Ecological & Human Health Assessment Report

Project Name: Integrated Mosquito and Vector Management Programs for Nine Districts

Date: June 2013

Prepared for:

Alameda County Mosquito Abatement District
Alameda County Vector Control Services District
Contra Costa Mosquito and Vector Control District
Marin/Sonoma Mosquito Vector Control District
Napa County Mosquito Abatement District
Northern Salinas Valley Mosquito Abatement District
San Mateo County Mosquito and Vector Control District
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Acronyms

µg/cm^2	microgram per square centimeter
2,4-D	2,4-Dichlorophenoxy acetic acid
AA-ITU	Ae. aegypti International Toxic Unit
ACMAD	Alameda County Mosquito Abatement District
ACVCSD	Alameda County Vector Control Services District
ae	acid equivalent
AI	Active ingredient
ALS	acetolactate synthesis
AMPA	aminomethylphosphonic acid
APEs	Alkylphenol ethoxylates
ATP	adenosine triphosphate
BCF	bioconcentration factor
BMP	Best management practice
Bti	*Bacillus thuringiensis* subspecies *israelensis*
Cal/EPA	California Environmental Protection Agency
CCMVCD	Contra Costa Mosquito and Vector Control District
CDPH	California Department of Public Health
CDPR	California Department of Pesticide Regulation
CEQA	California Environmental Quality Act
CFU	colony forming units
CNS	central nervous system
DDVP	Dichlorvos
DOD	Department of Defense
EEC	expected environmental concentrations
EPSP	enzyme 5-enolpyruvylshikimate 3-phosphate
IPM	Integrated Pest Management
ITUs	International Toxic Units
LC	lethal concentration
LC50	median lethal dose
LD	lethal dose
LOAEC	lowest observed adverse effect concentration

MSMVCD	Marin/Sonoma Mosquito Vector Control District
MVCAC	Mosquito and Vector Control Association of California Coastal Region
NCMAD	Napa County Mosquito Abatement District
NCMAD	Napa County Mosquito Abatement District
NOAEC	no observed adverse effect concentration
NOAELs	No observed adverse effect level
NOEC	No Observable Effect Concentration
NSVMAD	Northern Salinas Valley Mosquito Abatement District
PBO	Piperonyl Butoxide
PEIR	Programmatic Environmental Impact Report
ppm	parts per million
Programs	Integrated Mosquito and Vector Management Programs
REDs	reregistration eligibility decisions
RUP	restricted-use pesticide
SCCVCD	Santa Clara County Vector Control District
SCMAD	Solano County Mosquito and Vector Control District
SMCMVCD	San Mateo County Mosquito and Vector Control District
TBEE	triclopyr butoxyethyl ester
TEA	triethylamine
TPA or di-acid	tetrachloroterephthalic acid
ULV	Ultra Low Volume
USEPA	U.S. Environmental Protection Agency
USFWS	U.S. Fish and Wildlife Service
UV	ultraviolet

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1 Executive Summary

The nine northern California Districts (members of the Mosquito Vector Control Association of California Coastal Region [MVCAC]) participating in this CEQA Compliance effort engage in mosquito and other vector control activities to protect the public health in their respective Program Areas. These activities are being evaluated for compliance with the California Environmental Quality Act (CEQA) based on current CEQA statutes and Guidelines and recent case law. This draft technical report provides an evaluation of the potential hazards (and estimated risks) of the pesticide application activities used, and in some cases, planned, for vector control. The information provided by the Districts has been synthesized and evaluated to identify any potential environmental concerns due to use of potentially hazardous chemicals. This evaluation was based on a review of the documented characteristics of each chemical, including the efficacy, mode of action, candidate target species, reported toxicity to humans and wildlife, and likely fate and transport under application conditions. The information in this report is intended to be used to support the preparation of a Programmatic Environmental Impact Report (PEIR). The term pesticide as used herein refers to all modes of chemical and biological control, including insecticides, herbicides, and adjuvants unless otherwise indicated.

Each of the nine Districts provided extensive information about their pesticide use in support of this Human and Ecological Health Impact Assessment including:

> Pesticides used by each District

> Types of application sites (e.g., habitat types)

> Number of treatments per application site

> Total amount used per treatment for each application site, based on seasonal uses

This information is summarized in Attachment A. This draft technical report provides results of the review and evaluation of 46 active ingredients used and potentially considered for future use by the Districts. The objective was to identify those that may pose potential human health or ecological concern when used by the Districts. Documented toxicity and environmental fate of the pesticides were reviewed and evaluated, based primarily on the active ingredient, and the results are summarized in Section 4-(Table 4-1).

The pesticide application scenarios that result in reasonable efficacy with minimal unwanted estimated risk are preferred and are the basis of Integrated Pest Management (IPM) approaches practiced by the Districts.

Using the available information about the active ingredients reviewed, there were several overarching parameters that are known to adversely impact risk. Primary factors considered include the inherent toxicity and mode of action of the chemical. Other important factors that are considered include the possible transport and fate of the chemical in various media, the reported likely exposure routes, and documented ecological and human studies supporting the toxicity data. Several important parameters, such as the retention time (half-life) in various media are also considered, but are dependent on specific conditions at the time of application. Based on these criteria, several pesticides received additional discussion during the MVCAC workshop with the Districts on February 20, 2013.

Using the approach discussed above, select active ingredients were identified (Table 1-1) and discussed during the workshop to supplement the information contained in Chapter 4-and relevant to the evaluation of potential risk. Each of these pesticides exhibits at least one parameter that appears to drive potential risk, and the Districts provided additional information on measures employed to minimize potential risk.

The toxicity and adverse effects information collected, reviewed and critiqued for each of the pesticide products evaluated in this document is based primarily on results of laboratory studies that are extrapolated to appropriate potential receptor species In assessing the toxicity information in this document, it should be remembered that most toxicity data are derived from rigidly controlled laboratory animal studies designed to determine the potential adverse effects of the chemical under several possible routes of exposure. In these studies, the species of interest is exposed to 100% chemical at several doses to determine the lowest concentration resulting in a predetermined adverse effect (LOAEL) on numerous physiological and behavioral systems. The second component of these tests is to determine the highest concentration of chemical that results in no measurable adverse effect (NOAEL).

However, these laboratory tests are designed to document the effects of the chemical when there is a continuous, controlled exposure and do not realistically reflect the likely exposures or toxicity in the field application scenarios. In the field, animals can move around, are able to make selections of food and prey and often avoid sprayed areas completely. As such, the toxicity information is intended as guidance for determining the “safe” levels of applications that would not adversely impact non-target species. Because the applications are conducted under rigid BMPs, using the minimum effective pesticide application concentrations and do not result in continuous exposures, these laboratory derived estimates of potential risk are not appropriate for the actual exposures and effects in the field.

Table 1-1 Active Ingredients Identified for Discussion

Active Ingredient	Vector	Potential Issue
Methoprene	Mosquitoes	Prevalent use; toxicity to aquatics and insects
Etofenprox	Mosquitoes	Toxicity to aquatic organisms; no synergist required
Bti	Mosquitoes	Prevalent use; public concerns
Pyrethrins	Mosquitoes	Prevalent use; requires synergist (PBO)
Resmethrin	Mosquitoes	Requires synergist (e.g., PBO); potential endocrine disruptor
Vegetable Oil (coconut oil)/mix	Mosquitoes	Contains low percentage of petroleum distillate
Permethrin	Mosquitoes/yellow jacket wasps	Toxicity to aquatic organisms; potential endocrine disruptor
Lambda-cyhalothrin	Yellow jacket wasp	Toxicity to aquatic organisms; potential to bioaccumulate
Bromadiolone	Rats	Toxicity to non-target organisms including mammals, birds, aquatics
Difethialone	Rats	Toxicity to non-target organisms including mammals, birds, aquatics
Alkylphenol ethoxylates	Weeds	Toxicity to aquatic organisms; Moderately bioaccumulative
Glyphosate	Weeds	Prevalent use; possible endocrine disruptor
Diuron	Weeds	Prevalent use; toxicity to freshwater fish
Benfluralin	Weeds	Toxicity to aquatics; potential for bioaccumulation/endocrine disruption

This document provides information in tables and appendices about the parameters used to evaluate 46 active ingredients and a summary of the ecological and human health issues that may indicate a potential concern when used for vector control.

2 Introduction

This report provides a Human and Ecological Health Assessment of pesticides and herbicides contained in the Integrated Mosquito and Vector Management Programs (Programs) for nine mosquito abatement and/or vector control districts in northern California. The nine districts are: Alameda County Mosquito Abatement District (ACMAD), Alameda County Vector Control Services District (ACVCSD), Contra Costa Mosquito and Vector Control District (CCMVCD), Marin/Sonoma Mosquito Vector Control District (MSMVCD), Napa County Mosquito Abatement District (NCMAD), Northern Salinas Valley Mosquito Abatement District (NSVMAD), San Mateo County Mosquito and Vector Control District (SMCMVCD), Santa Clara County Vector Control District (SCCVCD), and the Solano County Mosquito Abatement District (SCMAD). The Programs provide for mosquito and/or vector control activities within each District's Program Area. The nine District Program Areas include both the Program Areas within the Districts and the surrounding counties where the Districts may provide mosquito and/or other vector management services when requested.

The immediate nine District Program Areas are located in the following nine counties of the state: Alameda, Contra Costa, Marin, Monterey, Napa, San Mateo, Sonoma, Solano, and Santa Clara. Control activities may also be provided in areas adjacent to the District Program Areas upon request of the adjacent jurisdictions to protect the health and safety of residents in adjacent jurisdictions. Actions that would be taken outside of the nine Districts' Program Areas are the same types of actions undertaken within the Districts' Program Areas and in similar types of habitats or sites. Therefore, the nine District Program Areas addressed in this report also include the nine additional surrounding counties: Mendocino, Merced, Lake, Sacramento, San Benito, San Joaquin, Santa Cruz, Stanislaus, Yolo, and the portion of Monterey County south of the NSVMAD.

A health assessment has been conducted to evaluate the potential risks posed by the chemical treatments/pesticide and herbicide formulations to non-target organisms, including humans and sensitive ecological receptors. Pesticides and herbicides are handled separately but in parallel during the evaluation. The first level of investigation is a toxicity/hazards evaluation comprised of a comprehensive literature review for the active ingredients contained in the chemical products and formulations used for vector control. This evaluation was performed to support the Programmatic Environmental Impact Report (PEIR) for each District. Reviewed toxicity literature included peer-reviewed publications, California Department of Pesticide Regulation (CDPR) data and reports, United States Environmental Protection Agency (USEPA) reports, and California Department of Public Health (CDPH) reports. The processes employed for the evaluation and the selection of active ingredients to focus on are described below. Although this document is intended to address the active ingredients most likely to engender some perceived or real concern by the public and regulators, the report includes hazard information about all active ingredients representing products in use or planned by the Districts represented in this report.

In general, application scenarios employed by the Districts represented in this report include several basic, yet critical, Best Management Practices (BMP) to minimize and often negate any potential exposures that might result in unwanted adverse effects to non-target species. Additional integrated pest management (IPM) and BMP practices are utilized by each District.

The results of this assessment are based on assumptions about toxicity and mode of action derived from available information and data, including the published literature. In addition to the documented efficacy (toxicity) to target species and vectors, this assessment also considers the potential risk to sensitive non-target organisms inventoried in a variety of habitat types in the Northern California Coastal Region and presented in the MVCAC Biological Resources Technical Report. Application scenarios and other data were evaluated to estimate the potential for exposures to sensitive non-target ecological receptors. Some of the potential pesticide applications that might result in unacceptable estimated risk have been

identified. While the majority of the active ingredients representing products used by the Districts are openly available and do not suggest an unacceptable risk, this review addresses the information and assumptions about possible use patterns and possible exposure issues.

2.1 General Issues Associated with the Current Pesticide Use

The Districts currently employ a combination of methods in their IPM programs for vector control, including the application of a variety of pesticide formulations by several mechanisms. These formulations, registered by the USEPA and California Environmental Protection Agency (Cal/EPA) are methodically applied by the Districts to minimize potential impacts to non-target receptors. However, the potential for impact to humans and ecological receptors from these chemicals was examined in order to provide defensible evidence to support conclusions reached about their safety and proper use scenarios.

Application scenarios employed by the Districts represented in this report include several basic, yet critical, BMPs to minimize and often negate any potential exposures that might result in unwanted adverse effects to non-target species. Pesticide application safety is maximized by the Districts by:

> applications according to strict adherence to label instructions

> restricting applications to low wind conditions to minimize drift

> using Ultra Low Volume (ULV) applications whenever possible

> applications late at night when non-target species (e.g. bees, etc.) are not active

> observation and documentation of nearby water sources and adherence to buffer zones

> use of appropriate PPE by applicators and field crews

> careful reporting and tracking of all pesticide uses by the District

> applications only on an “as needed” or “as appropriate” basis

Additional IPM and BMP practices tailored for each District are also utilized.

The objective of this report is to address and evaluate the potential for human and ecological hazards of application scenarios of the active ingredients contained in a variety of products and formulations used by the Districts in their efforts to control and abate mosquito and other vector infestations. The review of toxicity literature and environmental fate information focused on developing a scientifically defensible summary of the safety of these applications. If some level of concern or perception about unintended effects of the applications results from the evaluation, approaches will be developed to mitigate or prevent real or perceived adverse effects, including BMPs.

2.2 Human and Ecological Health Evaluation

2.2.1 Human Health Impact Evaluation

Pesticides used by the nine Districts were investigated to provide a preliminary toxicity assessment related to potential impacts to humans. Pesticide formulations, label recommendations, and application procedures were also reviewed to evaluate the potential likelihood for bioaccumulation and/or food item biomagnification. For each pesticide and herbicide evaluated, written explanations are provided regarding the physiochemical characteristics of the product, including absorption, metabolism, and elimination; and any other specific reported evidence of reproductive, developmental, or carcinogenic effects.

2.2.2 Ecological Health Impact Evaluation

A general hazard evaluation was conducted for the pesticides and herbicides used by the nine Districts and reported application scenarios (alternatives). The potential impacts to representative invertebrates,

wildlife, fish, and aquatic plants were considered, and then evaluated using a hazard evaluation of the active ingredients.

The evaluation included a review and evaluation of the current toxicity literature (available field and lab studies) relevant to ecological receptor impacts, and information relevant to the ultimate environmental fate for these active ingredients. The behavior of these materials in the environment (including fate and transport) are evaluated for chemicals reportedly handled by Districts' staffs. The potential effects could be caused by the active ingredients' mechanism of action (potential toxicity), as well as the potential for bioconcentration, bioaccumulation, or biomagnification.

The hazard and risk evaluations of the pesticides and herbicides used by the nine Districts were conducted in separate, but related phases. In the first phase, the objective was to determine and document the chemical characteristics and potential for adverse impacts when used as intended by the Districts. During the subsequent evaluation for the PEIR, this will be accomplished using a tiered sorting technique in which each chemical (active ingredient) will be ranked according to several criteria: 1) The documented toxicity of the active ingredient as indicated in the open literature; 2) The target species by vector; 3) The non-target species and biota that are likely to be exposed; 4) The likely media to be exposed for each chemical; and 5) Possible sensitive seasonal exposure conditions (Figure 1).

2.3 Initial Approach

The evaluations conducted for each active ingredient provide a general indication of the potential for human or ecological risk and possible adverse effects to non-target organisms.

This approach was used to develop the list of chemicals used by each District that should be of little or no concern when used according to product labels and to identify those (if any) that may be problematic in certain use scenarios. Pesticides are first reviewed for target vector efficacy, based on both documented laboratory and field studies. Pesticide efficacy is of prime importance in the evaluations, but efficacy is contrasted to potential adverse impacts in the determination of the safety of use. To provide an indication of the possible adverse effects of each pesticide, the characteristics of its application scenarios are scored for relative "safety".

> Pesticides proposed for use with low potential exposure to people including sensitive populations (i.e., children, the elderly).

> Pesticides proposed for use with very low or minimal toxicity (hazard) based on the above analyses.

> Pesticides with the least potential for toxicity when used in or near important habitats for sensitive or non-target species.

> Pesticides showing little or no extraordinary seasonal potential impacts.

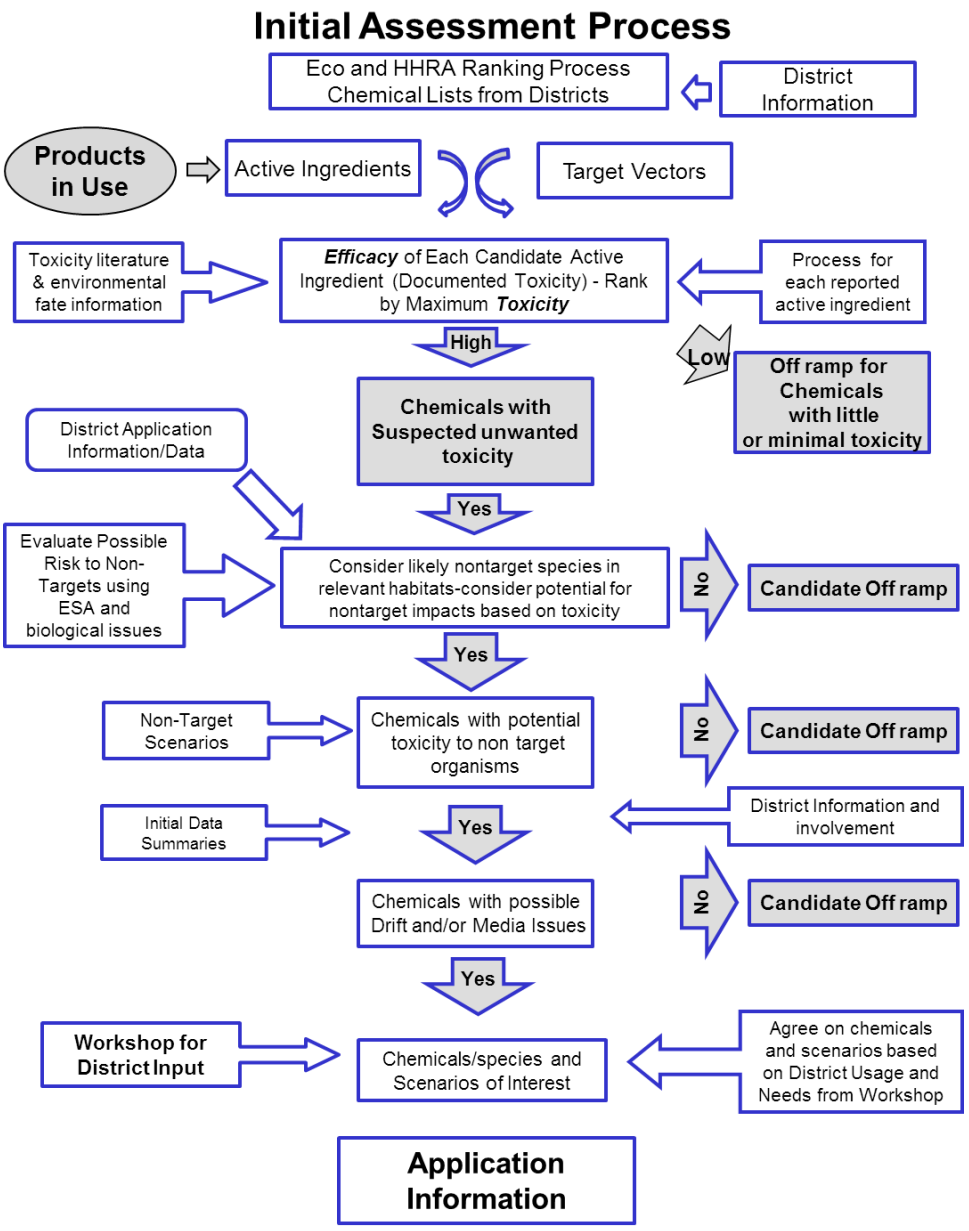

Figure 1 Process to Identify Chemicals for Further Discussion

2.3.1.1 Components of a Review

2.3.1.1.1 Problem Formulation

> Ecological effects characterization

> Identification of the environmental setting and pesticide of interest

> Characterization of pesticide mechanism of action

> Possible transport pathways

> Categories of receptors likely affected

> Identification of application scenarios provided by the Districts

2.3.1.1.2 Exposure Estimates

> Assumptions about potential exposures, including extent, timing and quantity

> Assumptions about potential species that might be exposed

2.3.1.1.3 Basic Risk Estimates

This information was used to:

> Identify likely uncertainties in the exposures

> Develop ranges of potential effects using “what if” parameter estimates.

> Determine estimate of risk needed to collect more information

The results of the evaluation for each of the pesticides of interest were used to qualitatively assess the potential for adverse effects of each active ingredient and select candidates for additional evaluation and characterization. These results are summarized immediately following discussion of environmental fate and toxicity for each pesticide throughout Section 4. Final conclusions and recommendations are included in Section 5.

The pesticide application scenarios that should result in low or “acceptable” results for the estimated risk will remain in the suite of potential control options while those with higher estimated risk estimates are identified. The process is graphically described in the following flow diagram (Figure 2).

At the conclusion of the initial evaluations, and in conjunction with the District managers, the results and possible recommendations for use scenarios based on acceptable risk estimates were the topic of a workshop conducted on February 20, 2013. The objective of the workshop was to discuss, review, and agree to the list of the active ingredients that should be included in the report and information about many of the BMPs used.

Safety evaluations generally follow the USEPA guidance (Figure 2) for pesticide evaluations, with a focus on the relevant uses and exposures identified and agreed upon during the Workshop.

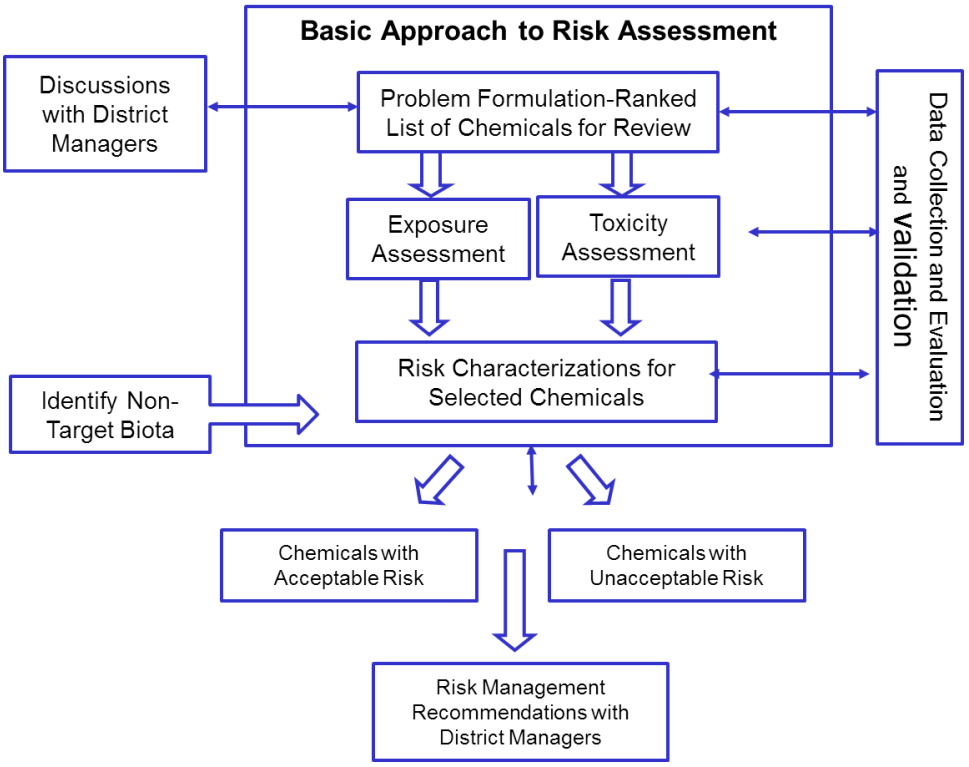

Figure 2 Process Used by USEPA to Evaluate Potential Risk for Chemicals

2.4 Approach to Refinement

The pesticide application scenarios that result in low or acceptable estimated risk as determined in the initial review of hazard information were categorized as lower potential risk, while others with higher potential estimated risk are identified and discussed with a focus on District-specific information about BMP employed by staff and applicators. The process includes the following:

> Development of the relationships that describe the type of pesticides/herbicides used by the Districts against the target pest(s), the locations of intended uses (water bodies, grasses, forests, urban, residential, etc.), and the likely human and wildlife populations that might be inadvertently exposed.

> Evaluation of the inherent hazard (toxicity) of the pesticide(s)/herbicide(s) to non-target receptors (humans and wildlife).

> As a subsequent task in the PEIR, consider the potential exposure of humans and non-target species to each of the pesticides/herbicides based on the application scenarios, the concentrations used, and the likelihood that the application will reach or contact any populations of concern.

> The information in the PEIR provides estimates of potential “risk” and possible safety issues for each of the typical pesticide application scenarios.

3 Vector Control Chemical Categories

The chemical products reported by the Districts, application scenarios, and potential new products and formulations for future use were obtained during project initiation and preparation of the PEIR Project Description. Application information provided by the Districts included the number of treatments and total amount applied of each product to specific habitat types during four quarters from Summer 2011 through Spring 2012 (see Attachment A). These data were integral for elucidating the estimated loading and potential exposures to different habitats that support non-target organisms. The evaluation focused on the active ingredients of the products/formulations. The target organisms and primary modes of action for the pesticide and herbicide active ingredients are described below. Tables 3-1 and 3-2 present the pesticide (non-herbicide) and herbicide products, respectively that are currently in use containing active ingredients reported here and the number of Districts using that Product.

Table 3-1 Pesticide Products Containing Reported Active Ingredients

Product	Active Ingredient	Vector	Number of Districts
Agnique MMF	Biodegradable Alcohol Ethoxylated Surfactant	Mosquito	6
Agnique MMF G	Biodegradable Alcohol Ethoxylated Surfactant	Mosquito	3
Altosid Briquets	Methoprene	Mosquito	4
Altosid Briquets 30-Day	Methoprene	Mosquito	2
Altosid Liquid Larvicide	Methoprene	Mosquito	2
Altosid Liquid Larvicide Concentrate	Methoprene	Mosquito	4
Altosid Liquid SR5	Methoprene	Mosquito	3
Altosid Liquid SR20	Methoprene	Mosquito	1
Altosid Pellets	Methoprene	Mosquito	7
Altosid Pellets WSP	Methoprene	Mosquito	3
Altosid SBG Single Brood Granule	Methoprene	Mosquito	2
Altosid XR-Briquets	Methoprene	Mosquito	7
Altosid XR-G	Methoprene	Mosquito	4
Astro®, Ortho® products, Bonide® products, Tengard® products, etc.	Permethrin	Yellow jacket wasp	1
Bell Terad 3 Blox	Cholecalciferol	Rat	1
BVA-2	Petroleum Distillate	Mosquito	7
Clarke Biomist 4-+ 12 ULV	Permethrin and Piperonyl Butoxide (PBO)	Mosquito	1
Contrac 8 oz blk	Bromadiolone	Rat	1
Contrac All-Weather Blox	Bromadiolone	Rat	2
Contrac Super Blox	Bromadiolone	Rat	2
Delta Dust	Deltamethrin	Yellow jacket wasp	2

Table 3-1 Pesticide Products Containing Reported Active Ingredients

Product	Active Ingredient	Vector	Number of Districts
Ditrac Blox	Diphacinone	Rat	2
Ditrac Tracking Powder	Diphacinone	Rat	1
Drione	Pyrethrin and Piperonyl Butroxide and Amorphous Silica Gel	Yellow jacket wasp	5
EcoExempt IC2	Rosemary Oil	Mosquito	1
FirstStrike Soft Bait	Difethialone	Rat	2
FourStar 180 Bs/Bti	*Bacillus sphaericus and Bacillus thuringiensis israelensis*	Mosquito	4
FourStar 45 Bti	*Bacillus sphaericus and Bacillus thuringiensis israelensis*	Mosquito	4
FourStar 90 briq	*Bacillus sphaericus and Bacillus thuringiensis israelensis*	Mosquito	2
FourStar SBG	Dithiopyr	Weed	1
Golden Bear 1111	Aliphatic Petroleum Hydrocarbons	Mosquito	4
Kontrol 4-4	Permethrin and PBO	Mosquito	1
MetaLarv SP-T	Methoprene	Mosquito	1
MGK Pyrocide 7396	Pyrethrins and PBO	Mosquito	4
Natular 2EC	Spinosad	Mosquito	2
Natular G30	Spinosad	Mosquito	3
Natular XRT	Spinosad	Mosquito	1
Permanone	Permethrin and PBO	Mosquito	1
Pyrenone 25-5	Pyrethrins and PBO	Mosquito	5
Pyrocide Mosquito Adulticiding Concentrate for ULV Fogging 7067	Pyrethrins and PBO	Mosquito	1
Scourge 18% + 12%*	Resmethrin and PBO	Mosquito	1
Skeeter Abate	Temephos	Mosquito	2
Spectracide Pro®	Tetramethrin and Permethrin and PBO	Yellow jacket wasp	1
Spectracide®	Prallethrin and Lambda-cyhalothrin	Yellow jacket wasp	1
Summit B.T.I. Briquettes	*Bacillus thuringiensis israelensis*	Mosquito	1
Teknar HP-D	*Bacillus thuringiensis israelensis*	Mosquito	2
Teknar SC	*Bacillus thuringiensis israelensis*	Mosquito	1
VectoBac 12AS	*Bacillus thuringiensis israelensis*	Mosquito	5
VectoBac G	*Bacillus thuringiensis israelensis*	Mosquito	5
VectoBac GS Biological Larvicide	*Bacillus thuringiensis israelensis*	Mosquito	1

Table 3-1 Pesticide Products Containing Reported Active Ingredients

Product	Active Ingredient	Vector	Number of Districts
VectoBac Technical Powder Biological Larvicide	*Bacillus thuringiensis israelensis*	Mosquito	1
VectoBac WDG Biological Larvicide	*Bacillus thuringiensis israelensis*	Mosquito	1
VectoLex CG	*Bacillus sphaericus*	Mosquito	7
VectoLex WDG	*Bacillus sphaericus*	Mosquito	4
VectoLex WSP	*Bacillus sphaericus*	Mosquito	5
VectoMax CG	*Bacillus sphaericus and Bacillus thuringiensis israelensis*	Mosquito	3
VectoMax WSP	*Bacillus sphaericus and Bacillus thuringiensis israelensis*	Mosquito	2
Wasp-Freeze	Phenothrin and Trans Allethrin	Yellow jacket wasp	3
Zenivex E20	Etofenprox	Mosquito	1

Table 3-2 Herbicide Products Containing Reported Active Ingredients

Product	Active Ingredient	Vector	Number of Districts
Alligare Dithiopyr 40	dithiopyr	Weed	1
Alligare Glyphosate 4-Plus	glyphosate	Weed	1
Alligare Glyphossate 5.4	glyphosate	Weed	1
Alligare Imazapyr 2 SL	imazapyr	Weed	1
Alligare Oryzalin 4	oryzalin	Weed	1
Alligare Triclopyr 3	triclopyr	Weed	1
AMVAC Dacthal	DCPA	Weed	1
Aquamaster	Glyphosate	Weed	3
Balan	Benefin	Weed	1
Blazon Pattern Indicator	Polymeric Colorant (proprietary)	Weed	2
Buccaneer	Glyphosate	Weed	1
BullsEye Pattern Indicator	Proprietary Colorant	Weed	2
Competitor	Modified Vegetable Oil	Weed	2
Dacthal	DCPA	Weed	1
Dimension Ultra 40WP	dithiopyr	Weed	1
Ecomazapyr 2 SL	imazapyr	Weed	1
Garlon-3A	Triclopyr	Weed	1
Green Light Amaze XL 2G	Benefit, Oryzalin	Weed	2
Habitat	Imazapyr	Weed	2

Table 3-2 Herbicide Products Containing Reported Active Ingredients

Product	Active Ingredient	Vector	Number of Districts
Imazapyr 4-SL	imazapyr	Weed	1
Karmex XP	Diuron	Weed	1
Liberate	Lecithin, methyl esters of fatty acids, alcohol ethoxylate	Weed	1
Monterey Nutgrass “Nihilator”	Bentazon (sodium salt)	Weed	1
Monterey Turflon Ester, * Turflon, * Garlon 3®, * Renovate®	Triclopyr	Weed	1
Monterey Weed Whacker	2,4-DP (dimethylamine salt)	Weed	1
MOR-ACT	Paraffin base petroleum oil	Weed	1
MSO	Methylated seed oil of Soybean	Weed	1
No Foam A	Alkyl Phenol Ethoxylate / Isopropanol	Weed	1
No Foam Defoamer	Polydimethylsiloxane & Silicon	Weed	1
Oust XP	Sulfometuron Methyl	Weed	2
Pennant Magnum	Metolachlor	Weed	1
Polaris	Imazapyr	Weed	2
Pro-Spreader Activator	Alkyl Phenol Ethoxylate / Isopropanol	Weed	2
R-11 Spreader Activator	Alkyl Phenol Ethoxylate / Butyl alcohol	Weed	1
Renovate 3	triclopyr	Weed	1
Reward	Diquat dibromide	Weed	1
Roundup Pro Max®	Glyphosate	Weed	2
Roundup Pro®	Glyphosate	Weed	3
Roundup®	Glyphosate	Weed	1
Roundup®, Rodeo®	Glyphosate	Weed	1
RoundupMax	Glyphosate	Weed	1
Scotts Halts Crabgrass Preventer	Pendimethalin	Weed	1
Trimec Lawn Weed Killer, * Spectricide® Weed Stop®, * Ortho® Weed-b-Gon®, * Weed Killer for Lawns, * Bayer Advanced™ Southern Weed Killer	2,4-D (2,4-dichlorophenoxy acetic acid)	Weed	1
Tripleline Foam-Away	Polydimethylsiloxane	Weed	1
Turf Trax Blue	Polymeric Colorant (proprietary)	Weed	1
Turfgro NIS	Ethanol 2,2-oxybis	Weed	1
Vigoro Crabgrass Preventer, * Monsanto Dimension®	Dithiopyr	Weed	1

3.1 Mosquito Control

3.1.1 Adulticides

Adulticides are generally applied as aerosols using ultra-low-volume (ULV) techniques. Aerial and ground application techniques are used to distribute the insecticides. Adulticide treatments are most frequently timed to correspond with mosquito activity (flying) when exposure is greatest to the insecticide aerosol mist (dusk to dawn) and granular material. In addition, residual barrier treatment applications are used in mosquito resting areas and migratory stops. These treatments are usually applied as large liquid droplets with a sprayer during daylight hours. The primary objective of this type of treatment is the temporary prevention of re-infestation.

3.1.1.1 Pyrethroids

Pyrethroids are synthetic analogs of pyrethrins (from the Chrysanthemum plant) and have similar neurological effects on target organisms. These compounds cause rapid mortality of adult mosquitoes by interfering with sodium channel function in the nervous system.

3.1.2 Larvicides

Larvicides are used to manage immature life stages of mosquitoes including larvae and pupae in aquatic habitats. Temporary aquatic habitats are usually targeted because permanent water bodies generally support natural mosquito predators such as fish. The larvicides are applied using ground application equipment, fixed wing aircraft and rotary aircraft.

3.1.2.1 Contact Pesticides

(S)-Methoprene is a hormone analogue that interferes with insect larval development (growth regulator). This chemical does not exhibit the nonspecific target effects of neurological toxins such as pyrethrin.

Spinosad is a natural insecticide derived from the fermentation of a common soil microorganism, *Saacharopolyspora spinosa*. Spinosad alters nicotine acetylcholine receptors in insects causing constant involuntary nervous system impacts ultimately leading to paralysis and death.

3.1.2.2 Surface Active Agents

Petroleum- and plant-based (ethoxylated isotearyl alcohols) oils are used as surface-active agents effective against larvae and pupae. These oils are effective against these immature life stages when inhaled at the water surface or by physically forming a surface film that drowns the mosquito. These treatments may also be effective against adult mosquitoes during adult emergence.

3.1.2.3 Stomach Toxins

Bacterial larvicides such as Bti (*Bacillus thuringiensis israelensis*) and Bs (*Bacillus sphaericus*) are highly selective (for mosquitoes) microbial pesticides that when ingested, produce gut toxins that cause destruction of the insect gut wall leading to paralysis and death. These microbial agents are delivered as endospores in granular, powder, or liquid concentrate formulations.

3.2 Other Vector Control

There are a variety of pesticides used for the control of vectors including rats, ticks, yellow jackets wasps, and weeds.

3.2.1 Rats

Toxic baits may be used to achieve adequate control of rats when populations become too large to impact using traps. Federal EPA changes to rodenticide regulations occurred in 2011 (http://www.epa.gov/pesticides/mice-and-rats/) in an effort to reduce the hazard to wildlife, pets and children. The use of baits is

confounded by the potential for food web transfer of the bait to other trophic level receptors that might also encounter and eat the raw bait or predate an animal that has ingested the bait.

3.2.1.1 Anticoagulant Rodenticides

Anticoagulant rodenticides cause fatal internal bleeding by thinning the animal's blood and preventing clotting. Two groups of anticoagulants exist including the older "first-generation" compounds effective if consumed over multiple doses and the newer "second-generation" compounds, which are fatal after a single dose. The acute toxicity of second-generation rodenticides presents a greater hazard to wildlife, pets, and children. Products containing second-generation active ingredients are no longer permitted to be sold to the general public. These products remain available to professional pest control personnel, however.

3.2.1.2 Other Rodenticides

Three other rodenticides are available for use in California. Bromethalin and cholecalciferol are chronic rodenticides and achieve successful results similar to those of anticoagulants. Multiple feeding doses are required to induce mortality of rodents. Bromethalin is a neurotoxin, which damages the central nervous system (CNS). Cholecalciferol produces hypercalcemia leading to renal failure and CNS depression, among other generalized symptoms of toxicity. Zinc phosphide is an acute toxicant and causes death within a few hours of consumption. Often, use of this compound requires "pre-baiting" prior to addition of the chemical to rat bait in order to achieve adequate bait acceptance. Zinc phosphide is used to lessen impact on predators in the food web.

3.2.2 Yellow Jacket Wasps

Aerosol insecticides can be effective when applied directly to yellow jacket wasp nest openings. Most conventional pesticides are either pyrethrin or pyrethroids. Synthetic pyrethroid insecticides act as sodium channel modulators and very effective when used against wasps. Pyrethrin compounds act as paralytics and will immobilize the insect temporarily and may cause mortality.

Short-residual pyrethroids include allethrin, penothrin, resmethrin, sumithrin, and tetramethrin. Longer-lasting pyrethroid insecticides include lambda-cyhalothrin, deltamethrin, and permethrin.

3.2.3 Tick Control

Although tick surveillance is the recommended method to monitor this vector, there are several pesticides that can be useful if an unwanted tick infestation should occur. Ticks (e.g., deer ticks) act as vectors for bacterial pathogens, such as *Borrelia burgdorferi*, the agent of Lyme disease. Currently, deltamethrin is the only active ingredient employed for tick control.

3.2.4 Weed Control

Herbicides are classified in several ways. Pre-emergent herbicides are applied to the soil to prevent seedlings from germinating and emerging. Post-emergent herbicides are applied after seedlings have emerged and control actively growing plants via contact damage or systemic impacts. Contact herbicides cause physical injury to the plant upon contact. Systemic herbicides damage the internal functioning of the plant.

3.2.4.1 Herbicides for Broadleaves

Herbicides for use against annual broadleaf weeds are generally post-emergent applications that affect the plant systemically. 2,4-Dichlorophenoxy acetic acid (2,4-D), imazapyr, triclopyr, sulfometuron methyl, bentazon, diuron, oryzalin, DCPA, dithiopyr, and pendimethalin are examples of broadleaf herbicides.

3.2.4.2 Herbicides for Grass Weeds

Herbicides used against annual grasses (e.g. crabgrass, foxtail, etc.) are pre-emergent applications containing ingredients such as pendimethalin. Weed grasses can be treated with post-emergent applications; however, these tend to be less effective than pre-emergent treatments. Some can be eliminated with spot treatments of potent, nonselective herbicides such as glyphosphate, which act systemically by inhibiting the synthesis of the aromatic amino acids phenylalanine, tyrosine, and tryptophan.

