RISK ASSESSMENT OF PESTICIDES AND FERTILIZERS PROPOSED FOR USE AT PROVOLT SEED ORCHARD

Grants Pass (Josephine County), OR
Medford District, U.S. Bureau of Land Management

Prepared for
U.S. Bureau of Land Management
Oregon State Office

by

LABAT-ANDERSON INCORPORATED
Contract HAC002016

March 18, 2002

TABLE OF CONTENTS

TABLES

FIGURES

1.0 INTRODUCTION

A glossary of terms and abbreviations is located in Section 10 of this document.

The purpose of this assessment is to analyze the risks to human health and non-target species from using pesticides and fertilizers at the Provolt Seed Orchard (Provolt) in Grants Pass, OR, located in the U.S. Bureau of Land Management (BLM) Medford District. Provolt proposes to use insecticides, a fungicide, and herbicides to control weeds, insect pests, and diseases in orchards and other managed areas of the grounds; and fertilizers to optimize seed production in the orchards. This assessment describes the methods for analyzing hazards, exposures, and risks from the pesticides and fertilizers proposed for use at the seed orchard, and presents the estimated risks to human health and non-target species for each chemical. The following chemicals are examined in this risk assessment:

Insecticides
- acephate
- chlorpyrifos
- diazinon
- dimethoate
- esfenvalerate
- horticultural oil
- permethrin
- propargite (miticide)

Fungicide
- chlorothalonil

Herbicides
- dicamba
- glyphosate
- hexazinone
- picloram
- triclopyr

Fertilizers
- ammonium sulfate
- ammonium phosphate
- ammonium nitrate
- potassium nitrate

Pesticides are not currently in use at Provolt, and have not been used in recent years. The full range of potential pest management issues was considered in selecting the pesticides to be included in the proposed program, so that these options will be available to the seed orchard manager if the need arises. The potential applications include many alternative pesticides and application methods to give the seed orchard manager flexibility in selectively and appropriately addressing observed pest management problems as they occur. Some of the proposed pesticides or application methods may be implemented only rarely, if ever.

Fertilizers are currently in use at Provolt. Future applications of fertilizers are anticipated to be unchanged from the current program, and are described in Section 2.0 of this risk assessment.

In addition to the active ingredients in a pesticide formulation, there are other ingredients, formerly referred to as “inert” ingredients. The U.S. Environmental Protection Agency (EPA) has classified these other ingredients into four categories, based on the degree of toxicity posed by the chemical, as follows (EPA 2000a) :

- List 1: Inerts of toxicological concern.
- List 2: Potentially toxic inerts, with high priority for testing
- List 3: Inerts of unknown toxicity
- List 4: Inerts of minimal concern

To include consideration of potential risks from these chemicals, any other ingredients in the proposed pesticide formulations that appear on either List 1 or List 2 are included in this quantitative risk assessment, along with the active ingredient in the formulation. Accordingly, the following other ingredients are included in the human health and non-target species risk assessments:

- Cyclohexanone: present in Digon® 400 formulation of dimethoate.

- Ethylbenzene: present in the Asana® XL formulation of esfenvalerate and the Pounce® 3.2 EC formulation of permethrin.

- Light aromatic solvent naphtha: present in the Pounce® 3.2 EC formulation of permethrin.

- Petroleum distillates: present in the Digon® 400 formulation of dimethoate.

- Xylene: present in the Asana® XL formulation of esfenvalerate and the Pounce® 3.2 EC formulation of permethrin.

1.1 Organization of this Report

This risk assessment report is organized into ten sections, as follows:

- Section 1 presents the purpose, describes the structure, and outlines the methodology of the risk assessment.

- Section 2 describes the proposed pesticide and fertilizer usage at the seed orchard. This includes pesticide application rates and schedules, types of application equipment, and other relevant factors specific to the applications to be considered in this risk assessment.

