrano, A., R. A. Relyea, M. Tejedo, and M. Torralva. 2012. Understanding of the impact of chemicals on amphibians: a meta-analytic review. Ecology and Evolution 2:1382–1397.

Elliott, J. E., S. Hindmarch, C. A. Albert, J. Emery, P. Mineau, and F. Maisonneuve. 2014. Exposure pathways of anticoagulant rodenticides to nontarget wildlife. Environmental monitoring and Assessment 186:895–906.

Elliott, J. E., B. A. Rattner, R. F. Shore, and N. W. Van Den Brink. 2016. Paying the pipers: mitigating the impact of anticoagulant rodenticides on predators and scavengers. Bioscience 66:401–407.

Environmental Protection Agency (EPA). 2011. A set of scientific issues being considered by the Environmental protection Agency regarding: scientific conclusions supporting EPA's FIFRA Section 6(B) notice of intent to cancel twenty homeowner rodenticide bait products. EPA Office of Chemical Safety and Pollution Prevention, Washington DC, USA.

Erickson, W., and D. Urban. 2004. Potential risks of nine rodenticides to birds and nontarget mammals: a comparative approach. Washington, DC: US Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances.

Fleischli, M. A., J. C. Franson, N. J. Thomas, D. L. Finley, and W. Riley. 2004. Avian mortality events in the United States caused by anticholinesterase pesticides: a retrospective summary of National Wildlife Health Center records from 1980 to 2000. Archives of Environmental Contamination and Toxicology 46:542–550.

Franklin, A. B., P. C. Carlson, A. Rex, J. T. Rockweit, D. Garza, E. Culhane, S. F. Volker, R. J. Dusek, V. I. Shearn-Bochsler, M. W. Gabriel, and K. E. Horak. Grass is not always greener: rodenticide exposure of a threatened species near marijuana growing operations. BMC Research Notes 11:94

Fraser, D., A. Mouton, L. E. K. Serieys, S. Cole, S. Carver, S. Vandewounde, M. Lappin, S. P. D. Riley, and R. Wayne. 2018. Genome-wide expression reveals multiple systematic effects associated with detection of anticoagulant poisons in bobcats (*Lynx rufus*). Molecular Ecology 27:1170–1187.

Gabriel, M. W., L. V. Diller, J. P. Dumbacher, G. M. Wengert, J. M. Higley, R. H. Poppenga, and S. Mendia. 2018. Exposure to rodenticides in northern spotted and barred owls on remote forest lands in northwestern California: evidence of food web contamination. Avian Conservation and Ecology 13.

Gabriel, M. W., L. W. Woods, R. Poppenga, R. A. Sweitzer, C. Thompson, S. M. Matthews, J. M. Higley, S. M. Keller, K. Purcell, R. H. Barrett, G. M. Wengert, B. N. Sacks, and D. L. Clifford. 2012. Anticoagulant rodenticides on our public and community lands: spatial distribution of exposure and poisoning of a rare forest carnivore. PLoS ONE 7:e40163.

Gabriel, M. W., L. W. Woods, G. M. Wengert, N. Stephenson, J. M. Higley, C. Thompson, S. M. Matthews, R. A. Sweitzer, K. Purcel, R. H. Barrett, S. M. Keller, P. Gaffney, M. Jones, R. Poppenga, J. E. Foley, R. N. Brown, D. L. Clifford, and B. N. Sacks. 2015. Patterns of natural and human-caused mortality factors of a rare forest carnivore, the fisher (*Pekania pennanti*) in California. PLoS ONE 10:e0140640.

Gordon, C. J. 1994. Thermoregulation in laboratory mammals and humans exposed to anticholinesterase agents. Neurotoxicology and Teratology 16:427–453.

Grue, C. E., P. L. Gibert, and M.E. Seeley. 1997. Neurophysiological and behavioral changes in non-target wildlife exposed to organophosphate and carbamate pesticides: thermoregulation, food consumption, and reproduction. American Zoologist 37:369–388.

Grue, C. E., and B. K. Shipley. 1984. Sensitivity of nestling and adult starlings to dicrotophos, an organophosphate pesticide. Environmental Research 35:454–465.

Hallmann, C. A., R. P. B. Foppen, C. A. M. van Turnhout, H. de Kroon, and E. Jongejans. 2014. Declines in insectivorous birds are associated with high neonicotinoid concentrations. Nature 511:341–344.

Hayes, T. B., A. Collins, M. Lee, M. Mendoza, N. Noriega, A. A. Stuart, and A. Vonk. 2002. Hermaphroditic, demasculinized frogs after exposure to the herbicide atrazine at low ecologically relevant doses. Proceedings of the National Academy of Sciences 99:5476–5480.