3.2.4.3 Herbicides for Sedges

Spot treatments with glyphosphate are also useful in eliminating sedges (e.g. yellow and purple nutsedge, green kyllinga, etc.). Pre-emergent materials such as DCPA are effective at killing seeds of green kyllinga, but ineffective against nutsedges.

3.2.4.4 Aquatic Invasive Species

Imazapyr is an imidazolinone herbicide (e.g., Habitat®) that inhibits acetolactate synthesis (ALS), an enzyme necessary for the production of essential amino acids in plants. This class of chemicals includes systemic, nonselective, pre- and post-emergent herbicides used for the control of terrestrial and aquatic weeds (e.g., imidazolinoes, pyrimidinyl thiobenzoates, sulfonylureas, sulfonyl amino carbonyl triazolinone, and triazolopyrimidines). In California, these compounds have been used to combat the invasive purple loosestrife plant in aquatic environments. Unfortunately, this species and others appear to have developed resistance to the ALS-inhibiting family of chemicals. In addition, the non-target impacts of these compounds may cause negative effects to threatened or endangered plants.

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4 Evaluation of Active Ingredients-Results

This section presents the supporting information and results of the evaluation that will be incorporated into the MVCAC EIR. Information below includes a description of the chemical compound, general pesticide use/application techniques, mode of action, toxicity, environmental fate and transport, and potential impact assessment.

Several insecticides were previously evaluated in the Monitoring Plan for Mosquito Larvicides and Adulticides (2011) prepared by MVCAC. Descriptions of these compounds, pesticide use patterns, and environmental fate and transport were updated in this document as needed. Source information for the fate and transport data include USEPA reregistration eligibility decisions (REDs), USEPA risk evaluations, DPR fate reviews, fate reviews from the scientific community, and data provided by manufacturers. Toxicity information was included for select compounds, including details relevant to ULV applied mosquito adulticides. Table 4-1 provides a summary of the toxicity and fate and transport information associated with the active ingredients. In addition, toxicity values for a variety of receptors (human and ecological) are presented later in Chapter 6 in Table 6-1.

The toxicity data included in the numerous tables and charts in this document are generally derived from rigidly controlled laboratory animal studies designed to determine the potential adverse effects of the chemical under several possible routes of exposure. In these studies, the species of interest is exposed to 100 percent chemical at several doses to determine useful information such as the lowest concentration resulting in a predetermined adverse effect (LOAEL) on numerous selected physiological and behavioral systems. The second component of these tests is to determine the highest concentration of chemical that results in no measurable adverse effect (NOAEL).

However, these, and other, coordinated and focused laboratory tests are designed to document the effects of the chemical when a continuous, controlled, exposure exists and do not realistically reflect the likely exposures or toxicity in the District field application scenarios. As such, the toxicity information is intended as an overview of potential issues and guidance for understanding the completely “safe” maximum exposure levels of applications that would not adversely impact humans or nontarget plant and animal species.

Although the regulatory community uses this basic information to provide a relative comparison of the potential for a chemical to result in unwanted adverse effects and this information is reflected in the approved usage labels and MSDSs, in actual practice, the amounts actually applied in the District’s Program Area are substantially less than the amounts used in the laboratory toxicity studies. Because of the large safety factors used to develop recommended product label application rates, the amount of chemical resulting in demonstrated toxicity in the laboratory is much higher than the low exposure levels associated with an actual application. The application concentrations consistent with the labels or MSDSs are designed to be protective of the health of humans and other nontarget species (i.e., low enough to not kill them, weaken them, or cause them to fail to reproduce). However adverse effects may still occur to some non-target organisms.

Chapter 4-provides the results of our review, evaluation, and synthesis of data for each of the selected pesticides in use by the nine Districts. The analysis provides informative results for the Districts that are interested in an evaluation of the potential efficacy and effects of their respective pesticide treatment scenarios. In each case, the evaluations include consideration of four primary parameters:

> Efficacy to target vectors;

> Documented acute and chronic ecological and human toxicity (where available);

> Known media/habitat use scenarios for each pesticide and for each District; and

> Evaluation of potential non-target biota that might be adversely impacted and the associated exposure level.

Using these parameters, the process results in a functional evaluation of the likelihood that each pesticide application scenario (scenarios) could be used safely or otherwise. At the conclusion of each evaluation process, those scenarios that appear to result in potential unwanted (adverse) impacts were subjected to additional evaluation based on generally accepted “risk evaluation” guidelines.

4.1 Pyrethrin, Pyrethroids, Pyrethroid-like Compounds and Synergists

Pyrethroid insecticides are synthetic compounds that are chemically similar to the pyrethrins but have been modified to increase stability and activity against insects. Some synthetic insecticides are similar to pyrethroids, such as etofenprox, but have a slightly different chemical composition. First generation or “Type I” photosensitive pyrethroids include d-allethrin, phenothrin (sumithrin), prallethrin, resmethrin, and tetramethrin. Typically, these pyrethroids are used indoors and around residential areas. The newer second-generation pyrethroids are mostly “Type II” pyrethroids. Chemically, Type II pyrethroids are distinguished from Type I pyrethroids by the presence of an α-cyano group in their structure. The active ingredients that fall into this group include deltamethrin, esfenvalerate, lambda-cyhalothrin, and permethrin. Type II pyrethroids are more toxic (than Type I pyrethroids) because they are less photosensitive and persist longer in the environment.

Pyrethroids affect insect neuroactivity by binding to a protein at the nerve fiber that regulates the voltage-gated sodium channel. This can delay the closing of sodium channels and/or cause a persistent activation of the sodium channels. This often results in repetitive activity (Type I pyrethroid) or blockage of nerve conduction (Type II pyrethroid).

4.1.1 Pyrethrins

Pyrethrins are naturally occurring products distilled from the flowers of Chrysanthemum species. Pyrethrins are composed of a mixture of six compounds: pyrethrin I and II, cinerin I and II, and jasmolin I and II. Pyrethrins are contact poisons that can quickly penetrate the neural system. Pyrethrins act by causing a persistent activation of the sodium channels on insect neurons. Although pyrethrins have an effective “knockdown” action (induction of temporary paralysis), they do not necessarily have high killing properties alone. In order to delay the metabolic action (inhibition of microsomal enzymes) so that a lethal dose is assured, the synergist piperonyl butoxide (PBO) is added to mosquito adulticides (USEPA 2006f).

Pyrethrins were first registered in the U.S. for use as an insecticide in the 1950s. Pyrethrins are used on many agricultural crops; on livestock and animal husbandry premises; for treatment of commercial and industrial facilities and storage areas where raw and processed food/feed commodities are stored or processed; and for wide-area mosquito abatement in areas that include aquatic habitats. They are also used on outdoor household areas, pastureland, aquatic area or standing water, and for hospitals, recreational areas, ULV applications, and mosquito abatement programs (USEPA 2006f, CDPR 2010a).

4.1.1.1 Environmental Fate

The major routes of dissipation for pyrethrins in the environment are photolysis (both in water and soil, with half lives of less than 1 day in both cases) and to a lesser degree, aerobic soil metabolism (Table 4-2). Hydrolysis under alkaline conditions is an important route of dissipation for pyrethrins in water (half-life at pH 9 is 14-to 17 hours); however, this reaction appears to be relatively slow under neutral or acidic conditions, which are more likely to occur in the environment. Pyrethrins are likely to persist under anaerobic conditions. Pyrethrins quickly adsorb to suspended solids in the water column, and partition into the sediment. They adsorb strongly to soil surfaces and are generally considered immobile in soils; therefore, the potential to leach into groundwater is considered low (USEPA 2006f).

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Pyrethrins	> na	> Major route of dissipation is photolysis. > Quickly adsorb to suspended solids in the water column and partition to the sediment.	> Major route of dissipation is photolysis. > Pyrethrins are likely to persist under anaerobic conditions. > Generally immobile in soils, therefore the potential to leach to groundwater is low.	> Insecticide: > Naturally occurring products distilled from Chrysanthemum spp. flowers. > Contact poisons that act by causing persistent activation of the sodium channels on insect neurons resulting in “knock-down” agent. > The synergist PBO is added to ensure a lethal dose.	> Low to moderate acute toxicity via the oral, dermal, and inhalation routes. > Chronic exposure effects include neurobehavioral, thyroid, and liver effects.	> Very highly toxic to freshwater fish and invertebrates. > Practically nontoxic to birds.
Allethrins and d-trans allethrin	> When used in coils and mats, allethrins are released into the air where they will be degraded by sunlight or be distributed in low concentrations to nearby surfaces.	> Not water soluble. > Photolysis half-life is <8 hrs.	> Adheres moderately to soil containing organic matter.	> Insecticide: Synthetic pyrethroid structurally similar to cinerin I in naturally occurring pyrethrum. > d-trans stereoisomer is the most insecticidally active. > Typically used as a “knock-down” agent. > Synergists such as PBO are added to ensure a lethal dose.	> Slightly to moderately acute toxicity via oral, dermal, and inhalation routes. > Toxicity varies with the amounts of different isomers present. > Not known to cause reproductive, teratogenic, mutagenic, or carcinogenic effects to mammals.	> Highly toxic to fish and invertebrates. > Very toxic to non-target insects. > Practically nontoxic to birds. > Bioaccumulation potential is unknown.
Phenothrin (sumithrin or d-phenothrin)	> na	> Low water solubility. > Major routes of dissipation are photolysis and anaerobic metabolism.	> High affinity for binding to soils and moderate persistence in surface soils. > Low leaching potential, therefore phenothrin is relatively immobile in soils or sediments. > Moderately persistent under aerobic conditions and persistent under anaerobic conditions.	> Insecticide: Adult mosquitoes.	> Low acute toxicity by oral, dermal, and inhalation routes. > Mild eye irritant but not a skin irritant.	> Highly toxic to fish and freshwater invertebrates. > Practically nontoxic to birds.
Prallethrin	> na	> Major route of dissipation is photolysis.	> Major route of dissipation is photolysis. > Readily sorbs to soils and sediments.	> Insecticide: Synthetic pyrethroid with fast “knock-down” activity against insect pests. > Has a neural exciting effect on mosquitoes. > In California, prallethrin is combined with phenothrin in the product Duet (the only prallethrin adulticide used in California).	> Low to moderate acute toxicity via oral, dermal, or inhalation routes. > Mild eye irritant but not a skin irritant.	> Highly toxic to fish and freshwater invertebrates. > Practically nontoxic to birds. > Very toxic to honey bees. > Low toxicity to algae.
Deltamethrin	> na	> Degrades via hydrolysis, photolysis, and microbial action. > May persist in aquatic environments, particularly in the sediment.	> Most persistent in soils with high clay or organic matter content. > Moderately to highly persistent in terrestrial environments.	> Insecticide: Synthetic pyrethroid. > Induce long-lasting inhibition of the sodium ion channel activation gate, resulting in repetitive nerve signals in sensory organs, nerves, and muscles.	> Low to moderate acute toxicity via oral, dermal, or inhalation routes. > Chronic exposure of humans results in choreoathetosis, hypotension, prenatal damage, and shock. > No reported teratogenic, mutagenic, or carcinogenic effects.	> Very highly toxic to fish and invertebrates. Potential bioaccumulation in fish. > Practically nontoxic to birds. > Nonselective insecticide and is highly toxic to non-target insects, including honey bees.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Esfenvalerate	> na	> Practically insoluble in water. > Extremely hydrophobic. > When present in surface water, expected to be bind to suspended particulates and organic matter. > Degrades via photolysis and aerobic metabolism.	> Strong tendency to bind to soil. > Relatively immobile in soil and has low tendency to leach. > Degrades via photolysis and aerobic metabolism.	> Insecticide: > Broad spectrum, nonselective, voltage-dependent sodium-channel agonist. > Causes repetitive firing of neurons.	> Moderately toxic via acute routes. > Possible endocrine-disruptor.	> Highly toxic to fish and aquatic invertebrates. Bioaccumulates rapidly in fish. > Moderately toxic to birds. > Highly toxic to honey bees.
Lambda-cyhalothrin*	> na	> Extremely hydrophobic and rapidly adsorbs to soils and sediments. > Primary degradation pathways include photolysis and aerobic metabolism.	> Primary degradation pathways include photolysis and aerobic metabolism.	> Insecticide: Synthetic pyrethroid. > Induces long-lasting inhibition of the sodium ion channel activation gate, resulting in repetitive nerve signals in sensory organs, nerves, and muscles.	> Moderately toxic via acute oral, dermal, and inhalation routes. > Mild eye irritant but not a skin irritant.	> Highly toxic to fish. Potential to bioaccumulate in fish. > Low toxicity to birds. > Highly toxic to honey bees.
Resmethrin	> na	> Primary degradation pathways include photolysis and aerobic metabolism.	> Primary degradation pathways include photolysis and aerobic metabolism. > Low mobility in soil/sediments. > Environmentally persistent in absence of light.	> Insecticide: Synthetic pyrethroid. > Induces long-lasting inhibition of the sodium ion channel activation gate, resulting in repetitive nerve signals in sensory organs, nerves, and muscles.	> Low toxicity via acute oral, dermal, and inhalation routes. > Possible endocrine-disruptor.	> Highly toxic to fish and aquatic invertebrates.
Tetramethrin	> na	> Not persistent in the environment. > Decomposes rapidly by photolysis and hydrolysis in shallow, nonturbid water.	> Slightly mobile in soil.	> Insecticide: Synthetic pyrethroid. > Induces long-lasting inhibition of the sodium ion channel activation gate, resulting in repetitive nerve signals in sensory organs, nerves, and muscles.	> Slightly toxic via acute oral and dermal routes. > Possible human carcinogen.	> Highly toxic to fish and aquatic invertebrates. > Practically nontoxic to birds. > Highly toxic to honey bees.
Permethrin*	> na	> Hydrophobic with low water solubility. > Primary degradation pathways include photolysis and aerobic metabolism.	> Tends to partition to soil and sediment.	> Insecticide > Synthetic pyrethroid. > Induces long-lasting inhibition of the sodium ion channel activation gate, resulting in repetitive nerve signals in sensory organs, nerves, and muscles.	> Slightly toxic via acute oral and dermal routes. > Acute ingestion exposure causes nausea, vomiting, headache, dizziness, anorexia, and hypersalivation. > Possible endocrine-disruptor.	> Highly toxic to fish and aquatic invertebrates. > Practically nontoxic to birds.* > Highly toxic to honey bees. > Dermal exposure can cause life-threatening effects to cats.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Etofenprox	> na	> Virtually insoluble in water. > Stable to hydrolysis. > Susceptible to photolysis.	> Not likely to persist.	> Insecticide: Pyrethroid-like chemical. > Acts on ion channels of the insect nervous system.	> Low toxicity via acute oral, dermal, and inhalation routes.	> Highly toxic to fish and aquatic invertebrates.
Piperonyl butoxide (PBO)	> na	> Degrades by photolysis. > Moderately mobile in soil-water systems.	> Degrades rapidly in the environment by photolysis and metabolism by soil microbes. > Moderately mobile in soil-water systems.	> Synergist: added to insecticides. > Enhances the pesticidal properties of other active ingredients, such as pyrethrins and synthetic pyrethroids, by directly binding to microsomal enzymes in the target organism, thereby inhibiting the breakdown of the other pesticides.	> Low toxicity via acute oral, dermal, and inhalation routes. > Possible endocrine-disruptor.	> Moderately toxic to most fish, highly toxic to some fish. > Highly toxic to aquatic invertebrates.
Naled	> Readily degraded in air. > Can volatilize.	> Readily degraded in water. > Degrades by photolysis. > Low water solubility.	> Readily degraded in soil under aerobic and anaerobic conditions. > Most mobile in soil of low organic content such as sandy loam.	> Insecticide: Organophosphate for control of adult mosquitoes.	> Moderately toxic via acute oral, dermal, and inhalation routes. > Rapidly absorbed by oral and inhalation exposure and distributes quickly to all tissues.	> Moderately toxic to fish and aquatic invertebrates. > Lethal effects found for birds and honey bees.
Temephos	> na	> Breaks down via photolysis and microbial degradation. > Extremely hydrophobic with low solubility.	> Adsorbs rapidly to organic material in water and binds strongly to soils.	> Insecticide: Cholinesterase inhibitor for control of mosquito larvae.	> Moderately toxic via acute oral, dermal, and inhalation routes.	> Slightly to moderately toxic to fish. > Highly toxic to aquatic invertebrates. > Toxic to stoneflies and mayflies.
Bacillus sphaericus (Bs)	> na	> Dormant spores persist for several weeks or months. > The δ-endotoxins generally persist for 2 to 4-weeks but are degraded by sunlight and soil microbes.	> Does not percolate through the soil and readily binds to sediments when in water column.	> Mosquito larvicide. > Bacterium contains microscopic protein pro-toxins which paralyzes the gut of larvae when consumed, resulting in starvation.	> Not pathogenic and does not demonstrate any systemic toxicity.	> Not acutely toxic to birds, mammals, fish or invertebrates. > Mosquito predators not affected by secondary exposure.
Bacillus thuringiensis israelensis (Bti)		> Degrade rapidly after exposure to UV light. > The δ-endotoxins are degraded by sunlight and soil microbes.	> Spores may persist in soil for several months.	> Mosquito larvicide. > Bacterium containing microscopic protein pro-toxins that paralyzes the gut.	> Not pathogenic and does not demonstrate any systemic toxicity.	> Not acutely toxic to birds, mammals, fish or invertebrates.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Spinosad	> Persists for a few hours in air.	> Persists for a few hours in water. > Binds readily to organic matter in water.	> Binds readily to organic matter in soil. Readily photo degrades. > Unlikely to leach to groundwater. > Quickly metabolized by soil microbes under aerobic conditions.	> Insecticide: Biologically derived from fermentation of a naturally occurring soil microbe. > Activates the CNS of insects through interaction with neuro-receptors causing continuous stimulation of the nervous system.	> Acute toxicity is low by all routes of exposure. > Not carcinogenic.	> Acute toxicity is low for fish, aquatic invertebrates, amphibians, and birds. > Very highly toxic to moths and butterflies.
Methoprene and s-Methoprene	> na	> Rapidly degrades in aqueous solution. > Degrades via photolysis and microbial metabolism.	> Relatively immobile in soil. > Metabolized in soil under aerobic and anaerobic conditions via photolysis and microbial metabolism.	> Insecticide: Long chain hydrocarbon ester. > Interferes with normal maturation process during insect life cycles, preventing reproduction.	> Very low toxicity via all acute routes.	> Moderately toxic to fish. > Very highly toxic to aquatic invertebrates. > Practically nontoxic to birds and amphibians.
Alcohol Ethoxylated Surfactant (monomolecular film)	> na	> Half-life in water is from 5 to 22 days.	> na	> Larvicide > Spread a thin film on the surface of the water that makes it difficult for larval pests to attach to the water surface, causing them to drown.	> na	> No observable effects to amphibians, fish, or non-target aquatic organisms (e.g., shrimp, snails, worms, mayfly naiad). > Surface-breathing insects may be temporarily impacted.
Aliphatic solvents (mineral oils, aliphatic hydrocarbons, petroleum distillates)	> Very low vapor pressure. > Low potential for volatility.	> Very low solubility. > Breakdown in 2 to 3 days.	> High sorption to organic matter.	> Larvicide > Creates a top-coating on water to drown larvae, pupae, and emerging adult mosquitoes.	> No deaths due to any acute doses.	> Practically nontoxic to fish, birds, and honey bees. > Rapid breakdown minimizes impact to non-target organisms.
Potassium Salts (soap salts)	> na	> na	> Degrade quickly by microbes and do not persist.	> Insecticide: > Fatty acids penetrate insect body coverings, disrupting cell membranes and causing dehydration and death.	> Low oral and dermal toxicity, but may cause stomach upset. > May be irritating to the skin and eyes.	> Slightly toxic to fish. > Highly toxic to aquatic invertebrates. > Practically nontoxic to birds.
Chlorophacinone	> Volatizes slowly.	> Low water solubility. > Degrades slowly by acid hydrolysis.	> Degrades slowly by photo degradation. > Moderately persistent and immobile. > Major route of dissipation is aerobic metabolism.	> Rodenticide: > First-generation anticoagulant. Acts by blocking vitamin the K cycle, resulting in the inability to produce blood-clotting factors. Damages capillaries, causing diffuse internal hemorrhaging. Death occurs from hypovolemic shock or severe anemia.	> Highly toxic by all acute exposure routes.	> Toxic to wildlife and fish. > Toxic via primary and secondary ingestion routes.
Diphacinone	> na	> Low water solubility. > Stable to photolysis.	> Volatizes slowly from water to soil. > Susceptible to aerobic soil metabolism. > Binds tightly to soil.	> Rodenticide: > First-generation anticoagulant. Acts by blocking vitamin the K cycle, resulting in the inability to produce blood-clotting factors. Damages capillaries, causing diffuse internal hemorrhaging. Death occurs from hypovolemic shock or severe anemia.	> Highly toxic by all acute exposure routes.	> Slightly to moderately toxic to fish and aquatic invertebrates. Does not accumulate in fish > Slightly toxic to birds. > Possible secondary risk to avian predators and scavengers.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Brodifacoum	> Nonvolatile.	> Low water solubility. > Stable to hydrolysis.	> Relatively persistent. > Immobile in soil.	> Rodenticide: > Second-generation anticoagulant. Acts by blocking vitamin the K cycle, resulting in the inability to produce blood-clotting factors. Damages capillaries, causing diffuse internal hemorrhaging. Death occurs from hypovolemic shock or severe anemia.	> Highly toxic by all acute exposure routes.	> Very highly toxic to fish. > Toxic to birds via primary and secondary ingestion exposure. > Nontoxic to honey bees.
Bromadiolone*	> na	> Stable to hydrolysis.	> Moderately persistent in soil. > Immobile in soil with high organic and clay content. > Susceptible to aerobic soil metabolism.	> Rodenticide > Second-generation anticoagulant. Acts by blocking vitamin the K cycle, resulting in the inability to produce blood-clotting factors. Damages capillaries, causing diffuse internal hemorrhaging. Death occurs from hypovolemic shock or severe anemia.	> Highly toxic by all acute exposure routes.	> Moderately toxic to fish. > Toxic to birds via primary and secondary ingestion exposure.
Difethialone*	> na	> Adsorbs to suspended solids and sediments. > Can slowly volatize from water surfaces.	> Immobile in soil.	> Rodenticide > Second-generation anticoagulant. Acts by blocking vitamin the K cycle, resulting in the inability to produce blood-clotting factors. Damages capillaries, causing diffuse internal hemorrhaging. Death occurs from hypovolemic shock or severe anemia.	> Highly toxic by all acute exposure routes. > No genotoxic or carcinogenic effects have been noted.	> Highly toxic to fish and aquatic invertebrates. > Very likely toxic to most mammals. > Likely to adversely affect snakes, non-target rodents, carnivorous mammals. > Highly toxic to birds via primary and secondary routes.
Cholecalciferol (vitamin D)	> Expected to be nonvolatile.	> Essentially insoluble.	> Immobile in soil.	> Rodenticide > Ingestion results in hypercalcemia from mobilization of calcium from bone matrix to blood plasma leading to metastatic calcification of soft tissues.	> Toxic by all acute exposure routes.	> Considered of low hazard to avian and canine species. > May impact non-target rodents. > Not expected to bioconcentrate in mammals because it is metabolized.
Sulfur (fumigant)	> na	> na	> Elemental sulfur becomes incorporated into the natural sulfur cycle. Oxidizes into sulfate and reduces into sulfide. Mediated by microbes.	> Rodenticide > Fumigant. Ignited cartridges produce toxic gases, displacing oxygen in burrows, and causing asphyxiation.	> Low toxicity by acute exposure routes. > No known oncogenic, teratogenic, or reproductive effects.	> Practically nontoxic to fish and aquatic invertebrates. > Nontoxic to birds. > Nontoxic to bees.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Sodium Nitrate (fumigant)	> na	> na	> Sodium nitrates are naturally occurring substances.	> Rodenticide > Fumigant. Pyrolysis of cartridge products results in simple organic and inorganic compounds, such as nitrous oxide and carbon monoxide, which diffuse through burrows causing organisms to die of asphyxiation.	> Low acute oral toxicity. > May cause eye irritation and slight dermal irritation.	> Any non-target organism in the burrow at treatment time will likely be killed. > USEPA recommends that applicators observe signs around burrows carefully for presence of non-targets.
Imazapyr	> Nonvolatile.	> Degradation by photolysis. > Stable to hydrolysis. > Stable to aerobic and anaerobic aquatic metabolism.	> Persistent in soil. > Mobile in soil. > Stable to aerobic and anaerobic soil degradation. > Leaches to groundwater.	> Herbicide > Prevents the synthesis of branched-chain amino acids.	> Slightly toxic via acute oral, dermal, and inhalation routes. > No evidence of carcinogenicity or mutagenicity.	> Practically nontoxic to birds, fish, aquatic invertebrates, and honey bees. > Poses a risk to non-target vascular plants. > Not expected to bioaccumulate.
Glyphosate*	> na	> Highly water soluble. > In aquatic systems, sediment appears to be the major sink for glyphosate residue. > Broken down by microbial degradation.	> Resistant to chemical degradation and sunlight and is fairly unleachable. > Relatively immobile in soil and does not move vertically below the six inch soil layer. > Low tendency to runoff. > Inactivated and biodegraded by microbes under aerobic and anaerobic conditions.	> Herbicide > Plants: Works via the shikimic pathway by inhibiting the enzyme EPSP synthase. Results in stunted growth, malformation, tissue death, etc. > Animals: Shikimic pathway absent in mammals.	> Very low toxicity via oral and dermal routes. > No evidence of carcinogenic or mutagenic effects. > Possible endocrine-disruptor.	> Practically nontoxic to birds, honey bees, fish, and freshwater invertebrates. > No evidence of bioaccumulation.
Triclopyr	> Nonvolatile.	> Highly soluble. > Primary loss via photodegradation. > Triclopyr triethylamine (TEA) rapidly dissociates in water to the acid/anion and triethanolamine. > Triclopyr butoxyethanol ester (TBEE) rapidly hydrolyses to the triclopyr acid/anion and butoxyethanol.	> Slightly mobile with sorption to soil increasing with time. > Primary loss via microbial degradation. > Moderately persistent, with persistence increasing with soil depth and anaerobic conditions.	> Herbicide > Pyridine-based synthetic auxin, which causes the plant to overdose on auxin resulting in epinasty, abnormal leaf formation, stem swelling, and death.	> (Technical triclopyr acid) Slightly toxic via acute oral, dermal, and inhalation routes. > (TEA and TBEE) slightly toxic by acute oral and dermal routes. Practically nontoxic by inhalation. > Not carcinogenic.	> (Triclopyr acid) Slightly toxic to birds and practically nontoxic to insects, fish, and aquatic invertebrates. > (TEA) Practically nontoxic to birds and invertebrates. Slightly toxic to fish. > (TBEE) Slightly toxic to birds, moderately to highly toxic to fish and invertebrates. > Does not bioaccumulate rapidly. > Triclopyr has low toxicity to grasses, but can injure conifers.
2,4-D (2,4-dichlorophenoxy acetic acid)	> na	> Found as a free anion in aqueous environments.	> Dissipation due to oxidative microbial mineralization, photodegradation, and leaching.	> Herbicide > Auxin-mimic. > Phenoxy or phenoxyacetic acid which acts as an herbicide, plant growth regulator, and fungicide.	> Low toxicity via oral and dermal routes. > Dose-dependent damage to eyes, thyroid, kidney, adrenals, ovaries, and testes have been observed in chronic studies of rats. > Possible endocrine disruptor.	> Slightly to moderately toxic to birds. > Some formulations highly toxic to fish. Bioconcentrates in fish. > Practically nontoxic to honey bees.

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Sulfometuron methyl	> Low potential to volatilize.	> Hydrolysis, photolysis, and microbial degradation are major routes of transformation. > Low tendency to sorb to sediments.	> Hydrolysis, photolysis, and microbial degradation are major routes of transformation. > Potential to leach.	> Herbicide > Inhibits acetolactate synthase, which inhibits the production of amino acids required for cells growth. Retards shoot and root development.	> Low toxicity via oral, dermal, and inhalation routes.	> Nontoxic to birds, aquatic invertebrates, and bees. > Slightly toxic to fish. > Low potential to bioaccumulate. > Phytotoxic to duckweed and a broad range of terrestrial plants.
Bentazon	> na	> Photolysis, and microbial degradation are major routes of dissipation.	> Photolysis, microbial degradation, leaching, and runoff are major routes of dissipation. > Low binding affinity to soil.	> Herbicide: > On contact, bentazon interferes with the ability of plants to use sunlight for photosynthesis by inhibiting electron transport.	> Slightly toxic via oral, dermal, and inhalation routes.	> Slightly toxic to birds and small mammals. > Practically nontoxic to fish and aquatic invertebrates. > Low risk to aquatic plants.
Diuron*	> na	> Major routes of dissipation are microbial degradation.	> Sorption highly correlated with soil organic matter. > Mobile and persistent. > Potential to leach to groundwater and contaminate surface waters.	> Herbicide > Substituted urea that inhibits photosynthesis by limiting the production of ATP, and other necessary metabolic processes. > One of the most commonly used herbicides in California.	> Low toxicity via oral, dermal, and inhalation routes. > Metabolism occurs through hydroxylation and dealkylation. > Known/likely carcinogen based on bladder cancer in rats.	> Slightly to practically nontoxic to birds. > Practically nontoxic to bees. > Moderately to highly toxic to fish and aquatic invertebrates. > Low bioaccumulation potential.
Benfluralin (benefin)*	> Volatizes rapidly.	> Major routes of dissipation are photolysis and anaerobic metabolism.	> Low mobility and variable persistence.	> Herbicide > Inhibits growth by acting as a mitotic disruptor.	> Practically nontoxic by acute oral and dermal routes. > Possible endocrine disruptor.	> Practically nontoxic to birds, small mammals, and honey bees. > Highly toxic to fish and aquatic invertebrates. > Considered to be bioaccumulative.
Oryzalin	> na	> Primary degradation process is photolysis.	> Primary degradation process is photolysis. > Not mobile. > Anaerobic conditions in soil cause chemical reduction.	> Herbicide > Disrupts growth processes during germination by inhibiting cell division in plants.	> Practically nontoxic by acute oral route. > Moderately toxic by acute dermal and inhalation routes. > Possible human carcinogen.	> Slightly toxic to practically nontoxic to birds. > Moderately toxic to fish and freshwater invertebrates. > Practically nontoxic to honey bees. > Does not accumulate in fish.
DCPA (chlorthal dimethyl) [metabolite is tetrachloroterephthalic acid (TPA)]	> Volatilization from soil a major route of dissipation.	> Stable to hydrolysis and photolysis.	> Low persistence and mobility. > The DCPA metabolite TPA is unusually mobile and persistent and will leach to groundwater.	> Herbicide > Kills germinating seeds by disrupting microtubule formation in exposed cells, causing abnormal cell division.	> Slightly toxic to practically nontoxic by all acute exposure routes. > Possible human carcinogen. > Possible endocrine disruptor.	> Practically nontoxic to birds on an acute basis, but persistent enough to result in chronic exposure to birds. > Practically nontoxic to bees. > Slightly toxic to practically nontoxic to fish and aquatic invertebrates.
Dithiopyr	> Volatilization contributes more to dissipation than any other route.	> Degrades slowly in water. > Resistant to photolysis and hydrolysis.	> Immobile in soil.	> Herbicide	> Low acute toxicity. > No known mutagenic or carcinogenic effects.	> na

Table 4-1 Summary Characteristics of Active Ingredients

Active Ingredient: Chemical or Biological	Fate & Transport			Mode of Action	Human Health[1]	Ecological Health[1]
	Air	Water	Soil			
Metalochlor	> na	> Stable under hydrolysis.	> Degradation dependent on microbially-mediated and abiotic processes. Photolysis in soil. > Moderately persistent and mobile. Potential to leach to groundwater.	> Herbicide > Inhibits seedling development by acting as a growth inhibitor by suppressing synthesis of chlorophyll, proteins, fatty acids/lipids, isoprenoids, and flavonoids.	> Slightly toxic via acute routes. > Possible endocrine disruptor.	> Practically nontoxic to birds. > Moderately toxic to fish. > Slightly toxic to aquatic invertebrates. > Low potential for bioaccumulation.
Pendimethalin	> Volatilizes from soil.		> Dissipates into the environment by binding to soil, microbially-mediated metabolism, and volatilization. > Persistence decreases with increased temperature and moisture and/or decreased soil organic carbon.	> Herbicide > Disrupts microtubules.	> Low acute toxicity. > Possible human carcinogen.	> Slightly toxic to birds. > Practically nontoxic to honey bees. > Highly toxic to fish and aquatic invertebrates. > High potential to bioaccumulate.
Alkylphenol ethoxylate (APE)*	> na	> Degrades faster in water than in soil.	> Bind strongly to particulates and are persistent in sediments.* > Aerobic conditions facilitate biotransformation.	> Adjuvant: > Enhance activity of active ingredients in herbicides or offset any problems associated with spray application. > Toxicity of APEs to aquatic organisms increases with alkyl chain length.	> Nonylphenol is of low acute and dermal toxicity. > Possible estrogen-mimics.	> Nonylphenol is persistent in the environment, moderately bioaccumulative, and extremely toxic to aquatic organisms.
Polydimethylsiloxane Fluids	> Volatile.	> Insoluble.	> Typically sorb to particulate matter and become associated with soils and sediments. > Degradation is slow in moist soils and quick in dry soils.	> Adjuvant > Enhance activity of active ingredients in herbicides or offset any problems associated with spray application.	> na	> Appear to be relatively nontoxic to benthic invertebrates. > Exhibits little bioaccumulation potential.
Modified Vegetable Oils and Methylated Seed Oil				> Adjuvant > Enhance activity of active ingredients in herbicides or offset any problems associated with spray application.		> Slightly toxic to fish (Competitor™). > Practically nontoxic to Daphnia (Competitor™). > Generally inert or essentially nonphytotoxic.
Lecithin				> Adjuvant > Enhance activity of active ingredients in herbicides or offset any problems associated with spray application.		> Slightly toxic to fish (Liberate™). > Moderately toxic to Daphnia (Liberate™).

[1] Toxicity levels (e.g., slight, low, moderate, high, etc.) are used prevalently in the published literature but are not standardized or representative of specific criteria. They qualitatively describe toxicity in relative terms.

Table 4-2 Degradation of Pyrethrins

Degradation Method	Half-life	Reference
Hydrolysis, pH 9 (water)	14 to 17 Hours	USEPA 2006b
Hydrolysis, neutral or acidic	Slow	USEPA 2006b
Photolysis (water and soil)	<1 Day	USEPA 2006b, Gunasekara 2005
Volatilization (soil)	1.8 to 97 Days	Gunasekara 2005
Aerobic metabolism (soil)	10.5 Days	USEPA 2006b
Anaerobic metabolism (soil)	86.1 Days	USEPA 2006b

4.1.1.2 Human Toxicity

Pyrethrins have low to moderate acute toxicity via the oral, dermal, and inhalation routes (Category III and IV). They are a moderate eye irritant (Category III), a mild dermal irritant (Category IV), and not a skin sensitizer. The oral median lethal concentration (LC50) was found to be 1400 mg/kg in rats, the dermal LC50 was found to be greater than 2000 mg/kg in rabbits, and the inhalation LC50 was found to be 3.4 mg/L in rats (USEPA 2006f) (Table 6.1).

The critical toxicological effects of pyrethrins are (1) neurobehavioral effects (tremors, labored breathing, hyperactivity, secretory signs, matted coats), following acute, short-term, and chronic exposure, with nervous system lesions observed in the rat and mouse following acute exposure; (2) thyroid effects, following chronic exposure in the rat and dog; and (3) liver effects, following short- and long-term exposure in the rat, dog, and mouse. Following inhalation exposure, neurobehavioral effects were observed initially, and respiratory tract lesions were observed at all dose levels. The neurobehavioral effects and the mode of action on the sodium channel are considered relevant to humans because the effects are observed in both the rat and mouse, and the mode of action affects a basic function of the nervous system that is common to all animals (USEPA 2006f) (Table 6.1).

4.1.1.3 Ecological Toxicity

The results of the toxicity testing with the technical grade active ingredient suggest that pyrethrins are very highly toxic to freshwater fish (LC50 = 5.1 µg/L) and invertebrates (median effect concentration [EC50] = 11.6 µg/L), as well as to estuarine/marine fish (LC50 = 16.0 µg/L) and invertebrates (LC50 = 1.4-µg/L) on an acute basis. Chronic toxicity studies show that pyrethrins impair growth (length and weight) of freshwater fish (lowest observed adverse effect concentration [LOAEC] of 3.0 µg/L) and reproduction of freshwater invertebrates (LOAEC of 2.0 µg/L). The chronic no observed adverse effect concentrations (NOAECs) for freshwater fish and invertebrates were reported as 1.9 and 0.86 µg/L, respectively (USEPA 2006f).

Pyrethrins were practically nontoxic to avian species on an acute oral and dietary basis (oral LD50 >2,000 mg/kg bw; dietary LC50 >5,620 mg/kg diet) (USEPA 2006f).

SWRCB has evaluated freshwater aquatic life toxicity data from USEPA's Office of Pesticides' Ecotoxicity Database and has identified the lowest LC50 for pyrethrins as 1.4- µg/L. This value is based on toxicity to scud and mysid during 96-hour tests (SWRCB 2012) (Table 6.1).

4.1.1.4 Ecological Toxicity Associated with ULV Application for Mosquito Abatement

The active ingredients used for control of adult mosquitoes have been deliberately selected for lack of persistence and minimal effects on non-target organisms when applied at label rates for ULV mosquito

control. The products applied as ULV sprays for adult mosquito control are not formulated for persistence, because their purpose is to kill active adult mosquitoes in flight.

Three of the studies discussed below investigated aquatic toxicity following ULV applications of pyrethrins. One study using laboratory toxicity tests on samples after aerial application found no significant mortality to *Ceriodaphnia dubia* following aerial application, and inconclusive results for *Hyalella azteca* (sediment collected prior to application was toxic to *H. azteca*) (Weston et al. 2006). Another study found no significant mortality in caged mosquito larvae or mosquitofish after truck application, and no significant difference in macroinvertebrate abundance, biomass, or species diversity (Jensen et al. 1999). Another study (Lawler et al. 2008) used caged organisms (*Daphnia magna* and *Callibaetis californicus*) to evaluate toxicity after multiple applications, and found no significant difference in mortality.

Following aerial applications of Evergreen Crop Protection EC 60-6 (6 percent pyrethrins, 60 percent PBO) in Sacramento for West Nile virus, Larry Walker and Associates (2006) reported results of water testing on samples from 10 waterways within the treatment area. Treated areas were sprayed nightly for 3 days. One additional application occurred 9 days prior to the 3-day event at selected locations. Samples were taken immediately after application (within 1 to 6 hours), and the next day (16 to 23 hours after the application). Pyrethrins concentrations were detected between 0.234-to 3.77 µg/L from 9 of 26 samples collected immediately after the application. The average concentration for samples collected 1 to 6 hours after application was 0.270 µg/L. Pyrethrins were not detected (<0.2 µg/L) 16 to 23 hours after each spray event.

Testing was also carried out by Weston et al. (2006) following the same applications. Prior to aerial spraying, pyrethrins were not detected in water or sediment samples. Pyrethrins were not detected in water samples taken 10 to 34-hours after the spray applications; however, pyrethrins were detected in sediment samples after aerial spraying at concentrations ranging from 93.1 to 403 micrograms per kilogram [µg/kg] in 4-of 6 samples. Neither water nor sediment was tested at later intervals, so the duration of persistence could not be determined in this study. Laboratory tests were conducted to determine the effects of short-term chronic exposure of *Ceriodaphnia dubia* to water collected after the spray events, following USEPA protocol. No significant differences in mortality were observed. In addition, sediment toxicity tests were performed with the amphipod *Hyalella azteca,* and toxicity was observed in samples collected both before and after application. The authors concluded that pyrethrins should present little risk to aquatic organisms due to the low toxicity and lack of long-term persistence.

Water and soil deposition of pyrethrins following aerial applications was evaluated at two sites in California by Schleier et al. (2008). Water was sampled after aerial applications of pyrethrins and PBO in irrigation ditches at one site (Princeton) and in static ponds at another (Colusa). Pyrethrins were not detected following spray events at either site (the reporting limit was 0.5 µg/L or less). The authors concluded that the amounts of pyrethrins and PBO deposited on the ground and in water after aerial ULV insecticide applications are probably lower than those estimated by previously published studies to predict exposure and risk.