- Section 3 covers the environmental fate and transport modeling of the chemicals. The modeling was used to estimate potential concentrations in surface water and leachate. The results of the modeling were used in the exposure analysis for both the human health and non-target species risk assessments.

- Section 4, the human health hazard assessment, summarizes and discusses the toxic properties of each chemical.

- Section 5, the human health exposure assessment, describes the methods used to estimate levels of exposure and resulting doses to the public and workers.

- Section 6, the human health risk characterization, uses the results of the hazard and exposure assessments to draw inferences about human health risks (including cancer risks), based on estimated daily and lifetime doses to the public and workers.

- Section 7 describes the results of the problem formulation for the non-target species risk assessment, which identifies the ways that pesticide use at Provolt may result in risks to non-target species, and the non-target species and ecosystems potentially affected.

- Section 8, the non-target species analysis section, characterizes the exposures and possible types of effects to terrestrial and aquatic wildlife species.

- Section 9, the non-target species risk characterization, estimates and describes the risks based on evaluation of the data described in Section Eight.

- Section 10 presents a glossary of technical terms for reader reference.

1.2 Overview of the Human Health Risk Assessment

To assess the risk of human health effects from using pesticides and fertilizers at Provolt, it was necessary to estimate the human exposures that could occur as a result of the proposed applications and associated activities, and to estimate the probability and extent of adverse health effects that could occur as a result of those exposures. This risk assessment employs the three principal analytical elements that the National Research Council (1983) described and EPA (1989, 2000b) affirmed as necessary for characterizing the potential adverse health effects of human exposures to existing or introduced hazards in the environment: hazard assessment, exposure assessment, and risk characterization.

Hazard assessment requires gathering information to determine the toxic properties of each chemical and its dose-response relationship. Human hazard levels are derived primarily from the results of laboratory studies on animals. The goal of the hazard assessment is to identify acceptable doses for noncarcinogens, and identify the cancer potency of potential carcinogens.

Exposure assessment involves estimating doses to persons potentially exposed to the pesticides or fertilizers. In the exposure assessment, dose estimates were made for typical, maximum, and accidental exposures. These exposures are defined as follows:

- *Typical:* Typical exposure reflects the average dose an individual may receive if all exposure conditions are met. Typical exposure assumptions include the application rate usually used at the seed orchard, usual number of applications per year, and other similar assumptions.

- *Maximum:* Maximum exposure defines the upper bound of credible doses that an individual may receive if all exposure conditions are met. Maximum exposure assumptions include the maximum application rate according to the label, maximum number of applications per year, and other similar assumptions.

- *Accidental:* The possibility of error exists with all human activities. Therefore, it is possible that during seed orchard operations, accidents could expose individuals to unusually high levels of pesticides or fertilizers. To examine these potential health effects, several accident scenarios were evaluated for health effects to members of the public and workers.

It is important to note that these exposure scenarios estimate risks from clearly defined types of exposure. If all the assumptions in an exposure scenario are not met, the dose will differ from that estimated here, or may not occur at all.

Risk characterization requires comparing the hazard information with the dose estimates to predict the potential for health effects to individuals under the conditions of exposure. The risk characterization also identifies uncertainties (such as data gaps where scientific studies are unavailable) that may affect the magnitude of the estimated risks.

1.3 Overview of the Non-Target Species Risk Assessment

The non-target species risk assessment follows the steps of problem formulation, analysis, and risk characterization, as described in the U.S. Environmental Protection Agency's Guidelines for Ecological Risk Assessment (EPA 1998). This risk assessment also identifies uncertainties that are associated with the conclusions of the risk characterization. The discussion that follows briefly describes these elements. A detailed description of ecological risk assessment methodology is contained in EPA (1998).