Henny, C. J., E. J. Kolbe, E. F. Hill, and L. J. Blus. 1987. Case histories of bald eagles and other raptors killed by organophosphorus insecticides topically applied to livestock. Journal of Wildlife Disease 23:292–295.

Herring, G., C. A. Eagles-Smith, and J. Buck. 2017. Characterizing golden eagle risk to lead and anticoagulant rodenticide exposure: a review. Journal of Raptor Research 51:273–292.

Hosea, R. C. 2000. Exposure of non-target wildlife to anticoagulant rodenticides in California. Proceedings of the Vertebrate Pest Conference 19.

Huang, A. C., J. E. Elliott, S. Hindmarch, S. L. Lee, F. Maisonneuve, V. Bowes, K. M. Cheng, and K. Martin. 2016. Increased rodenticide exposure rate and risk of toxicosis in barn owls (*Tyto alba*) from southwestern Canada and linkage with demographic but not genetic factors. Ecotoxicology 25:1061–1071.

Hunt, K. A., D. M. Bird, P. Mineau, and L. Shutt. 1992. Selective predation of organophosphate-exposed prey by American kestrels. Animal Behavior 43:971–976.

International Union of Pure and Applied Chemistry (IUPAC). 2019. Global availability of information on agrochemicals. Accessed from: https://sitem.herts.ac.uk/aeru/iupac/atoz.htm (May 2019).

Kiesecker, J. M. 2002. Synergism between trematode infection and pesticide exposure: a link to amphibian limb deformities in nature? Proceedings of the National Academy of Sciences 99:9900–9904.

Lawrence, D., N. McLinskey, S. Huff, and C. P. Holstege. 2009. Toxin-induced neurologic emergencies. Pages 30-46 in Clinical Neurotoxicology: Syndromes, Substances, Environments. Saunders Elsevier, Philadelphia, PA, USA.

Lima, L. L., and T. P. Salmon. 2010. Assessing some potential environmental impacts from agricultural anticoagulant uses. Proceedings of the Vertebrate Pest Conference 24.

Littrell, E. E. 1990. Effects of field vertebrate pest control on nontarget wildlife (with em-

phasis on bird and rodent control). Proceedings of the Fourteenth Vertebrate Pest Conference 55.

Martin, P. A., and K. R. Solomon. 1991. Acute carbofuran exposure and cold stress: interactive effects in mallard ducklings. Pesticide Biochemistry and Physiology 40:117–127.

McMillin, S., M.S. Piazza, L.W. Woods, and R.H. Poppenga. 2016. New rodenticide on the block: Diagnosing bromethalin intoxication in wildlife. Proceedings of the Vertebrate Pest Conference 27:419–421.

Mineau, P., and C. Palmer. 2013. The impact of the nation's most widely used insecticides on birds. American Bird Conservancy.

Murray, M. 2011. Anticoagulant rodenticide exposure and toxicosis in four species of birds of prey presented to a wildlife clinic in Massachusetts, 2006-2010. Journal of Zoo and Wildlife Medicine 42:88–97.

National Drug Intelligence Center (NDIC). 2007. Domestic cannabis cultivation assessment 2007. United States Department of Justice, Washington D.C., USA.

Pimentel, D. 2005. Environmental and economic costs of the application of pesticides primarily in the Unites States. Environment, Development and Sustainability 7:229–252.

Proulx, G., and N. MacKenzie. 2012. Relative abundance of American badger (*Taxidea taxus*) and red fox (*Vulpes vulpes*) in landscapes with high and low rodenticide poisoning levels. Integrative Zoology 7:41–47.

Rattner, B. A., and J. C. Franson. 1983. Methyl parathion and fenvalerate toxicity in American kestresl: acute physiological responses and effects of cold. Canadian Journal of Physiological Pharmacology 62:787–792.

Relyea, R. A. 2003. Predator cues and pesticides: a double dose of danger for amphibians. Ecological Applications 13:1515–1521.

Relyea, R. A. 2012. New effects of Roundup on amphibians: predators reduce herbicide mortality; herbicides induce antipredator morphology. Ecological Application 22:634–647.

Relyea, R. A., and N. Diecks. 2008. An unforeseen chain of events: lethal effects of pesticides on frogs at sublethal concentrations. Ecological Application 18:1728–1742.

Riley, S. P. D., R. M. Sauvajot, T. K. Fuller, E. C. York, D. A. Kamradt, C. Bromley, and R. K. Wayne. 2003. Effects of urbanization and habitat fragmentation on bobcats and coyotes in southern California. Conservation Biology 17:566–576.