Deposition of pyrethrins following truck-mounted application was evaluated in large seasonal wetlands in California (Jensen et al. 1999). Pyrethrins were not detected (<20 µg/L) in surface waters one hour after ULV applications. The authors found no significant differences in macroinvertebrate abundance and biomass or species diversity in areas treated with any of the materials when compared with untreated ponds. No mortality occurred in mosquitofish held in water (in sentinel cages in treated ponds). Similarly, no difference in mortality was observed for mosquito larvae held in water (in sentinel cages in treated ponds) when compared with untreated ones. The authors concluded that ULV applications for adult mosquito control were not likely to significantly affect aquatic insects or fish in these habitats.

Lawler et al. (2008) evaluated pyrethrins and PBO in sediment following multiple applications of pyrethrins from truck-mounted equipment in the Colusa and Sacramento National Wildlife Refuges in California. Stock tanks were filled with a layer of soil overlain with 1,150 liters of water. Zooplankton (*Daphnia magna*) were held in sentinel cages in the water column and mayfly larvae (*Callibaetis californicus*) were

placed in cages at the bottom of each tank, where they were in contact with sediment. ULV applications of pyrethrins were made from truck-mounted equipment twice weekly for six weeks. Pyrethrins concentration in sediments and sentinel survival were evaluated after 5 and 11 spray event applications. Pyrethrins were found at low concentrations (23.1 and 33.1 µg/kg) in 2 of 6 tanks after five spray events, but there was no evidence of accumulation in sediments. After 11 spray events, sediment in 4 of 6 tanks (including one that had held residues after spray 5) contained no detectable amount of pyrethrins (<2 µg/kg), one tank had pyrethrins concentrations at 4 µg/kg, and another at 34.5 µg/kg. There was no significant difference in mortality for mayfly larvae held in sentinel cages on the sediment. Likewise, there was no significant difference in mortality seen in *D. magna* held in the water column. PBO-synergized pyrethrins had no detectable effect on the survival of *D. magna* held in tanks in the spray area, even after 11 biweekly spray events. They concluded that applications of pyrethrins and PBO at rates used for mosquito control did not have detectable effects on the indicator species.

Several papers were published documenting that ULV-applied mosquito adulticides do not accumulate in water or sediment during repeated applications. Chemical testing was conducted following multiple spray events by Amweg et al (2006). There was no increase in the level of pyrethrins or PBO following multiple daily spray events, and the concentration had returned to background level when samples taken one week after the last application were tested. Similarly, Lawler et al. (2008) reported that the concentration of pyrethrins and PBO in tanks within a treated area were not significantly higher after 11 applications than in samples taken after the fifth application. In many cases, the concentrations were actually lower following the 11th spray event than after the fifth spray event.

4.1.1.5 Summary of Toxicity and Potential Effects

Pyrethrins readily degrade in water and soil, but may persist under anoxic conditions. They tend to strongly adsorb to soil surfaces, and hence have low potential to leach into groundwater. These chemicals may have low to moderate acute toxicity to mammals; however, proper personal protective equipment would alleviate potential for human exposure, especially when delivered via ULV techniques. Pyrethrins may be highly toxic to fish (freshwater, estuarine, marine) and invertebrates, although, exposures would likely be low during and following ULV applications, which are designed to prevent environmental persistence and potential impacts to non-target ecological receptors.

Pyrethrin is used for both mosquito (five Districts) and yellow jacket wasp (three Districts) control. For yellow jacket wasp control, pyrethrin (1 percent of the formulation) was applied around parks, landscaping, and directly into ground nests. A single product was applied several hundred times throughout the reporting year at approximately one-ounce doses. For mosquito control, pyrethrin is applied to manmade and natural sites including ditches, and moving and standing water. Three products containing pyrethrin (5 percent) were applied several hundred times throughout the reporting year.

4.1.2 Allethrins and *d-trans* allethrin

Allethrins are synthetic pyrethroids that are structurally very similar to cinerin I in naturally occurring pyrethrum. There are three asymmetric carbons and, thus, eight potential isomers; however, four isomers are present in the greatest concentration for product formulations. One of the stereoisomers, d trans of d isomer (d-trans allethrin), is recognized as being the most insecticidally active and toxicologically important of the four isomers. Allethrins are typically used as a “knock-down” agent and a different, residual pesticide is co-formulated with allethrins in the end-use products to kill the target pests (USEPA 2009b). D-trans allethrin is usually combined with synergists such as PBO.

Allethrins are used to control flying and crawling insects in a number of commercial, horticultural and residential applications. Commercial applications include space, broadcast and crack and crevice treatments in a variety of commercial, industrial, residential, and institutional sites. Horticultural applications include foliar and fogger treatment on nonfood plants. Residential uses include pest control in

homes and outdoor domestic structures, on gardens, and direct application to pets. Allethrins are also approved for use in commercial animal premise (indoor) misting systems (USEPA 2009b).

4.1.2.1 Environmental Fate

Allethrins were the first pyrethroids developed and they differ from more recently developed pyrethroids in their high photolability (USEPA 2009b). The photolysis half-life is less than 8 hours (WHO 1989). Allethrins (and the d-trans allethrin component) are not soluble in water and are expected to adhere moderately to soil containing organic matter. When used in mosquito coils and mats, allethrins are released into the air where they will either be degraded by sunlight or be distributed in low concentrations to nearby surfaces.

4.1.2.2 Human Toxicity

The toxicity of allethrin varies with the amounts of different isomers present. The LD50 of allethrin in male rats is 1,100 mg/kg (685 mg/kg in female rats) while the LD50 of *d-trans* allethrin in rats is 860 mg/kg. Allethrin is slightly toxic to moderately toxic by dermal absorption and ingestion. The dermal LD50 of allethrin in rabbits is 11,332 mg/kg. Dermal exposure results in itching, burning, tingling, and numbness. Large doses by any route can cause physical symptoms such as nausea, vomiting, diarrhea, tremors, convulsions, and coma. A chronic dosage of 50 mg/kg/day for two years produced no detectable effect in dogs. Allethrin is not known to cause reproductive, teratogenic, mutagenic, or carcinogenic effects to mammals (EXTOXNET 1993a) (Table 6.1).

4.1.2.3 Ecological Toxicity

The chemical is practically nontoxic to birds but highly toxic to fish and invertebrates with the *d-trans* isomer exhibiting greater toxicity to non-target insects than allethrin (EXTOXNET 1993a). The LC50 for fish ranges from 0.0026 to 0.08 mg/L (USEPA 2009b). The bioaccumulation potential of allethrin is unknown (Table 6.1).

4.1.2.4 Summary of Toxicity and Potential Effects

Allethrins readily degrade via photolysis especially when released into the air following coil deployment. Residual released material may deposit to soil surfaces and moderately adhere to organic matter. Allethrins may be highly toxic to fish, invertebrates, and non-target insects, but they are unstable in the environment and likely do not pose unacceptable risk to ecological receptors.

Allethrins, including *d-trans* allethrin are intermittently used to target yellow jacket wasp nests. Allethrin (*d-trans* isomer) is combined with another active ingredient, phenothrin in a single product used by two Districts for wasp and yellow jacket control. This product was used in 12 applications of 37.5 ounces (volume) (<0.1 ounces of active ingredients) during the summer of 2011 (of the reporting year). Because allethrins are used in localized, low-volume applications, environmental persistence is not expected nor is unwanted exposure to non-target ecological or human receptors.

4.1.3 <u>Phenothrin (sumithrin or d-phenothrin)</u>

Phenothrin has been registered by the EPA since 1976, and is used to control adult mosquitoes, and as an insecticide in transport vehicles such as aircraft, ships, railroad cars, and truck trailers. It is also used as an insecticide and miticide in commercial, industrial, and institutional nonfood areas, in homes and gardens, in greenhouses, and in pet quarters and on pets, and is used in urban areas, outdoor residential areas, around buildings and structures, at recreational areas, golf courses, zoos, and for agricultural crops (CDPR 2010a).

4.1.3.1 Environmental Fate

Phenothrin has a relatively high affinity for binding to soils, is moderate persistence in surface soils, and has low solubility. Its low leaching potential means that it is likely to remain immobile once it binds to soil sediments. The major routes of dissipation of phenothrin in the environment are photolysis in water (half-life at 6.5 days) and aerobic metabolism (in soil from 18.6 to 25.8 days, and in aquatic environments at 36.1 days) (Table 4-3). Even though phenothrin is likely to undergo photolysis in water, its high affinity for binding to particulate matter makes photolysis less likely to happen, except during the brief period in which the chemical is suspended in water before binding to sediment. Phenothrin is moderately persistent under aerobic conditions and is persistent under anaerobic conditions (USEPA 2008b).

Table 4-3 Degradation of Phenothrin

Degradation Method	Half-life	Reference
Hydrolysis, all pH levels	Stable	USEPA 2008a
Photolysis (water)	6.5 Days	USEPA 2008a
Aerobic metabolism (water)	36.1 Days	USEPA 2008a
Aerobic metabolism (soil)	18.6 to 25.8 Days	USEPA 2008a
Anaerobic metabolism (water)	173.3 Days	USEPA 2008a

4.1.3.2 Human Toxicity

Phenothrin is not known to be acutely toxic at high exposure levels to humans or mammals. Phenothrin exhibits low acute toxicity by oral (Category III), dermal (Category III), and inhalation (Category IV) routes of exposure. Phenothrin is mild eye irritant (Category III) but is not a skin irritant or a skin sensitizer. The oral LC50 was found to be greater than 5,000 mg/kg in rats, the dermal LC50 was found to be greater than 2000 mg/kg in rats and the inhalation LC50 was found to be greater than 2.1 mg/L in rats (USEPA 2008b).

Neurotoxic effects were observed in developmental toxicity studies but not observed in other acute, chronic, and subchronic toxicity studies done in rats and dogs up to the limit dose of 20,000 mg/kg/day. Maternal toxicity in rats was evidenced by the appearance of generalized clinical effects in dosed individuals; these effects included decreased maternal weight gain and decreased food consumption at the highest dosage tested of 3000 mg/kg/day (USEPA 2008b) (Table 6.1).

4.1.3.3 Ecological Toxicity

Phenothrin technical grade active ingredient is highly toxic on an acute basis, with the LC50 ranging from 15.8 to 18.3 µg/L for freshwater fish. Phenothrin is also highly toxic to estuarine/marine fish on an acute basis. The LC50 for estuarine and marine fish ranges from 38.3 to 94.2 µg/L. Phenothrin is very highly toxic to freshwater invertebrates. The EC50 for freshwater invertebrates is 4.4-µg/L. Chronic data for phenothrin show adverse reproductive effects for freshwater invertebrates at a NOAEC of 0.47 µg/L. This indicates a potential for chronic reproductive effects to freshwater invertebrates as a result of phenothrin exposure. Additional chronic effects to estuarine and marine invertebrates are expected based on the chronic reproductive toxicity to freshwater invertebrates and the acute effects to estuarine and marine invertebrates (USEPA 2008b).

Based on studies of avian acute dietary toxicity, phenothrin can be classified as practically nontoxic to avian species. The LC50 for avian dietary toxicity is above 5,000 parts per million (ppm) (USEPA 2008b).

SWRCB has evaluated freshwater aquatic life toxicity data from USEPA's Office of Pesticides' Ecotoxicity Database and has identified the lowest LC50 for phenothrin as 0.025 µg/L. This value is based on toxicity to mysid during a 96 hour test (SWRCB 2012) (Table 6.1).

4.1.3.4 Ecological Toxicity associated with ULV Application for Mosquito Abatement

Davis and Peterson (2008) measured family diversity, richness, and evenness at 1, 7, 14, and 28 days after truck application of phenothrin applied as Anvil 10+10 ULV. Most response variables showed no significant treatment effect, although there were some reductions in number of individuals. The authors concluded that the reductions in aquatic non-target populations did not suggest any trends or persistent deleterious biological effects following a single adulticide application.

New York City Department of Health sampled 32 locations for phenothrin and PBO before and after spray events during mosquito adulticide applications that occurred during July through September 2000. Out of the 68 post-application samples collected by the city, only two had concentrations of either phenothrin or PBO greater than the 0.5 µg/L reporting limit: 1.10 µg/L for phenothrin on August 18, 2000, at Mt. Loretto Pond on Staten Island; and 1.03 µg/L for PBO and 0.55 µg/L for phenothrin for a sample collected on August 5, 2000, at Alley Park Pond in Queens (Suffolk County 2006).

Zulkosky et al. (2005) evaluated phenothrin applied as Anvil. In 2002, phenothrin was not detected in either spray event (detection limit of 0.0005 µg/L). In 2003, phenothrin was detected at 0.0011 µg/L immediately after spray application, but was not detected in samples collected 1 to 10 days after spraying Anvil.

The Massachusetts Department of Agricultural Resources (2010) conducted a study where phenothrin was applied aerially as Anvil 10+10 ULV to six sites. There were no detections of phenothrin during this study (Massachusetts Department of Agricultural Resources 2010).

4.1.3.5 Summary of Toxicity and Potential Effects

Phenothrin is generally applied using ULV techniques, which encourages dissipation rather than persistence in the environment. It is not expected to pose unacceptable risk to human or ecological receptors, because it is handled in small amounts using proper personal protective equipment (by the applicator) and its low potential for exposure to non-targets.

As stated above, phenothrin and *d-trans* allethrins are used in conjunction for yellow jacket wasp control. One product containing *d-trans* allethrins was used in limited amounts by two districts during the reporting year. Phenothrins are used in localized, low-volume applications, therefore environmental persistence and meaningful exposure to non-target ecological receptors is not expected.

4.1.4 Prallethrin

Prallethrin is a synthetic pyrethroid with fast knock-down activity against household insect pests. It is used in household insecticide products against mosquitoes, houseflies, and cockroaches. Prallethrin also has veterinary uses in the treatment of domestic pets. Prallethrin has been applied in urban areas, outdoor residential areas, recreational areas, golf courses, around building and structures and at areas of standing water (CDPR 2010a). Prallethrin has an exciting effect on mosquitoes, and is added to Duet (the only prallethrin-containing adulticide product used in California) primarily for this property rather than its inherent toxicity. The other active ingredient in Duet is phenothrin.

4.1.4.1 Environmental Fate

Prallethrin readily sorbs to soils and sediments. The major route of dissipation of prallethrin in the environment is photolysis in both water (half-life at 13.6 hours) and soil (at 25 days) (Sumitomo Chemical 2009).

4.1.4.2 Human Toxicity

Prallethrin has low to moderate acute toxicity via the oral, dermal, and inhalation routes (Category II, III and IV). It is a moderate eye irritant (Category III), not a dermal sensitizer, and is nonirritating to skin. The oral LC50 was found to be 460 to 640 mg/kg to rats, the dermal LC50 was found to be greater than 5000 mg/kg, and the inhalation LC50 was found to be 288 to 333 mg/m^3 (USEPA 2003a) (Table 6.1).

4.1.4.3 Ecological Toxicity

Prallethrin is highly toxic to fish (LC50 of 17.6 µg/L based on a 96 hour acute toxicity test to zebrafish (*Danio rerio*) and aquatic invertebrates (EC50 of 19 µg/L based on a 48 hour acute toxicity test to *Daphnia magna*). Prallethrin has low toxicity to algae (EC50 of 4.9 mg/L based on a 72 hour acute toxicity test to *Scenedesmus subspicatus*) and birds (LD50 of 1171 mg/kg for bobwhite quail. It is very toxic to bees (Agro-allianace Pty Ltd nd).

SWRCB has evaluated freshwater aquatic life toxicity data from USEPA's Office of Pesticides' Ecotoxicity Database and has identified the lowest LC50 for prallethrin as 3.9 µg/L. This value is based on toxicity to mysid during a 96 hour test (SWRCB 2012) (Table 6.1).

4.1.4.4 Summary of Toxicity and Potential Effects

Like other Type 1 pyrethroids, prallethrin is readily degraded via photolysis and is less environmentally persistent than the Type 2 variety. Prallethrin is used to treat domestic pets and is therefore, not expected to cause significant mammalian toxicity. Prallethrin is practically nontoxic to birds but is highly toxic to non-target organisms including, fish, aquatic invertebrates, and honey bees.

Prallethrin is intermittently used to target yellow jacket and paper wasp nests. Localized applications are generally completed during the fall and sometimes winter months. Because this active ingredient is used in localized, low-volume applications, it is not expected to persist in the environment or pose unwanted toxicity to non-target ecological or human receptors.

4.1.5 Deltamethrin

Deltamethrin is a pyrethroid that kills insects on contact and through ingestion. Type II pyrethroids such as deltamethrin induce long-lasting inhibition of the sodium ion channel activation gate. This results in prolonged permeability of the nerve to sodium and produces a series of repetitive nerve signals in sensory organs, nerves, and muscles. The mechanism is the same for target and non-target organisms (National Pesticide Information Center 2010). The primary use of deltamethrin (approximately 85 percent of the total production) is for crop protection. Deltamethrin is also used to protect stored commodities such as cereals, grains, and coffee beans. Other uses include insect control for public health concerns, pest control in forestry, pest control in animal facilities, parasite control on animals, and as a wood preservative (CDPR 2000). Deltamethrin is used as a mosquito adulticide to a limited extent in California, but public health uses typically target other vectors such as yellow jackets. It is used as a barrier application and not a ULV application and it is not used over or adjacent to water bodies. Formulations used for mosquitoes include Suspend SC Insecticide (primarily used), and K-Othrine SC Insecticide.

4.1.5.1 Environmental Fate

Deltamethrin degrades via hydrolysis, photolysis, and microbial action (Table 4-4) and is more persistent in soils with a high clay or organic matter content. The half-life of deltamethrin is approximately 25 to 33 days under aerobic conditions (CDPR 2000, FAO-WHO 2002a).

Table 4-4 Degradation of Deltamethrin

Degradation Method	Half-life	Reference
Hydrolysis, pH 8	31 Days	FAO-WHO 2002
Hydrolysis, pH 9	2.5 Days	FAO-WHO 2002
Photolysis (water)	<21 Days	FAO-WHO 2002
Photolysis (soil)	48 Days	FAO-WHO 2002
Aerobic metabolism (soil)	22 to 25 Days	FAO-WHO 2002

Table 4-4 Degradation of Deltamethrin

Degradation Method	Half-life	Reference
Anaerobic metabolism (soil)	32 to 36 Days	FAO-WHO 2002
Field conditions (soil)	14 to <150 Days	FAO-WHO 2002

4.1.5.2 Human Toxicity

Deltamethrin is of low to moderate acute toxicity. The oral LD50 for rats is 30 mg/kg in an oil vehicle or >5,000 mg/kg in a water vehicle. The LD50 for dogs is 300 mg/kg (EXTOXNET 1995a). The acute dermal LD50 for rabbits is >2,000 mg/kg and no skin irritation and slight eye irritation were reported (EXTOXNET 1995a). Symptoms of acute exposure in humans include ataxia, convulsions, dermatitis, edema, diarrhea, headache, irritability, among others (EXTOXNET 1995a). Symptoms of chronic exposure of humans to deltamethrin include choreoathetosis, hypotension, prenatal damage, and shock (EXTOXNET 1995a). Deltamethrin has no reported teratogenic, mutagenic, or carcinogenic effects. Mice fed doses of deltamethrin during gestation showed no changes in the number of implants, fetal mortality, fetal weight, or malformations (EXTOXNET 1995a) (Table 6.1).

4.1.5.3 Ecological Toxicity

Deltamethrin is very highly toxic to fish and aquatic invertebrates. It is practically nontoxic to birds (USEPA 2010a). Of particular importance when using pyrethroids in general is to note that non-target insects may have the same approximate sensitivity as mosquito larvae (Mian and Mulla 1992). These include mayflies, stoneflies, whirligig beetle, caddisflies, and the snipefly. The water boatman and backswimmer have low sensitivity to some pyrethroids. See Table 2 of Mian and Mulla (1992). Deltamethrin is very highly toxic to honey bees (USEPA 2010a) (Table 6.1).

4.1.5.4 Summary of Toxicity and Potential Effects

Deltamethrin may be persistent in high organic matter soils and aquatic sediments. It is nonselective and therefore, may post risk to non-target organisms such as honey bees. Deltamethrin is highly toxic to fish (and bioaccumulative) and invertebrates, however, it is generally not applied to aquatic systems. It is not expected to pose risk to aquatic receptors under the prescribed application usage by the MVCAC districts.

One product containing deltamethrin (0.05 percent) is used by two Districts for yellow jacket wasp control. It is primarily used in the summer months to specifically target yellow jacket wasp ground nests. It was applied almost 300 times during the summer of 2011. There are a range of limited exposure and localized and limited usage patterns and potential unwanted effects are dependent on the use.

4.1.6 <u>Esfenvalerate</u>

Esfenvalerate is a broad-spectrum nonselective insecticide applied as needed for the control of a wide selection of arthropod pests. Esfenvalerate is a mixture of four stereoisomers, enriched with the S,S-isomer, the most insecticidally active isomer. (The parent mixture, fenvalerate, is a mixture of the same four isomers in relatively equal proportions.) Esfenvalerate containing products registered for use in California are applied for home/garden consumer use, commercial pesticide application use, and agricultural production use (Kelley 2003). Esfenvalerate is a voltage-dependent sodium-channel agonist. Esfenvalerate works against the insect nervous system, resulting in repetitive firing of neurons.

4.1.6.1 Environmental Fate

Esfenvalerate is practically insoluble in water, extremely hydrophobic and has a strong tendency to bind to soil particles. Esfenvalerate, as a result of these characteristics, is relatively immobile in soil and to

show a low tendency to leach. Esfenvalerate, when present in surface waters, is expected to be bound to suspended particulates (clay, soil, and sediment particles) and to organic matter (Kelley 2003). Primary degradation pathways include photolysis and aerobic metabolism (Table 4-5). (FAO-WHO 2002b)

Table 4-5 Degradation of Esfenvalerate

Degradation Method	Half-life	Reference
Hydrolysis, pH 5–9	64 to 130 Days	FAO-WHO 2002b, Kelley 2003
Photolysis (water)	6 to 17.2 Days	FAO-WHO 2002b, Kelley 2003
Photolysis (soil)	3 to 15.8 Days	FAO-WHO 2002b, Kelley 2003
Aerobic metabolism (water)	4 to 72.3 Days	Kelley 2003
Aerobic metabolism (soil)	35 to 546 Days	FAO-WHO 2002b, Kelley 2003
Anaerobic metabolism (water)	65 to 79 Days	Kelley 2003
Anaerobic metabolism (soil)	104 to 203 Days	Kelley 2003

4.1.6.2 Human Toxicity

Esfenvalerate is a relatively new compared to other pesticides on the market; therefore, the usage history for this compound is incomplete. The oral LD50 of esfenvalerate in rats is 458 mg/kg. The dermal LD50 in rabbits is 2,000 mg/kg (considered moderately toxic). The inhalation LC50 in rats is greater than 2.93 mg/L (EXTOXNET 1994). Esfenvalerate has not been implicated in cancer or birth defects in mammal studies. Esfenvalerate is included in the final list of candidate chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.1.6.3 Ecological Toxicity

Esfenvalerate is moderately toxic to birds. The acute oral LD50 for mallard ducks is 9,932 mg/kg. The compound is highly toxic to fish and aquatic invertebrates. Esfenvalerate is highly toxic to bees. The compound tends to repel bees for a day or two after application. Most intoxicated bees die in the field before they can return to the hive (EXTOXNET 1994). The 96-hr LC50 is 0.00026 mg/L for bluegill and rainbow trout and 0.00024-mg/L for *Daphnia magna* (EXTOXNET 1994). Esfenvalerate rapidly bioaccumulates in fish. The bioaccumulation factor in rainbow trout is about 400 times the background esfenvalerate water concentrations. The chemical is also highly toxic to bees (EXTOXNET 1994) (Table 6.1).

4.1.6.4 Summary of Toxicity and Potential Effects

Esfenvalerate is insoluble in water and tends to bind to organic matter in soils and sediment with low leaching potential. Degradation occurs through photolysis and aerobic metabolism, therefore it does not appear to persist in the environment. This pesticide is generally deployed in bait stations above the ground, which limits its release to the soil surface and aquatic systems. Esfenvalerate is considered moderately toxic to mammals and birds; and highly toxic to aquatic invertebrates and honey bees. In addition, it is both highly toxic to and bioaccumulative in fish.

Esfenvalerate was not used by the MVCAC districts from during the reporting year. As a result, potential loading scenarios to different habitats types could not be determined. Potential non-target biological receptors could also not be surmised due to the lack of habitat-specific application data. However, as noted above, honey bees could be at risk from this pesticide. There is a lack of persistence potential and the Districts did not use esfenvalerate products during the reporting year.

4.1.7 Lambda-cyhalothrin

Lambda-cyhalothrin is a synthetic pyrethroid insecticide used for controlling pest insects in agriculture, public health, and in construction and households. Lambda-cyhalothrin is a 1:1 mixture of two of the four enantiomers, which constitute cyhalothrin. Insecticidal products containing lambda-cyhalothrin have been widely used to control insect pests in agriculture, public health, and homes and gardens (He et al. 2008).

4.1.7.1 Environmental Fate

Lambda-cyhalothrin is an extremely hydrophobic compound and has rapid and strong adsorption to soils and sediments. Lambda-cyhalothrin residues dissolved in water decrease rapidly if suspended solids and/or organic materials are present because lambda-cyhalothrin molecules are strongly adsorbed by particulates and plants (He et al. 2008). Primary degradation pathways include photolysis and aerobic metabolism (FAO-WHO 2008).

Table 4-6 Degradation of Lambda-cyhalothrin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5	Stable	FAO-WHO 2008
Hydrolysis, pH 7	454 to Days	FAO-WHO 2008
Hydrolysis, pH 9	7.3 Days	FAO-WHO 2008
Photolysis	24 Days	FAO-WHO 2008
Aerobic metabolism (soil)	22 to 83 Days	FAO-WHO 2008
Microcosm (water)	<1 Day	He, et al. 2008

4.1.7.2 General Toxicity

In 2005, a pyrethroid insecticide analysis report was completed for the Sacramento, San Joaquin, and Central valleys to summarize existing information on pyrethroid usage patterns, fate and transport and toxicity (Oros and Werner 2005). The report identified lambda-cyhalothrin as one of the top five pyrethroids used in these areas to manage pests in a variety of settings and applications. According to Oros and Werner (2005), lambda-cyhalothrin has been found in sediments at levels that are known to be toxic to the several representative invertebrate species in the Sacramento, San Joaquin, and Central valleys.

4.1.7.3 Human Toxicity

Lambda-cyhalothrin is moderately toxic via the acute oral, dermal, and inhalation routes (NPIC 2001).The median lethal oral dose (LD50) of lambda-cyhalothrin has been reported at 56 to 79 mg/kg for female and male rats, respectively or as high as 144-mg/kg (Ray 1991). Technical-grade lambda-cyhalothrin is less toxic when exposure occurs dermally, given its relatively poor absorption by this route; dermal LD50s of 632 mg/kg and 696 mg/kg for male and female rats have been cited. One of the formulated products, Karate®, can cause significant skin and eye irritation (Ray 1991).

Acute exposure to lambda-cyhalothrin has been linked with changes in neurological function when administered at a single dose of 0, 2.5, 10, or 35 mg/kg-d, a result consistent with its action on sodium channel permeability (USEPA 2002). Rats exposed for 4-hours to an aerosol of cyhalothrin at concentrations of 3.68 to 68 mg/m^3 exhibited a concentration-dependent increase in signs of neurotoxicity. Effects ranged from lethargy and salivation at the lowest concentration, to death (shortly after termination of exposure) at the highest concentration (Curry and Bennet 1985).

Chronic studies of lambda-cyhalothrin and cyhalothrin have been repeatedly and consistently documented decreased body weight gain and reduced food consumption exposure levels as low as

0.9 mg/kg/day, with numerous study results yielding NOAELs of 1 to 2.5 mg/kg/day. Signs of neurotoxicity and changes in organ weights are also common effects of chronic exposure to lambda-cyhalothrin and cyhalothrin (USEPA 2002, 2004a, 2007b, a).

Although little research on the developmental toxicity of lambda-cyhaolthrin is publically available, the information provided by the USEPA indicates that the maternal NOAEL was 10 mg/kg-d for both species (USEPA 2002, 2004a). The developmental NOAEL was the highest dose tested in each study; in rats, it was 15 mg/kg-d, and in rabbits, 30 mg/kg-d. A study of the reproductive effects of this compound over three generations indicated a LOAEL of 5.0 mg/kg-d which elicited adverse effects in both the parents and pups, with toxicity manifested as reduced body weight and body weight gain in the parents, and reduced pup weight and reduced pup weight gain during lactation (USEPA 2002, 2004a). No genotoxicity data for cyhalothrin or lambda-cyhalothrin were identified in recent USEPA pesticide tolerance documents (USEPA 2002, 2004a, 2007b, a). A chronic feeding study of cyhalothrin in the diets of rats resulted in no oncogenic effects (USEPA 2002) (Table 6.1).

4.1.7.4 Ecological Toxicity

Lambda-cyhalothrin is of low toxicity to birds. The oral LD50 for the mallard duck is >3,950 mg/kg and the dietary LC50 for the bobtail quail is 5,300 mg/kg (WHO 2007). A 1-year neurotoxicity study on the dog where lambda-cyhalothrin was administered by gavage, derived a NOAEL of 0.5 mg/kg bw per day and a LOAEL of 3.5 mg/kg bw per/day. Systemic neurotoxicity (i.e., ataxia, tremors, and occasionally convulsions) was observed, with an overall NOAEL of 0.5 mg/kg bw per day. Signs of systemic neurotoxicity were observed from the first week and generally occurred within a few hours after treatment (PMRA 2003). In 2007, the USEPA released a revised Pesticide Tolerance for lambda-cyhalothrin, published in the Federal Register Volume 72, Number 157, indicating that the dog is known as the most vulnerable to toxic effects.

Lambda-cyhalothrin is highly toxic to aquatic organisms, including fish, shellfish, shrimp, crabs and clams (He et al. 2008). The 96-hr LC50 is 0.21 µg/L for bluegill (WHO 2007) and 0.24-µg/L for rainbow trout (He et al. 2008). The 48-hr LC50 for *Daphnia magna* is 0.36 µg/L (He et al. 2008).

Lambda-cyhalothrin is known to be toxic to honey bees. As presented by IPCS, honey bees have an oral LD50 of 0.97 µg/bee. Additionally, He et al. (2008) reported an oral LD50 over 48-hours and contact LD50 of 0.038 µg/bee and 0.909 µg/bee (Table 6.1).

4.1.7.5 Summary of Toxicity and Potential Effects

Lamda-cyhalothrin may be persistent in the absence of light and has been found at concentrations known to be toxic to aquatic invertebrates. The potential for persistence of this chemical and its toxicity to mammals, aquatic organisms (vertebrates and invertebrates), and non-target insects such as honey bees is of concern from a potential human and ecological risk perspective.

Lambda-cyhalothrin is available to the public in commonly-used products for residential wasp control. Lambda-cyhalothrin is used by one district for targeted application to yellow jacket and paper wasp nests. This product (0.01 percent lambda-cyhalothrin) is used throughout the year and exceeded 2,000 ounces (volume) of product and less than one ounce of active ingredient during the reporting year. Some of the Districts use products containing this active ingredient as a courtesy to the public to assist with wasp control at residences (restricted to yards, gardens, and home exteriors). The amount applied directly to wasp nests (by the public and the Districts) is minute and there is little to no potential for non-target organism exposures. The potential for human exposure (public and trained professionals [e.g., District staff]) is extremely low when use product label instructions are properly followed.

Although there is a potential for environmental persistence and exposure to domestic pets and non-target receptors, this a.i. is readily available as an insect spray and the uses by the Districts are generally focused, and very localized to minimize or eliminate those exposures.

4.1.8 Resmethrin

Resmethrin has been registered by the EPA since 1967, and is used to control flying and crawling insects in the home, lawn, garden, and industrial sites. It can also be used to control insects on ornamental plants (outdoor and greenhouse use), on pets and horses, and as a mosquitocide. Resmethrin is also used at commercial and industrial areas, warehouses, urban areas, and golf courses, and on aquatic areas or standing water, and selected agricultural crops. Because of its toxicity to fish, resmethrin is a restricted-use pesticide (RUP) for the purpose of public health mosquito abatement, and is available for this use only by certified pesticide applicators or persons under their direct supervision. Resmethrin works by interacting with sodium channels in the peripheral and central nervous system of target organisms (USEPA 2006g).

4.1.8.1 Environmental Fate

Resmethrin degrades rapidly when exposed to light; however, when not subject to photolysis, resmethrin tends to be environmentally persistent (Table 4-7). Reported half-lives in water range from 22 minutes (photolysis in seawater) to 37 days (aerobic metabolism). Resmethrin has low mobility and has a high affinity to bind to soils/sediments and organic carbon (USEPA 2006g).

Table 4-7 Degradation of Resmethrin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5 – 9	>89 Days	USEPA 2006c
Photolysis (distilled water)	47 minutes	USEPA 2006c
Photolysis (seawater)	22 minutes	USEPA 2006c
Aerobic metabolism (water)	37 Days	USEPA 2006c
Aerobic metabolism (soil)	198 Days	USEPA 2006c
Anaerobic metabolism (soil)	Stable	USEPA 2006c

4.1.8.2 Human Toxicity

Resmethrin has low acute toxicity via the oral (Category III), dermal (Category III), and inhalation (Category IV) routes of exposure. It is not an eye or skin irritant nor is it a skin sensitizer. The oral LC50 was 4639 to 6091 mg/kg in rats, the dermal LC50 was found to be greater than 2000 mg/kg in rabbits, and the inhalation LC50 was found to be 5.28 mg/L in rats (USEPA 2006g). Resmethrin is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.1.8.3 Ecological Toxicity

Resmethrin is moderately toxic to birds. The oral LD50 for red-winged blackbirds is 75 mg/kg. Resmethrin technical grade active ingredient is very highly toxic to freshwater fish and invertebrates and to estuarine/marine fish and invertebrates. The LC50 or EC50 ranges from 0.28 to 11 µg/L for rainbow trout (*Oncorhynchus mykiss*), water flea (*Daphnia magna*), sheepshead minnow (*Cyprinodon variegates*), and pink shrimp (*Penaeus duorarum*). Both freshwater fish and estuarine/marine fish early life-stage chronic toxicity tests were used to evaluate the chronic toxicity of resmethrin. Results from the freshwater fish early life-stage toxicity test indicated a NOAEC of 0.32 µg/L and an LOAEC of 0.59 µg/L (EPA 2006b). Resmethrin is very highly toxic to honey bees (LD50 = 0.063 µg/bee) (USEPA 2006g).

SWRCB has evaluated aquatic life toxicity data from USEPA's Office of Pesticides' Ecotoxicity Database and has identified the lowest LC50 for resmethrin of 0.28 µg/L (SWRCB 2012). This value is based on toxicity to rainbow trout during a 96-hour test (Table 6.1).

4.1.8.4 Ecological Toxicity associated with ULV Application for Mosquito Abatement

Abbene et al. (2005) evaluated deposition of resmethrin in formulation with PBO (Scourge) following truck-mounted applications in fresh and salt water marshes at 6 sites. Resmethrin was not detected in water samples from any site (<0.005 µg/L). Deposition of resmethrin following aerial applications by helicopter was assessed in the same report (Abbene et al. 2005). Applied materials were detected in some water samples taken within 30 minutes of the application. The average concentration of resmethrin following helicopter applications was 0.037 µg/L. The highest concentrations were found in some samples collected from surface water within 1 hour of helicopter applications (0.293 µg/L resmethrin). The authors carried out a series of sample collections after two spray events to evaluate the persistence of the material in water. Resmethrin displayed an exponential decrease and was not detected (<0.005 µg/L) within 9 hours of the application. One site included two repeat weekly applications of resmethrin follow an application of methroprene the prior week. Concentrations of resmethrin and PBO measured after the second application were lower than those measured after the first application.

The same study included effects of aerial applications of resmethrin and PBO on two aquatic organisms: the sheepshead minnow (*Cyprinodon variegatus*) and the estuarine grass shrimp (*Palaemonetes pugio*) (Suffolk County 2006). The field study faced problems with low dissolved oxygen and high temperature, which compromised their ability to detect toxicity that may have been due to pesticide exposure. Therefore, dosing experiments and prey capture tests were conducted in the laboratory to measure toxicity of the applied products. These tests demonstrated that the doses used in the spray were not directly toxic to grass shrimp and did not affect their ability to capture prey under controlled conditions. Further laboratory experiments demonstrated that all of the mortality seen in the field could have been caused by low dissolved oxygen alone, using a USEPA time-to-death approach. Furthermore, their data showed that the chemicals used had very low persistence in the water column, as discussed above. Resmethrin was never detected in sediment and was not detected in samples from surface water taken more than 2 hours after the spray.

A related study evaluated benthic community structure, and found that benthic population differences could not be attributed to the application of pesticides, but were more likely due to environmental differences (Suffolk County 2006).

Zulkosky et al. (2005) sampled freshwater ponds, salt marshes, tidal inlets and embayments, and marine coastal water off Staten Island, New York within an hour after mosquito control applications of resmethrin (Scourge). In 2002, resmethrin was detected in five of ten locations at concentrations ranging from 0.0017 to 0.98 µg/L (detection limit of 0.0005 µg/L). No information was provided on application methods at each site.

4.1.8.5 Summary of Toxicity and Potential Effects

Resmethrin may also be persistent in environments free of light (e.g., bound to organic matter in anoxic soils and sediments). Due the potential for persistence and high toxicity to both aquatic and estuarine/marine fish and invertebrates, as well as the potential for endocrine disruption, this RUP may be of concern from a potential ecological risk perspective.

Resmethrin is contained in one product (18.5 percent) used by one of the Districts. It is applied to tree holes, residential areas near reclaimed marshes, and industrial areas for mosquito control. Seven applications during the spring and summer (2012 and 2011, respectively) resulted in the use of almost two gallons of product (<0.5 gallons of resmethrin). Studies have shown rapid dissipation/low persistence and no observed aquatic fish and invertebrate toxicity following aerial ULV applications.

Scourge® is being phased out of the District's program and replaced with a nonresmethrin alternative, making this product less problematic.

4.1.9 Tetramethrin

Tetramethrin is part of the pyrethroid class of pesticides and was first registered in 1968. It is a broad spectrum, nonsystemic, synthetic pyrethroid used to control flying and crawling insects in a number of commercial, horticultural and residential applications. Commercial applications include space, broadcast and crack-and-crevice treatment in a variety of commercial, industrial, residential, and institutional sites. Horticultural applications include foliar and fogger treatment on nonfood plants. Residential uses include pest control in homes and outdoor domestic structures, on gardens and direct application to cats, dogs and horses. Tetramethrin is a mixture of four stereoisomers designated as 1R-trans, 1R-cis, 1S-trans, and 1S-cis in an approximate ratio of 4:1:4:1. The first two isomers are the most insecticidally active (USEPA 2010c).

4.1.9.1 Environmental Fate

Tetramethrin is not a persistent pyrethroid in the environment (Table 4-8). It may be co-formulated with synergists, other active ingredients such as pyrethrins and pyrethroids, and growth inhibitors. These other ingredients are more persistent than tetramethrin and provide residual activity against insects not initially exposed. Tetramethrin decomposes rapidly by photolysis and hydrolysis in shallow, nonturbid water. Tetramethrin is slightly mobile in soil (USEPA 2010c).

Table 4-8 Degradation of Tetramethrin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5	15.9 to 19.7 Days	USEPA 2010b
Hydrolysis, pH 7	0.89 to 1.06 Days	USEPA 2010b
Hydrolysis, pH 9	13 to 20 minutes	USEPA 2010b
Photolysis (air)	30 minutes	USEPA 2010b

4.1.9.2 Human Toxicity

The USEPA considers tetramethrin to be slightly toxic via the oral and dermal routes (Category III or IV) and classifies it as a Category III eye irritant. The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. Tetramethrin meets the criteria for classification as a possible human carcinogen (USEPA 2010c) (Table 6.1).