In *problem formulation*, the purpose of the assessment is provided, the problem is defined, and a plan for analyzing and characterizing risk is determined. The potential stressors (in this case, pesticides and fertilizers), the ecological effects expected or observed, the receptors, and ecosystem(s) potentially affected are identified and characterized. Using this information, the three products of problem formulation are developed: (1) assessment endpoints that adequately reflect management goals and the ecosystem they represent, (2) conceptual models that describe key relationships between a stressor and assessment endpoint, and (3) an analysis plan that includes the design of the assessment, data needs, measures that will be used to evaluate risk hypotheses, and methods for conducting the analysis phase of the assessment.

Analysis is a process that examines the two primary components of risk–exposure and effects–and the relationships between each other and ecosystem characteristics. The assessment endpoints and conceptual models developed during problem formulation provide the focus and structure for the analysis. Exposure characterization describes potential or actual contact or co-occurrence of stressors with receptors, to produce a summary exposure profile that identifies the receptor, describes the exposure pathway, and describes the intensity and extent of contact or co-occurrence. Ecological effects characterization consists of evaluating ecological effects (e.g., ecotoxicity) data on the stressor of interest, as related to the assessment endpoints and the conceptual models, and preparing a stressor-response profile.

Risk characterization uses the results of the analysis phase to develop an estimate of the risks to ecological entities, describes the significance and likelihood of any predicted adverse effects, and identifies uncertainties, assumptions, and qualifiers in the risk assessment.

1.4 References

EPA. See U.S. Environmental Protection Agency.

National Research Council. 1983. *Risk Assessment in the Federal Government: Managing the Process.* National Academy Press. Washington, DC.

U.S. Environmental Protection Agency. 1989. Risk assessment guidance for Superfund—Volume 1: Human health evaluation manual (part A). Interim final. EPA/540/1-89/002. Office of Emergency and Remedial Response. Washington, DC.

U.S. Environmental Protection Agency. 1998. Guidelines for ecological risk assessment. Risk Assessment Forum. Washington, DC.

U.S. Environmental Protection Agency. 2000a. Lists of other (inert) pesticide ingredients. Office of Pesticide Programs. Washington, DC. http://www.epa.gov/opprd001/inerts/lists.html

U.S. Environmental Protection Agency. 2000b. Risk characterization handbook. EPA 100-B-00-002. Science Policy Council. Washington, DC.

2.0 PROGRAM DESCRIPTION

This section describes the chemical pest management, fertilization program, and risk evaluation approach for Provolt. The following sections provide a description of the chemical pesticide application methods; application rates, timing, and potentially treated areas; and health and environmental protection measures.

2.1 Application Methods

Pesticides may be applied using several methods. For some pesticides, different combinations of pesticide and application method are being proposed, to give the seed orchard flexibility in addressing the specific management needs that may occur, including:

- high-pressure hydraulic sprayer
- hydraulic sprayer with hand-held wand
- tractor-pulled spray rig with small boom
- backpack sprayer
- hand-held wick
- capsule implantation
- broadcast spreader

Only ground-based application methods are being proposed; aerial application is not part of this pest management program. Each method is described briefly in the following paragraphs.

2.1.1 High-Pressure Hydraulic Sprayer

High-pressure hydraulic sprayers consist of a powered pump and tank carried by truck or tractor, and hand-held nozzles for dispersing the solution upward into the tree. These sprayers could be used to treat individual mature trees with the insecticides chlorpyrifos, diazinon, esfenvalerate, horticultural oil, permethrin, or propargite; or with the fungicide chlorothalonil.

2.1.2 Hydraulic Sprayer with Hand-Held Wand

A spray tank is mounted on a truck, tractor, or all-terrain vehicle, and may be used to apply herbicides around trees in orchard units, along fencelines, and as a spot treatment in fallow fields, orchard units, and administrative areas. The sprayer may be operated by one worker, who drives and stops to spray; or by two workers, with one driving and the other spraying. This method may be used to apply the insecticides chlorpyrifos, diazinon, dimethoate, esfenvalerate, permethrin, or propargite; the fungicide chlorothalonil; or the herbicides dicamba, glyphosate, hexazinone, picloram, or triclopyr.