Riley, S. P. D., C. Bromley, R. H. Poppenga, F. A. Uzal, L. Whited, and R. M. Sauvajot. 2007. Anticoagulant exposure and notoedric mange in bobcats and mountain lions in urban southern California. Journal of Wildlife Management 71:1874–1884.

Rohr, J. R., T. R. Raffel, S. K. Sessions, and P. J. Hudson. 2008. Understanding the net effects of pesticides on amphibian trematode infections. Ecological Application 18:1743–1753.

Rudd, J. L., S. C. McMillin, M. W. Kenyon, and D. L. Clifford. 2018. Prevalence of first and second-generation anticoagulant rodenticide exposure in California mountain lions (*Puma concolor*). Pages 254-257 in D. M. Woods, editor. Proceedings of the Vertebrate Pest Conference 28. University of California, Davis, CA, USA.

Serieys, L. E. K., T. C. Armenta, J. G. Moriarty, E. E. Boydston, L. M. Lyren, R. H. Poppenga, K. R. Crooks, R. K. Wayne, and S. P. D. Riley. 2015. Anticoagulant ro-

denticides in urban bobcats: exposure, risk factors and potential effects based on a 16-year study. Ecotoxicology 24:844–862

Serieys, L. E. K., A. J. Lea, M. Epeldegui, T. C. Armenta, J. Moriarty, S. VandeWoude, S. Carver, J. Foley, R. K. Wayne, S. P. D. Riley, and C. H. Uittenbogaart. 2018. Urbanization and anticoagulant poisons promote immune dysfunction in bobcats. Proceedings of the Royal Society B: Biological Sciences 285:20172533

Soderlund, D. M. 2010. Toxicology and mode of action of pyrethroid insecticides. Pages 1665-1686 in Hayes' Handbook of Pesticide Toxicology. Academic Press, Cambridge, MA, USA.

Stromborg, K. L. 1986. Reproduction of bobwhites fed different dietary concentrations of an organophosphate insecticide, methamidophos. Archives of Environmental Contamination and Toxicology 15:143–147.

State Water Resources Control Board (SWRCB). 2018. Proposed updates to the cannabis cultivation policy, principles and guidelines for cannabis cultivation. Available from https://www.waterboards.ca.gov/water_issues/programs/cannabis/cannabis_policy.html (May 2019).

Stone, W. B., J. C. Okoniewski, and J. R. Stedelin. 1999. Poisoning of wildlife with anticoagulant rodenticides in New York. Journal of Wildlife Diseases 35:187–193.

Stone, W. B., J. C. Okoniewski, and J. R. Stedelin. 2003. Anticoagulant rodenticides and raptors: recent findings from New York, 1998 – 201. Bulletin of Environmental Contamination and Toxicology 70:34–40.

Sweitzer, R. A., V. D. Popescu, C. M. Thompson, K. L. Purcell, R. H. Barrett, G. M. Wengert, M. W. Gabriel, and L. W. Woods. 2016. Mortality risks and limits to population growth of fishers. The Journal of Wildlife Management 80:438–451.

Thompson, C., R. Sweitzer, M. Gabriel, K. Purcell, R. Barrett, and R. Poppenga. 2013. Impacts of rodenticide and insecticide toxicants from marijuana cultivation sites on fisher survival rates in the Sierra National Forest, California. Conservation Letters 7:91–102.

van den Brink, N. W., J. E. Elliott, R. F. Shore, and B. A. Rattner. 2018. Anticoagulant rodenticides and wildlife: concluding remarks. USDA National Wildlife Research Center- Staff Publications. 2097.

Van Lier, R. B., and L. D. Cherry. 1988. The toxicity and mechanism of action of bromethalin: a new single-feeding rodenticide. Fundamental and Applied Toxicology 11:664-672.

Vidal, D., V. Alzaga, J. J. Luque-Larena, R. Mateo, L. Arroyo, and J. Viñuela. 2009. Possible interaction between a rodenticide treatment and a pathogen in common vole (*Microtus arvalis*) during a population peak. Science of the Total Environment 408:267–271.

Warner, R. E. 1994. Agricultural land use and grassland habitat in Illinois: future shock for midwestern birds? Conservation Biology 8:147–156.

Watt, B. E., A. T. Proudfoot, S. M. Bradberry, and J. A. Vale. 2005. Anticoagulant rodenticides. Toxicological Reviews 24:259–269.

Wolansky, M. J., and J. A. Harrill. 2008. Neurobehavioral toxicology of pyrethroid insecticides in adult animals: a critical review. Neurotoxicology and Teratology 30:55–78.