4.1.9.3 Ecological Toxicity

The USEPA evaluated the potential ecological risk posed by use of tetramethrin both indoors and outdoors and concluded that exposure to non-target organisms is unlikely. Tetramethrin is considered practically nontoxic to birds and terrestrial mammals. The oral LD50 for bobwhite quail is >2,250 mg/kg bw (USEPA 2010c). Tetramethrin is considered highly toxic to aquatic organisms. The 96-hr LC50 of tetramethrin for rainbow trout is 3.7 µg/L. The 48-hr EC50 for immobilization of *Daphnia magna* is 45 µg/L (USEPA 2010c). Tetramethrin is also highly toxic to honey bees. The contact 48-hr LD50 is 0.155 µg/bee (Table 6.1).

4.1.9.4 Summary of Toxicity and Potential Effects

Tetramethrin is not persistent in the environment and degrades rapidly (photolysis and hydrolysis) in surface waters. It is only slightly mobile in saturated soils and is highly toxic to fish and aquatic invertebrates, as well as honeybees. Tetramethrin does not appear to pose a risk to humans; however, it has been classified as a possible human carcinogen.

Tetramethrin is used by one district during the spring, summer, and fall for yellow jacket and paper wasp control. One A single product (containing 0.1 percent tetramethrin) was applied directly containing

0.1 percent tetramethrin was to more than 80 nests, corresponding to times directly to wasp nests, which corresponded to approximately 2,000 ounces (volume) of product, or approximately 2 ounces of active ingredient used during the reporting year. Although there is a potential for effects pm fish and aquatic invertebrates, the uses by the Districts are generally focused, and very localized to minimize or eliminate those exposures.

4.1.10 Permethrin

Permethrin is a Type I pyrethroid (i.e., it lacks a cyano group at the α carbon position of the alcohol moiety) that primarily targets the nervous system of insects, causing muscle spasms, paralysis, and death (USEPA 2006d). Permethrin has been registered by the EPA since 1979, and is currently registered and sold in a number of products such as household insect foggers and sprays, tick and flea sprays for yards, flea dips and sprays for cats and dogs, termite treatments, agricultural and livestock products, and mosquito abatement products. Permethrin is also used at urban areas, household gardens, recreation areas, golf courses, hospitals, zoos, pastureland, and animal husbandry areas (CDPR 2010a).

4.1.10.1 Environmental Fate

Permethrin has very low mobility, is moderately persistent and has a high affinity to bind to soils/sediments and organic carbon. The relatively low water solubility and hydrophobic nature of permethrin leads to strong soil adsorption and a tendency to partition to sediment in aquatic systems. It is also slow to hydrolyze and biodegrade. Reported half-lives in surface water range from 1.8 hours to <2.5 days (Imgrund 2003). Major degradation pathways include photolysis and microbial metabolism (Table 4-9).

Table 4-9 Degradation of Permethrin

Degradation Method	Half-life	Reference
Hydrolysis	Stable (pH 3-6), 125–350 Days (pH 9)	DPR 2010, USEPA 2009a
Photolysis, ponds (water)	19.6 to 27.1 Hours	Imgrund 2003
Photolysis (soil)	104 to 324-Days	Imgrund 2003
Aerobic metabolism (soil)	3.5 to 113 Days	Imgrund 2003
Anaerobic metabolism (water)	113 to 175 Days	USEPA 2009a
Anaerobic metabolism (soil)	<3 to 197 Days	Imgrund 2003
Sediment/seawater degradation	<2.5 Days	Imgrund 2003
Streams, pH 7.0 to 7.5, 13 to 15°C	1.8 to 20.4 Hours	Imgrund 2003

4.1.10.2 Human Toxicity

Acute oral studies conducted with rats by the Department of Defense (DOD 1977) showed that exposure to permethrin caused tremors, weight loss, and increased liver and kidney weights starting at 185 mg/kg. The NOAELs in the DOD studies ranged from 92 to 210 mg/kg.

Oral LD50 values in rats range from 220 mg/kg to 8900 mg/kg and in mice, from 230 mg/kg to 1,700 mg/kg (IPCS 1999). The lethal dose of permethrin depended both on the vehicle in which permethrin was administered, as well as the cis/trans composition of the mixture. Permethrin is only slightly toxic via the dermal route, with an LD50 >2,000 mg/kg in rabbits (Braun and Killeen 1975b, Sauer 1980a). Permethrin of various cis/trans formulations has caused only very mild irritation when applied to either intact or abraded skin of rabbits (Braun and Killeen 1975b, a, Sauer 1980c, b). Dermal exposure in humans can cause tingling and pruritus with blotchy erythema on exposed skin, and has caused transient paresthesia (ATSDR 2003).

In humans, acute effects observed subsequent to ingestion of permethrin included nausea, vomiting, abdominal pain, headache, dizziness, anorexia, and hypersalivation. Reports of severe poisoning are rare and usually follow ingestion of substantial, but poorly described, amounts of permethrin. Symptoms of severe poisoning include impaired consciousness, muscle fasciculation, convulsions, and noncardiogenic pulmonary edema (ATSDR 2003). Dermal exposure in humans can cause tingling and pruritus (itchy sensation) with blotchy erythema (reddening of the skin) on exposed skin. Systemic effects are similar to those seen in acute and chronic ingestion with prolonged contact or contact with high concentrations of permethrin. Acute toxicity to permethrin via inhalation has been shown to be very small. The 4-hour LC50 was 23.5 mg/L for inhalation in rats (Kidd and James 1991).

The USEPA (2006a) has classified permethrin as category III for acute oral and acute dermal toxicity; category III for eye irritation potential, and category IV for dermal irritation potential. Technical grade permethrin is not considered a skin sensitizer (USEPA 2006a). Permethrin is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.1.10.3 Ecological Toxicity

Permethrin can be toxic to wildlife at high doses and it should not be applied or allowed to drift to crops or weeds where active foraging takes place (USEPA 2006d). However, in controlled toxicity tests with mammals permethrin is considered to have low mammalian toxicity (Nowak et al. 2000). Permethrin has low toxicity to dogs (Richardson 1999), gerbils, guinea pigs, hamsters, mice and rats (Sutton et al. 2007); however, dermal exposure in cats of 100 mg/kg of permethrin (equivalent to 1 mL of a 45 percent PSO in a 4.5 kg cat) has resulted in life-threatening effects (Hansen 2006).

Permethrin is practically nontoxic to birds (USEPA 2006d). The acute 5-day dietary LC50 for mallard ducks is >10,000 mg/kg/day.

Permethrin is very highly toxic to fish and aquatic invertebrates. The 96-hr LC50 for bluegill sunfish is 0.79 µg/L. The EC50 for *Hexagenia bilineauta* is 0.1 µg/L (USEPA 2006d).

Permethrin is highly toxic to bees in laboratory conditions from contact exposure. Acute contact and oral toxicity reported by USEPA (USEPA 2006d), was an LD50 of 0.13 µg/bee and 0.024-µg/bee, respectively. Theiling and Croft (1988) indicate that severe losses may be expected if bees are present at the time of treatment, or within a day thereafter. However, when used properly, permethrin has a strong repellent effect in the environment and has been considered to pose little risk to bees (USEPA 2006d) (Table 6.1).

4.1.10.4 Ecological Toxicity associated with ULV Application for Mosquito Abatement

Deposition of permethrin following truck-mounted application was evaluated in large seasonal wetlands in California (Jensen et al. 1999). Permethrin was not detected (<20 µg/L) in surface waters 1 hour after ULV applications. The authors found no significant differences in macroinvertebrate abundance and biomass or species diversity in areas treated with any of the materials when compared with untreated ponds. No mortality occurred in mosquitofish held in sentinel cages in treated ponds. Similarly, no difference in mortality was observed for mosquito larvae held in sentinel cages in treated ponds when compared with untreated ones. The authors concluded that ULV applications for adult mosquito control were not likely to significantly affect aquatic insects or fish in these habitats.

Davis and Peterson (2008) measured family diversity, richness, and evenness at 1, 7, 14, and 28 days after truck application of permethrin. Most response variables showed no significant treatment effect, although there were some reductions in number of individuals. The authors concluded that the reductions in aquatic non-target populations did not suggest any trends or persistent deleterious biological effects following a single adulticide application. Significant differences for the pond study were found on the dates closest to the spray event.

Pierce et al. (2005) evaluated deposition after two permethrin ULV applications made with truck-mounted equipment on Key Largo, Florida. They collected samples in the Atlantic Ocean and Florida Bay on either side of the treated area, including measurement of pesticide residues on glass fiber pads set on floats above the water surface, and water collected from the surface microlayer and 20 centimeters below the surface. Water was sampled from a canal running through the treated area following a third application. With the exception of a 0.07 µg/L sample from the bay, permethrin was not detected in the offshore samples; however, was detected in samples of the water surface microlayer taken from the canal. Detection of permethrin occurred in samples of the surface microlayer taken 2 to 4-hours after the applications (5.1 to 9.4-µg/L). Samples taken below the water surface did not contain detected residues. Within 12 hours of the application, permethrin was undetected in either surface microlayer or subsurface water. The application was carried out shortly before the arrival of a hurricane, and droplet size was not reported. This is the only published study in which significant amounts of pesticide were detected following an application by truck-mounted equipment. This study did not measure PBO concentrations.

4.1.10.5 Summary of Toxicity and Potential Effects

Permethrin may also be persistent in environments free of light (e.g., bound to organic matter in anoxic soils and sediments). Due the potential for persistence and high toxicity to both aquatic and estuarine/marine fish and invertebrates, as well as the potential for endocrine disruption, this RUP may be of concern from a potential ecological risk perspective.

Permethrin is used by three Districts for mosquito or yellow jacket wasp control during the spring, summer, and fall. Four products containing permethrin were used during the reporting year. Some Districts reported permethrin use in volume while others reported use by weight. For the reporting year, approximately 3 ounces (weight) and approximately 20 ounces (volume) of permethrin were applied. These products were used in reclaimed marshes, around residences, and applied directly to ground nests. Three of the products used contain between 2.5 and 4.6 percent permethrin. The fourth and most commonly used product contains 0.25 percent permethrin.

Studies have shown rapid dissipation/low persistence and no observed aquatic fish and invertebrate toxicity following aerial ULV applications; however, these studies are limited and inconclusive. Based on its potential for endocrine disruption, usage patterns, as well as the availability of safer alternatives for wasp control this product is generally used with careful and strict BMP applications.

4.1.11 Etofenprox

Etofenprox is a pyrethroid-like insecticide registered by the EPA since 2001. Similar to pyrethroids, etofenprox acts on ion channels of the insect nervous system. It is used as an insecticide with contact and stomach action against many pests on a broad range of crops. Etofenprox differs in structure from pyrethroids in that it lacks a carbonyl group and has an ether moiety, whereas pyrethroids contain ester moieties. It is used as an indoor nonfood crack and crevice insecticide, a spot treatment for pets, and as an outdoor fogger to control a variety of insect pests. Etofenprox is used in backyards, patios, barns, picnic areas, and other areas where flying and crawling insects are a problem. It is also used as a mosquito adulticide.

4.1.11.1 Environmental Fate

Etofenprox is virtually insoluble in water, stable to hydrolysis, and is rapidly degraded with light (Table 4-10). In water/sediment systems, etofenprox degrades relatively quickly. Residues of etofenprox are not likely to persist in the environment (FAO-WHO 2011).

Table 4-10 Degradation of Etofenprox

Degradation Method	Half-life	Reference
Hydrolysis	Stable	FAO-WHO 2011
Photolysis (water)	1.7 to 7.9 Days	Central Life Sciences 2009, FAO-WHO 2011
Photolysis (soil)	4.4 Day	Central Life Sciences 2009
Water/sediment systems (water)	1 to 10 Days	FAO-WHO 2011
Water/sediment systems (sediment)	6 to 20 Days	FAO-WHO 2011

4.1.11.2 Human Toxicity

Etofenprox has low acute toxicity via the oral, dermal, and inhalation routes. It is not an acute eye or skin irritant and is not a dermal sensitizer, however etofenprox does cause skin irritation after repeated exposure (USEPA 2008a).The acute oral and dermal LD50 values in rats are both greater than 2,000 mg/kg. The acute oral LD50 value in the dog is greater than 5,000 mg/kg. The acute 4-hour inhalation LC50 value in the rat is greater than 5.88 mg/L. Etofenprox was not irritating to rabbit skin or rabbit eyes. Etofenprox was not a skin sensitizer in the guinea-pig maximization test (FAO-WHO 2011). The major target organs of etofenprox are the liver, thyroid, kidney, and hematopoietic system (EPA 2008d). In rats the target organs are the liver and thyroid. The NOAEL for chronic toxicity is 3.7 mg/kg/day for male rats. The target organ in mice is the kidney. The NOAEL is 3.1 mg/kg/day for mice (Wellmark International 2010) (Table 6.1).

4.1.11.3 Ecological Toxicity

Etofenprox is toxic to aquatic organisms, including fish and invertebrates. The LC50 for rainbow trout is 3.3 µg/L and the LC50 for bluegill is 8.5 µg/L. Product formulations are toxic to bees exposed to direct treatment on blooming crops and weeds (Wellmark International 2010).

SWRCB has evaluated freshwater aquatic life toxicity data from USEPA Office of Pesticides' Ecotoxicity Database and has identified the lowest LC50 for etofenprox as 0.019 µg/L. This value is based on toxicity to mysid during a 96 hour test (SWRCB 2012) (Table 6.1).

4.1.11.4 Summary of Toxicity and Potential Effects

Etofenprox does not tend to persist in the environment or appear to pose a risk to mammals as it is frequently applied to backyards and patios, and sometimes directly to domestic pets. It does exhibit some toxicity to fish and aquatic invertebrates; however, it degrades rapidly in surface waters thereby reducing the potential for long-term exposures and adverse effects.

Etofenprox was applied as a single application to a waste treatment plant in both fall and summer by one District during the reporting year. Approximately 14-ounces (volume) of etofenprox was used for the two treatments. It is generally applied during the nighttime hours when sensitive receptors such as honeybees are not active. Etofenprox is available in a new product, Zenivex that does not require synergists such as PBO. Therefore, it likely exhibits less toxicity than others that require co-application with other chemicals, including synergists to increase its efficacy for mosquito control. Based on toxicity, environmental fate, and usage patterns, etofenprox, using BMP is not likely to result in unwanted adverse impacts.

4.1.12 Piperonyl Butoxide

PBO was first registered in the 1950s and acts as a synergist. Synergists are chemicals that primarily enhance the pesticidal properties of other active ingredients, such as pyrethrins and synthetic pyrethroids. PBO is a registered active ingredient in products used to control many different types of flying and

crawling insects and arthropods, although there are no products that contain only PBO. It is registered for use in agricultural, residential, commercial, industrial, and public health sites. PBO interferes with the insect's ability to detoxify pyrethrins and pyrethroids, thus enhancing the product's effectiveness. PBO inhibits microsomal enzymes in target organisms by direct binding to these enzymes and inhibits the breakdown of other pesticides including pyrethrins and pyrethroids (USEPA 2006e).

4.1.12.1 Environmental Fate

PBO degrades rapidly in the environment by photolysis in water and is metabolized by soil microorganisms (Table 4-11). Other tested routes of degradation, such as hydrolysis, aerobic and anaerobic aqueous metabolism, are very slow or have questionable rates due to experimental difficulties, as in the case of soil photodegradation. PBO is moderately mobile in soil-water systems (USEPA 2006e).

Table 4-11 Degradation of Piperonyl Butoxide

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable	USEPA 2006b, FAO-WHO 2002c
Photolysis (water)	8.4 Hours	USEPA 2006b
Aerobic metabolism (water/sediment)	>30 Days	FAO-WHO 2002c
Anaerobic metabolism (water/sediment)	>181 Days	FAO-WHO 2002c
Terrestrial dissipation (soil)	1 to 14 Days	FAO-WHO 2002c

4.1.12.2 Human Toxicity

PBO has a low acute toxicity by oral, inhalation and dermal routes. It has been assigned toxicity USEPA Category III by oral and dermal and Category IV by inhalation exposure routes. In the acute studies, PBO has been identified as minimally irritating to eyes and skin, and is a dermal sensitizer. The oral LC50 was 4,570 to 7,220 mg/kg in rats, the dermal LC50 was found to be greater than 2,000 mg/kg in rabbits, and the inhalation LC50 was found to be greater than 5.9 mg/L in rats (USEPA 2006e). The major target organ for PBO is the liver. Subchronic studies in rats showed PBO treatment caused increases in liver weight and clinical parameters such as cholesterol and enzyme activity compared to controls (USEPA 2006e). PBO is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.1.12.3 Ecological Toxicity

PBO is moderately toxic to freshwater fish on an acute basis (LC50 = 1.9 mg/L) based on studies of rainbow trout (*Oncorhynchus mykiss*). PBO ranges from moderately toxic (LC50 = 12.0 mg/L) to highly toxic (LC50 = 0.51 mg/L for waterflea, *Daphnia magna*) to freshwater invertebrates on an acute basis. PBO is moderately toxic to estuarine/marine fish (LC50 = 3.94-mg/L) based on observed effects to sheepshead minnow (*Cyprinodon variegatus*) on an acute basis. PBO is highly toxic to estuarine invertebrates (LC50 = 0.49 mg/L) based on studies with mysid shrimp (*Mysidopsis bahia*). PBO is highly toxic to amphibians on an acute basis (LC50 = 0.21 mg/L) based on studies with western chorus frog tadpole (*Pseudacris triseriata*) (USEPA 2006e).

A chronic early life stage of fish study with fathead minnow evaluated embryo survival at hatch and length and weight of larvae. This study found a LOEC of 0.11 mg/L. A study with water fleas found a LOEC of 0.047 mg/L (USEPA 2006e)

SWRCB has evaluated toxicity data from U.S. EPA's Office of Pesticides' Ecotoxicity Database for PBO when applied in formulation with pyrethrins. The lowest LC50 was 0.14-µg/L, based on toxicity to mysid during a 96-hour test. Toxicity data was also evaluated for PBO when applied in formulation with

resmethrin. SWRCB identified the lowest LC50 as 1.3 µg/L based on toxicity to pink shrimp during a 96-hour test. For PBO applied in formulations other than pyrethrins or resmethrin, SWRCB identified the lowest LC50 as 490 µg/L based on toxicity to mysid during a 96-hour test (SWRCB 2012). PBO is practically nontoxic to honey bees on an acute oral basis (LD50 >25 µg/bee) (USEPA 2006e) (Table 6.1).

4.1.12.4 Ecological Toxicity associated with ULV Application for Mosquito Abatement

Following aerial applications of Evergreen Crop Protection EC 60-6 (6 percent pyrethrins, 60 percent PBO) in Sacramento for West Nile virus, Larry Walker Assoc. (2006) reported results of water testing on samples from 10 waterways within the treatment area. Treated areas were sprayed nightly for 3 days. One additional application occurred 9 days prior to the 3-day event at selected locations. Samples were taken immediately after application (within 1 to 6 hours), and the next day (16 to 23 hours after the application). Piperonyl butoxide was detected in water from 14-of the 25 samples collected after the application. Concentration of PBO ranged from <1.0 to 20 µg/L (average 2.036 µg/L) immediately after application. PBO concentrations ranged from <1.0 to 4.2 µg/L with an average of 0.853 µg/L in samples taken between 16 and 23 hours after application. Of the 31 samples taken between 16 and 23 hours after application, PBO was detected in 11 samples. Water samples were tested eight days following aerial applications, from four sites. No PBO was detected in any of these samples, therefore, the duration of persistence of PBO appears to be greater than 16 hours, but less than 1 week.

Testing was also carried out by Weston et al. (2006) following the same applications. Prior to aerial spraying, PBO was not detected in sediment samples; however, PBO was detected at 0.2 µg/L in 2 of 4 water samples. PBO was detected in water (0.44-to 3.92 µg/L, all 7 samples) and sediment (16 to 61.4 µg/kg, for 4-of 6 samples) at 10 to 34-hours after application. Neither water nor sediment was tested at later intervals, so the duration of persistence could not be determined in this study. Laboratory tests were conducted to determine the effects of short-term chronic exposure of *Ceriodaphnia dubia* to water collected after the spray events, following USEPA protocol. No significant differences in mortality were observed. In addition, sediment toxicity tests were performed with the amphipod *Hyalella azteca,* and toxicity was observed in samples collected both before and after application. The authors concluded that pyrethrins and PBO should present little risk to aquatic organisms due to the low toxicity and lack of long-term persistence, but that PBO had the potential to enhance toxicity of other pesticides, especially pyrethroids, already present in the environment. Weston et al. performed additional laboratory tests to determine the effect of PBO on toxicity of pyrethroids present on sediment, and found that even by removing 80 percent of the overlying water and replacing it with fresh PBO solution daily, within 24-hours, over 30 percent of PBO is lost, most likely to photo degradation. The results indicated that most sediments present at the creeks used for this study already contained concentrations of pyrethroids acutely lethal to *H. azteca* from urban uses not related to mosquito control activities.

Water and soil deposition of pyrethrins and PBO following aerial applications was evaluated at two sites in California (Schleier III et al. 2008). Water was sampled after aerial applications of pyrethrins and PBO in irrigation ditches at one site (Princeton) and in static ponds at another (Colusa). PBO was detected at low levels and decreased exponentially with time. Average PBO concentrations were 0.0125 to 0.0199 microgram per square centimeter (µg/cm^2) on ground deposition pads and 0.1723 to 1.274-µg/L in water samples, immediately following the applications. Within 36 hours of the applications, PBO had decreased to background levels in water. Concentrations of PBO decreased 77 percent between 1 and 12 hours after the spray event. The authors concluded that the amounts of pyrethrins and PBO deposited on the ground and in water after aerial ULV insecticide applications are probably lower than those estimated by previously published studies to predict exposure and risk.

Lawler et al. (2008) evaluated pyrethrins and PBO in sediment following multiple applications of pyrethrins formulated with PBO from truck-mounted equipment in the Colusa and Sacramento National Wildlife Refuges in California. Stock tanks were filled with a layer of soil overlain with 1,150 liters of water. Zooplankton (*Daphnia magna*) were held in sentinel cages in the water column and mayfly larvae

(*Callibaetis californicus*) were placed in cages at the bottom of each tank, where they were in contact with sediment. ULV applications of pyrethrins formulated with PBO were made from truck-mounted equipment twice weekly for six weeks. Concentration in sediments and sentinel survival were evaluated after application 5 and 11. PBO concentrations ranging from 8.37 to 14.9 µg/kg were seen in 5 of 6 tanks after five applications, but in only 2 of 6 tanks after 11 applications (1.93 and 2.55 µg/kg). There was no significant difference in mortality for mayfly larvae held in sentinel cages on the sediment. Likewise, there was no significant difference in mortality seen in *D. magna* held in the water column. They concluded that applications of pyrethrins and PBO at rates used for mosquito control did not have detectable effects on the indicator species. The persistence of PBO in sediment was not evaluated in this study. PBO-synergized pyrethrins had no detectable effect on the survival of *D. magna* held in tanks in the spray area, even after 11 biweekly spray events.

Amweg et al. (2006) evaluated deposition of PBO in water and sediment following truck-mounted applications of synergized pyrethrins to a freshwater wetland in Colusa County in 2004. PBO was detected in 2 of 18 sediment samples above the reporting limit of 2.0 µg/kg, at 3.27 and 3.0 µg/kg, respectively. PBO was detected in 3 of 10 samples of water at concentrations above the reporting limit of 0.01 µg/L, ranging from 0.04-to 0.08 µg/L. The highest concentrations of PBO were observed in samples obtained within 12 hours of spraying; concentrations in water and sediment were below the reporting limit in samples taken one week after the last ULV application (Amweg et al. 2006).

Several papers were published documenting that ULV-applied mosquito adulticides do not accumulate in water or sediment during repeated applications. Chemical testing was conducted following multiple spray events in 2006 by Amweg et al. There was no increase in the level of pyrethrins or PBO following multiple daily spray events, and the concentration had returned to background level when samples taken one week after the last application were tested. Similarly, Lawler et al. (2008), reported that the concentration of pyrethrins and PBO in tanks within a treated area were not significantly higher after 11 applications than in samples taken after the fifth application. In many cases, the concentrations were actually lower following the 11th spray event than after the fifth spray event. Accumulation of PBO was evaluated by Amweg et al. (2006). PBO did not accumulate in water or sediment, even after eight biweekly applications by truck-mounted equipment over the course of two months.

ULV applications of the resmethrin formulated with PBO in Suffolk County New York have been evaluated (Abbene et al. 2005). Deposition of resmethrin and PBO following truck-mounted applications in fresh and salt water marshes was assessed at 6 sites. PBO was detectable at low levels (0.008 µg/L and 0.017 µg/L) in 2 of 6 water samples taken immediately after the application. Deposition of resmethrin and PBO following aerial applications by helicopter was assessed in the same report (Abbene et al. 2005). Applied materials were detected in some water samples taken within 30 minutes of the application. PBO was detected more frequently than resmethrin, and detection of PBO was more common after helicopter applications (83 percent) than following those carried out by truck (33.3 percent). The average concentration of PBO was 4.361 µg/L. The highest concentrations were found in some samples collected from surface water within 1 hour of helicopter applications (59.8 µg/L PBO). The authors carried out a series of sample collections after two spray events to evaluate the persistence of the materials in water. PBO was not detected (<0.005 µg/L) in samples taken 96 hours after the application (Abbene et al. 2005). One site included two repeat weekly applications of resmethrin follow an application of methroprene the prior week. Concentrations of resmethrin and PBO measured after the second application were lower than those measured after the first application.

The same study included effects of aerial applications of resmethrin and PBO on two aquatic organisms: the sheepshead minnow (*Cyprinodon variegatus*) and the estuarine grass shrimp (*Palaemonetes pugio*) (Suffolk County 2006). The field study faced problems with low dissolved oxygen and high temperature, which compromised their ability to detect toxicity that may have been due to pesticide exposure. Therefore, dosing experiments and prey capture tests were conducted in the laboratory to measure toxicity of the applied products. These tests demonstrated that the doses used in the spray were not

directly toxic to grass shrimp and did not affect their ability to capture prey under controlled conditions. Further laboratory experiments demonstrated that all of the mortality seen in the field could have been caused by low dissolved oxygen alone, using a USEPA time-to-death approach. Furthermore, their data showed that the chemicals used had very low persistence in the water column, as discussed above. PBO was last detected in samples taken 48 hours after the spray.

Another related study evaluated benthic community structure, and found that benthic population differences could not be attributed to the application of pesticides, but were more likely due to environmental differences (Suffolk County 2006).

Zulkosky et al. (2005) sampled freshwater ponds, salt marshes, tidal inlets and embayments, and marine coastal water off Staten Island, New York within an hour after mosquito control applications of resmethrin formulated with PBO (Scourge). PBO was detected in all but one location at concentrations ranging from 0.0006 to 15 µg/L. PBO was still present at three locations in samples collected three days after a Scourge spray. No information was provided on application methods at each site. Zulkosky et al. (2005) also evaluated phenothrin (in formulation with PBO) applied as Anvil. In 2002, PBO was detected in all samples at concentrations ranging from 0.0003 to 0.0007 µg/L. In 2003, PBO was detected at 0.020 µg/L immediately after spraying Anvil and was found at concentrations ranging from <0.0005 to 0.007 µg/L 10 days later.

New York City Department of Health sampled 32 locations for phenothrin formulated with PBO before and after spray events during mosquito adulticide applications that occurred during July through September 2000. Out of the 68 post-application samples collected by the city, only one sample had concentrations of PBO greater than the 0.5 µg/L reporting limit: 1.03 µg/L for PBO for a sample collected on August 5, 2000, at Alley Park Pond in Queens (Suffolk County 2006).

The Massachusetts Department of Agricultural Resources conducted a study where phenothrin formulated with PBO was applied aerially as Anvil 10+10 ULV to six sites. There were no detections of phenothrin during this study; however, PBO was detected at 0.12 µg/L. (Massachusetts Department of Agricultural Resources 2010).

Davis and Peterson (2008) also evaluated phenothrin formulated with PBO and applied as Anvil 10+10 ULV. The authors concluded that the reductions in aquatic non-target populations did not suggest any trends or persistent deleterious biological effects following a single adulticide application.

4.1.12.5 Summary of Toxicity and Potential Effects

PBO has been an effective synergist used in mixtures with other insecticidal active ingredients since the 1950s. It degrades rapidly in soil and water and, therefore, does not tend to persist in the environment. PBO may be highly toxic to some species of fish and aquatic invertebrates and is being evaluated as a possible endocrine disruptor.

PBO is contained as a secondary ingredient along with pyrethrin, resmethrin, and permethrin in several products used by eight Districts. These products are used throughout the year in manmade and natural sites with standing and moving water, as well as tree holes, ditches, and residential areas. There were several hundred applications during the reporting year. It is generally applied using ULV techniques, which are designed for low chemical persistence and toxicity to non-target receptors. PBO is not expected to pose risk to aquatic organisms especially when applied using ULV techniques; however, it has been shown to have the potential to enhance toxicity of other pesticides and should be considered when it is an additive to a pesticide formulation.

4.2 Organophosphate Insecticides

4.2.1 Naled

Naled is an organophosphate insecticide that has been registered since 1959 for use in the U.S. It is used in rotation with pyrethrins or pyrethroids for control of adult mosquitos to avoid the development of resistance. In addition to use for controlling adult mosquitoes, naled also has indoor and outdoor general use, and is used on food and feed crops, farms, dairies, pastureland, and in greenhouses and over standing water (CDPR 2010a). Dichlorvos (DDVP), a registered OP insecticide, is a metabolite of naled (USEPA 2006c).

4.2.1.1 Environmental Fate

Naled is readily degraded in water, under sunlight, in soil under aerobic and anaerobic conditions (Table 4-12), in the air, and on plants. On plant surfaces, naled is degraded to DDVP. Naled is more mobile in soil of low organic content such as sandy loam when compared with other soil types (CDPR 1999). Naled has low water solubility and can volatilize (CDPR 1999, 2001).

Table 4-12 Degradation of Naled

Degradation Method	Half-life	Reference
Hydrolysis, pH 5 (water)	96 Hours	DPR 1999
Hydrolysis, pH 7 (water)	15.4 to 17 Hours	DPR 1999
Hydrolysis, pH 9 (water)	1.6 to 1.7 Hours	DPR 1999
Photolysis (water)	3.7 to 4.4-Days	DPR 1999
Photolysis (plant surfaces)	<5 Days	DPR 1999
Aerobic metabolism (soil)	3 Days	DPR 1999
Anaerobic metabolism (soil)	6 Days	DPR 1999

4.2.1.2 Human Toxicity

Naled is rapidly absorbed by all routes (oral, inhalation, and intraperitoneal) and distributes to all tissues in the rat, chicken, goat, and cow. The oral LC50 for naled technical grade active ingredient is 81 to 336 mg/kg in rats or mice, the dermal LC50 is 354-to 800 mg/kg in rats or rabbits, and the inhalation LC50 is 3.1 to 156 mg/L in rats or mice (CDPR 1999) (Table 6.1).

4.2.1.3 Ecological Toxicity

Naled technical grade active ingredient was found to be moderately toxic to highly toxic to wide range of species including rainbow trout (LC50 = 0.08 mg/L), blue gill (LC50 = 0.33 mg/L), sheephead minnow (LC50 = 1.2 mg/L), mullet (LC50 = 0.55 mg/L), daphnia (LC50 = 0.35 µg/L), pink shrimp (EC50 = 5.5 µg/L), grass shrimp (LC50 = 8.9 mg/L), and eastern oyster (EC50 = 0.19 mg/L). Lethal effects were also found in honey bees (LD50 = 0.48 µg/bee) and mallards (LD50 = 52 mg/kg) (CDPR 1999).

SWRCB has evaluated the U.S. EPA's Office of Pesticides' Ecotoxicity Database to access toxicity of naled to freshwater aquatic life and has identified LC50 values that range from 0.14-to 3,300 µg/L (SWRCB 2012) (Table 6.1).

4.2.1.4 Ecological Toxicity associated with ULV Application for Mosquito Abatement

Tucker et al. (1987) evaluated deposition and non-target effects for truck-mounted and aerial applications of naled. The maximum concentration of naled in water samples following truck applications (0.71 µg/L)

occurred 15 minutes after the application. The concentration in water decreased exponentially after this; detected concentrations persisted for 4-hours. No significant mortality was observed in copepods or fish exposed from truck-mounted applications. The same study evaluated deposition of these materials following applications made from aircraft (Tucker et al. 1987). The maximum concentration of naled in water samples following aerial applications (20.15 µg/L) occurred 27 minutes after the application. The concentration in water decreased exponentially after this; detected concentrations persisted for 9 hours. Deposition rates for naled from aerial applications were much higher (47 to 68 percent) than those resulting from ground applications (21 to 22 percent). The authors reported significant mortality in copepods held in sentinel cages in the treated area and exposed to naled by aerial application. No significant mortality was observed for juvenile fish held in the treated area. This is the only report of significant mortality in aquatic organisms following a ULV application. The size of droplets released is not given and the amount of material recovered from glass filter pads placed on the ground was unusually high. Perhaps the conditions of the applications resulted in a greater proportion of the product reaching the ground.

In what may have been the same study, Wang et al. (1987) also investigated the fate of naled after aerial ULV applications of mosquito adulticides at a salt marsh in Florida. Approximately 30 minutes after application, the concentration of naled in the water was 20.15 µg/L, decreasing to 0.2 µg/L at 6.45 hours, and was not detected at 12.45 hours (detection limit of 0.05 µg/L). The peak concentration of dichlorvos (a breakdown product of naled) was 2.22 µg/L approximately 30 minutes after application, and was still detectable at 12.45 hours (0.28 µg/L).

Deposition of naled during aerial applications was also evaluated (Pierce et al. 2005). Naled was detected in low concentrations (0.19 µg/L) in the water surface microlayer at 1 of 18 sites. It was not detected in subsurface water (detection limit 0.05 µg/L). Residues were not detectable in the water surface microlayer 12 hours after the application. Dichlorvos, a breakdown product of naled, and itself a registered pesticide, was detected at 2 to 4-hours after the application. Trace amounts were still detectable at 10 to 12 hours post-treatment.

4.2.1.5 Summary of Toxicity and Potential Effects

Naled has low water solubility and is mobile in some soils. It is moderately toxic to mammals, fish, and aquatic vertebrates. Naled was not used by the Districts during the reporting year.

4.2.2 Temephos

Temephos is a cholinesterase inhibitor registered by the EPA in 1965 to control mosquito larvae (USEPA 2000). Temephos is the only organophosphate with larvicidal use and is often used to help prevent mosquitoes from developing resistance to the bacterial larvicides. Temephos is used on lakes, ponds, reservoirs, swamps, marshes, tidal areas, intermittently flooded areas, catch basins, drainage systems, irrigation systems, ornamental ponds, wastewater, polluted and stagnant water, and is applied by mosquito abatement Districts (CDPR 2010a). MVCAC member agencies primarily apply temephos to manmade sources such as tire piles, utility vaults, and cemetery urns. Mosquito control products containing temephos are not labeled for application to agricultural lands or pasture and are not used in such sites. Temephos provides effective control of mosquito larvae in highly polluted water (containing high levels of decaying organic matter, such as rotting leaves or manure).

Temephos is a broad-spectrum insecticide and has also been used operationally to control midges and black flies for many years. However, the concentration that effectively controls mosquito larvae is well below that needed for control of other insects. In addition, midges and black flies are found in different habitats than larval mosquitoes. The larval stage of most midges develop in sediment at the bottom of water bodies, while black flies develop attached to hard surfaces in swift moving rivers and streams. Materials commercially available for midge control are heavy and designed to release their active ingredients on the floor of the water body and those for control of black flies are placed in flowing streams and allowed to move down with the current.

4.2.2.1 Environmental Fate

The presence of microorganisms in aquatic environments and exposure to sunlight are likely to be the predominant means of transformation/dissipation of temephos (Table 4-13; (USEPA 1998a)). Temephos is an extremely hydrophobic material with low solubility. It adsorbs rapidly onto organic material in the water and binds strongly to soils. Temephos breaks down in water through photodegradation and bacterial degradation (USEPA 2000).

Table 4-13 Degradation of Temephos

Degradation Method	Half-life	Reference
Hydrolysis	>86 Days	USEPA 1998a
Photolysis (water)	15 Days	USEPA 1998a
Aerobic metabolism (water)	17.2 Days	USEPA 1998a
Anaerobic metabolism (water)	12.2 to 27.2 Days	USEPA 1998a

The RED cites a study submitted by the registrant in which temephos was monitored in sediments following field applications for mosquito control over a 3-year period. The active ingredient became undetectable in sediment after 24-hours (USEPA 2000). Lores et al. (1985) found that concentrations in water of 15 to 60 ppb immediately following the application declined to 2 to 5 ppb within 24-hours. Sanders et al. (1981) reported similar results. Pierce et al. (1989) examined aerial application of liquid formulation of temephos to a mangrove swamp in Florida, and found the material had become undetectable 4-hours after the application in intertidal water. It persisted in simulated intertidal pools for 72 hours. The liquid and BG formulation products are designed to deliver the active ingredient to the water surface in order to maximize exposure of mosquito larvae.

4.2.2.2 Human Toxicity

Temephos is moderately toxic via the oral, dermal, and inhalation routes. The oral LD50 in rats is 444-mg/kg. The dermal LD50 in rabbits is 970 mg/kg. The inhalation LC50 in rats is 1.3 mg/L (USEPA 2012) (Table 6.1).

4.2.2.3 Ecological Toxicity

Temephos has low toxicity for vertebrates at the levels used for mosquito control (USEPA 2000). However, it is toxic to insects and some other invertebrates (Brown et al. 1996), and the margin of safety between concentrations effective for mosquito control and levels at which non-target impacts occur is much narrower than that of s-methoprene or the bacterial larvicides (Brown et al. 1999, Lawler et al. 1999, Hurst et al. 2007).

Temephos is slightly to moderately toxic to fish (USEPA 2000); however, field applications result in concentrations of temephos far lower than that at which fish are affected. Field studies have repeatedly demonstrated a lack of impact on fish inhabiting treated sites. Mulla et al. (1964) reported that temephos was nontoxic to mosquito fish that were confined in screened cages for one week in artificial ponds treated with 0.1 pound per acre AI. Similarly, no significant mortality was observed in juvenile snook (*Centroponzis undecimalis*) or sheepshead minnow (*Cyprinodon*) caged in a mangrove swamp treated with aerial applications of liquid temephos (Pierce et al. 1989). Tietze et al. (1991) demonstrated laboratory tests that liquid formulations of temephos were nontoxic to young mosquitofish (3 to 5 days old) at field application rates. Mosquitofish exhibited no mortality when exposed to up to 100 times field application rates.

Temephos is highly toxic to aquatic invertebrates, but many groups are only impacted at concentrations far above those used for mosquito control applications (USEPA 2000). Von Windeguth and Patterson (1966) conducted laboratory tests on temephos and fenthion (another organophosphate) to determine margin of safety for treatment of midges in a lake. The dose of fenthion used for midges was above that which caused mortality in shrimp and amphipods. Abate (temephos) was less toxic to most aquatic non-target organisms than fenthion and not toxic to shrimp (*Palomonetes paladosus*) and amphipods (*Hyalella azteca*) at concentrations used for mosquito control applications (LD50 was 1 mg/L and 0.65 mg/L, respectively). Neither product was toxic to fish at levels necessary to kill midge larvae (0.25 lb active ingredient per acre). In field tests, they reported that no noticeable mortality was observed for Odonates (dragonflies), copepods, ostracods, or shrimp (Von Windeguth and Patterson 1966).

Temephos does have an immediate impact on some groups of planktonic crustaceans, with copepods and brachiopods (*cladocera*) being more sensitive than amphipods or ostracods. Fortin et al. (1987) studied the impact of temephos on non-target organisms in rectangular manmade pods. Application of temephos resulted in an immediate reduction in populations of copepods and cladocerans, but populations began to recover within 3 days and had reached pre-treatment levels within 2 to 3 weeks. Ostracopods in the ponds were not affected. Helgen et al. (1988) also reported sharp reductions in populations of calanoid copepods (*Diaptomus leptopus*) and cladocreans (*Daphnia pulex*, *Simocephalus* sp., and Chydoridae) following applications of temephos. Copepods exhibited varying degrees of recovery. However, some cladocerans remained absent from the treated area for up to 35 days. In an open field setting, Lawler et al. (1999) reported that aerial applications of temephos to a mangrove swamp in Florida resulted in no observable effect on survival of amphipods (Talitridae), the primary non-target organism present.