2.1.3 Tractor-Pulled Spray Rig with Boom

This method may be used to apply herbicides for control of weeds in orchard units, in roadways, or in fallow areas. Equipment consists of a hydraulic spray tank pulled by a tractor or heavy-duty pickup truck, with a spray boom attached to the tank to release the herbicide. At Provolt, this method may be used to apply the herbicides dicamba or glyphosate.

2.1.4 Backpack Sprayer

A backpack sprayer consists of a plastic tank containing the pesticide that is strapped to the applicator's back. A hand-operated hydraulic pump forces the liquid from the tank through a nozzle in a hand-held wand. At Provolt, a backpack sprayer could be used to apply the insecticides dimethoate, esfenvalerate, permethrin, or propargite; or the herbicides dicamba, glyphosate, hexazinone, picloram, or triclopyr for treatment of unwanted vegetation in orchard units and along fencelines.

2.1.5 Hand-Held Wick

A hand-held wick consists of a stick containing diluted herbicide in contact with an absorbent material (a rope or wiper pad), which is then wiped directly on the foliage of target vegetation. At Provolt, this method may be used to apply dicamba or glyphosate for spot treatment of weeds.

2.1.6 Capsule Implantation

The insecticide acephate may be implanted into individual trees for long-term control of insect pests in the form of a capsule. One small hole is drilled into a tree for every 4 inches of its diameter at breast height (DBH), and a capsule is inserted.

2.1.7 Broadcast Spreader

Fertilizers may be distributed over the ground using a spreader pulled by a truck, or mounted on a tractor o r ATV.

2.2 Application Rates, Timing, and Potential Treated Areas

Table 2-1 summarizes the details of possible pesticide applications that may be made at Provolt. At Provolt, pesticides will not be used on a planned schedule, but only as needed to control insect pests, weeds, and disease. The timing and frequencies listed in the table indicate what could be expected if control using that particular pesticide was indicated by observed seed orchard conditions.

Table 2-1. Pesticide and Fertilizer Application Summary

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Insecticides				
Acephate: Acecap® 97 (97% a.i. in an implant capsule)				
Implants	Individual trees in any orchard unit	1 capsule/4 inches circumference 1 application to 100 trees on Apr 15	1 capsule/4 inches circumference 1 application to 300 trees on Apr 15	Mar - Apr
Chlorpyrifos: Dursban 50W (50% a.i. as a wettable powder in water-soluble packets)				
High-pressure hydraulic sprayer -or- Hydraulic sprayer with hand-held wand	Individual trees in any orchard unit	1 lb a.i./acre, in water at 100 gal/acre (0.02 lb a.i./tree) 1 application to 300 trees on Jun 1	2 lb a.i./acre, in water at 100 gal/acre (0.04 lb a.i./tree) 1 application to 300 trees on Jun 1 and an additional application to 150 trees on Jul 1	May - Sep
Diazinon: Diazinon 50W (50% a.i. as a wettable powder)				
High-pressure hydraulic sprayer -or- Hydraulic sprayer with hand-held wand	Individual trees in any orchard unit	0.015 lb a.i./tree, in water at 3 gal/tree 1 application to 100 trees on 20 acres on Jun 1	0.075 lb a.i./tree, in water at 5 gal/tree 1 application to 300 trees on 20 acres on Jun 1 and an additional application to 150 trees on 10 acres on Jul 1	Apr - Sep
Dimethoate: Digon 400 (43.5% a.i. as a liquid concentrate)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Individual trees in any production orchard unit	0.13 lb a.i./tree, in water at 2 gal/tree 1 application to 500 trees on May 1	0.34 lb a.i./tree, in water at 4 gal/tree 2 applications to 500 trees on May 1 and Jun 1	Apr - Sep

Table 2-1. Pesticide and Fertilizer Application Summary (continued)