Several studies have evaluated effects on non-target insects. A field study of repeated applications of temephos to a saltmarsh in New Jersey concluded that species richness, diversity, and community structure of aquatic insects was unaffected (Campbell and Denno 1976). Stoneflies and mayflies are particularly susceptible to temephos and the label carries a prohibition against applying Abate in habitats containing these organisms.

Among the materials available for control of mosquito larvae, temephos has the narrowest margin of impact and the greatest potential for effects to non-target organisms. However, it is an effective method of control in isolated sources that may be difficult to treat by other means, such as sources with high concentrations of organic material, and ones in which other less toxic alternatives have failed to produce adequate levels of control. Temephos was in widespread use in California for control of larval mosquitoes from 1965 into the mid-1980s. The microbial pesticides, methoprene, and surface oils are used much more frequently now and have largely replaced temephos as the method of choice for larval sources in water of the U.S. Temephos is more widely used in other parts of the U.S. such as Delaware, New Jersey, New York, Maryland, and Florida (Table 6.1).

4.2.2.4 Summary of Toxicity and Potential Effects

Temephos has extremely low water solubility and binds strongly to soils. It is moderately acutely toxic to mammals and fish, but highly toxic to non-target aquatic invertebrates (e.g., stoneflies, mayflies). The USEPA (2000) states that there is likely no exposure of people to temephos in drinking water or from residential use. It is not expected to have direct impact on terrestrial animals and the use of temephos has declined over time (USEPA 2000). Temephos was used in one product by two Districts during the reporting year. It is typically applied in all four quarters of the year resulting in the use of over 1,000 pounds of product. Active ingredient concentration in this product is 5 percent resulting in a total use of about 50 pounds of AI. Based upon the environmental fate, toxicity, and BMP approaches, the use patterns for temephos should not result in unwanted adverse effects.

4.3 Mosquito Larvicides

4.3.1 *Bacillis sphaericus* (Bs)

Microbial larvicides are bacteria that are registered as pesticides for control of mosquito larvae in outdoor areas such as irrigation ditches, flood water, standing ponds, woodland pools, pastures, tidal water, fresh- or saltwater marshes, and stormwater retention areas (USEPA 2007c). The microbial larvicides concentrates registered for use in California include *Bacillus sphaericus* (Bs) and *Bacillus thuringiensis* subspecies *israeliensis* (Bti). These concentrates include fermentation solids, bacterial spores, and insecticidal toxins. Their mode of action requires that they be ingested to be effective, which means they cannot be used to control mosquitoes at some life stages (late 4th instar larvae and pupae). Bs spores contain a protein that damages and paralyzes the gut of mosquito larvae that ingest the *spores*, thus starving the larvae (USEPA 1999). A standard bioassay similar to that used for Bti has been developed to determine preparation potencies.

Bs was first registered by the EPA in 1991 for use against mosquito larvae. Bs can control mosquito larvae in highly organic aquatic environments, including sewage waste lagoons, animal waste ponds, and septic ditches. Bs is used on rice, fruit trees, walnuts, almonds, corn, asparagus, cotton, dates, and other crops. It is also applied to alfalfa, pastures, agricultural drainage systems, animal drinking water, fodder grasses, irrigation systems, swimming pools, ornamental ponds and fountains, catch basins, wastewater, bilge water, industrial processing water, industrial waste disposal systems, solid wastes sites, garbage dumps, and on tidal areas, swamps, marshes, bogs, intermittently flooded areas, standing water, and by mosquito abatement Districts (CDPR 2010a).

4.3.1.1 Environmental Fate

Dormant Bs spores may persist in the environment for several weeks to months; however, the δ-endotoxins generally persist for 2 to 4-weeks following application. Factors affecting its persistence include the formulation of the *B. sphaericus* product, agitation of the waterbody, receiving water quality and temperature. The δ-endotoxins produced by Bs degrade rapidly in sunlight as a result of exposure to ultraviolet radiation and are degraded by soil microorganisms. Bs, as with other soil microbes, does not percolate through the soil and readily binds to sediments within the water column, and is, therefore, not available to contaminate ground water (Maine Department of Environmental Protection 2010). Field evaluations of VectoLex-CG (a commercial formulation of *B. sphaericus*) have shown environmental persistence for several weeks (Mulla et al. 1988).

4.3.1.2 Human Toxicity

Bs is not pathogenic and does not demonstrate any systemic toxicity. An acute oral toxicity/pathogenicity study was conducted with Bs technical material in rats. An oral dose of approximately 1 x 10^8 colony forming units (CFU) administered to rats resulted in no mortalities, no evidence of pathogenicity or treatment related toxicity in rats given an oral, intratracheal installation or intravenous dose. In an acute oral toxicity study, Bs technical material caused no deaths in rats given a dose of 5,000 mg/kg; therefore the acute oral LD50 was greater than 5,000 mg/kg. There was no mortality in rabbits over the 14-day observation period following a 2,000 mg/kg dermal application for 24-hours; thus, the acute dermal LD50 was greater than 2,000 mg/kg. In a 4-hour acute inhalation toxicity study in rats, the maximum attainable concentration was 0.09 mg/L, with 13.3 percent of the particles having a mass median aerodynamic diameter of >10 microns. Since there was no mortality or no clinical signs during exposure or the 14-day observation period, the 4-hour inhalation LC50 was greater than 0.09 mg/L. Dermal irritation of Bs technical material was moderately irritating to rabbit skin at 72 hours. Irritation and iridal effects following a 100 mg aliquot of Bs placed in the eye of rabbits were no longer present at day 10 post-treatment (USEPA 1997a) (Table 6.1).

4.3.1.3 Ecological Toxicity

Available literature indicates that Bs is not acutely toxic to non-target species, including birds, mammals, fish and invertebrates. Bs has a very low toxicity for fish, and all aquatic invertebrates. Amounts that effective control mosquito larvae are many levels of magnitude below those, which affect other organisms. Acute aquatic freshwater organism toxicity tests were conducted on bluegill sunfish, rainbow trout and daphnids. The 96-hour LC50 and No Observable Effect Concentration (NOEC) value for bluegill sunfish and rainbow trout was greater than 15.5 mg/L; the 48-hour EC 50 and NOEC value for daphnids was greater than 15.5 mg/L. Acute aquatic saltwater organism toxicity tests were conducted on sheepshead minnows, shrimp and oysters. The 96-hour LC50 value for both sheepshead minnows and shrimp was 71 mg/L, while the NOEC value was 22 mg/L for sheepshead minnows and 50 mg/L for shrimp. The 96-hour EC50 value for oysters was 42 mg/L with an NOEC of 15 mg/L. The LC50 and NOEC value for immature mayflies was 15.5 mg/L. Additional studies on various microorganisms and invertebrates, specifically cladocerans, copepods, ostracods, mayflies, chironomid midges, water beetles, backswimmers, water boatmen, giant water bugs, and crawfish, have shown no adverse effects or negative impacts (Miura et al. 1981, Holck and Meek 1987, Key and Scott 1992, Tietze et al. 1993). Furthermore, Ali (1991) states that although *B. sphaericus* is known to be highly toxic to mosquito larvae, Bs does not offer any potential for midge control.

Applications of Bs also leave populations of mosquito predators intact and do not cause secondary effects when treated larvae are consumed by other insects. Key and Scott (1992) conducted laboratory studies with Bs on the grass shrimp *Palaemonetes pugio* and the mummichog *Fundulus heteroclitus*. Their study indicated that both Bti and Bs larvicides have large margins of safety. In a study by Aly and Mulla (1987), aquatic mosquito predators were fed with *Cx. quinquefasciatus* 4th instar larvae intoxicated with either Bti or Bs preparations. Although the mosquito larvae contained large amounts of the bacterial preparations in their gut, no effect upon longevity or ability to molt was observed in the backswimmer *Notonecta undulata*, in naiads of the dragonfly *Tarnetrum corruptum*, or in naiads of the damselfly *Enallagma civile*. Equally, the reproduction of *N. undulata* and the predation rate and ability to emerge normally in *T corruptum* and *E. civile* were not affected by ingestion of large amounts of bacterial toxins.

Bs has not been found to have adverse effects on chironomids or any other aquatic species at levels used for mosquito control (Table 6.1).

4.3.1.4 Summary of Toxicity and Potential Effects

Bs is an effective microbial pesticide specifically targeted at mosquito larvae. A common member of microbial communities and a natural biological enemy of mosquito larvae, Bs does not exhibit toxicity or risk to non-target organisms. This microbial active ingredient is used by eight of the Districts, typically throughout the year, including during each quarter of the reporting year. Bs is contained in nine products, which are applied to both standing and moving water at natural and manmade sites. Several thousand applications occurred during the reporting year. Concentrations of the active ingredient in these products range from 6 to 51.2 percent.

Based upon the environmental fate, toxicity, and use patterns by the districts during the reporting year, it should not result in unwanted adverse effects.

4.3.2 *Bacillus thuringiensis* subspecies *israelensis* (Bti)

Bti concentrates are made up of the dormant spore form of the bacterium and an associated pure toxin. The toxin disrupts the gut in mosquito larvae by binding to receptor cells (USEPA 2007c). *Bti* organisms produce five different microscopic protein pro-toxins packaged inside one larger protein container or crystal. The crystal is commonly referred to as delta (δ-) endotoxin. This toxin consists of five proteins that are released only under extremely alkaline conditions. Mosquitoes are unique in having very alkaline conditions within the midgut (the stomach of vertebrates contains acid). When a mosquito larva ingests the δ -endotoxin, the five proteins are released in the alkaline environment of the insect larval gut. The

five proteins are converted into five different toxins by specific enzymes present in the gut of mosquito larvae. Once converted, these toxins destroy the gut wall, which leads to paralysis and death of the larvae. Bti is toxic to larval stages of all genera of mosquitoes, and to black flies (*Simuliidae*). The dependence on alkaline conditions and the presence of specific enzymes gives this material a high degree of specificity for mosquitoes and black flies. Bti is also used for control of chironimids, but much higher levels are needed for effective control.

An isolate of Bti was first registered by the EPA in 1961 for use as an insecticide (USEPA 1998f). The subspecies israelensis (Bti) was first registered as an insecticide in 1983. One formulation of Bti is used in California for controlling knats on primarily in greenhouse crops, including peppers, tomatoes, celery, cabbage, leafy vegetables, cauliflower, walnuts, almonds, dates, corn, asparagus, bananas, fruit trees, and other crops. It is applied for mosquito control on rice, alfalfa, pastures, animal drinking water, ornamental nurseries, ornamental ponds, irrigation systems, swimming pools, drainage systems, lakes, streams, swamps, marshes, tidal areas, standing water, polluted or stagnant water, sewage systems, intermittently flooded areas, catch basins, domestic dwellings, and by mosquito abatement Districts and by ULV application (CDPR 2010a).

4.3.2.1 Environmental Fate

Bti toxins degrade rapidly in the phyllosphere as a result of exposure to ultraviolet (UV) light. Bti toxins may persist in soil for several months, yet a half-life for typical Bti products on foliage is approximately 1 to 4-days (USEPA 1998g).

Generally, Bti persists in the environment for periods measured in days. Factors that affect persistence and efficacy of Bti in the environment may include, but are not limited to, the formulation of the Bti product, agitation of the waterbody, receiving water quality and temperature. Solid and granule formulations, which act through a slow release action, generally persist for longer periods than liquid formulations. Agitation of sediments in the water column acts to resuspend Bti and, therefore, causes the bacterium to persist as an available pesticide for longer periods. Waters with higher organic content generally require higher doses of Bti due to lower ingestion rates by mosquito larvae. Similarly, lower water temperatures reduce the feeding rate of mosquito larvae and, therefore, may result in a longer persistence of the solid and granule formulations (Maine Department of Environmental Protection 2010).

Toxins produced by Bti degrade rapidly in sunlight as a result of exposure to ultraviolet radiation. Persistence of Bti is low in the environment, usually lasting 1 to 4-days due to sensitivity to UV light. The δ-endotoxins produced by Bti degrade by soil microorganisms with soil half-lives of 3 to 6 days. The bacterium is moderately persistent in soil with a half-life of 4-months. Bti, as with other soil microbes, does not percolate through the soil and readily binds to sediments within the water column, and is, therefore, not available to contaminate ground water (Maine Department of Environmental Protection 2010).

4.3.2.2 Human Toxicity

No known mammalian health effects have been demonstrated in any infectivity/pathogenicity study. Studies for acute oral toxicity have found no adverse toxic effects, infectivity, or pathogenicity at doses up to 4.7×10^{11} spores/kg. Studies on acute pulmonary toxicity have found no adverse toxic effects, infectivity, or pathogenicity at doses up to 2.6×10^{7} spores/kg. Studies on acute intraperitoneal toxicity have found Bti to be nontoxic at dose levels below 10^{8} CFU per animal (USEPA 1998g) (Table 6.1).

4.3.2.3 Ecological Toxicity

Due to the relatively short insecticidal half-life of Bti spores and crystals, the exposure and subsequent risk to non-target wildlife is limited to the time immediately after application. Toxicity and infectivity risks due to δ-endotoxins effects to non-target avian, freshwater fish, freshwater aquatic invertebrates, estuarine and marine animals, arthropod predators/parasites, honey bees, annelids and mammalian wildlife will be minimal to nonexistent at the label use rates of registered Bti active ingredients.

Bti δ-endotoxin has a direct adverse effect on the target insect orders (*Lepidoptera, Coleoptera, Diptera*), but susceptibility varies widely among individual species. Any one registered product has a narrow susceptible insect range (USEPA 1998g).

The amount of toxins contained within Bti products is reported indirectly as the result of at least two different bioassays, and is difficult to equate to one another. Prepared volumes of toxins are applied to living mosquito larvae and the resulting mortality produces through formulae numerical measures known as International Toxic Units (ITUs) and *Ae. aegypti* International Toxic Units (AA-ITUs). These measures are only roughly related to observed efficacy in the field, and are therefore inappropriate to consolidate and report on like other toxicants (active ingredients). There is currently no chemical test that will differentiate Bti from mosquito control products from other spore forming bacilli existing in the environment.

Bti applied at label rates has virtually no adverse effects on applicators, livestock, or wildlife, including beneficial insects, annelid worms, flatworms, crustaceans, mollusks, fish, amphibians, reptiles, birds, or mammals (Garcia et al. 1981, Holck and Meek 1987, Gharib and Hilsenhoff 1988); (Miura et al. 1980, Mulla et al. 1982, Reish et al. 1985, Siegel and Shadduck 1987, Knepper and Walker 1989, Merrit et al. 1989, Tietze et al. 1991, Molloy 1992, Tietze et al. 1992, Tietze et al. 1993, La Clair et al. 1998). However, non-target activity on larvae of some insect species closely related to mosquitoes and found with mosquito larvae in aquatic habitats has been observed. There have been reported impacts in larvae belonging to the midge families Chironomidae, Ceratopogonidae, and Dixidae (Mulla et al. 1990, Molloy 1992, Anderson et al. 1996). These non-target insect species, taxonomically closely related to mosquitoes and black flies, apparently contain the necessary gut pH and enzymes to activate delta-endotoxins. However, the concentration of Bti required to cause these effects is 10 to 1,000 times higher than maximum allowed label rates for mosquito control.

Bacterial spores of Bti are uniquely toxic to nematoceran Diptera (mosquitoes, midges, blackflies, psychodids, and ceratopogonids) (Lacey and Mulla 1990). That result was reported after reviewing Bti studies conducted using a variety of Bti formulations, and under a variety of test conditions. Lacey and Mulla (1990) concluded that Bti was a highly selective larvicide that produced minimal adverse impact on the environment. Garcia et al. (1981) tested a total of 23 species of aquatic organisms other than mosquito larvae using various formulations of Bti in his laboratory. No mortality was observed for these species with the exception of *Chironomus maturus* and a *Simulium* sp. (black fly), which showed a degree of susceptibility similar to that of mosquito larvae. Miura et al. (1980) found Bti at rates used for mosquito control to be very safe to organisms associated with mosquito breeding habitats. A total of 28 species or species groups were treated with the bacterium under simulated or field conditions, with no adverse effects observed, except for chironomid larvae, which were slightly affected. However, the effect was so light that the population in the field continuously increased after the treatment. Miura et al. (1981) found Bti and Bs, when applied at rates used for mosquito control, was very safe to organisms associated with mosquito breeding habitats, including the natural enemies of mosquito larvae. When various aquatic organisms were exposed to the bacteria under laboratory or field conditions, no adverse effect was noted on the organisms, with the exceptions of chironomid and psychodid larvae. Chironomid larvae were slightly affected by Bti treatment at a rate used for mosquito control, but pyschodid larvae were only affected at the higher concentration (50 mg/L).

Exposure of brook trout (*Salvelinus fontinalis)* fry to 4,500 and 6,000 milligrams per liter (mg/L) Teknar (a liquid formulation of Bti) (more than 50 times the allowed label rate for mosquito control) for 45 minutes resulted in 20 and 86.4-percent mortality, respectively (Fortin et al. 1986). Some species of chironomids are also susceptible to Bti, but at doses much higher than those used to control mosquito larvae (Mulla et al., 1990). Bti has been used extensively for control of mosquitoes in Germany without affecting populations of chironomids (Becker and Margalit 1993)

A number of Bti fermentation-based products tested at high-dose levels have shown intrinsic toxicity to non-target organisms. Investigations conducted to determine the source of the non-target activity have

implicated heat-labile soluble substances contaminating the technical material. Toxic effects have been seen in aquatic invertebrate *Daphnia magna*, the honeybee, some beneficial insects and fish (rainbow trout, bluegill) studies, with Daphnia being the most sensitive indicator of toxicity. The impurities are found in the supernatant fluids separate from the delta-endotoxins. The toxicity does not appear to be due to the heat-stable β-exotoxin, because autoclaving of the test material renders the supernatant fluids innocuous. The heat-labile, soluble toxic impurities have thus far been seen in Bti subspecies *kurstaki*, *aizawai*, and *israelensis*, but may possibly be present in other Bti varieties. Damgaard (1995) reported varying levels of at least one soluble exotoxin in all commercial Bti products tested (Damgaard 1995). Bti subspecies *aizawai*-based products show the greatest negative effects on non-target organisms. With Bti subspecies *kurstaki*, the manifestation of the toxin(s) appears to be at least partly related to production methodology, especially the composition of the growth media used in industrial fermentation. In response to concerns, the manufacturer of VectoBac has completed continuous 10-day exposure tests on *Daphnia magna* with the active ingredients found in VectoBac products (fermentation solids and solubles produced by *Bti* strain AM65-52). Results indicated that the LC50 is higher than 50 mg/L for *Daphnia magna* when exposed continuously for 10 days. Based on maximum label rates of VectoBac products, expected environmental concentrations (EEC) of active ingredients do not exceed 1 mg/L immediately following application, based on a conservative assumption of a water depth of 10 cm. Therefore, application of VectoBac at label rates will not result in active ingredient concentrations approaching 10 percent of the LC50 for *Daphnia magna* (DeChant 2010).

Evidence indicates that chironomid larvae (which are closely related to mosquitoes) are the only non-target aquatic species that may be affected at concentrations of Bti used for mosquito control. Observed effects on chironomids were slight and populations in the field continuously increased after the treatment (Table 6.1).

4.3.2.4 Summary of Toxicity and Potential Effects

Bti is an effective microbial pesticide specifically targeted at mosquito larvae. Bti is a natural enemy of mosquito larvae and, therefore does not pose risk to non-target organisms, including humans and ecological receptors. This microbial larvicide is used by eight of the districts, typically throughout the year (all quarters during the reporting year). Bti is contained in 12 products used by the Districts during the reporting year. Proper PPE is used during handling, loading, and applying of the liquid form of Bti, such as spray treatments. Bti is an important and safe component of any IPM program for mosquito larvae control.

It is important to distinguish this subspecies from Bt, which is frequently used in corn. In addition, Bti is a microbial gut toxin product and not a “live” bacterium. Bti is considered one of the safest natural forms of mosquito control.

4.3.3 Spinosad

Spinosad was first registered for use in California in 1996 for use as an agricultural insecticide, and more recently, registration has been approved for the use of mosquito control in California in areas such as dormant rice fields, wastewater, and temporary standing water (CDPR 2010b). Spinosad is used on a variety of crops, ornamental plants, greenhouses, ornamental lawns, and gardens; rangeland, pastures, animal husbandry premises, dairy barns, silos, and cattle; industrial sites, cracks and crevices, rights-of-way, recreation areas, golf courses, outdoor buildings and structures, and household or domestic dwellings (CDPR 2010a).

Spinosad is a biologically derived insecticide produced from the fermentation of *Saccharopolyspora spinosa*, a naturally occurring soil organism. Spinosad is a mixture of spinosyn A and spinosyn D; commercial formulations contain a spinosyn A to spinosyn D ratio of approximately 85:15. Spinosad activates the central nervous system of insects through interaction with neuro-receptors and causes continuous stimulation of the insect nervous system (Kollman 2002, Clarke Mosquito Control 2009). The EPA has classified spinosad as a “reduced risk” compound because it is an alternative to more toxic, organophosphate insecticides (CDPR 2002).

4.3.3.1 Environmental Fate

The routes of spinosad dissipation and transformation in the environment include photodegradation and biotransformation on plant surfaces, aqueous photolysis, photodegradation on soil, and biotransformation via soil microorganisms (Table 4-14). Aqueous photolysis is rapid in natural sunlight, and is the primary route of degradation in aquatic systems exposed to sunlight. In the soil environment, spinosad adsorbs strongly to soil particles and is unlikely to leach to great depths. It is photodegraded quickly on soil exposed to sunlight, but the degradation rate is decreased at longer exposure times. Spinosad is quickly metabolized by soil microorganisms under aerobic condition. Under anaerobic conditions, the degradation rate is slower (Kollman 2002). Photolysis results in degradates that are orders of magnitude less toxic than Spinosad.

Table 4-14 Degradation of Spinosad

Degradation Method	Half-life	Reference
Hydrolysis, pH 7-9 (water)	Stable	Kollman 2002
Photolysis (water)	0.84 to 0.96 Day	Kollman 2002
Photolysis (soil)	8.68 to 9.44-Days	Kollman 2002
Aerobic metabolism (soil)	14.5 to 17.3 Days	Kollman 2002
Anaerobic metabolism (soil)	161 to 250 Days	Kollman 2002

4.3.3.2 Human Toxicity

Spinosad is of low acute toxicity by all exposure routes. The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rabbits is >2,800 mg/kg. The inhalation LC50 for rats is >5.18 mg/L (USDA 1999). There has been no evidence of mutagenic or carcinogenic effects in chronic studies (Table 6.1).

4.3.3.3 Environmental Toxicity

Spinosad is slightly to moderately toxic to fish and most aquatic invertebrates (USDA 1999). Acute LC50 values for bluegill and sheepshead minnow are greater than 5,000 µg/L and 7,000 µg/L, respectively, and the chronic NOAEC values for trout and sheepshead minnow are both greater than 1,000 µg/L (Goudie 2010). Hertlein et al. (2010) stated that no negative impacts were observed for individual mosquito fish held in water containing up to 50,000 µg/L of spinosad. This material also has low acute toxicity for fresh and saltwater invertebrates, with an acute EC 50 of greater than 10,000 µg/L for daphnia (Goudie 2010). The acute EC50 for oysters was greater than 300 µg/L (Goudie 2010). Laboratory studies demonstrate some toxicity for some aquatic invertebrates under chronic exposure, but residues dissipate rapidly and are rapidly degraded by photolysis with a half-life in water of less than half a day (Goudie 2010). Stark and Vargas (2003) reported a decline in *Daphnia pulex* when exposed to Spinosad in the laboratory. However, the organisms were held in a continuous renewal system, with fresh Spinosad added every 24 hours. Mortality also occurred in daphnia held in Plexiglas enclosures at a field site during applications of Spinosad (Duchet et al. 2008). However, mortality occurred immediately after the applications and the authors also noted that the Spinosad dissipated rapidly from the water column and was detected at 4 to 13 percent of the initial concentrations (8 to 33 µg/L) in water 4-days after its application (Duchet et al. 2008). Hertlein et al. (2010), reporting an unpublished study by Laddoni (2006, no citation available) noted slight impacts on nonmosquito insects (*Dyticsidae, Hesteridae, Libelluledae, Notonectidae*) were observed in an artificial pond treated with 50 ppb or 50 g/ha of spinosad. However, this is far below field use rates and the authors concluded that Spinosad was minimally disruptive to non-targets when applied near field use rates (15 to 25 ppb).

Spinosad is practically nontoxic to birds. The acute oral LD50 for bobwhite quail and mallard ducks is >2,000 mg/kg (USDA 1999).

While high doses and/or chronic exposure of Spinosad may adversely affect some aquatic invertebrates, the short-term exposure at levels used for mosquito control is unlikely to have unwanted effects (Table 6.1).

4.3.3.4 Summary of Toxicity and Potential Effects

Spinosad readily degrades by a number of chemical and biological processes and is not environmentally persistent. Although toxicity is low to mammals, fish, invertebrates, and birds, non-target insects (e.g., some species of moths and butterflies) could be at risk. However, low amounts typically used for mosquito control would not likely pose a significant risk to potential ecological receptors.

Spinosad is used by four of the Districts throughout the year, including during each quarter of the reporting year. It is applied in three different products (0.5 to 20 percent Spinosad) to standing and moving water in natural and anthropogenic sites. These products were applied several thousand times throughout the reporting year.

Based on the environmental fate, human and ecological toxicity, and usage patterns, using BMP application practices, spinosad should not result in unwanted adverse effects.

4.3.4 Methoprene and s-Methoprene

Methoprene is a long chain hydrocarbon ester classified as an insect growth regulator and selective larvicide. Methoprene consists of two enantiomers: S-methoprene and R-methoprene. S-methoprene is the biologically active enantiomer in the racemic compound (FAO-WHO 2005). (Fate and transport characteristics of the s-enantiomer and the mixture are similar, but toxicity differs.) Methoprene is used principally against mosquitoes, but is effective against a range of insects, including the orders Diptera, Lepidoptera and Coleoptera. Methoprene is an insect growth regulator that interferes with the normal maturation process of insects, preventing them from completing their life cycle and reaching adulthood, thus ultimately preventing them from reproduction (Csondes 2004).

Methoprene was first registered by the EPA in 1975 (USEPA 1991e). Methoprene is used indoors and outdoors at domestic dwellings, in flea and tick treatments for cats and dogs, for crack and crevice treatments, and on outdoor buildings and structures, recreation areas, swimming pools, golf courses, ornamental lawns, ornamental ponds, and shrubs. Methoprene is used at animal husbandry premises, on cattle, barnyards, rangeland, pastures, fallow land, and in animal drinking water. It is used at industrial sites, on highway rights-of-way, industrial waste disposal systems, industrial/commercial ponds, wastewater, and bilge water. Methoprene can be applied to irrigation systems, orchards, crops, berries, fruit trees, and rice. It is also used in drainage systems, swamps, marshes, intermittently flooded areas, catch basins, polluted stagnant water, sewage systems, and applied by mosquito abatement Districts (CDPR 2010a).

Methoprene products used in mosquito control are applied as briquets, pellets, sand granules, and liquids. The liquid and pelletized formulations can be applied by helicopter and fixed-wing aircraft or ground-based equipment. Methoprene is applied either in response to observed high populations of mosquito larvae at a site, or as a sustained-release product that can persist for 4-months or longer if a site has limited accessibility and has regularly produced immature mosquitoes in the past.

4.3.4.1 Environmental Fate

When methoprene is released into water, it sorbs to suspended solids and sediments. When applied to soil, methoprene is relatively immobile, tending to reside in the top few centimeters of the soil (Csondes 2004). Methoprene (and s-methoprene) is a very short-lived material in nature (Table 4-15). It rapidly degrades in aqueous solution and on inert surfaces by photolysis. It is metabolized in soil under both

aerobic and anaerobic conditions. Degradation in the aquatic environment is due to both microbial metabolism and photolysis (USEPA 1991e).

Table 4-15 Degradation of Methoprene/s-Methoprene

Degradation Method	Half-life	Reference
Hydrolysis (water)	Stable	FAO-WHO 2005
Photolysis (thin film)	6 Hours	FAO-WHO 2005
Photolysis (water/sediment system)	<1 Day	FAO-WHO 2005
Aerobic metabolism (pond water)	30 to 40 Hours	FAO-WHO 2005
Aerobic metabolism (soil)	10 Days	FAO-WHO 2005; USEPA 1991b
Anaerobic metabolism (soil)	14 Days	USEPA 1991b
Field dissipation (pond water)	13 Days	Csondes 2004

4.3.4.2 Human Toxicity

Methoprene is of very low acute toxicity by all routes (USEPA 1991c). The oral LD50 for rats is >10,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The inhalation LC50 for rats is >210 mg/L (USEPA 1991c, 2001) (Table 6.1).

4.3.4.3 Ecological Toxicity

Methoprene is practically nontoxic to birds. The oral LD50 for mallard ducks is >2,000 mg/kg. It is moderately toxic to freshwater fish. The 96-hr LC50 for bluegill sunfish is 1.52 µg/L. Methoprene is highly toxic to aquatic invertebrates. The 48-hr EC50 of technical methoprene for *Daphnia magna* is 89 µg/L (USEPA 1991c).

Methoprene is applied at very low concentrations for mosquito control. The manufacturer has developed a number of formulations to maintain an effective level of the active material in the mosquito habitat (0.5 to 3.0 parts per billion [ppb]; (Scientific Peer Review Panel 1996) for a practical duration, thus minimizing the cost and potential impacts associated with high-frequency repeat applications (see Table 2-4). Rate of release and data generated under laboratory and field conditions with methoprene mosquito product formulations, including slow release briquet formulations, indicate a maximal rate of release of ≤4-ppb (EPA 2001). Ross et al. (1994) conducted microcosm studies, which applied 5 sustained release methoprene formulations at maximum label application rates to tanks containing water 6 inches deep. Methoprene concentrations were measured 1, 2, 4, 7, 14, 21, 28, and 35 days after treatment, and the highest methoprene concentration measured was 6 ppb.

Exhaustive reviews of the published literature on this material attest to its lack of adverse environmental impact ((Mian and Mulla 1992, Scientific Peer Review Panel 1996, Glare and O'Callaghan 1999, State of Minnesota 1999, USEPA 2001). The acute, short-term toxicity of ZR-515 (methoprene) was also tested on 35 aquatic organisms, including Protozoa, Platyhelminths, Rotatoria, Annelida, Arthropoda, Mollusca, Chordata and Thallophyta, and LC50 values of 0.9 to 5.0 mg/L were calculated (250 to 1,000 times label rates) (Miura and Takahashi 1973). Dosages used for larval mosquito control produced no adverse effect on the organisms tested, except for some sensitivity in the aquatic Diptera (flies) in the families Chironomidae, Ephydridae, and Psychodidae.

Bircher and Ruber (1988) assessed the toxicity of methoprene to all lifecycle stages of the salt marsh copepod (*Apocyclops spartinus*) at concentrations ranging from 100 to 10,000 µg/L. In general, the copepods were resistant to concentrations of methoprene used to control mosquitoes, but early larval stages did show some mortalities (the calculated 48-hour LC50, adjusted for control mortality, was

800 µg/L). Christiansen et al. (1977) showed a reduction in survival of larvae of the mud-crab *Rhithropanopeus harrisii* (Gould) in the laboratory under a range of salinity and temperature conditions, when exposed to 10, 100, and 1,000 µg/L methoprene, levels 5 to 500 times field application rates. McKenney and Mathews (1988) reported that larval survival, growth, and energy metabolism of an estuarine shrimp *Palaemonetes pugio* were altered by exposure to 100 µg/L of methoprene (50 times greater than application rates). However, Wirth et al. (2001) reported no observed differences in the percent successfully hatching or larval mortality 3 days post hatch in *P. pugio* exposed for 96 hours to 1,000 µg/L. In addition, in 2005, Suffolk County conducted 4-day static renewal toxicity tests on grass shrimp (*Palaemonetes pugio*) using water collected 30 minutes after aerial application of methoprene for mosquito control and observed no toxicity. Similar investigations have been carried out with *Leander tenuiconis*, an estuary shrimp that occurs in Australian intertidal marshes. Methoprene was nontoxic at field application levels in 96-hour toxicity tests (Brown et al. 1996). The LC50 of methoprene for *L tenuiconis* (14,320 µg/L) in these tests was 1,790 times field concentrations when applied at label rates. The authors concluded that methoprene could be safely applied in situations where the shrimp were present and that no mortality of shrimp was likely at the levels applied for mosquito control. Further laboratory work (Brown et al. 2000) found that the dose lethal to mosquitoes (*Culex annulirostris*) was 3,000 times below the LC_{95} for shrimp (*Caradina indistincta)*. Zulkosky et al. (2005) investigated potential effect of methoprene runoff to larval lobsters (*Homarus americanus*) in continuous flow-through systems for 48 hours: methoprene was not toxic at the highest concentration tested (10 micrograms per liter [µg/L] or 10 µg/L). Laboratory studies with fish demonstrated that methoprene had no effect on the survival of adult and juvenile rainbowfish (*Melanotaenia duboulayi*) (Brown et al. 2002). No effect was observed on swimming performance of rainbowfish when exposed to up to ten times effective field concentrations of applications made for mosquito control (Hurst et al. 2007).

Methoprene does not have adverse effects on amphibians. Tests conducted on various life stages of different amphibian species (*Bufo woodhousei*, *Rana catesbeiana* and *Rana pipiens*) found no adverse effects from acute or chronic exposures at the highest dose tested. Acute studies on *R. catesbeiana* and *R. pipiens* larvae indicate LC50 values >10,000 µg/L and *B. woodhousei* adult LC50 values >1,000 µg/L (highest dose tested). Chronic studies on *B. woodhousei* indicate a 22-day LC50 >1,000 µg/L and LC50 >1,000 µg/L for *R. catesbeiana* and *R. pipiens* (USEPA 2001).

One early field study assessing applications of technical (pure powdered) methoprene on a Louisiana coastal marsh yielded ambiguous results (Breaud et al. 1977). Highly significant declines were observed in the occurrence of 14-invertebrates immediately following the application, including selected life stages and species of amphipods, shrimp, mayflies, dance flies, midges, freshwater snails, damselflies and dragonflies, and water beetles. However, the abundance of five other invertebrates significantly increased including water boatmen, moth flies, two species of crawfish, and predaceous diving beetles. No statistically significant difference was seen between the test and control populations of another 28 aquatic organisms. Interpretation of this study is difficult in part because of the mixed nature of the results, which may simply indicate the complexity of ecosystem dynamics in marshlands. Also, the application rate (28 gm active ingredient/ha technical powder) was at least twice the highest label rate of active ingredient allowed today, and was effectively much higher when the encapsulation and other coatings on modern formulations are considered. The relevance of Breaud et al.'s entire experiment as a legitimate field study may be called into question, as the properties of technical grade methoprene powder render it unfit for any type of direct field application under current label restrictions.

Since the publication of Breaud et al. (1977), there have been numerous field studies using currently available mosquito control products containing methoprene, in which no detectable effect was observed in aquatic invertebrates. For example, no detectable mortality occurred in Talitridae amphipods exposed to aerial applications of Altosid to a Florida mangrove swamp in 1999 (Lawler et al. 1999). A similar study assessed applications of a sustained release formulation of methoprene and a combined liquid formulation of *Bti* and methoprene (duplex) to tidal wetlands of San Francisco Bay. No difference was seen in growth or

development of corixid beetles, and no difference in the number of non-target insects inhabiting treated versus untreated plots (Lawler et al. 2000). The authors also monitored brine flies at treated and untreated sites using sentinel cages, and sampled populations with sweep nets. No decline was observed in flies relative to controls collected by sweep nets. Caging of sentinels was unsuccessful at assessing impacts, since none of the caged flies survived at untreated sites or treated ones.

Aerial applications of liquid methoprene on saltmarsh habitat have also been assessed in Australia (Russell et al. 2009). Changes in assemblages of invertebrates through time were observed in both treated and untreated (control) plots. No significant effects were seen on arthropods in ephemeral pools. There was no significant difference in abundance of nonmosquito dipterans (flies), heteropterans (true bugs), and hymenopterans (primarily ants) in treated versus untreated sites. Some differences were observed in copepod populations during the treatment period, but these were short-term or inconsistent between localities or between sampling method. The authors concluded that applications of *Bti* and methoprene to salt marshes do not affect the structure or composition of assemblages of non-target arthropods (Russell et al. 2009).

Published studies on non-target impacts of methoprene for mosquito control were reviewed recently (Davis 2007, Davis and Peterson 2008). The authors also carried out an ecological risk evaluation of mosquito larvicides in a series of ponds at the Benton Lakes National Wildlife Refuge in Montana. *Bti* and methoprene were applied directly to water as liquids, and aquatic arthropods were sampled following the applications. No overall treatment effects were observed on aquatic non-target invertebrates collected in D-shaped net samples. A linear model was then fitted to each of the response variables to determine multivariate treatment effects. Data indicated a possible acute impact on amphipods immediately following application, but no significant effect at 7 to 28 days. No trend was seen across dependent groups of non-target organisms, and there were no persistent biological effects.

Careful review of these and other studies, and the recent reviewers listed above leads to the conclusion that: 1) applications of methoprene (especially technical powder) at rates significantly higher than allowed by the label can adversely impact a number of aquatic animals; 2) animal species are not extirpated (locally eliminated) by repeated methoprene use except at application rates far higher than those used for mosquito control; 3) emergence of adults of some fly species (specifically, some types of midges) can be temporarily reduced at application rates similar to those used for mosquito control; 4) larval flies affected by methoprene are not killed at label application rates, but are prevented from becoming adults; 5) for species that are affected by methoprene, recolonization and reestablishment of populations from neighboring sites is fast once intense control was relaxed; 6) the patchy distribution of mosquito larvae leads to maintenance of untreated refugia for non-targets, speeding recolonization; and 7) no bioaccumulation of methoprene has been seen in animals that have eaten mosquito or midge larvae treated with methoprene.

The concentrations of methoprene applied for mosquito larvae control are unlikely to affect non-target aquatic species, except for some fly species closely related to mosquitos. For species that are affected by methoprene, recolonization and reestablishment of populations from neighboring sites is fast once intense control is relaxed (Table 6.1).

4.3.4.4 Summary of Toxicity and Potential Effects

Methoprene readily degrades in soil and water by a variety of processes. It may exhibit toxicity to fish and aquatic invertebrates, as well as non-target insects including moths, butterflies, and beetles.

Methoprene is longer-lasting than some of the other larvicides on the market and, therefore requires fewer applications of low amounts. Methoprene is effective at much lower concentrations than alternative larvicide products, which correlates with reduced acute exposures to non-target organisms, as well as potential effects to a lower diversity of midges and chironomids. Extended release forms including granular and briquette varieties are also available, including 90-day briquettes, This product may be more residual in the

environment, however, the methoprene active ingredient in this formulation has a short half-life in water and does not migrate through soil, significantly reducing the potential for groundwater impacts.

Considered the safest of all larvicide alternatives, methoprene is used prevalently by the nine Districts during each season, including all four quarters of the reporting year. There are 12 methoprene-containing products (0.2 to 8.62% methoprene) that are applied to natural and anthropogenic standing and moving water bodies. Eleven of these products are Altosid formulations (Wellmark) and one is MetaLarv SP-T (Table 3-1). These products were applied several thousand times during the reporting year.

Liquid and granular forms are most prevalently used in residential and ornamental pond application scenarios. Treatments to wetlands including marshes require the granular form (e.g. Altosid XRG with *Bacillus sphaericus*) to penetrate dense aquatic vegetation including cattails and tules. Methoprene is also sometimes co-applied with Bti. Drift is almost irrelevant for hand and aerial (e.g., helicopter) applications since treatments are restricted at moderate to high wind speeds. Methoprene is highly effective against mosquitoes at low concentrations and degrades quickly in the environment, thereby reducing the potential exposure and risk to non-target organisms. When handled and applied using appropriate BMP, methoprene is one of the safest (human and ecological) and most effective mosquito control products used by the Districts. Based on toxicity, environmental fate, and usage patterns, methoprene, using BMP is not likely to result in unwanted adverse impacts

4.3.5 Alcohol Ethoxylated Surfactant (monomolecular film)

The monomolecular film used in California for the control of mosquito larvae is alpha-isooctadecyl-omega-hydroxypoly(oxyethylene). Agnique is the trade name for this recently reissued surface film larvicide. Monomolecular films are alcohol ethoxylated surfactants, which are low-toxicity pesticides that spread a thin film on the surface of the water that makes it difficult for mosquito larvae, pupae, and emerging adults to attach to the water's surface, and cause them to drown (USEPA 2007c). It also disrupts larval respiration of some other classes of air-breathing aquatic insects.

Monomolecular films are used on ornamental ponds, pastures, irrigation systems, drainage systems, drinking water systems, intermittently flooded areas, catch basins, lakes, ponds, reservoirs, tidal areas, marshes, and standing water, industrial waste disposal systems, polluted and stagnant water, and sewage systems (CDPR 2010a).

4.3.5.1 Environmental Fate

Reported half-lives of monomolecular films in water range from 5 to 22 days and Agnique has an average persistence in the environment of 5 to 21 days at label application rates (Oester 2010).

4.3.5.2 Human Toxicity

Because of the mode of action and likely exposure scenarios of these products, there is little to no indication of potential for adverse effects to humans.

4.3.5.3 Ecological Toxicity

A number of efficacy and non-target studies had been conducted on this material when it was registered under the name Aerosurf. Minor proprietary changes in preparation did not apparently change any of the material's potential environmental impacts; therefore, the earlier literature is referenced.

Most published studies conducted with this larvicide tested application rates of 3 to 100 times the maximum label rate. At these rates, no observable effect on mortality or development was noted in tests on green tree frogs, seven species of fresh and salt water fish, two species of shrimp, five species of water beetle, or one species each of fairy shrimp, crayfish, snail, polychaete worm, mayfly naiad, copepod, ostracod, or midge. In addition, no effect was seen on five species of plants. Air (surface) breathing insects were temporarily adversely impacted. Waterboatmen, backswimmers, and one species

of water beetle exhibited increased mortality at application rates above label limits. In addition, a clam shrimp, a crab, an amphipod, and one species of isopod exhibited minor to significant increases in mortality at levels several times the highest application rate allowed by the label (Oester 2010).

Although evidence indicates that application of monomolecular films and petroleum distillates may result in reductions to populations of surface-breathing insects at the time of treatment, it is unlikely that overall populations of invertebrate species are affected as populations recover quickly due to recolonization from neighboring sites (Table 6.1).

4.3.5.4 Summary of Toxicity and Potential Effects

Alcohol ethoxylated surfactants exert no lasting or observable effect on most non-target organisms, so that , using BMP application practices, these chemicals should not result in unwanted adverse effects. Aliphatic Solvents (Mineral Oils and Aliphatic Petroleum Hydrocarbons)

Specially-derived aliphatic solvents (e.g., mineral oils and aliphatic petroleum hydrocarbons such as GB-1111) are used to form a coating on top of water to drown larvae, pupae, and emerging adult mosquitoes. They are the product of petroleum distillations processes, and thus, are complex mixtures of long-chain aliphatic compounds. Aliphatic solvents have been used for many years nationwide to kill aphids on crops and orchard trees, and to control mosquitoes (USEPA 2007d). They are applied to a wide variety of crops, trees and ornamental plants; to swamps, marshes and intermittently flooded areas; are used as an adjuvant for pesticides; and are applied by mosquito abatement Districts (CDPR 2010a). Dormant oils are widely used in the Central Valley on tree crops.

4.3.5.5 Environmental Fate

Petroleum distillates are effective in many situations in which monomolecular films do not give sufficient control. These materials also break down much more rapidly than monomolecular films (2 to 3 days as opposed to 21 days) which decreases their impact to non-target organisms.

4.3.5.6 Human Toxicity

These chemicals have a low degree of acute toxicity to mammals. There was no mortality in rats at an acute oral dose of 28,000 mg/kg bw. They are virtually nontoxic via dermal and inhalation routes (USEPA 2007d) (Table 6.1).

4.3.5.7 Ecological Toxicity

The safety of petroleum distillates for non-targets has been demonstrated by both laboratory and field studies. Three studies (Tietze et al. 1991, Tietze et al. 1992, Tietze et al. 1994) tested three species of fish (Inland Silversides, Mosquitofish, and Sheepshead Minnows), and a range of microorganisms and concluded that petroleum distillate formulation GB-1111 is not toxic to the tested organisms at label application rates. Mulla and Darwazeh (1981) tested with GB-1111 in small experimental ponds and found that benthic invertebrates (including mayflies, dragonflies, and damselflies) were unaffected, while populations of surface-breathing insects were temporarily reduced, following application of this larvicide. Miles et al. (2002) completed an independent study of non-target effects of GB-1111, with financial assistance from the U.S. Fish and Wildlife Service (USFWS), on the tidal marshes of the Don Edwards National Wildlife Refuge in San Francisco Bay near Newark, California, and observed the following effects: 1) surface-breathing insect populations were reduced at the time of treatment; 2) this effect did not persist beyond a few days (no residual pesticide effects); 3) those potentially affected animals with high mobility left the site, while some of those that could not leave died (especially water boatmen [*Corixidae*]); and 4) overall populations of invertebrate species were not affected, apparently because of recolonization from neighboring untreated sites.

Although evidence indicates that application of monomolecular films and petroleum distillates may result in reductions to populations of surface-breathing insects at the time of treatment, it is unlikely that overall

populations of invertebrate species are affected as populations recover quickly due to recolonization from neighboring sites (Table 6.1).

4.3.5.8 Summary of Toxicity and Potential Effects

Aliphatic solvents have very low water solubility and high sorption to organic matter. They are practically nontoxic to most non-target organisms and rapidly break down in the environment, reducing their impact on susceptible non-targets so that, using BMP application practices, these products should not result in unwanted adverse effects.

4.4 Other Insecticides

4.4.1 Potassium Salts

Potassium salts of fatty acids are used as insecticides, acaricides, herbicides and algaecides. They are used to control a variety of insects and mosses, algae, lichens, liverworts and other weeds, in or on many food and feed crops, ornamental flower beds, house plants, trees, shrubs, walks and driveways, and on dogs, puppies and cats. Potassium salts of fatty acids include potassium laurate, potassium myristate, potassium oleate and potassium ricinoleate. Once applied, however, these salts are degraded quickly in soil by microbes, and do not persist in the environment (USEPA 1992).

4.4.1.1 General Toxicity

Commonly referred to as “soap salts”. They are produced by adding potassium hydroxide to fatty acids found in plant or animal oils. Fatty acids are extracted from palm, coconut, olive, castor, and cottonseed plants (National Pesticide Information Center 2001). Fatty acids penetrate an insect’s body covering and disrupt the cell membranes. The insect dies of dehydration. Soft-bodied insects, such as aphids, are more susceptible as are immature insects.

4.4.1.2 Human Toxicity

Soap salts have low oral and dermal toxicity to mammals but may cause general stomach upset in humans. They may be irritating to the skin and eyes (USEPA 1992). These products are generally considered safe by the FDA. The USEPA classifies soap salts as Category IV (lowest level of toxicity) for acute effects (Table 6.1).

4.4.1.3 Ecological Toxicity

Soap salts are practically nontoxic to birds but slightly toxic to fish and highly toxic to aquatic invertebrates (USEPA 1992). Pesticides containing potassium salts of fatty acids are used in a wide array of outdoor sites; however, the compounds degrade very quickly in soil. Because soap salts are not applied directly to water, they pose little threat to sensitive aquatic invertebrates (USEPA 1992) (Table 6.1).

4.4.1.4 Summary of Toxicity and Potential Effects

Potassium salts degrade quickly in the environment. They are of low toxicity to birds and mammals, but highly toxic to fish aquatic non-target invertebrates. The Districts did not use potassium salt products during the reporting year; therefore, when needed, using BMP application practices, these products should not result in unwanted adverse effects

4.5 Rodenticides

These chemicals are for the control of mammal pests, particularly commensal rats and mice (e.g., Norway rat, roof rat, and house mouse) but also a variety of field rodents.

The anticoagulant rodenticides are typically grouped into “first-generation” (e.g., chlorophacinone, diphacinone) and “second-generation” (e.g., brodifacoum, bromadiolone, difethialone) compounds.

Second-generation anticoagulants tend to be more acutely toxic than are the first-generation anticoagulants, and they are retained much longer in body tissues of primary consumers. In contrast, the first-generation compounds are less acutely toxic and more rapidly metabolized and/or excreted (Housenger and Melendez 2012). Both classes have the same mode of action but second generation anticoagulants have a significantly longer liver half-life than first generation anticoagulants (Hartless and Jones 2011).

4.5.1 Chlorophacinone

Chlorophacinone is used to control a variety of vertebrate pests, mainly rodents, but also jackrabbits (lagomorphs), and moles (insectivores). It is a first-generation anticoagulant and is formulated as tracking powder, as loose-grain bait, paraffinized pellets, rat and mouse bait ready-to-use place packs, and paraffin blocks. Chlorophacinone is currently registered for the control of rodents in and around buildings, households and domestic dwellings, uncultivated agricultural and nonagricultural areas, commercial transportation facilities; industrial areas, and food processing, handling, and storage areas and facilities. Both general use and restricted use chlorophacinone products are currently registered (USEPA 1998e).

4.5.1.1 Environmental Fate

Chlorophacinone is readily degradable by photolysis in the environment. It has low water solubility, is very susceptible to direct photolysis in water and is moderately susceptible to photodegradation on soil (Table 4-16; USEPA, 1998c). Chlorophacinone volatilizes slowly from water and soil and degrades slowly by acid hydrolysis with no measurable hydrolysis at higher pHs. Chlorophacinone is considered to be moderately persistent and immobile in soil. The major route of dissipation in soil appears to be aerobic soil metabolism (USEPA 1998e, Hartless and Jones 2011).

Table 4-16 Degradation of Chlorophacinone

Degradation Method	Half-life	Reference
Hydrolysis, pH 5	232 Days	Hartless & Jones 2011
Hydrolysis, pH 7-9	Stable	USEPA 1998c, Hartless & Jones 2011
Photolysis (water)	37 minutes	USEPA 1998c
Photolysis (soil)	4 Days	USEPA 1998c
Aerobic metabolism (soil)	17 to 47.2 Days	USEPA 1998c, Hartless & Jones 2011

4.5.1.2 Human Toxicity

The USEPA classifies chlorophacinone as Category I (highly toxic) to mammals for oral, dermal, and inhalation toxicity for mammals (USEPA 1998e). The oral LD50 of chlorophacinone is 3.15 mg/kg for male rats and 0.329 mg/kg for male rabbits (USEPA 1998e). Human volunteers were able to tolerate a single dose of 20 mg active ingredient with an uneventful recovery and no treatment (EXTOXNET 1985c). The dermal LD50 for rabbits is 200 mg/kg. The inhalation LC50 for male rats is 7.0 µg/L. Chlorophacinone is not known to cause skin or eye irritation (USEPA 1998e) (Table 6.1).

4.5.1.3 Ecological Toxicity

Chlorophacinone is toxic to wildlife and fish. The 96-hr LC50 for rainbow trout is 450 µg/L. The use of food bait (aerial or ground broadcast or hand applied pellets) may present an exposure risk to seed-eating birds (USEPA 1998e). The oral LD50 is 258 mg/kg for bobwhite quail (USEPA 1998e). The oral LD50 for carnivorous mammals, including carnivores (e.g., mountain lions, bobcats, coyotes) is 2.1 to 50 mg/kg (Hosea 2000). Data are lacking to assess potential secondary risks to avian predators and scavengers,

which may feed on poisoned rodents. The USEPA presumes high risks to any non-target small mammals. Primary risks to larger mammals are reduced by proper use of bait stations. Secondary risk to predatory mammals such as coyotes has been demonstrated. The USEPA indicates that chlorophacinone poses minimal risk to freshwater organisms (USEPA 1998e) (Table 6.1).

4.5.1.4 Summary of Toxicity and Potential Effects

Chlorophacinone has low water solubility and is moderately persistent in soils. Loose-grain baits may present a risk to non-target foraging animals, including seed-eating birds. This first-generation rodenticide is highly toxic to mammals, including humans, domestic pets, and non-target mammalian wildlife. However, since it is generally applied as solid bait blocks, significant release and environmental impact is not anticipated. In addition, chlorophacinone was not used by the MVCAC Districts during the reporting year. For these reasons, and when needed, using BMP application practices, these products should not result in unwanted adverse effects

4.5.2 Diphacinone

Diphacinone and diphacinone salt products are first-generation anticoagulants formulated predominantly as food baits (loose bait, feeder boxes, place packs, or paraffinized bait blocks) for control of commensal rats (Norway rat, roof rat) and mice (house mouse). Food baits also are registered for controlling ground squirrels and pocket gophers. One product is registered as a tracking powder for control of rats and mice indoors and at burrows located along the periphery of buildings. Because diphacinone salt is highly soluble, it is also used to prepare water baits for indoor control of rats and mice. Use sites for rat and mouse food baits are predominantly in and around buildings and similar man-made structures. Some labels include sewers or other wet or damp sites such as dumps, irrigation ditches, along fences, gullies, and other such areas. Diphacinone salt has special local needs registration in California for control of deer mice, jackrabbits, chipmunks, muskrats, woodrats, voles, and commensal rats and mice (USEPA 1998e).

4.5.2.1 Environmental Fate

Diphacinone has low water solubility and volatilizes slowly from water and soil. Diphacinone is stable to hydrolysis at pH 7-9 and stable to photolysis. One clearly established route of transformation for diphacinone is aerobic soil metabolism (Table 4-17). Because diphacinone binds tightly to soil, most of the chemical would remain in the top soil layers and its potential to reach ground water is low (USEPA 1998e, Federoff and Lin 2011).

Table 4-17 Degradation of Diphacinone

Degradation Method	Half-life	Reference
Hydrolysis, pH 5	44 Days	USEPA 1998c
Hydrolysis, pH 7-9	Stable	USEPA 1998c
Photolysis (water)	Stable	Federoff & Lin 2011
Photolysis (soil)	Stable	Federoff & Lin 2011
Aerobic metabolism (water)	180 Days	Federoff & Lin 2011
Aerobic metabolism (soil)	28 to 32 Days	USEPA 1998, Federoff & Lin 2011

4.5.2.2 Human Toxicity

The USEPA has rated diphacinone as Category I for oral, dermal, and inhalation toxicity to mammals. Oral LD50s for rats were 2.3 mg/kg and 7.0 mg/kg in two separate studies (USEPA 1998e). The dermal LD50 for rabbits is 3.6 mg/kg. The inhalation LC50 for rats is <0.6 µg/L (USEPA 1998e). Diphacinone is

listed as Category III for eye irritation and Category IV for skin irritation (USEPA 1998e). Given the exclusively nonfood uses of diphacinone, chronic toxicity and carcinogenicity, USEPA has not conducted chronic toxicity and carcinogenicity studies (USEPA 1998e). The use of rodenticides can sometimes pose risks to domestic animals via primary or secondary exposure. The oral LD50 is 0.3 to 7.5 mg/kg for dogs and 14.7 mg/kg for cats. The oral LD50 for swine is 150 mg/kg (EXTOXNET 1993b) (Table 6.1).

4.5.2.3 Ecological Toxicity

The use of food bait (aerial or ground broadcast or hand applied pellets) may present an exposure risk to seed-eating birds (USEPA 1998e). Diphacinone is slightly toxic to birds. The oral LD50 is 3,158 mg/kg for mallard duck and 1,630 mg/kg for bobwhite quail (EXTOXNET 1993b). There is potential secondary risk to avian predators and scavengers, which may feed on poisoned rodents. The lowest observed lethal single dose to screech owls was 130 mg/kg bw (Rattner et al. 2012). The lowest observed lethal 7-day dose to screech owls was 0.82 mg/kg owl/day (Rattner et al. 2012). Primary risks to larger mammals are reduced by proper use of bait stations. The USEPA expects minimal risk to aquatic organisms from the current uses of diphacinone (USEPA 1998e). However, diphacione is slightly to moderately toxic to fish and invertebrates. The 96-hr LC50 is 7.6 mg/L for bluegill and 2.8 mg/L for trout. The 48-hr LC50 for *Daphnia magna* is 1.8 mg/L (USEPA 1998e). It is not known to bioaccumulate in fish readily (USEPA 1998e) (Table 6.1).

4.5.2.4 Summary of Toxicity and Potential Effects

Diphacinone technical material has low water solubility and is generally applied as food bait blocks; however, diphacinone salt is highly soluble and is used to prepare water baits for indoor control of rodents. Diphacinone salt has special local needs registration in California for control of deer mice, jackrabbits, chipmunks, muskrats, woodrats, voles, and commensal rats and mice (USEPA 1998e). This first-generation rodenticide is highly toxic to mammals, including humans, domestic pets, and non-target mammalian wildlife. However, since it is generally applied as solid bait blocks or in-home water treatments, significant release and environmental impact is not anticipated.

Diphacinone is used by three Districts in tree holes, burrows, creeks, and parks. Districts use two different products. Diphacinone is applied over 80 times a year and application occurs in all four quarters, including during the reporting year. Active ingredient concentrations in these products are 0.2 and 0.005 percent, respectively. Based on the limited use patterns by the Districts and low potential exposure to non-target species, it is not likely, using BMP application practices, that these products should not result in unwanted adverse effects For likely future uses, USEPA has released a list of new, more protective rodenticide products, including tamper-resistant and weather-resistant bait stations (http://www.epa.gov/pesticides/mice-and-rats/rodent-bait-station.html).

4.5.3 Brodifacoum

Brodifacoum is a second generation anticoagulant pesticide for rodent control against commensal rats and mice (Housenger and Melendez 2012). It is formulated as meal bait, paraffinized pellets, rat and mouse bait ready-to-use place packs, and paraffin blocks. Brodifacoum is currently registered for the control of rats and mice in and around farm structures, households and domestic dwellings, uncultivated agricultural and nonagricultural areas, inside transport vehicles, commercial transportation facilities, industrial areas, sewage systems, aircraft, ships, boats, railway cars, and food processing, handling, and storage areas and facilities. Only general-use brodifacoum products are currently registered (USEPA 1998e).

4.5.3.1 Environmental Fate

Brodifacoum has low solubility and is nonvolatile. It is stable to hydrolysis at pH 5, 7, and 9, relatively persistent in soil (half-life of 157 days), and immobile in soil columns. Photolysis by sunlight in aqueous media is potentially important, if exposure to aquatic environments occur. Brodifacoum is persistent in soil, but little, if any, contamination of surface and ground waters is expected because of its use pattern and immobility in soil (USEPA 1998e, Housenger and Melendez 2012).

4.5.3.2 Human Toxicity

Brodifacoum is listed as Category I for oral, dermal, and inhalation exposure by the USEPA. The oral LD50 for rats is 0.418 to 0.561 mg/kg. The dermal LD50 for rabbits is 3.16 to 5.21 mg/kg. The inhalation LC50 for rats is 3.05 to 4.86 µg/L. Brodifacoum is listed as Category III for eye irritation and is unlikely to cause skin irritation (USEPA 1998e) (Table 6.1).

4.5.3.3 Ecological Toxicity

Like other common rodenticides, brodifacoum is often found in tissues of wildlife. The LD50 for carnivores such as coyotes, foxes, and mountain lions is 0.27 to 25.0 mg/kg (Stone et al. 1999, Hosea 2000). Many non-target mammals are susceptible to fatal ingestion of brodifacoum. Eastern gray squirrels, white-tailed deer, raccoons, and red foxes have been recovered and determined to have died from exposure to anticoagulant rodenticides (first or second generation) (Stone et al. 1999). Domestic animals may accidentally ingest bait. The oral LD50 is 100 g of bait for cats and 355 to 1,000 g of bait for dogs (EXTOXNET 1985a).

The LD50 for wild birds, including birds of prey is 2 to 100 mg/kg (Stone et al. 1999, Hosea 2000). The acute oral LD50 for mallard ducks is 2.0 mg/kg (EXTOXNET 1985a). The LD50 for mallard ducks is 0.26 mg/kg (USEPA 1998e). Scavenging birds may also be exposed to brodifacoum. Howald (Howald 1997) reported common ravens removed and consumed bait blocks from bait stations on Langara Island during a rat eradication program. Bald eagles captured and tested have also shown blood plasma residues of brodifacoum (Howald et al. 1999). Similar results have been demonstrated for northwestern crows (Howald 1997, Howald et al. 1999) and eastern screech owls (Merson et al. 1984) with some fatalities recorded. The level of concern (LOC) for predatory birds is >100 ng/g ww (hepatic concentration) with >200 ng/g ww considered potentially lethal (Christensen et al. 2012). Brodifacoum (in combination with bromadiolone and difethialone) was indicated in the mortality of three red-tailed hawks in Manhattan in 2012 (New York State Department of Environmental Conservation 2012). In a recent study of raptors and owls in Denmark, 92 percent of all birds contained detectable hepatic concentrations of anticoagulant rodenticides, with second-generation anticoagulants (brodifacoum, bromadiolone, and difenacoum the most prevalent) (Christensen et al. 2012).

The California Department of Fish and Game Pesticide Investigations Unit identified wildlife losses possibly due to pesticide exposure (Hosea 2000). Clinical signs consistent with anticoagulant toxicosis were observed during necropsies of 43 percent of the animals with anticoagulant residues. Of the 74-animals examined in this study, 69 percent had been exposed to anticoagulant rodenticides, indicating that urban use of anticoagulant rodenticides may be important in California. The primary compound identified in this study was brodifacoum (61 percent of mammals and 55 percent of birds). This compound was only registered for use in, or adjacent to, structures. Due to the feeding behaviors of some of the exposed non-target wildlife (i.e., birds of prey do not eat pelletized or grain foods, bobcats and mountain lions are carnivores) the authors concluded that it was unlikely for these species to consume rodenticide baits directly. Raccoons, canids, kangaroo rats, and wild turkeys were thought to have been exposed via the primary route. Acute LD50 data indicated that brodifacoum has the highest toxicity of the four identified rodenticides.

Brodifacoum is also very highly toxic to aquatic organisms, but due to its extremely low solubility, the USEPA does not believe the chemical poses a hazard to non-target aquatic organisms. The 96-hr LC50 is for 0.025 mg/L for bluegill and 0.015 mg/L for rainbow trout (USEPA 1998e). Additionally, the USEPA has determined that brodifacoum does not pose a risk to honey bees (USEPA 1998e) (Table 6.1).

4.5.3.4 Summary of Toxicity and Potential Effects

Brodifacoum has low water solubility and is generally applied as food bait blocks or pellets. This second-generation rodenticide is highly toxic to mammals, including humans, domestic pets, and non-target mammalian wildlife. Brodifacoum is often found in the tissues of wildlife, including avian and mammalian

predators. Compared to other rodenticides reviewed herein, brodifacoum has the greatest acute toxicity and is one of the most commonly identified poisons in tissues of non-target wildlife. Brodifacoum was not used by the Districts during the reporting year. Due to its limited use by the Districts, brodifacoum does not appear to be an active ingredient of concern, and using BMP application practices, these products should not result in unwanted adverse effects

4.5.4 Bromadiolone

Bromadiolone is an anticoagulant rodenticide that is used to control Norway rats (*Rattus norvegicus*), roof rats (*Rattus rattus*), and house mice (*Mus musculus*) in and around buildings and in transport vehicles (ships, trains, and aircraft), alleys, and sewers. Formulation types include meal bait, pellets, ready-to-use place packs, and paraffinized blocks (USEPA 1998e, Sternberg et al. 2011).

4.5.4.1 Environmental Fate

Bromadiolone is moderately persistent in soil and is immobile in in soil with high organic and clay content. Bromadiolone is stable to hydrolysis at pH 5, 7, and 9. The major route of dissipation appears to be aerobic soil metabolism (half-life of 14-days).Two of the major degradates identified in the aerobic soil metabolism study are persistent (USEPA 1998e, Sternberg et al. 2011).

4.5.4.2 Human Toxicity

Bromadiolone is highly toxic to mammals by acute oral, dermal, and inhalation exposure. The oral LD50 is between 0.56 and 0.84-mg/kg for rats. The dermal LD50 is 1.71 mg/kg for rabbits. The inhalation LC50 is 0.43 µg/kg for rats. Bromadiolone is listed as Category III for eye irritation and IV for skin dermal irritation (USEPA 1998e) (Table 6.1).

4.5.4.3 Ecological Toxicity

Because bromadiolone is a rodenticide, risk is presumed for any small mammals that ingest bait containing the chemical (USEPA 1998e). Like other common rodenticides, bromadiolone is often found in tissues of wildlife. The LD50 for carnivores such as coyotes, foxes, and mountain lions is 1.125 to 25.0 mg/kg (Stone et al. 1999, Hosea 2000). Domestic animals may accidentally ingest bait. Acute toxicity for dogs (hemorrhages fatal if not treated) occurs at 10 mg/kg. For a 10 kg dog that would correspond to 100 mg of pure bromadiolone (2 kg bait at a typical application of 0.005 percent) (EXTOXNET 1985b). The maximum tolerated oral dosage for cats is 25 mg/kg (EXTOXNET 1985b).

The LD50 for wild birds, including birds of prey is 16.93 mg/kg (Stone et al. 1999, Hosea 2000). The LD50 is 138 mg/kg for bobwhite quail (USEPA 1998e). Bromadiolone (in combination with brodifacoum and difethialone) was indicated in the mortality of three red-tailed hawks in Manhattan in 2012 (New York State Department of Environmental Conservation 2012). In a recent study of raptors and owls in Denmark, 92 percent of all birds contained detectable hepatic concentrations of anticoagulant rodenticides, with second-generation anticoagulants (brodifacoum, bromadiolone, and difenacoum the most prevalent) (Christensen et al. 2012).

Bromadiolone bioconcentration factors (BCF) of 160X and 1,658X were determined for edible and nonedible tissues in bluegill sunfish, respectively (USEPA 1998e). The 96-hr LC50 is 0.24-mg/L for rainbow trout and 3.0 mg/L for bluegill (USEPA 1998e). The 24-hr LC50 for *Daphnia magna* is 8.8 mg/L (EXTOXNET 1985b). Due to the methods of bromadiolone application, little if any of the chemical is expected in water bodies. Additionally, bromadiolone is extremely insoluble and therefore is not expected to pose a major risk to aquatic organisms (USEPA 1998e) (Table 6.1).

4.5.4.4 Summary of Toxicity and Potential Effects

Bromadiolone is moderately persistent in soils and is generally applied as food bait blocks or pellets. This second-generation rodenticide is highly toxic to mammals, including humans, domestic pets, and non-target

mammalian wildlife. Bromadiolone is often found in the tissues of wildlife, including avian and mammalian predators. Mortalities of raptors have been associated with secondary bromadiolone poisoning.

Bromadiolone is being used in and around manmade and natural standing and moving water, including during the reporting year. There are currently four Districts using a total of five products that contain bromadiolone (0.005 percent) for rodent control. These products were applied during all four quarters of the reporting year. One product alone accounts for approximately 0.05 lbs AI applied per year (over 1,000 lbs of product). When deployed in sewers, bromadiolone blocks are sometimes attached to a string and hung below manhole covers. This method of bait deployment reduces the probability of exposure (by multiple routes) to humans and non-target wildlife, especially dietary exposure (ingestion route) to ground-foraging birds and mammals. In addition, this rodenticide causes rapid mortality of targeted rats, therefore poisoned individuals tend to expire in the sewers and not represent prey for secondary consumers in the terrestrial environment. Further, bromadiolone is usually wax-encased (e.g., Contrac Blox) in block form, which has exceptionally low water solubility and low leaching potential.

Outside of sewers, bromadiolone is typically contained in tamper-proof bait stations, which are most frequently deployed at residential locations per the request of homeowners, and not near aquatic systems, open lands, or woodlands. Residential treatments involve bait station deployment generally within 50 feet of homes. Bait stations are anchored to treatment locations (e.g., wires, stakes, etc.) to ensure that they cannot be dragged away by wildlife. In addition, bait stations have small openings that prevent the entrance and exposure to non-rodent mammals (e.g., squirrels, skunks, etc.). Residents are properly educated regarding the location of deployed tamper-proof bait stations and potential risks to children and pets.

Bromadiolone is a single-dose rodenticide that when used properly (such as in the absence of food competition), causes rapid knock-down of rat populations and very limited potential for impacting aquatic systems and resulting in exposure to humans and non-target wildlife. If use is expanded by the Districts in the future or additional issues arise regarding the use of this rodenticide, new, more protective rodenticide bait station alternatives reported by the USEPA could be considered (http://www.epa.gov/pesticides/mice-and-rats/rodent-bait-station.html). Based on toxicity, environmental fate, and usage patterns, bromadiolone, using BMP is not likely to result in unwanted adverse impacts.

4.5.5 Bromethalin

Bromethalin is often used to exterminate rodents resistant to first generation anticoagulant rodenticides. Bromethalin is easily confused with second generation anticoagulant rodenticides (brodifacoum, bromadiolone) due to naming similarities. After ingestion, bromethalin is rapidly absorbed and undergoes N-demethylation in the liver, forming desmethylbtomethalin, which is thought to be the major toxic metabolite. The plasma half-life of bromethalin is about six days in rats. Excretion occurs mainly in bile and enterohepatic resuspension is suspected. The mode of action is the uncoupling of oxidative phosphorylation, which leads to decreased cellular ATP production and failure of Na+, K+-ATPase pumps. The cells lose osmotic control and swell. Cerebral and spinal cord edema elevates cerebrospinal fluid pressures and leads to neurologic dysfunction. Bromethalin toxicosis in dogs manifests as either paralytic or convulsant syndrome. Cats develop paralytic syndrome at all doses (Dunayer 2003).

Some bromethalin products meet the USEPA's new, more protective risk reduction standards. When applied properly, these products present a lower risk of accidental exposure to children, pets, and wildlife. They are applied in tamper-resistant and weather-resistant bait stations (USEPA 2013).

4.5.5.1 Environmental Fate

Bromethalin is stable to hydrolysis and is persistent to aerobic soils. In addition, a major degradate, desnitrobromethalin, also appears to be persistent and its mobility has not been characterized. However, because bromethalin is formulated as pelleted food bait, total usage of the active ingredient is low and ground water leaching and surface runoff is expected to be minimal (USEPA 2011).

Table 4-18 Degradation of Bromethalin

Degradation Method	Half-life	Reference
Hydrolysis	Stable	USEPA 2011
Aerobic metabolism (soil)	178 Days	USEPA 2011

4.5.5.2 Human Toxicity

Bromethalin is classified as very highly toxic to mammals on an acute oral basis. The acute oral (14-day) LD50 of bromethalin is 2.11 mg/kg for rats (USEPA 2011). The oral LD50 is 2.38 to 5.6 mg/kg for dogs and 0.54-mg/kg for cats (USEPA 2011). The minimum lethal oral dose for cats is 0.45 mg/kg (USEPA 2011).

4.5.5.3 Ecological Toxicity

Bromethalin is classified by the USEPA as highly toxic to birds and mammals on an acute oral basis and as highly toxic to birds on a subacute dietary basis. The acute oral (14-day) LD50 for bobwhite quail is 4.56 mg/kg (USEPA 2011). Data are not available to characterize the toxicity of bromethalin to nontarget invertebrates such as honey bees. Very little research has been conducted to directly measure the secondary poisoning hazard of bromethalin. Aquatic exposure is expected to be negligible based on the use patterns of bromethalin and there are currently no data available on the toxicity of this rodenticide to fish or aquatic invertebrates. The USEPA has listed bromethalin as “may affect” and “likely to adversely affect” the federally threatened Alameda whipsnake and the endangered salt marsh harvest mouse in California (USEPA 2011).

4.5.5.4 Summary of Toxicity and Potential Effects

Due to its acute toxicity to rodents and mammals, and the potential for exposure to non-target pets and wildlife, use of bromethalin requires a thorough understanding of possible routes of exposure. Placement and amounts of bromethalin used are critical factors in reducing potential unwanted secondary exposures and effects. Many uses of this product include subterranean placement to poison moles and voles (usually in worm-like commercial products). Some reent bromethalin products meet the USEPA’s new, more protective risk reduction standards and using proper application techniques they can result in a lower risk of accidental exposure to children, pets, and wildlife. They can also be applied in tamper-resistant and weather-resistant bait stations. Use of these products should always include appropriate BMPs and prior evaluation of the potential predators and non-targets that might consume this product.

4.5.6 Difethialone

Difethialone is an anti-coagulant rodenticide that is registered for use only in baits for control of three commensal rodents: the Norway rat (*Rattus norvegicus*), the roof rat (*Rattus rattus*), and the house mouse (*Mus musculus*). Formulation types registered include pellets, pellet packs, blocks, mini blocks, paraffin blocks, meal, packs or pouches, paste and bait stations. Currently, labeled uses of difethialone include in and around homes, and agricultural, industrial and commercial buildings, transport vehicles and associated ports, alleys and sewers (Housenger and Melendez 2011).

Difethialone was introduced as a second-generation rodenticide in 1986 for the control of commensal rats and mice including those resistant to first-generation anticoagulants. Difethialone inhibits the vitamin K-dependent step in the synthesis of a number of blood coagulation factors and disrupts normal blood-clotting mechanisms and induces capillary damage (Housenger and Melendez 2011).

4.5.6.1 Environmental Fate

Difethialone adsorbs to suspended solids and sediment and is immobile in soil. Difethialone can slowly volatilize from water surfaces. The compound is relatively stable to hydrolysis and aerobic metabolism, but degrades rapidly by photolysis (Table 4-19).

Table 4-19 Degradation of Difethialone

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	154 to 211 Days	Housenger & Melendez 2011
Photolysis, pH 5-9 (water)	57 to 62 minutes	Housenger & Melendez 2011
Aerobic metabolism (soil)	204 Days	Housenger & Melendez 2011

4.5.6.2 Human Toxicity

Difethialone is very toxic to mammals by all acute exposure routes. The LD50 for male rats is 0.55 mg/kg bw. The LD50 for mice is 1.29 mg/kg bw. The dermal LD50 is 6.5 mg/kg bw for rats. The inhalation LC50 is ≥ 5.0 µg/L in 4 hrs but ≤19.3 µg/L in 4-hrs for rats (Annex I - Norway 2007). Difethialone is not known to cause skin or eye irritation. No genotoxic or carcinogenic effects have been noted (Annex I - Norway 2007) (Table 6.1).

4.5.6.3 Ecological Toxicity

Difethialone is likely very toxic to most mammals. Domestic animals are somewhat less susceptible than rats and mice. The LD50 is 11.8 mg/kg bw for dogs and ≥ 16 mg/kg bw for cats. A dog would have to ingest 400 g of bait for mortality to occur (Lechevin and Poche 1988). The LD50 for domestic pigs is between 2.0 and 3.0 mg/kg bw (Annex I - Norway 2007). Difethialone is also acutely toxic by dermal and inhalation exposure. The dietary LC50 for ferrets is 97.7 mg active ingredient/kg (Savarie 2005). USEPA studies concluded that, based on the best available information, difethialone is “likely to adversely affect” Alameda whipsnake, salt marsh harvest mouse, and San Joaquin kit fox (Housenger and Melendez 2011, Wagman and Shelby 2012). Risk evaluation indicated that the registered uses of difethialone exceed acute LOCs for the small mammalian weight class of salt marsh harvest mouse. Kit fox are likely to be affected via secondary exposure as well as indirectly from reduced prey availability (small mammals). The whipsnake may be affected as the acute Risk Quotients exceed the LOC for both primary and secondary exposure and habitat modification would also occur (fewer small mammal burrows) (Housenger and Melendez 2011, Wagman and Shelby 2012). The recommended action is that a formal consultation with USFWS under Section 7 of the ESA be initiated.

Difethialone is very highly toxic to birds on an acute oral and subacute dietary exposure basis (Housenger and Melendez 2011). Difethialone is acutely toxic to birds. The LD50 for bobwhite quail is 0.264-mg/kg bw (Annex I - Norway 2007). The dietary LC50 for magpies is 4.48 mg active ingredient/kg diet (Savarie 2005). Secondary exposure of birds of prey has been demonstrated. A study of barn owls gave a low LD100 between 0.27 and 0.39 mg/kg bw (Annex I - Norway 2007). There are no data to characterize chronic toxicity to birds and mammals (Housenger and Melendez 2011). Difethialone is highly toxic to aquatic organisms. The 96-hr LC50 for rainbow trout is 51 µg/L. The 48-hr EC50 for *Daphnia magna* is 4.4-µg/L. There are no data to characterize toxicity of difethialone to the honey bee (Table 6.1).

4.5.6.4 Summary of Toxicity and Potential Effects

Difethialone is persistent in soils and is generally applied as food bait blocks or pellets. This second-generation rodenticide is highly toxic to mammals, including humans, domestic pets, and non-target mammalian wildlife. Difethialone is often found in the tissues of wildlife, including avian and mammalian

predators. Difethialone has been categorized as “likely to adversely affect” several species of sensitive California wildlife and registered uses of difethialone exceed the LOC for both primary and secondary exposure. Indirect effects to habitat have been suggested for areas where difethialone is used for pest control (Housenger and Melendez 2011).

One product containing difethialone (0.0025 percent) is used by two Districts for rat control and is applied around creeks, parks, and landscaping. Application typically occurs in the fall, winter, and spring, including 19 applications during the reporting year.

Difethialone is used in areas frequented by humans and domestic animals (parks, landscaped areas) during much of the year. The availability of new, more protective rodenticide bait stations reported by the USEPA should be considered when available. (http://www.epa.gov/pesticides/mice-and-rats/rodent-bait-station.html).

4.5.7 Cholecalciferol

Cholecalciferol is used to control Norway rats (*Rattus norvegicus*), roof rats (*Rattus rattus*), and house mice (*Mus musculus*) in and around homes, industrial buildings and similar man-made structures, in and around agricultural buildings, including swine, poultry, cattle and dairy facilities, warehouses and food storage areas; in transport vehicles (ships, trains and aircraft) and in and around related port and terminal buildings; and in alleys. Formulation types include pellets and blocks (Clock-Rust and Sutton 2011). Cholecalciferol is a sterol (vitamin D3) and its ingestion results in hypercalcemia from mobilization of calcium from bone matrix into blood plasma leading to metastatic calcification of soft tissues (Clock-Rust and Sutton 2011).

4.5.7.1 Environmental Fate

The environmental fate of cholecalciferol is not well described. Based on physical/chemical properties of cholecalciferol, it is expected to be nonvolatile, essentially insoluble in water and immobile in soil (Clock-Rust and Sutton 2011). Information on biotic and abiotic degradation was not available.

4.5.7.2 General Toxicity

The parent compound and metabolites are fat soluble and stored in adipose tissue. Enterohepatic recirculation of cholecalciferol and metabolites occurs. After a massive intake of cholecalciferol, excess calcifediol is produced in the liver. Because of their high lipid solubility, cholecalciferol and its metabolites are eliminated from the body very slowly (primarily through bile and feces). Two mechanisms occur with consumption of large doses of cholecalciferol. First, more calcium is absorbed from the intestines. Second, cholecalciferol metabolites stimulate phosphorus transfer from bone to plasma. The increased plasma calcium concentrations result in vomiting, lethargy, and muscle weakness. Specific organ effects include acute renal tubular necrosis, gastrointestinal stasis, gastric acid secretion, decreased skeletal muscle responsiveness, and decreased neural tissue responsiveness. The increase in plasma calcium causes soft tissue mineralization resulting in loss of functionality of kidneys, cardiac muscle, etc. (Morrow 2001).

4.5.7.3 Human Toxicity

Cholecalciferol is acutely toxic to target rodents. The oral LD50 for cholecalciferol dissolved in corn oil is 42.5 mg/kg for mice and 43.6 mg/kg for rats (Marshall 1984). The dermal LD50 of the finished bait product (0.075 percent cholecalciferol) is 2,000 mg/kg for rabbits (Marshall 1984) (Table 6.1).