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Esfenvalerate: Asana® XL (8.4% a.i. as an emulsifiable concentrate)				
High-pressure hydraulic sprayer -or- Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Individual trees in any production orchard	0.001 lb a.i./tree, in water at 2 gal/tree 2 applications to 1,700 trees on May 1 and Jun 1	Cumulative maximum = 1.6 lb a.i./acre per year 0.002 lb a.i./tree, in water at 4 gal/tree 2 applications to 1,700 trees on May 1 and Jun 1	Apr - Jul
Horticultural Oil: Dormant Oil 435 (98.8% paraffinic hydrocarbon oil)				
High-pressure hydraulic sprayer	Individual trees in any orchard, as an additive to other insecticides, fungicides, or miticides; or alone as a dormant spray	0.03 gal oil/tree, in water at 3 gal/tree 2 applications to 350 trees on Jun 1 and Jul 1	0.05 gal oil/tree, in water at 5 gal/tree 2 applications to 350 trees on Jun 1 and Jul 1	Mar - Sep (as an additive) Sep - Jul (as a dormant oil)
Permethrin: Pounce® 3.2 EC (38.4% a.i. as an emulsifiable concentrate)				
High-pressure hydraulic sprayer	Individual trees in any production orchard unit	0.01 lb a.i./tree, in water at 5 gal/tree 2 applications to 1,700 trees on May 1 and Jun 1	0.02 lb a.i./tree, in water at 10 gal/tree 2 applications to 1,700 trees on May 1 and Jun 1	May - Jul
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Individual trees in any orchard unit	0.002 lb a.i./tree, in water at 1 gal/tree 2 applications to 250 trees on May 1 and Jun 1	0.006 lb a.i./tree, in water at 3 gal/tree 2 applications to 250 trees on May 1 and Jun 1	May - Jul

Table 2-1. Pesticide and Fertilizer Application Summary (continued)

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Propargite: Omite® CR (32 % a.i. as a wettable powder in water soluble bags)				
High-pressure hydraulic sprayer -or- Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Individual trees in any orchard unit	1.4 lb a.i./acre, in water at 100 gal/acre 1 application to 250 trees on May 31	2.4 lb a.i./acre, in water at 100 gal/acre 2 applications to 550 trees on May 31 and Sep 15	Apr - Oct
Fungicide				
Chlorothalonil: Bravo® 500 (40.4% a.i. as a liquid concentrate)				
High-pressure hydraulic sprayer -or- Hydraulic sprayer with hand-held wand	Individual trees in any orchard unit, individual plants in special use areas	2.1 lb a.i./acre, in water at 100 gal/acre 2 applications to 250 trees on May 1 and Jun 1	4.2 lb a.i./acre, in water at 100 gal/acre 3 applications to 550 trees on May 1, Jun 1, and Jun 30	Feb - Jun
Herbicides				
Dicamba: Banvel® (48.2% a.i. as a water-soluble liquid)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Spot or strip treatments of weeds along fences, along roads, within orchard units, within open fields, or around facilities	1 lb a.i./treated acre, in water at 10 to 100 gal/acre 1 application to 3 acres on Apr 30	2 lb a.i./treated acre, in water at 10 to 100 gal/acre 2 applications to 5 acres on Apr 30 and Jun 15	Apr - Jun
Tractor-pulled spray rig with fixed nozzles or small boom	Strip treatments along roads or fences	1 lb a.i./acre, in water at 10 to 100 gal/acre 2 applications to 3 acres on Apr 30 and Jun 15	2 lb a.i./acre, in water at 10 to 100 gal/acre 2 applications to 5 acres on Apr 30 and Jun 15	Mar - Jul

Table 2-1. Pesticide and Fertilizer Application Summary (continued)