4.5.7.4 Ecological Toxicity

Cholecalciferol is considered of low hazard to avian and canine species. The oral LD50 for dogs is 88 mg/kg. The oral LD50 for mallard ducks and bobwhite quail is 2,000 mg/L (Marshall 1984). When used in bait form, cholecalciferol may directly impact sensitive species such as non-target rodents (Clock-Rust and Sutton 2011). Cholecalciferol is not expected to bioconcentrate since it is metabolized in mammals (Clock-Rust and Sutton 2011)

4.5.7.5 Summary of Toxicity and Potential Effects

Cholecalciferol is essentially insoluble in water and immobile in soils. It is generally applied as food bait blocks or pellets. The mode of action of cholecalciferol differs from the other rodenticides examined herein in that it is not an anticoagulant. Rather, cholecalciferol baits deliver a toxic dose of vitamin D to pests. Although it is highly toxic to target rodents, cholecalciferol is considered of low hazard to non-targets such as birds or domestic dogs.

Cholecalciferol is used in one product (0.075 percent) by one District. It is used along creeks, parks, and waterfronts in the fall, winter, and spring. Cholecalciferol was used on 26 occasions accounting for total application of 37 ounces of product. Based on the reported usage, using BMP application practices, these products should not result in unwanted adverse effects

4.5.8 Sulfur (fumigant)

Elemental sulfur is a naturally occurring component of the earth's core and crust and is ubiquitous in the environment. Sulfur has been used as a pesticide in the United States since the 1920s, and is currently registered for use as an insecticide and fungicide on a wide range of field and greenhouse-grown food and feed crops, livestock (and livestock quarters), and indoor and outdoor residential sites. Sulfur is also one of the active ingredients in four fumigant (gas-producing) cartridge products, which are used for rodent control on lawns, golf courses, and in gardens. Carbon, sodium and potassium nitrates, sawdust, and sulfur are used in the pyrotechnic fumigant gas producing cartridge products. After the cartridges are ignited, they produce toxic gases that cause asphyxiation of the pests. These toxic gases, not the active ingredients, are the stressors for these products. The gases displace the oxygen in the burrows, creating an un-breathable atmosphere, causing asphyxiation of the target organisms (USEPA 2008d).

Elemental sulfur, when applied as a pesticide, will become incorporated into the natural sulfur cycle. The main processes and dissipation of elemental sulfur are oxidation into sulfate and reduction into sulfide. These processes are mainly mediated by microbes (USEPA 2008d).

4.5.8.1 Human Toxicity

Elemental sulfur is known to be of low toxicity and poses little, if any, risk to human health. The USEPA classifies sulfur as Category IV (least toxic) for acute oral toxicity (EXTOXNET 1995b). The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rats is also >2,000 mg/kg. Acute inhalation exposure can cause respiratory irritation. The inhalation LC50 for 98 percent sulfur in rats is >2.56 mg/L. Sulfur is not a skin sensitizer. No known risks of oncogenic, teratogenic, or reproductive effects are associated with the use of sulfur (EXTOXNET 1995b) (Table 6.1).

4.5.8.2 Ecological Toxicity

Sulfur is nontoxic to birds (EXTOXNET 1995b). The 8-day dietary LC50 for bobwhite quails is >5,620 mg/L for 95 percent sulfur wettable powder. Sulfur is of practically nontoxic to aquatic organisms (EXTOXNET 1995b). The 96-hr LC50 for bluegill and rainbow trout is >180 mg/L using a 99.5 percent sulfur dust formulation. The 48-hr LC50 for *Daphnia magna* is >5,000 mg/L using 90 percent sulfur. Sulfur is considered nontoxic to bees (EXTOXNET 1995b) (Table 6.1).

4.5.8.3 Summary of Toxicity and Potential Effects

Sulfur fumigants are of low toxicity prior to activation of sulfur-containing cartridges. Elemental sulfur will become incorporated back into the natural sulfur cycle after deployment. Sulfur fumigant cartridges are placed in pest burrow and produce toxic gases, which will negatively impact any animal in the burrow. Therefore, sulfur fumigants should not be applied when there is evidence of non-target animal presence. Sulfur fumigants were not used by the Districts during the reporting year and risk to non-targets is readily avoided by inspecting application sites thoroughly, therefore, using BMP application practices, these products should not result in unwanted adverse effects.

4.5.9 Sodium Nitrate (fumigant)

Sodium nitrate is used with other components as an active ingredient to control mammals such as woodchucks, ground squirrels, and coyotes in open fields, noncrop areas, rangelands, lawns and golf courses. End-use products containing sodium nitrate are used as fumigant gas cartridges designed to be placed in burrows. The sodium nitrate supports the combustion of charcoal in the formulation of each product. Pyrolysis of these sodium nitrate products results in simple organic and inorganic compounds, mostly in the form of gases such as nitrous oxide and carbon monoxide, which eventually diffuse through burrow openings or into the soil causing organisms to die of asphyxiation (USEPA 1991d).

4.5.9.1 Human Toxicity

Available acute toxicity studies indicate that sodium nitrate may cause eye irritation (Category II) and slight dermal irritation (Category IV) to mammals, but pose relatively low acute oral toxicity (Category III) hazard (USEPA 1991b). The only people exposed to sodium nitrates should be pesticide applicators and they should be exposed only minimally (USEPA 1991b). The USEPA believes that sodium nitrates, when used as indicated, do not present any unreasonable adverse effects to humans (Table 6.1).

4.5.9.2 Ecological Toxicity

Sodium nitrates are naturally occurring substances and exposure of the environment is limited and localized when the products are used as fumigants in burrows (USEPA 1991b). When used as indicated by the product label, any organism inside of a treated burrow would likely be killed by the toxic fumes. The nonselective nature of this pesticide is particularly problematic when protected species are present. Nontarget species such as burrowing owls, black-footed ferrets, kangaroo rats, or desert tortoises often inhabit pest burrows and may be at risk (Keefover-Ring 2009). USEPA recommends that applicators observe signs around burrows indicating the presence of non-target species and use caution (Table 6.1).

4.5.9.3 Summary of Toxicity and Potential Effects

Sodium nitrate fumigants are of low toxicity prior to activation of the cartridges. Sodium nitrates are naturally occurring substances. Sodium nitrate fumigant cartridges are placed in pest burrow and produce toxic gases, which will negatively impact any animal in the burrow. Therefore, sodium nitrate fumigants should not be applied when there is evidence of non-target animal presence. Sodium nitrate fumigants were not used by the Districts during the reporting year and risk to non-targets is readily avoided by inspecting application sites thoroughly; therefore, using BMP application practices, these products should not result in unwanted adverse.

4.6 Herbicides

4.6.1 Imazapyr

Imazapyr is part of the imidazolinone chemical class. Imazapyr is a systemic, nonselective, pre- and post-emergent herbicide used for the control of a broad range of terrestrial and aquatic weeds, and controls plant growth by preventing the synthesis of branched-chain amino acids. Imazapyr is applied either as an acid or as the isopropylamine salt. Imazapyr is used for pre- and post-emergence control of a broad range of weeds, including terrestrial annual and perennial grasses, broadleaf herbs, woody species, and riparian and emergent aquatic species. Agricultural uses of imazapyr include field corn and grass. Imazapyr is also registered for use on a variety of commercial and residential use sites, including forestry sites, rights-of-way, fence rows, hedge rows, drainage systems, outdoor industrial areas, outdoor buildings and structures, domestic dwellings, paved areas, driveways, patios, parking areas, walkways, various water bodies (including ponds, lakes, streams, swamps, wetlands, stagnant water, and urban areas) (USEPA 2006b).

4.6.1.1 Environmental Fate

Imazapyr is an anionic, organic acid that is nonvolatile and is both persistent and mobile in soil. Commercial formulations contain either imazapyr acid or the imazapyr isopropylamine salt, both of which are dissolved in a water solution. Imazapyr is mainly in ionic form at typical environmental pH levels, and the behavior of the acid and salt forms are similar. Upon direct application, or indirect release into surface water, photolysis is the only identified mechanism for imazapyr degradation in the environment (Table 4-20), with a half-life of approximately 3 to 5 days in surface water. Laboratory studies show imazapyr is essentially stable to hydrolysis, aerobic and anaerobic soil degradation, as well as aerobic and anaerobic aquatic metabolism. Field dissipation study observations are consistent with imazapyr's intrinsic ability to persist in soils and move via runoff to surface water and to leach to groundwater (USEPA 2006b).

Table 4-20 Degradation of Imazapyr

Degradation Method	Half-life	Reference
Hydrolysis	Stable	USEPA 2006a
Photolysis (water)	3 to 5 Days	USEPA 2006a
Aerobic metabolism (water and soil)	Stable	USEPA 2006a
Anaerobic metabolism (water and soil)	Stable	USEPA 2006a

4.6.1.2 Human Toxicity

Imazapyr is slightly toxic to mammals via oral, dermal, and inhalation exposure. The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The inhalation LC50 for rats is >1.3 mg/L. There is no evidence that imazapyr is carcinogenic or mutagenic (USDOE-Bonneville Power Administration 2000). The USEPA has determined that the risk to humans of dietary and incidental exposure is below the level of concern (USEPA 2006b). Imazapyr is classified as a Category I primary eye irritant (USEPA 2006b). The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The inhalation LC50 for rats is >1.3 mg/L (USDOE-Bonneville Power Administration 2000) (Table 6.1).

4.6.1.3 Ecological Toxicity

Imazapyr is practically nontoxic to birds, fish, *Daphnia*, honey bees. The oral LD50 for mallard ducks is >2,150 mg/kg. The 96-hr LC50 for rainbow trout is >100 mg/L. The 48-hr LC50 for *Daphnia magna* is >1,000 mg/kg. The LD50 for honey bees is >100 µg/bee (USDOE-Bonneville Power Administration 2000). Although there are no risks of concern to terrestrial birds, mammals, and bees or aquatic invertebrates and fish, imazapyr does pose an ecological risk to non-target terrestrial and aquatic vascular plants (USEPA 2006b). Imazapyr is not expected to bioaccumulate in aquatic organisms because it exists as an anion at typical environmental pHs (USEPA 2006b) (Table 6.1).

4.6.1.4 Summary of Toxicity and Potential Effects

Imazapyr is persistent in soil and also tends to leach to groundwater. It is of low acute toxicity to mammals and practically nontoxic to birds, fish, and invertebrates. Non-target plants may be at risk from imazapyr application. Based upon the toxicity and environmental fate of imazapyr, and using BMP application practices, these products should not result in unwanted adverse effects.

4.6.2 Glyphosate

Glyphosate is a nonselective, post-emergent, and systemic herbicide registered for use in agricultural and nonagricultural areas. It is applied to agricultural drainage systems, irrigation systems, sewage systems, forest trees, greenhouses, outside of household/domestic dwellings, and to a variety of feed and food

crops. When applied at lower rates, glyphosate is plant growth regulator (USEPA 1993). It works by inhibiting the synthesis of the enzyme 5-enolpyruvylshikimic acid-3-phosphate synthase (EPSP), which is needed for production of amino acids. These amino acids aid in synthesis of proteins that link primary and secondary metabolism. Glyphosate is not effective on submerged or mostly submerged foliage and therefore is only applied to control emergent foliage (Schuette 1998, Siemering 2005).

4.6.2.1 Environmental Fate

Glyphosate is highly water-soluble. Glyphosate is broken down by microbial degradation to its metabolite aminiomethylphosphonic acid (AMPA) and carbon dioxide. The rate of degradation in water is generally slower than the rate in soil because there are fewer microorganisms in water than in most soils. For all aquatic systems, sediment appears to be the major sink for glyphosate residue. Even though glyphosate is highly water soluble it appears that parent glyphosate and AMPA have a low potential to move to groundwater due to their strong soil adsorptive characteristics (USEPA 1993, Schuette 1998, Siemering 2005).

In the soil environment, glyphosate is resistant to chemical degradation, is stable to sunlight, is relatively nonleachable, and has a low tendency to runoff (except as adsorbed to colloidal matter and sediment). It is relatively immobile in most soil environments as a result of its strong adsorption to soil particles and does not move vertically below the 6 inch soil layer. Glyphosate's primary route of decomposition in the environment is through microbial degradation in soil (Table 4-21). The herbicide is inactivated and biodegraded by soil microbes at rates of degradation related to microbial activity in the soil and factors that affect this activity. The biological degradation process is carried out under both aerobic and anaerobic conditions by soil microflora (USEPA 1993, Schuette 1998).

Table 4-21 Degradation of Glyphosate

Degradation Method	Half-life	Reference
Hydrolysis	>35 Days, Stable	USEPA 1993b, Schuette 1998, FAO-WHO 2005a
Photolysis (water)	Stable (pH 5,7 and 9)	USEPA 1993b
Photolysis (soil)	Stable	USEPA 1993b
Aerobic metabolism (water)	7 Days	USEPA 1993b
Aerobic metabolism (soil)	1.85 to 25 Days	USEPA 1993b, FAO-WHO 2005a
Anaerobic metabolism (soil)	8.1 to 22.1 Days	USEPA 1993b, Schuette 1998
Field dissipation (soil)	44 to 60 Days	Schuette 1998
Streams, ponds, natural waters	1.5 to 63 Days	Schuette 1998

4.6.2.2 General Toxicity

Glyphosate is an herbicide designed to specifically affect plants via the shikimic acid pathway. Glyphosate inhibits the enzyme 5-enolpyruvylshikimate 3-phosphate (EPSP) synthase, which is absent in mammals (Miller et al. 2010). The resulting deficiency in ESPS production leads to reductions in aromatic amino acids necessary for plant protein synthesis and growth. Glyphosate is absorbed directly across the leaves and stems and is translocated throughout the plant, concentrating in the meristem (Miller et al. 2010). The effects of the herbicide are generally visible between 4 and 20 days post-application and include stunted growth, loss of pigmentation, malformation or wrinkling of leaves, and ultimately tissue death (Miller et al. 2010). There are several formulations of glyphosate, including an acid, monoammonium salt, diammonium salt, isopropylamine salt, potassium salt, sodium salt, and trimethylsulfonium or trimesium salt. The commonly used Roundup™ products are isopropylamine salt formulations. The salts do not

contribute to the weed control activity; therefore, the acid equivalent (ae) of glyphosate acid is the most accurate method of expressing and comparing concentrations (Table 6.1).

4.6.2.3 Human Toxicity

The shikimic acid pathway is specific to plants and some microorganisms; therefore, glyphosate is thought to have very low toxicity to mammals (USEPA 1993). The USEPA classifies glyphosate as Category III for oral and dermal toxicity (USEPA 1993). The oral LD50 for technical grade glyphosate for rats is 4,320 mg/kg (USEPA 1993). The dermal LD50 for technical grade glyphosate in rabbits is ≥2000 mg/kg (USEPA 1993). Technical grade glyphosate is nonvolatile and the LC50 for rats is ≥4.43 mg/L based on a 4-hr, nose-only inhalation study (USEPA 1993, Miller et al. 2010).

The isopropylamine and ammonium salts exhibit low toxicity to mammals via the oral and dermal routes. The oral LD50 for the isopropylamine salt in rats is ≥ 5,000 mg/kg. The oral LD50 for the ammonium salt form in rats is 4,613 mg/kg. The dermal LD50 for rabbits is ≥ 5,000 mg/kg for both salts (Miller et al. 2010). The salt formulations of glyphosate also exhibit low toxicity via the inhalation route. The 4-hr LC50 for rats exposed to the isopropylamine form is >1.3 mg/L air. The LC50 for rats exposed to the ammonium salt form was >1.9 mg/L in a whole-body exposure (Miller et al. 2010).

A one-year feeding study resulted in no chronic effects in beagle dogs at daily doses of 500 mg/kg (USEPA 1993). There is no scientific evidence indicating that glyphosate is carcinogenic or mutagenic (USEPA 1993). Experimental evidence has shown that neither glyphosate nor its major breakdown product (aminiomethylphosphonic acid [AMPA]) bioaccumulates in any animal tissue (Williams et al. 2000). Glyphosate is poorly biotransformed in rats and is excreted mostly unchanged in the feces and urine (Williams et al. 2000).

Despite the apparent lack of toxicity to mammals, concerns have been raised about the long-term safety of glyphosate. In one study, glyphosate has been shown to alter the respiratory and hepatic systems of rats and to cause damage to reproductive functions and fetal development (Clair et al. 2012). Additionally, a recent study found significant contamination in all urine samples taken from an urban human population in Germany. The levels of glyphosate in the subjects' urine were 5 to 20 times the maximum allowable limit for drinking water (Brandli and Reinacher 2012). In another study, rats and mice were fed a diet containing glyphosate for 13 weeks. The two highest dose groups of male rats (25,000 and 50,000 mg/kg of 99 percent pure glyphosate) had significant reductions in sperm concentrations (Chan and Mahler 1992). Female rats in the 50,000 mg/kg group had slightly longer estrus cycles than the control group (Chan and Mahler 1992). Although still in review, glyphosate is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a), which focuses on pesticide active ingredients and inert ingredients with relatively greater potential for human exposure (Table 6.1).

4.6.2.4 Ecological Toxicity

Glyphosate is practically nontoxic to birds. The oral LD50 for bobwhite quail is >2,000 mg/kg. It is also practically nontoxic to freshwater fish. The 48-hr LC50 for bluegill sunfish is >24-mg/L (USEPA 1993). Maximum bioconcentration factors were 0.52 times for whole fish (USEPA 1993). Technical grade glyphosate is slightly toxic to practically nontoxic to freshwater invertebrates with 48-hr LC50s ranging from 55 to 780 mg/L. LC50 values have also been obtained for several species of frogs and the American toad. The 24-hr LC50 for amphibians ranged from 6.6 to 18.1 mg/L (Howe et al. 2004). No significant acute toxicity to amphibians was observed with the technical material or the products (e.g., Roundup Original). Glyphosate is practically nontoxic to honey bees. The acute oral LD50 is >100 µg/bee (USEPA 1993) (Table 6.1).

4.6.2.5 Summary of Toxicity and Potential Effects

Products currently available containing glyphosate include Roundup, Rodeo, Pondmaster, ProMax, Proud3, and Alligare. Each has a formulation with a slight variation of toxicity and environmental characteristics. This summary is focused on the active ingredient, N-(phosphonomethyl) glycine.

Using BMP approaches, applications of glyphosate can be used safely when an adequate buffer to water sources is maintained. Although there has been some recent concerns expressed about possible sub-lethal effects of glyphosate products, it is virtually nontoxic to mammals and practically nontoxic to birds, fish, and invertebrates. Glyphosate has been identified as a candidate by USEPA for evaluation as a potential endocrine disruptor (USEPA 2009a). Based on these issues, it is likely that USEPA will provide an updated review of its potential risks in 2015, but until then, glyphosate products are effective, generally safe, products used for weed control. http://gmo-journal.com/2011/11/21/safety-review-of-glyphosate-herbicide-faces-tough-critics

4.6.3 Triclopyr

Triclopyr is a pyridine-based herbicide used for the control of woody plants and annual and perennial broadleaf weeds. The two registered formulations are triclopyr triethylamine salt (TEA) and triclopyr butoxyethyl ester (TBEE). Triclopyr TEA rapidly dissociates in water to the triclopyr acid/anion and triethanolamine. Triclopyr BEE rapidly hydrolyses in the environment to the triclopyr acid/anion and butoxyethanol. It is the triclopyr acid (known simply as triclopyr) that causes phytotoxicity. Triclopyr is used at railroad or other rights-of-way, for commercial and residential use, and on rice, pasture, and woodlands. Triclopyr is absorbed by leaves and roots and is moved throughout the plant. The triclopyr TEA formulation is also used to control aquatic plant species. Triclopyr is a pyridine-based herbicide that acts as a synthetic auxin, giving a plant an auxin overdose 1,000 times natural levels (Ganapathy 1997). Triclopyr is absorbed by leaves and roots and is moved through the plant into the foliage rapidly. The effects occur at the cellular level first when ethylene and protein production in the plant increases first, followed by epinasty, abnormal leaf formation, and stem swelling, and death. Triclopyr has low phytotoxicity to grasses, but can cause injury to conifers at high application rates (Ganapathy 1997). There are two formulations of triclopyr: the triethylamine salt (TEA) and the butoxyethyl ester (TBEE).

4.6.3.1 Environmental Fate

Triclopyr is nonvolatile and highly soluble. Triclopyr is “slightly mobile” with sorption to soil increasing with time. Triclopyr is moderately persistent, with persistence increasing as it reaches deeper soil levels and anaerobic conditions. The predominant degradation pathway for triclopyr in water is photodegradation and the predominant degradation pathway in soil is microbial degradation (Table 4-22; (Ganapathy 1997, USEPA 1998h).

Table 4-22 Degradation of Triclopyr

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	270 Days, Stable	USEPA 1998g, Ganapathy 1997
Photolysis (water)	0.36 to 1.7 Days	USEPA 1998g
Aerobic metabolism (water)	142 Days	USEPA 1998g
Aerobic metabolism (soil)	8 to 18 Days	USEPA 1998g
Anaerobic metabolism (water)	>365 Days	USEPA 1998g
Field dissipation (water)	0.5 to 3.5 Days	USEPA 1998g, Ganapathy 1997
Field dissipation (soil)	10.4 to 33 Days	USEPA 1998g, Ganapathy 1997

4.6.3.2 Human Toxicity

Triclopyr is slightly toxic to mammals by oral and dermal routes and has been classified as Category III by the USEPA (USEPA 1998c). The TBEE formulation is slightly toxic by the oral and dermal route (Category III) and practically nontoxic by inhalation (Category IV) (USEPA 1998c). The oral LD50 for technical triclopyr in rats is 630 mg/kg for females and 729 for males (USEPA 1998c). Triclopyr is not carcinogenic (Table 6.1).

4.6.3.3 Ecological Toxicity

Triclopyr is rapidly absorbed by animals and then excreted by the kidney, primarily in the unmetabolized form. Aquatic organisms are more susceptible to triclopyr. Triclopyr acid is slightly toxic to birds and practically nontoxic to, insects, freshwater fish, and aquatic invertebrates. The oral LD50 of triclopyr acid for mallard ducks is 1,698 mg/kg. The 96-hr LC50 for rainbow trout is 117 mg/L. Triclopyr does not bioaccumulate rapidly (Ganapathy 1997). The 96-hr LC50 for *Daphnia magna* is 132 mg/L. The LD50 for honey bees is 60.4-µg/bee (Ganapathy 1997). Triclopyr does not bioaccumulate rapidly (USEPA 1998c).

The TBEE formulation is slightly toxic to birds, moderately toxic to highly toxic to freshwater fish and slightly to moderately toxic to freshwater invertebrates. The 96-hr LC50 of TBEE to bluegill sunfish is 0.36 mg/L (Ganapathy 1997). The TEA formulation is practically nontoxic to birds and invertebrates and moderately to highly toxic to fish (USEPA 1998c) (Table 6.1).

4.6.3.4 Summary of Toxicity and Potential Effects

Triclopyr is highly soluble and slightly mobile in soil. Technical triclopyr, TEA, and TBEE have similar slight toxicity to mammals, birds, fish, and invertebrates. It also has low toxicity to non-target grasses, but can cause injury to conifers. Based upon the toxicity and environmental fate of triclopyr, and using BMP application practices, these products should not result in unwanted adverse effects.

4.6.4 <u>2,4-Dichlorophenoxy acetic acid (2,4-D)</u>

The compound 2,4-dichlorophenoxyacetic acid (2,4-D) is an herbicide in the phenoxy or phenoxyacetic acid family. Although it is classified as an herbicide, a plant growth regulator, and a fungicide, it is mainly used as a selective postemergence herbicide for the control of broadleaf weed species and aquatic weeds. 2,4-D is registered for use on pasture/rangeland, turf, wheat, corn, soybeans, fallow land, hay other than alfalfa, noncropland (roadways, rights-of-way, ditches, industrial sites, etc.), forestry, rice, sugarcane, pome fruits, stone fruits, nut orchards, filberts, grass grown for seed and sod, aquatic weed control, potatoes, asparagus, strawberries, blueberries, grapes, cranberries, and citrus (USEPA 2005).

2,4-D is generally not applied as the acid, but is applied as one of several formulations, which quickly break down into 2,4-D acid (e.g., chemical formulations of 2,4-D amine salts and 2,4-D esters). 2,4-D mimics the effect of auxins, or other plant growth regulating hormones, and thus stimulates growth, rejuvenates old cells, and overstimulates young cells leading to abnormal growth patterns and death in some plants (Walters 1999). 2,4-D is thought to increase cell-wall plasticity, biosynthesis of proteins and the production of ethylene. The abnormal increase in these processes results in uncontrolled cell division and growth which damages vascular tissue (USEPA 2005).

4.6.4.1 Environmental Fate

In the aqueous environment, 2,4-D is most commonly found as the free anion (the amine salt formulations dissociate to the anion and ester formulations hydrolyze to the anion, usually within 1 day) (Walters 1999). The dissipation of 2,4-D is dependent on oxidative microbial-mediated mineralization, photodegradation in water, and leaching (USEPA 2005). 2,4-D has low persistence in soil, primarily due to degradation by soil microbes, and microorganisms also readily degrade 2,4-D in aquatic environments (Table 4-23). In water, 2,4-D will biodegrade at a rate dependent upon the level of nutrients present,

temperature, availability of oxygen, and whether or not the water has been previously contaminated with 2,4-D or other phenoxyacetic acids (Walters 1999, Siemering 2005).

Table 4-23 Degradation of 2,4-D

Degradation Method	Half-life	Reference
Hydrolysis (water)	Stable	USEPA 2005
Hydrolysis (soil)	39 Days	Walters 1999
Photolysis (water)	12.9 to 13 Days	USEPA 2005, Walters 1999
Photolysis (soil)	68 to 393 Days	USEPA 2005, Walters 1999
Aerobic metabolism (water)	15 Days	USEPA 2005, Walters 1999
Aerobic metabolism (soil)	6.2 to 66 Days	USEPA 2005, Walters 1999
Anaerobic metabolism (water)	41 to 333 Days	USEPA 2005, Walters 1999
Field dissipation (soil)	59.3 Days	Walters 1999

4.6.4.2 Human Toxicity

The modes of toxicity to mammals from the acid, ester, and salt forms of 2,4-D are similar, although the acid and salt forms can also be eye irritants. The oral LD50 for rats ranges from 639 to 1,646 mg/kg, depending on the chemical form of 2,4-D used. All forms of 2,4-D are considered of low acute dermal toxicity. The dermal LD50 in rabbits ranges from 1,829 to >2,000 mg/kg. All forms of 2,4,-D are considered of low inhalation toxicity. The inhalation LC50 for rats ranges from 0.78 to >5.4-mg/L, depending on the formulation used (Gervais et al. 2008).

In mammals, 2,4-D is actively secreted by the proximal tubules of the kidney and toxicity appears to result when renal clearance capacity is exceeded. Dose-dependent toxic effects, including damage to the eyes, thyroid, kidney, adrenals, ovaries, and testes, have been observed in rats at 15 mg/kg/day(Charles et al. 1996). Additionally, reproductive toxicity, neurotoxicity, and developmental toxicity have also been observed (Gervais et al. 2008). Because 2,4-D has been associated with effects on the thyroid and gonads following exposure, it has been included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.6.4.3 Ecological Toxicity

2,4-D is slightly to moderately toxic to birds. The LD50 is 1,000 mg/kg in mallards (EXTOXNET 1996a). The LD50 for acute oral exposure for pheasants is 472 mg/kg (Gervais et al. 2008). Some formulations are highly toxic to fish while others are less toxic. The LC50 for cutthroat trout ranges from 1.0 to 100 mg/L, depending on the formulation tested (EXTOXNET 1996a). The 24-hr LC50 for honey bees has been estimated as between 104-and 115 µg/bee and therefore 2,4-D is considered practically nontoxic to bees (USEPA 2005). 2,4-D has been shown to accumulate in fish at up to 18X the ambient concentrations within two days of exposure (Wang et al. 1994) as cited by (Tu et al. 2001) (Table 6.1).

4.6.4.4 Summary of Toxicity and Potential Effects

2,4-D has low persistence in soil and leaches to groundwater. 2,4-D is of low to moderate toxicity to mammals and birds; however, some formulations are highly toxic to fish and invertebrates. In addition, 2,4-D has been associated with dose-dependent damage to eyes, thyroid, kidneys, adrenals, ovaries, and testes in chronic studies of rats. It has been included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a).. Although 2,4,D has been characterized and a potential candidate for the banned chemical list (based upon the high toxicity of 2,4-D

to some fish, the potential of 2,4-D to runoff, and other factors), USEPA has not yet banned it based on its efficacy and documented need by agriculture and industry Therefore, the status of 2,4, D should be monitored by the Districts for updates by the regulatory community.

4.6.5 Sulfometuron Methyl

Sulfometuron methyl was originally registered as a pesticide active ingredient in the U.S. in February 1982. Sulfometuron methyl is a broad-spectrum sulfonylurea herbicide recommended for preemergence and postemergence control of annual, biennial, and perennial grasses and broadleaf weeds. The herbicide is used for general weed control on industrial noncrop sites and for selective weed control on turf grasses on industrial sites. It is also used for selective weed control in forest site preparation and in the release of several types of pines and certain hardwoods (O'dell 1999). Similar to other sulfonylurea herbicides, sulfometuron's mode of action involves inhibiting the activity of the enzyme acetolactate synthase, which inhibits the production of amino acids required for cell growth in plants (USEPA 2008c). The result is growth inhibition followed by a decline in plant vigor, discoloration, chlorosis, and terminal bud death. Although seed development is not inhibited, sulfometuron methyl effectively retards or stops root and shoot development (O'dell 1999).

4.6.5.1 Environmental Fate

Hydrolysis, photolysis and microbially-mediated degradation are major routes of transformation of sulfometuron methyl in water, soil, and water-sediment systems (Table 4-24). The degradation in soil and water appears to be enhanced in the presence of an active microbial population (aerobic and anaerobic degradation both proceed more slowly under sterile conditions) (USEPA 2008c). Sulfometuron methyl has a low tendency to sorb to sediments. Partitioning of sulfometuron methyl and its breakdown products between water and sediment is dependent on pH and organic content of the solids (O'dell 1999). Sulfometuron methyl has the potential to leach to ground water and/or reach surface water during runoff events (USEPA 2008c).

Table 4-24 Degradation of Sulfometuron methyl

Degradation Method	Half-life	Reference
Hydrolysis, pH 5	5 to 14 Days	O'dell 1999
Hydrolysis, pH 7	>30 Days	O'dell 1999
Photolysis (water)	12 Days	O'dell 1999
Photolysis (soil)	11 Days	O'dell 1999
Aerobic metabolism (soil)	53 Days	O'dell 1999
Anaerobic metabolism (soil)	283 Days	O'dell 1999
Field dissipation (soil)	14 Days	O'dell 1999

4.6.5.2 Human Toxicity

Sulfometuron methyl is classified as Category IV to mammals for oral and inhalation toxicity and Category III for dermal toxicity by the USEPA (USEPA 2008c). The oral LD50 for rats is >5,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The inhalation LC50 for rats is >5.0 mg/L (USEPA 2008c) (Table 6.1).

4.6.5.3 Ecological Toxicity

Sulfometuron methyl is nontoxic to birds, slightly toxic to fish, and practically nontoxic to *Daphnia* and honey bees (EXTOXNET 1996c). The avian oral LD50 is >4,650 mg/kg. No sublethal effects have been observed during acute toxicity studies of birds (USEPA 2008c). The LC50 for fish >100 mg/L. The LC50 for *Daphnia*

magna is 125 mg/L (technical material) and >1,000 mg/L (dispersible granule). The 48-hr contact LC50 for honey bees is >100 µg active ingredient/bee (USEPA 2008c). Sulfometuron methyl has low potential to volatilize from soil or water or to bioaccumulate (USEPA 2008c).

Sulfometuron methyl is phytotoxic to duckweed (*Lemna gibba*) at concentrations of ≥ 0.59 µg/L but the effects appear to be reversible given sufficient recovery periods (USEPA 2008c). The chemical is toxic to a broad range of terrestrial plants. EC25 values have been established for sorghum, sugar beets, corn, and soybeans. In all cases, the most sensitive endpoints were seedling emergence and vegetative vigor (USEPA 2008c) (Table 6.1).

4.6.5.4 Summary of Toxicity and Potential Effects

Sulfometuron methyl tends to sorb to soils but has the potential to leach to groundwater. It is of low toxicity to mammals, birds, and bees. It is slightly toxic to fish. The chemical is phytotoxic to non-target aquatic plants such as duckweed. Based upon the toxicity and environmental fate of sulfometuron methyl**, and** using BMP application practices, these products should not result in unwanted adverse effects.

4.6.6 Bentazon

Bentazon, also known by its trade name Basagran, is a selective herbicide that is used after seedlings have emerged to control broadleaf weeds and sedges among food and feed crops including alfalfa, beans, corn, peanuts, peas, peppers, peppermint, rice, sorghum, soybeans and spearmint. Bentazon also is registered for use on ornamental lawns and turf. Most bentazon used in the U.S. (73 percent) is applied to soybean crops. Based on chemical affinities, bentazon is considered a member of the thiadiazine group, containing nitrogen and sulfur atoms. It is a benzothiadiazinone contact herbicide and photosynthetic electron transport inhibitor. Bentazon is formulated and used as the sodium salt alone or in combination with atrazine (USEPA 1994c). The chemical interferes with the ability of susceptible plants to use sunlight for photosynthesis and visible injury to the plants occurs within 4-to 8 hours of application followed by death of the plant.

4.6.6.1 Environmental Fate

Dissipation of bentazon is dependent on photolysis, microbe-induced degradation, leaching and surface water runoff (Table 4-25). Degradation in aquatic environments is dependent on photolysis. Degradation in soil is controlled by processes involving microbes in the presence of oxygen. Bentazon has a low binding affinity to soil and therefore may leach into ground water and runoff into surface waters. Leaching did not appear to be a major route of dissipation in field studies, however. Bentazon dissipates rapidly under typical use conditions. The soil degradates of bentazon include AIBA, which is very mobile but not persistent (half-life 1-10 days), and N-methylbentazon which is not mobile (USEPA 1994c).

Table 4-25 Degradation of Bentazon

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable	USEPA 1994c
Photolysis (water)	2.6 Days	USEPA 1994c
Photolysis (soil)	>39 Days	USEPA 1994c
Aerobic metabolism (water)	Stable	USEPA 1994c
Aerobic metabolism (soil)	24 to 98 Days	USEPA 1994c
Anaerobic metabolism (water)	Stable	USEPA 1994c
Anaerobic metabolism (soil)	89 Days	USEPA 1994c
Field dissipation (soil)	7 to 33 Days	USEPA 1994c

4.6.6.2 Human Toxicity

Bentazon is slightly toxic (Category III) to mammals via the oral, dermal, and inhalation routes and is a skin sensitizer (USEPA 1994a, EXTOXNET 1996b). The oral LD50 for rats is 1,100 mg/kg. The dermal LD50 for rabbits is 4,000 mg/kg. Bentazon has not been associated with carcinogenic effects but causes some developmental toxicity effects in rats and rabbits (USEPA 1994a) (Table 6.1).

4.6.6.3 Ecological Toxicity

Bentazon is slightly toxic to birds on an acute oral and subacute dietary basis and exceeds the level of concern for avian chronic reproductive effects. The risk to birds can be reduced by lowering the maximum seasonal application rate from four to two pounds per acre, as recommended by the USEPA (USEPA 1994a). The oral LD50 for bobwhite quail is 1,171 mg/kg. Bentazon is slightly toxic to small mammals and practically nontoxic to fish and invertebrates. It poses a low risk to aquatic plants but may present a hazard to terrestrial plants (USEPA 1994a) (Table 6.1).

4.6.6.4 Summary of Toxicity and Potential Effects

Bentazon has low binding affinity to soil and leaches to groundwater or tends to runoff. Bentazon is slightly toxic or practically nontoxic to mammals, birds, and bees, fish, aquatic invertebrates, and aquatic plants. Based upon its low toxicity**,** and using BMP application practices, these products should not result in unwanted adverse effects.

4.6.7 Diuron

Diuron is a substituted urea herbicide used for the control of a wide variety of annual and perennial broad leaved and grassy weeds on both crop and noncrop sites. The mechanism of herbicidal action is the inhibition of photosynthesis. It is rapidly translocated into the stems and leaves of plants. Diuron primarily acts by inhibiting the Hill reaction in photosynthesis, limiting the production of high-energy compounds like adenosine triphosphate (ATP) which are necessary for various metabolic processes. Diuron is registered for pre- and post-emergent herbicide treatment of both crop and noncrop areas, as a mildewcide and preservative in paints and stains, and as an algaecide in commercial fish production, residential ponds and aquariums. Products containing diuron are intended for both occupational and residential uses. Occupational uses include agricultural food (such as citrus, berries, asparagus, pineapple, and oranges) and nonfood crops (such as cotton); ornamental trees, flowers, and shrubs; paints and coatings; ornamental fish ponds, and catfish production; rights-of-way and industrial sites. Residential uses include ponds, aquariums, and paints (USEPA 2003b). Diuron is one of the most commonly used pesticides in California. It is often used in rights of way (Moncada 2004).

4.6.7.1 Environmental Fate

The major route of dissipation for diuron in the environment is microbial degradation in water (Table 4-26). Diuron also degrades through photolysis in both water and soil, but at a slower rate. Sorption of diuron to soil is highly correlated with soil organic matter. However, relative to other pesticides diuron is generally considered both mobile and persistent. Diuron has the potential to leach to ground and to contaminate surface waters (USEPA 2003b, Moncada 2004).

Table 4-26 Degradation of Diuron

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	>1,240 Days, Stable	USEPA 2003, Moncada 2004
Photolysis (water)	43 to 2,180 Days	USEPA 2003, Moncada 2004
Photolysis (soil)	173 Days	USEPA 2003, Moncada 2004
Aerobic metabolism (water)	33 Days	USEPA 2003

Table 4-26 Degradation of Diuron

Degradation Method	Half-life	Reference
Aerobic metabolism (soil)	372 Days	USEPA 2003, Moncada 2004
Anaerobic metabolism (water)	5 Days	USEPA 2003
Anaerobic metabolism (soil)	995 Days	USEPA 2003, Moncada 2004
Field dissipation (soil)	73 to 134 Days	USEPA 2003, Moncada 2004

4.6.7.2 Human Toxicity

Diuron has low acute toxicity (Category III or IV) to mammals by the oral, dermal, or inhalation exposure routes. It is not irritating to eyes or skin. Diuron is rapidly absorbed and metabolized by rats and is mostly excreted in the urine (USEPA 2003b). In mammals, metabolism occurs through hydroxylation and dealkylation (Moncada 2004). The oral LD50 for rats is 4,721 mg/kg for males and >5,000 mg/kg for females. The dermal LD50 is >2,000 mg/kg. The inhalation LC50 is >7.1 mg/L. Diuron has been classified as a “known/likely” human carcinogen based on urinary bladder carcinomas in rats. Tumors occurred at doses >600 mg/kg/day (USEPA 2003b) (Table 6.1).

4.6.7.3 Ecological Toxicity

Diuron is slightly to practically nontoxic to birds. The oral LD50 for mallard ducks is >2,000 mg/kg (USEPA 2003b). It is practically nontoxic to honey bees (48-hr LC50 = 145 µg/bee). Diuron is moderately toxic to most aquatic organisms; however, it is highly toxic to cutthroat trout. The 96-hr LC50 for bluegill sunfish is 5.9 mg/L but only 0.71 mg/L for cutthroat (USEPA 2003b). The 48-hr LC50 for *Daphnia magna* is 1.4-mg/L. The bioconcentration factor (BCF) for bentazon predicted from its water solubility indicates low bioaccumulation potential (EXTOXNET 1996b) (Table 6.1).

4.6.7.4 Summary of Toxicity and Potential Effects

Diuron is mobile and persistent in soil. It leaches to groundwater and can contaminate surface waters when transported from the application areas. It is of low toxicity to mammals and birds, practically nontoxic to bees, but moderately toxic to fish and aquatic invertebrates. In spite of these specific toxicity issues, Districts, when using BMP application practices for diuron should not encounter unwanted adverse effects when maintaining adequate buffer zones and care in applications.

4.6.8 Benfluralin (Benefin)

Benfluralin is a pre-emergent dinitroaniline herbicide used to control grasses on commercial and residential turf. Benfluralin also has four food/feed use sites that include lettuce, alfalfa, clover, and birdsfoot trefoil. Other nonfood/nonfeed sites include nonbearing fruit and nut trees, nonbearing berries, nonbearing vineyards, turf, ornamentals, rights of way, fence rows/hedgerows, and Christmas tree plantations. Benfluralin works by inhibiting growth (and acts as a mitotic disruptor) (USEPA 2004d).

4.6.8.1 Environmental Fate

Primary degradation pathways for benfluralin include photolysis and anaerobic metabolism in water (Table 4-27). Benfluralin has low mobility in soils. Benfluralin is of variable soil persistence with different mechanisms of degradation. Benfluralin volatilizes rapidly, but application practices and granular formulations are designed to slow volatilization (USEPA 2004d).

Table 4-27 Degradation of Benfluralin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable	USEPA 2004c
Photolysis (water)	5.5 to 9.9 Hours	USEPA 2004c
Photolysis (soil)	12.5 Days	USEPA 2004c
Aerobic metabolism (soil)	20 to 86 Days	USEPA 2004c
Anaerobic metabolism (water)	38 Hours	USEPA 2004c
Anaerobic metabolism (soil)	12 Days	USEPA 2004c
Field dissipation (soil)	22 to 79 Days	USEPA 2004c

4.6.8.2 Human Toxicity

Benfluralin is classified as practically nontoxic (Category IV) to mammals by acute oral and dermal routes and low toxicity (Category III) for skin and eye irritation. The chemical is toxic to the kidneys, liver, and thyroid in longer-term studies. It has not been assessed for carcinogenicity in humans. Benfluralin is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.6.8.3 Ecological Toxicity

Benfluralin is practically nontoxic to birds on an acute and sub-acute basis (LD50 >2,000 mg/kg), but has been associated with reproductive effects in chronic studies (USEPA 2004d). It is considered practically nontoxic to small mammals (LD50 >10,000 mg/kg) and honey bees (LD50 >10 µg/bee) but highly toxic to freshwater fish (USEPA 2004d). The LC50 for typical end-use product is <100 µg/L for bluegill sunfish (USEPA 2004d). Preliminary toxicity data indicates that benfluralin is highly toxic to estuarine and marine invertebrates (USEPA 2004b). Benfluralin is considered to be bioaccumulative. The BCF for whole fish is 1580 (USEPA 2004d) (Table 6.1).

4.6.8.4 Summary of Toxicity and Potential Effects

Benfluralin has low mobility and variable persistence in soils. It volatilizes rapidly, but application methods are meant to slow volatilization. Benfluralin is practically nontoxic to mammals, birds, and bees on an acute basis. It is highly toxic to fish and aquatic invertebrates and is bioaccumulative. Additionally, benfluralin has been included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a). When benfluralin is applied to water bodies, it generally binds to sediments. It also photodegrades when exposed to sunlight, and does not persist in soil and sediments. Benfluralin does not generally leach into groundwater from soil applications due to its low mobility in soil. Benfluralin when used according to label guidelines and BMP application techniques, should not result in unwanted adverse effects.

4.6.9 <u>Oryzalin</u>

Oryzalin is a selective, preemergent surface-applied herbicide used for control of annual grasses and small-seeded broadleaf weeds. Oryzalin is a dinitroaniline herbicide that controls weeds by disrupting the growth process during seed germination by inhibiting cell division in plants; it does not control established weeds. It is used to control annual grasses, broadleaf weeds, woody shrubs and vines in grapes, berries and orchard crops, including both fruits and nuts. It also is used on residential and commercial/industrial lawns and turf, golf course turf, ornamentals and shade trees, Christmas tree plantations, fencerows/hedgerows,

nonagricultural rights-of-way, and uncultivated areas including patios, paths, paved areas and power stations. Oryzalin is used most on turf, almond orchards and grapes (USEPA 1994d).

4.6.9.1 Environmental Fate

The primary degradation pathways for oryzalin is photolysis (Table 4-28), otherwise oryzalin biodegrades slowly with a half-life of approximately 2 months. Oryzalin is not mobile under field conditions and most of the applied oryzalin either binds to soil or is fully mineralized. Oryzalin is most mobile in coarse, wet, alkaline soils with little organic matter. However, oryzalin would not be stable if it were to leach to groundwater. Anaerobic conditions below the soil surface would cause the chemical reduction of the compound (USEPA 1994d).

Table 4-28 Degradation of Oryzalin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable	USEPA 1994d
Photolysis (water)	1.4 Hours	USEPA 1994d
Photolysis (soil)	3.9 Days	USEPA 1994d
Aerobic metabolism (water)	Moderate	USEPA 1994d
Aerobic metabolism (soil)	2.1 Months	USEPA 1994d
Anaerobic metabolism (water)	Moderate	USEPA 1994d
Field dissipation (soil)	58 to 146 Days	USEPA 1994d

4.6.9.2 Human Toxicity

Formulations include granular, wettable powder, water dispersible granules, emulsifiable concentrate, flowable concentrate, and liquid. In acute toxicity studies using laboratory animals, oryzalin is practically nontoxic by the oral route (USEPA 1994b). It is of moderate dermal and inhalation toxicity and causes slight eye irritation (USEPA 1994b). Oryzalin is generally of moderate acute toxicity, but is carcinogenic in animal studies; therefore, oryzalin has been classified as a possible human carcinogen. The oral LD50 for rats is >10,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The inhalation LC50 is >3.7 mg/L (Table 6.1).

4.6.9.3 Ecological Toxicity

Oryzalin is slightly toxic to practically nontoxic to birds. The oral LD50 for bobwhite quail is 506.7 mg/kg. The dietary LC50 for mallard ducks is >5,000 mg/kg. Oryzalin is moderately toxic to fish and freshwater invertebrates. The 96-hr fish is between 2.88 and 3.26 mg/L. The 48-hr LC50 for *D. magna* is 1.4-mg/L. Oryzalin is practically nontoxic to honey bees. The 48-hr contact LD50 for honey bees is >11 µg/bee (USEPA 2004c). Oryzalin does not accumulate significantly in fish. The BCF is 66.1 in whole bluegill sunfish (USEPA 2004c) (Table 6.1).

4.6.9.4 Summary of Toxicity and Potential Effects

Oryzalin is immobile in soils and is not of concern for ground or surface water contamination (USEPA 2004c). It is practically nontoxic to mammals, birds, and bees. It is moderately toxic to fish but does not accumulate in them. Oryzalin is a possible human carcinogen; however, proper personal protective equipment is thought to be sufficient to protect handlers from the chemical. Based upon the low toxicity and environmental fate of oryzalin**,** and using BMP application practices, these products should not result in unwanted adverse effects.

4.6.10 DCPA

DCPA (or chlorthal dimethyl) is a pre-emergent herbicide used to control annual grasses and broadleaf weeds on ornamental turf and plants, strawberries, seeded and transplanted vegetables, cotton, and field beans. Use practice limitations prohibit applying DCPA directly to water or wetlands (swamps, bogs, marshes, and potholes) or through any type of irrigation system (USEPA 1998d). This herbicide kills germinating seeds by disrupting microtubule formation in exposed cells, causing abnormal cell division.

4.6.10.1 Environmental Fate

DCPA is stable to hydrolysis and photolysis. Biodegradation is the primary dissipation process for DCPA (Table 4-29). Under laboratory conditions, the half-life is approximately 15-30 days, but longer half-lives have been reported in the field. DCPA is not especially persistent or mobile. Volatilization from soil is also a major dissipation route for DCPA. Tetrachloroterephthalic acid (TPA or di-acid) is the only significant DCPA metabolite. TPA is unusually mobile and persistent in the field. Data suggest that TPA will leach to groundwater wherever DCPA is used, regardless of soil properties (USEPA 1998d).

Table 4-29 Degradation of DCPA

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable	USEPA 1998e
Photolysis (water)	Stable	USEPA 1998e
Photolysis (soil)	Stable	USEPA 1998e
Aerobic metabolism (soil)	18 to 37 Days	USEPA 1998e
Anaerobic metabolism (soil)	37 to 59 Days	USEPA 1998e
Field dissipation (soil)	44 to 126 Days	USEPA 1998e

4.6.10.2 Human Toxicity

DCPA has been classified as practically nontoxic for acute-oral toxicity and dermal irritation. DCPA has been classified as slightly toxic for dermal LD50, inhalation LC50, and eye sensitivity. The chemical has been classified as a possible human carcinogen based on increased incidence of thyroid tumors and liver tumors in rats (USEPA 1998b). The oral LD50 for rats is ≥ 5,000 mg/kg. The oral LD50 for beagle dogs is >10,000 mg/kg. The dermal LD50 for rabbits is >2,000 mg/kg. The 4-hr inhalation LC50 for rats is >4.48 mg/L (USEPA 1998d). DCPA is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.6.10.3 Ecological Toxicity

DCPA is more practically nontoxic to birds on an acute basis. The oral LD50 for bobwhite quail is >2,250 mg/kg; however, DCPA is persistent enough to result in chronic exposure to birds (USEPA 1998d). It is practically nontoxic to bees (LD50 >230 µg/bee) (USEPA 1998d). DCPA is slightly toxic to practically nontoxic to fish and aquatic invertebrates. The 96-hr LC50 for rainbow trout is between 30 and >180 mg/L, depending on the study (USEPA 1998d) (Table 6.1).

4.6.10.4 Summary of Toxicity and Potential Effects

DCPA is not mobile in soil and has low persistence; however, the metabolite TPA is unusually mobile and persistent and will leach to groundwater. DCPA is of low acute toxicity to most receptors, but is classified as a possible human carcinogen and a possible endocrine disruptor. Despite the potential for chronic

effects from DCPA exposure, its low persistence and low toxicity and using BMP application practices, DCPA applications should not result in unwanted adverse effects.

4.6.11 Dithiopyr

Dithiopyr is a pre-emergent and post-emergent herbicide used for control of annual grasses and broad leaf weeds in established ornamental turf (USEPA 1991a).

4.6.11.1 Environmental Fate

Dithiopyr degrades slowly in water. Hydrolysis is not a significant route of degradation. Dithiopyr is slightly mobile to relatively immobile in soil. Photodegradation is not a significant route of degradation in soil. Volatization contributed more to dissipation than soil aerobic metabolism. Field dissipation for turf grass had a half-life of 17-61 days (USEPA 1991a).

4.6.11.2 Human Toxicity

Dithiopyr has low acute toxicity to mammals. The oral LD50 and 24-hr dermal LD50 for rats is >5,000 mg/kg. The 4-hr inhalation LC50 for rats is 5.98 mg/L (Ward 1993). The NOELs for systemic and reproductive toxicity in rats are 25 and 2,500 mg/L, respectively (Ward 1993). Dithiopyr is not known to have mutagenic or carcinogenic effects(Table 6.1).

4.6.11.3 Ecological Toxicity

No information is readily available on the potential ecological effects of dithiopyr (University of California Agriculture and Natural Resources 2012).

4.6.11.4 Summary of Toxicity and Potential Effects

Dithiopyr degrades slowly in water and is immobile in water. It is of low acute toxicity to mammals and has not been associated with carcinogenic or mutagenic effects. Little is known about the environmental impacts of dithiopyr use. Due to the lack of reported, documented effects of dithiopyr, and using BMP application practices, proper application of dithiopyr should not result in unwanted adverse effects.

4.6.12 Metolachlor

Metolachlor, a broad-spectrum herbicide, was first registered in 1976 for general weed control in noncrop areas. Since first registered for use on turf, it is now also registered for use on corn, cotton, peanuts, pod crops, potatoes, safflowers, sorghum, soybeans, stone fruits, tree nuts, nonbearing citrus, nonbearing grapes, cabbage, peppers (bell, chili, Cubanelle, tabasco), buffalograss, guymon bermudagrass for seed production, nurseries, hedgerows/fencerows and landscape plantings. Metolachlor's major use sites are corn, soybeans, and sorghum. Metolachlor is a chloracetanilide herbicide that inhibits seedling development (USEPA 1995). When absorbed through the roots and shoots just above the seed of the target weeds, it acts as a growth inhibitor by suppressing synthesis of chlorophyll, proteins, fatty acids and lipids, isoprenoids (including gibberellins), and flavonoids (including anthocyanins) (Rivard 2003).

4.6.12.1 Environmental Fate

Metolachlor degradation appears to be dependent on microbially-mediated and abiotic processes (Table 4-30). Metolachlor is stable to hydrolysis under normal environmental conditions, but subject to photolysis in soils (USEPA 1995). Metolachlor ethane sulfonic acid and metolachlor oxanilic acid are the two most common degradates of metolachlor (Rivard 2003). Metolachlor is moderately persistent and mobile. Extensive leaching can occur in soils with low organic carbon content, and is greatest if soil texture is coarse (Rivard 2003). Substantial amounts of metolachlor could be available for runoff to surface water for several months post-application (USEPA 1995).

Table 4-30 Degradation of Metolachlor

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	Stable, >30 Days	USEPA 1995
Photolysis (water)	70 Days (natural sunlight), 4 Hours (artificial sunlight)	USEPA 1995
Photolysis (soil)	8 Days (natural sunlight) 37 Days (artificial sunlight)	USEPA 1995, Rivard 2003
Aerobic metabolism (water)	47 Days	USEPA 1995
Aerobic metabolism (soil)	26 to 67 Days	USEPA 1995, Rivard 2003
Anaerobic metabolism (water)	78 Days	USEPA 1995
Anaerobic metabolism (soil)	37 to 81 Days	USEPA 1995, Rivard 2003
Field dissipation (soil)	7 to 292 Days	USEPA 1995, Rivard 2003

4.6.12.2 Human Toxicity

The chemical has displayed low-level toxicity in acute tests. It is slightly toxic (Category III) via the oral, dermal, and inhalation routes but is nonirritating to eyes and skin (Category IV) (USEPA 1995). Metolachlor is included in the final list of chemicals for screening under the USEPA Endocrine Disruptor Screening Program (USEPA 2009a) (Table 6.1).

4.6.12.3 Ecological Toxicity

Metolachlor is practically nontoxic to birds on an acute basis. The oral LD50 for mallard ducks is 4,640 mg/kg (USEPA 1995). Technical metolachlor is moderately toxic to freshwater fish. The LC50s for fish range from 3.9 to 10 mg/L. Metolachlor is slightly toxic to aquatic invertebrates (EC50 = 25.1 mg/L) (USEPA 1995). The chemical has low potential to bioaccumulate in fish with a whole fish BCF of 69X and whole body elimination after 14-days depuration (USEPA 1995) (Table 6.1).

4.6.12.4 Summary of Toxicity and Potential Effects

Metalochlor is moderately persistent in soil and mobile, potentially leaching to groundwater. It is slightly toxic to mammals and a potential endocrine disruptor. It is moderately toxic to fish and has low potential for bioaccumulation. Based upon the low toxicity**,** and using BMP application practices, these products should not result in unwanted adverse effects.

4.6.13 Pendimethalin

Pendimethalin is a selective herbicide registered for control of broadleaf weeds and grassy weed species. It is used on various agricultural and nonagricultural sites in crop and noncrop areas. It is applied to soil pre-plant, pre-emergence, and post-emergence with ground and aerial equipment (USEPA 1997b). It is also used in aquatic rice culture and in nonagricultural, residential outdoor weed controls, such as grounds plantings, ornamentals, and turf grass (e.g., residential, golf course, landscape, sod farms) (CDPR 1994). Pendimethalin acts as a microtubule disruptor (USEPA 1997b).

4.6.13.1 Environmental Fate

Pendimethalin dissipates in the environment by binding to soil, microbially-mediated metabolism, and volatilization (Table 4-31). Persistence decreases with increased temperature, increased moisture and decreased soil organic carbon. Pendimethalin residues are tightly bound to soil and sediment particles. Pendimethalin has a low potential to leach to ground water in most soils (USEPA 1997b).

Table 4-31 Degradation of Pendimethalin

Degradation Method	Half-life	Reference
Hydrolysis, pH 5-9	28 to >30 Days	USEPA 1997, DPR 1999b
Hydrolysis (water with soil fungi)	10 to 11 Days	USEPA 1997
Photolysis (water)	16.5 to 60 Days	USEPA 1997, DPR 1999b
Photolysis (soil)	Stable	USEPA 1997
Aerobic metabolism (soil)	42 to 1,322 Days	USEPA 1997
Anaerobic metabolism (water)	6 to 105 Days	USEPA 1997
Anaerobic metabolism (soil)	>60 Days	USEPA 1997
Field dissipation (soil)	34 Days	USEPA 1997

4.6.13.2 Human Toxicity

Pendimethalin has low acute toxicity to mammals. It is listed as Category III for oral toxicity and Category IV for dermal and inhalation exposure. It is nonirritating to skin and slightly irritating to eyes. The oral LD50 for rats is between 1,050 and 1,250 mg/kg. The dermal LD50 for rabbits is >5,000 mg/kg. The inhalation LC50 for rats is >320 mg/L. The LOEL for reproductive effects in rats is 346 mg/kg/day for male rats and 436 mg/kg/day for female rats (USEPA 1997b). Pendimethalin has been classified as a possible human carcinogen because it has caused thyroid follicular cell adenomas in rats (USEPA 1997b) (Table 6.1).

4.6.13.3 Ecological Toxicity

Pendimethalin is slightly acutely toxic to birds. The oral LD50 for mallard ducks is 1,421 mg/kg. Avian chronic toxicity studies have not yet been completed. Pendimethalin is practically nontoxic to honey bees (LD50 >49.7 µg/bee). It is highly toxic to fish and has high potential to bioaccumulate in fish (USEPA 1997b). The LC50 for rainbow trout is 0.138 mg/L (technical pendimethalin) and 0.52 mg/L (formulated pendimethalin product) (USEPA 1997b). Reproductive effects to fish (reduced egg production, reduced hatch success) occur at exposure >6.3 µg/L. Technical pendimethalin is also highly toxic to aquatic invertebrates (LC50 for *D. magna* is 0.28 µg/L). The formulated product is moderately toxic to these organisms (LC50 for *D. magna* is 5.1 µg/L) (Table 6.1).

4.6.13.4 Summary of Toxicity and Potential Effects

Pendimethalin is of varying persistence in soil, depending on temperature and moisture. It is of low toxicity to mammals, birds, and bees. It is toxic to fish and aquatic invertebrates and has a potential to bioaccumulate. Pendimethalin is classified as a possible human carcinogen; however, the USEPA has determined that all uses of pendimethalin (as prescribed) will not cause unreasonable risks to humans or the environment (USEPA 1997b). Based upon this evaluation and the USEPA literature, using BMP application practices with these products should not result in unwanted adverse effects.

4.7 Adjuvants

An adjuvant is any compound that is added to an herbicide formulation or tank mix to facilitate the mixing, application, or effectiveness of that herbicide. Adjuvants can either enhance activity of an herbicide's active ingredient (activator adjuvant) or offset any problems associated with spray application, such as adverse water quality or wind (special purpose or utility modifiers). Activator adjuvants include surfactants, wetting agents, sticker-spreaders, and penetrants. Nonionic alkylphenol ethoxylate-based and silicone-based adjuvants are wetter/spreaders. Adjuvants can also be primarily oil-based. Oil

additives function to increase herbicide absorption through plant tissues and increase spray retention. Oil adjuvants are made up of either petroleum, vegetable, or methylated vegetable or seed oils plus an emulsifier for dispersion in water (Bakke 2007).

Adjuvants are not under the same registration guidelines as are pesticides. EPA regulates the inclusion of certain ingredients in adjuvant formulations, but it does not stringently test and regulate the manufacture and use of adjuvant products (as they do for pesticides). As such, there is little information on the effects of different adjuvants, other than that provided by the manufacturer or published by the scientific community (Tu et al. 2001, Bakke 2007). DPR does require the registration of adjuvants that are considered to increase the action of the pesticide it is used with (Bakke 2007).

The long-term fates of most adjuvants in soils and elsewhere in the environment are largely unknown, partially because of the lack of long-term monitoring data, but also because the ingredients in most adjuvants are not disclosed (Tu et al. 2001).

4.7.1 Alkylphenol Ethoxylate

Alkylphenol ethoxylates (APEs) can be used as detergents, wetting agents, dispersants, emulsifiers, solubilizers and foaming agents. Alkylphenol ethoxylates are widely in domestic detergents, pesticide formulations and industrial products. Industrial applications include pulp and paper, textiles, coatings, agricultural pesticides, lube oils and fuels, metals and plastics. Primary degradation of alkylphenol ethoxylates in the environment generates more persistent shorter chain alkylphenol ethoxylates and alkylphenols (i.e., nonylphenol, octylphenol, and mono- to triethoxylates), some of which may mimic natural hormones and disrupt endocrine function in wildlife and humans (Ying et al. 2002).

4.7.1.1 Environmental Fate

Alkylphenol ethoxylates degraded faster in the water column than in sediment. Alkylphenol ethoxylates bind strongly to aquatic particles in river and coastal environments and are persistent in sediments. Aerobic conditions further facilitate biotransformation of alkylphenol ethoxylate metabolites as compared to anaerobic conditions (Ying et al. 2002).

4.7.1.2 Human Toxicity

Nonylphenol (NP) is of low acute oral and dermal toxicity but is highly irritation and corrosive to the skin and eyes (USEPA 2010b). Concern exists regarding the estrogen-mimicking behaviors of alkyl phenol ethoxylate (USEPA 2010b). The compounds nonylphenol (NP) and nonylphenol ethoxylate (NPE) are of particular interest and concern to the public and the EPA. NPs and NPEs are produced in large volumes and are widely used (Table 6.1).

4.7.1.3 Ecological Toxicity

NP is persistent in the environment, moderately bioaccumulative, and extremely toxic to aquatic organisms. NPE, though less toxic than NP, is also highly toxic to fish, aquatic invertebrates, and aquatic plants (USEPA 2010b). Toxicity of APEs to aquatic organisms increases with alkyl chain length. The toxicity of a variety of APEs are discussed in detail by Bakke (2003) (Table 6.1).

4.7.1.4 Summary of Toxicity and Potential Effects

APEs include a broad range of chemicals that tend to bind strongly to particulates and persist in sediments. Nonylphenol and short-chain NPEs are moderately bioaccumulative and extremely toxic to aquatic organisms. Aside from use in agricultural herbicide mixtures, APEs are commonly present in detergents, cleaners, food packaging, and cosmetics. The acute toxicity of APEs to mammals is low. They are possible estrogen-mimics. Nonylphenol has been detected in human breast milk, umbilical cord blood, and urine (USEPA 2010b).

The USEPA (USEPA 2010b) has recently recommended that this suite of chemicals be evaluated further due to their wide-spread use (past and present), persistence, and possible estrogen-mimicking behavior.

4.7.2 Polydimethylsiloxane Fluids

Most polydimethylsiloxane fluids are nonvolatile polymeric organosilicon materials consisting of ([CH3]2SiO) structural units. Various polydimethylsiloxane fluids ranging from low to high viscosity are used in a wide range of industrial applications, such as manufacturing textiles, paper, and leather goods and serve as antifoams, softeners, or water repellents. In consumer applications, polydimethylsiloxane fluids can be found in personal-, household- and automotive care products. They are used as softeners in skin care products, conditioners in hair care, additives in polish formulations, and as waterproofers and other surface treatments. Some polydimethylsiloxane materials are also sold as end products (usually in the industrial market), such as transformer dielectric fluids and heat transfer liquids (Dow Corning Corp. 1998).

4.7.2.1 Environmental Fate

Polydimethylsiloxane fluids are insoluble in water and have a high adsorption coefficient. Volatile, low molecular weight dimethyl siloxanes will evaporate into the atmosphere where they undergo indirect photolytic degradation. However, high molecular weight polydimethylsiloxanes typically sorb to particulate matter when in water and become associated with soil and sediments (Griessbach and Lehmann 1999).

Polydimethylsiloxanes degrade into lower molecular weight siloxanols and finally into dimethylsilanediol. Significant degradation to lower molecular weight compounds have been noted after a few weeks' soil contact. The actual rate and extent of degradation vary as a function of soil moisture content and clay type (Dow Corning Corp. 1998). The degradation rate of polydimethylsiloxanes is highly influenced by soil moisture. Degradation is slow on moist soils (3 percent within 6 months) but quite rapid on dry soil (50 percent within several days) (Griessbach and Lehmann 1999).

4.7.2.2 General Toxicity

PMDS appears to be relatively nontoxic to benthic invertebrates and exhibits little bioaccumulation potential (Henry et al. 2001) (Table 6.1).

4.7.2.3 Summary of Toxicity and Potential Effects

Polydimethylsiloxanes are insoluble in water and typically sorb to particulates. Degradation time varies depending on moisture in soils. These chemicals appear to be relatively nontoxic to most organisms, but data is lacking. Although there is a paucity of information regarding the toxicity and environmental fate of polydimethylsiloxanes, using BMP application practices, these products should not result in unwanted adverse effects.

4.7.3 Modified Vegetable Oil and Methylated Seed Oil

Vegetable-derived oils (from soybeans, cottonseeds, etc.) decrease surface tension, but they are not as effective as other surfactants at increasing spreading, sticking, or penetration. Vegetable oils are generally of two types: triglycerides or methylated oils. Triglycerides are essentially oil-surfactant hybrids, and are generally called "seed oils." These seed soils are extracted from plants by pressing or solvent extraction, and tend to have higher viscosities than methylated oils. Methylated seed oils are better solvents than petroleum-based oils. Triglyceride oils usually contain only 5 to 7 percent surfactant emulsifier, while methylated seed oils contain 10 to 20 percent surfactant (Tu et al. 2001). Oil adjuvants can increase the penetration of oil-soluble herbicides into plants. These adjuvants are commonly used in hot, dry conditions (Tu et al. 2001). Attachment of the methanol to the oil alters the hydrophilic/lipophilic balance of the oil to an optimum level (Hartzler 2001).

4.7.3.1 General Toxicity

Adjuvants are generally considered inert or essentially nonphytotoxic (Tu et al. 2001).Toxicity information is available for the product, Competitor™ (modified vegetable oil, polyethylene glycol fatty acid ester, polyoxyethylene sorbitan fatty acid ester) (Washington State Department of Agriculture 2009). The 96-hr LC50 for rainbow trout is 95 mg/L (slightly toxic). The 48-hr EC50 for daphnids is >100 mg/L (practically nontoxic) (Table 6.1).

4.7.3.2 Summary of Toxicity and Potential Effects

Modified vegetable oils and methylated seed oils are essentially nontoxic to most organisms, including plants. Little is known of the environmental fate of these adjuvants. Although there is paucity of toxicity and environmental fate information for these oils, using BMP application practices, these products should not result in unwanted adverse effects.

4.7.4 Lecithin

Lecithin (phosphatidylcholine) is a commonly used amphoteric surfactant, which is derived from soybeans. Amphoteric surfactants contain both a positive and negative charge and typically function similarly to nonionic surfactants. There is little published research on the use and efficacy of amphoteric surfactants (Tu et al. 2001).

4.7.4.1 General Toxicity

Lecithin is a general term used to describe yellow-brownish fatty substances occurring in animal and plant tissues. When used with herbicide applications, lecithin acts as an amphoteric surfactant and functions similarly to nonionic surfactants (Tu et al. 2001). Toxicity information exists for the product, Liberate™ (lecithin, alcohol ethoxylate, modified vegetable oil) (Washington State Department of Agriculture 2009). The 96-hr LC50 for rainbow trout is 17.6 mg/L (slightly toxic) and the 48-hr LC50 for daphnids is 9.3 mg/L (moderately toxic). Little is known about the fate of lecithins in the environment or their effect on non-target organisms (Tu et al. 2001) (Table 6.1).

4.7.4.2 Summary of Toxicity and Potential Effects

Little is known about the toxicity or environmental fate of lecithins. Lecithins are naturally occurring phospholipids in biological cell membranes (Bakke 2007). Although there is a paucity of information on these products, using BMP application practices, use of these products should not result in unwanted adverse effects.

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5 Evaluations of Active Ingredients

While the majority of the active ingredients reviewed do not suggest an unacceptable risk when used properly, some became the focus of additional discussion concerning actual use patterns and BMPs employed. The results of the assessments are based on preliminary assumptions about toxicity and mode of action derived from available information and data, published by the manufacturers, researchers, and other published literature.

Using the available information about the active ingredients reviewed, there were several overarching parameters that are known to adversely impact risk. Primary factors considered include the inherent toxicity and mode of action of the chemical that can imply toxicity to non-target species. Other important factors that are considered include the possible transport and fate of the chemical in various media, the reported likely exposure routes, and documented ecological and human studies supporting the toxicity data. Several important parameters, such as the retention time (half-life) in various media are considered, but are based only on available information about regional conditions. Several pesticides received additional discussion during the MVCAC workshop on February 20, 2013.

Using the approach discussed above, select active ingredients were identified (Table 5-1) and discussed during the workshop to supplement the information relevant to the evaluation of potential risk. Each of these pesticides exhibits at least one parameter that appears to drive potential risk.

Table 5-1 Active Ingredients Identified for Discussion

Active Ingredient	Vector	Potential Issue
Methoprene	Mosquitoes	Prevalent use; toxicity to aquatics and insects
Etofenprox	Mosquitoes	Toxicity to aquatic organisms; no synergist required
Bti	Mosquitoes	Prevalent use; public concerns
Pyrethrins	Mosquitoes	Prevalent use; requires synergist (PBO)
Resmethrin	Mosquitoes	Requires synergist (e.g., PBO); potential endocrine disruptor
Vegetable Oil (coconut oil)/mix	Mosquitoes	Contains low percentage of petroleum distillate
Permethrin	Mosquitoes/ yellow jacket wasps	Toxicity to aquatic organisms; potential endocrine disruptor
Lambda-cyhalothrin	Yellow jacket wasp	Toxicity to aquatic organisms; potential to bioaccumulate
Bromadiolone	Rats	Toxicity to non-target organisms including mammals, birds, aquatics
Difethialone	Rats	Toxicity to non-target organisms including mammals, birds, aquatics
Alkylphenol ethoxylates	Weeds	Toxicity to aquatic organisms; Moderately bioaccumulative
Glyphosate	Weeds	Prevalent use; possible endocrine disruptor
Diuron	Weeds	Prevalent use; toxicity to freshwater fish
Benfluralin	Weeds	Toxicity to aquatics; potential for bioaccumulation/endocrine disruption

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6 Toxicity Summary: All Active Ingredients

Toxicity information gathered from the published literature and regulatory sources is included in Table 6.1 below. The table includes information such as LD50 LC50s USEPA toxicity rating and other relevant toxicity information.

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Table 6-1 Toxicity Values Reported in the Literature for Active Ingredients

Active Ingredient	Mammalian Oral LD50 (mg/kg)[A]	Mammalian Dermal LD50 (mg/kg)[B]	Mammalian Inhalation LC50 (mg/L)[A]	USEPA Tox Rating	Avian LD50 (mg/kg)[C]	Fish LC50 (mg/L)[D]	Aquatic Invert EC50 (µg/L)[E]	Honeybee LD50 (µg/bee)	Other Receptors
Pyrethrins	1400	>2,000	3400	oral and dermal (III), inhalation (IV)	>5,620	0.0051	11.6	no data, likely toxic	no data
Allethrins and *d-trans* allethrin	685 (allethrin, F) 860 (d-trans)	11,332 (allethrin)	no data	no data	>2,000 (allethrin) >5,620 (d-trans)	0.0026 to 0.08	no data	3 to 9	Dog dietary NOEL = 50 mg/kg/Day for 2 years.
Phenothrin (sumithrin or d-phenothrin)	>5,000 (no deaths)	>2,000 (no deaths)	>2.1 (no deaths)	oral and inhalation (IV), dermal (III)	>5,000	0.0158	4.4	no data, likely toxic	no data
Prallethrin	460	>5,000	0.66	no data	1,171	0.012	6.2	0.028	no data
Deltamethrin	>5,000	>2,000	no data	no data	no data	no data	no data	no data	no data
Esfenvalerate	458	2,000	2.93		9,932	0.00026	0.00024	highly toxic	no data
Lambda-cyhalothrin	56 to 144	632 to 696 (rats)	no data	no data	>3,950	0.0021	0.36	0.97	no data
Resmethrin	4,639	>2,000	5.28	no data	75 (blackbird)	.00028	0.063	0.963	no data
Tetramethrin	>5,000	>2,000	no data	oral (III), dermal (IV)	>2,250	0.0037	45	0.155	no data
Permethrin	2280	>2,000	no data	oral and dermal (III), inhalation (IV)	>10,000	0.00079	0.1 (mayfly)	0.13	toxic to cats via dermal route
Etofenprox	>2,000	>2,100	>5.9	no data	>2,000	0.0027	no data	0.27 (oral), 0.13 (contact)	Dog oral LD50 >5,000 mg/kg
Piperonyl butoxide (PBO)	4,570	>2,000	>5.9	oral and dermal (III), inhalation (IV)	>2,250	1.9	510	>25	Tadpole LC50 = 0.21 mg/L
Naled	81 to 85	354	0.19	all acute (II)	52	.087 (lake trout), 2.2 (bluegill)	0.3	0.48	no data
Temephos	444	970	1.3	oral and dermal (II), inhalation (III)	no data	3.49	10	no data	no data
Bacillus sphaericus (Bs)	no data	no data	no data	no data	no data	>15.5	15,500	no data	no data
Bacillus thuringiensis (Bti)	>4,7x10^{11} spores/kg	>2.67x10^{11} spores/kg	4,7x10^{11} spores/kg	all acute (IV)	no data	no data	50,000 (10 Days)	no data	no data
Spinosad	>5,000	>2,800	>5.18	no data	>2,000	5.9	92,700	11,500	Butterfly/moth LD50 = 0.022 mg/kg. No effect on amphibians.
Methoprene and s-Methoprene	>10,000	>2,000	>210	oral and inhalation (IV), dermal (III)	>2,000	>50	89	no data	Frog LC50 >10,000 µg/L
Alcohol Ethoxylated Surfactant (monomolecular film)	no data	no data	no data	no data	no data	No observable effects	No observable effects to shrimp, snails, worms, or mayfly naiads	no data	No observable effects to amphibians.
Aliphatic solvents (mineral oils, aliphatic hydrocarbons, petroleum distillates)	>28,000 (no deaths observed)	>5,000	3.9	no data	>2,250	no effect	<900	no effect	no data
Potassium Salts (soap salts)	no data	no data	no data	all acute effects (IV)	no data	no data	no data	no data	no data
Chlorophacinone	3.15 (rats)	0.329	.007	all acute effects (II)	258	0.45	640	no data	Carnivorous mammals LD50 = 2.1 to 50 mg/kg

Table 6-1 Toxicity Values Reported in the Literature for Active Ingredients

Active Ingredient	Mammalian Oral LD50 (mg/kg)[A]	Mammalian Dermal LD50 (mg/kg)[B]	Mammalian Inhalation LC50 (mg/L)[A]	USEPA Tox Rating	Avian LD50 (mg/kg)[C]	Fish LC50 (mg/L)[D]	Aquatic Invert EC50 (µg/L)[E]	Honeybee LD50 (µg/bee)	Other Receptors
Diphacinone	2.3 to 7.0	3.6	<0.0006	all acute effects (I)	400 to 2000	2.6	1800	no data	Dog oral LD50 = 7.5 mg/kg
Brodifacoum	0.418 to 0.561	3.16 to 5.21	0.00305 to 0.00486	all acute effects (I)	0.26	0.015	980	no risk	Carnivorous mammals LD50 = 0.27 to 25 mg/kg
Bromadiolone	0.56 to 0.84	1.71	0.00043	all acute effects (I)	138 to 170	0.24	240 to 2000	no data	Carnivorous mammals LD50 = 1.125 to 25 mg/kg
Bromethalin	9.1 to 10.7	2,000	0.024	oral and inhalation (I), dermal (II)	4.6 to 11	0.038 to 0.598	2.0	no data	Dog oral LD50 2.38 to 5.6 mg/kg bw. Cat oral LD50 0.54-mg/kg bw
Difethialone	0.55	6.5 (rats)	.005	no data	0.264	0.051	4.4	no data	Dog oral LD50 = 11.8 mg/kg bw. Cat oral LD50 ≥16 mg/kg bw
Cholecalciferol (vitamin D)	43.6	2,000 (finished bait)	no data	no data	2,000	no data	no data	no data	Dog oral LD50 = 88 mg/kg
Sulfur (fumigant)	>5,000	>2,000	>2.56	no data	>5,620	>180	>5,000,000	nontoxic	no data
Sodium Nitrate (fumigant)	3,700	<2,000	no data	oral (III)	no data	no data	no data	no data	Any non-targets in burrow susceptible.
Imazapyr	>5,000	>2,000	>1.3	no data	>2,150	>100	>1,000,000	>100	no data
Glyphosate	4,320 (technical) ≥5,000 (salt forms)	≥2,000 (tech) ≥5,000 (salts)	≥4.43 (tech) >1.3 (salts)	oral and dermal (III)	>2,000	>24	55,000 to 780,000	>100 (practically nontoxic)	No acute toxicity to frogs.
Triclopyr	630	>2000	>2.6	oral and dermal (III)	1,698 (technical)	117 (technical) 0.36 (TBEE)	132,000	60.4	no data
2,4-D (2,4-dichlorophenoxy acetic acid)	639 to 1,646	1,829 to 2,000	0.78 to >5.4	no data	472 (pheasant)	1 to 100 (cutthroat trout)	132,000	104-to 115	no data
Sulfometuron methyl	>5,000	>2,000	>5.0	oral and inhalation (IV), dermal (III)	>4,650	>100	>100,000	>100	EC25 values available for many non-target plants
Bentazon	1,100	>2500	no data	all acute (III)	1171	practically nontoxic	practically nontoxic	>100	no data
Diuron	4,721	>2,000	>7.1	all acute (III or IV)	>2,000	0.71 (cutthroat trout) 5.9 (bluegill)	1,400	145	no data
Benfluralin (benefin)	>10,000 (small mammals)	>5000	>2.3	all acute (IV)	>2,000 (sub-acute)	<0.1	2180	>10 (practically nontoxic)	no data
Oryzalin	>10,000	>2,000	>3.7	oral (IV), dermal and inhalation (III)	506.7	2.88 to 3.26	1,400	>11 (practically nontoxic)	no data
DCPA (chlorthal dimethyl) [metabolite is tetrachloroterephthalic acid (TPA)]	>5,000	>2,000	>4.48	dermal and inhalation (III), oral (IV)	>2,250	30 to >180	practically nontoxic	>230 (practically nontoxic)	Dog oral LD50 = 10,000 mg/kg

Table 6-1 Toxicity Values Reported in the Literature for Active Ingredients

Active Ingredient	Mammalian Oral LD50 (mg/kg)[A]	Mammalian Dermal LD50 (mg/kg)[B]	Mammalian Inhalation LC50 (mg/L)[A]	USEPA Tox Rating	Avian LD50 (mg/kg)[C]	Fish LC50 (mg/L)[D]	Aquatic Invert EC50 (µg/L)[E]	Honeybee LD50 (µg/bee)	Other Receptors
Dithiopyr	>5,000	>5,000	5.98	no data	no data	no data	no data	no data	no data
Metalochlor	2,780	10,000	1.75	all acute (III)	4,640	3.9 to 10	25,100	no data	no data
Pendimethalin	1,050 to 1,250	>5,000	>320	oral (III), dermal and inhalation (IV)	1,421	0.138 (technical) 0.58 (formulated product)	0.28 (technical) 5.1 (product)	>49.7 (practically nontoxic)	no data
Alkylphenol ethoxylate (APE)	600 to >10,000	>0.22	>2,000	no data	no data	1.5 to 6.4-(differs by chain length)	460 to 740, depending on which AE	no data	No effects on frogs
Polydimethylsiloxane Fluids	no data	no data	no data	no data	no data	no data	no data	no data	Relatively nontoxic to benthic invertebrates.
Modified Vegetable Oils and Methylated Seed Oil	no data	no data	no data	no data	no data	95 (Competitor™)	>100 (Competitor™)	no data	no data
Lecithin	no data	no data	no data	no data	no data	17.6 (Liberate™)	9.3 (Liberate™)	no data	no data

A. Unless otherwise specified, values are for rats.
B. Unless otherwise specified, values are for rabbits.
C. Unless otherwise specified, values are for mallard duck or bobwhite quail.
D. Unless otherwise specified, values are for rainbow trout or bluegill sunfish.
E. Values are for *Daphnia* or similar species.

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ATTACHMENT

A

CHEMICAL USE DATA SUBMITTED
BY DISTRICTS

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Table A35. Pesticide Product Key – SCCVCD