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Dicamba: Banvel® (48.2% a.i. as a water-soluble liquid) (continued)				
Hand-held wick	Spot treatments in orchard units, open fields, near sensitive areas, near facilities, along fencelines and roadsides	24.1% a.i. solution wiped on individual weed plants 2 applications to weeds on 2 acres on Apr 30 and Jun 15	24.1% a.i. solution wiped on individual weed plants 2 applications to weeds on 3 acres on Apr 30 and Jun 15	Mar - Jul
Glyphosate: Rodeo® (53.8% a.i. as isopropylamine salt; water-soluble liquid)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Spot or strip treatments of weeds along fences, along roads, within orchard units, within open fields, or around facilities	4 lb a.i./acre, in water at 10 to 40 gal/acre 1 application to 3 acres on Apr 30	5 lb a.i./acre, in water at 10 to 40 gal/acre 2 applications to 5 acres on Apr 30 and Jun 15	Apr - Aug
Tractor-pulled spray rig with fixed nozzles or small boom	Strip treatments along roads or fences	1 lb a.i./acre, in water at 50 to 100 gal/acre 1 application to 3 acres on Apr 30	4 lb a.i./acre, in water at 50 to 100 gal/acre 1 application to 5 acres on Apr 30	Apr - Aug
Hand-held wick	Spot treatments in orchard units, open fields, near sensitive areas, near facilities, along fencelines and roadsides	17.9 % a.i. solution wiped on individual weed plants 2 applications to weeds on 2 acres on Apr 30 and Jun 15	17.9 % a.i. solution wiped on individual weed plants 2 applications to weeds on 3 acres on Apr 30 and Jun 15	Apr - Aug

Table 2-1. Pesticide and Fertilizer Application Summary (continued)

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Hexazinone: Velpar® (90% a.i. as a soluble powder)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Spot or strip treatments of weeds along fences, along roads, within orchard units, within open fields, or around facilities	1 lb a.i./acre, in water at 25 to 100 gal/acre 1 application to 3 acres on Jun 1	7.2 lb a.i./acre, in water at 25 to 100 gal/acre 1 application to 5 acres on Jun 1	Apr - Jun
Picloram: Tordon® 22K (24.4% a.i. as a liquid concentrate)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Spot or strip treatments of weeds along fences, along roads, within orchard units, within open fields, or around facilities	0.25 lb a.i./acre, in water at 10 to 50 gal/acre 1 application to 3 acres on Apr 30	1 lb a.i./acre, in water at 10 to 50 gal/acre 2 applications to 5 acres on Apr 30 and Jun 15	Apr - Jun
Triclopyr: Garlon® 4 (61.6% as a liquid concentrate)				
Hydraulic sprayer with hand-held wand -or- Backpack sprayer	Spot or strip treatments of weeds along fences, along roads, within orchard units, within open fields, or around facilities	1.5 lb a.i./acre, in water at 10 to 100 gal/acre 1 application to 3 acres on Apr 30	8 lb a.i./acre, in water at 10 to 100 gal/acre 2 applications to 5 acres on Apr 30 and Jun 15	Apr - Jun

Table 2-1. Pesticide and Fertilizer Application Summary (continued)

Application Method	Location	Typical Application Rate, Area, and Date	Maximum Label Application Rate, Maximum Area and Date	Application Date Range
Fertilizers				
Ammonium sulfate (21-0-0-24), ammonium phosphate (11-52-0), ammonium nitrate (34-0-0), or potassium nitrate (14-0-45)				
Broadcast spreader pulled by tractor or ATV	All orchards	175 lb/acre ammonium sulfate -plus- 175 lb/acre ammonium phosphate -plus- 20 lb/acre ammonium nitrate -plus- 35 lb/acre potassium nitrate 1 application to 120 acres starting on Feb 1	175 lb/acre ammonium sulfate -plus- 175 lb/acre ammonium phosphate -plus- 20 lb/acre ammonium nitrate -plus- 35 lb/acre potassium nitrate 1 application to 120 acres starting on Feb 1	Feb - Mar

3.0 ENVIRONMENTAL FATE AND TRANSPORT

This section summarizes the environmental fate and transport of the pesticides and fertilizers proposed for use at Provolt. Environmental fate profiles of the chemicals are presented in Section 3.1. Modeling approaches, calculational methods, and results for runoff, leaching, and associated water concentrations are described in Section 3.2. Section 3.3 presents the approach and results used to evaluate off-target drift of the pesticides, and Section 3.4 lists the references cited in this section.

3.1 Environmental Fate Profiles

The following paragraphs present the chemical and physical properties that were used in characterizing the environmental fate and transport of the pesticides and fertilizers. Table 3-1 summarizes the chemical properties of the pesticides used in the runoff and leaching modeling.

3.1.1 Acephate

Acephate has a high water solubility of 790,000 mg/L at 20 °C and a calculated organic carbon partition coefficient (K_{oc}) of 3, both indicating high potential mobility (Extoxnet 2000, HSDB 2001).

Aerobic soil metabolism is the main degradation pathway for acephate, producing methamidophos which is rapidly biodegraded to CO_2 (EPA 2000a). Acephate's half-life in soil is 0.5 to 4 days for most soil types (HSDB 2001). Its foliar half-life ranges from 0.7 to 8.2 days (HSDB 2001).

Acephate is unlikely to bioconcentrate, with a predicted bioconcentration factor (BCF) of 0.3 (EPA 1984).

3.1.2 Chlorothalonil

Chlorothalonil is almost insoluble in water, with a value of 0.6 mg/L at 25 °C (EFDB 2001). Log K_{oc}s of 2.9, 3.0, 3.1 (K_{oc} = 1,259), and 3.8 mL/g were measured in sandy soil, sandy loam, silty clay loam, and silt, respectively (Caux et al. 1996). EPA (1999a) indicated that it is not generally been considered a highly mobile pesticide, and is more likely to be found in runoff from treated areas.

Chlorothalonil is transformed principally by aerobic and anaerobic microbial action (EPA 1999a). Its main breakdown product is the 4-hydroxy metabolite. The half-life ranged from 10.3 days in sandy loam soils to 36.5 days in silty clay loam soils (Caux et al. 1996). EPA (1999a) reported that terrestrial dissipation half-lives range from 4 to 90 days, with a value of 30 days considered representative. The foliar half-life on grape leaves was measured as 10 to 15 days, and as 3.6 to 21.31 days on potato plants (Caux et al. 1996).

Reported BCFs range from 16 (catfish) to 264 (bluegill sunfish) for whole fish (Caux et al. 1996).

Table 3-1. Chemical Properties of Pesticides and Other Ingredients

Chemical	Water Solubility (mg/L)	Half-life (days) Soil	Half-life (days) Foliar	Washoff Fraction*	K_{oc}	BCF***
Pesticides						
Acephate	790,000	4	8.2	0.70	3	0.3
Chlorothalonil	0.6	36.5	21.3	0.50	1,259	264
Chlorpyrifos	2	120	7	0.65	31,000	2,729
Diazinon	40	39	5.3	0.9	191	542
Dicamba	6,500	16	9	0.65	2.2	28
Dimethoate	25,000	20	3.6	0.95	18	2.3
Esfenvalerate	0.002	75	14	0.4	5,300	1,400
Glyphosate	12,000	60	8	0.60	4,900	0.52
Hexazinone	33,000	154	30	0.90	43	2
Horticultural Oil	100	42	2	0.50	1,000	46
Permethrin	0.04	38	10	0.30	63,096	480
Picloram	740,000	167	8	0.60	17	0.54
Propargite	0.63	78	13	0.20	31,061	775
Triclopyr Ester	6.8	46	15	0.70	780	1.08
Other Ingredients						
Cyclohexanone	23,000	5	2.5	0.90**	17	3.6
Ethylbenzene	161.2	71	35	0.60**	164	15
Light aromatic solvent naphtha	0.03	48	24	0.50**	1,000	1,000
Petroleum distillates	100	42	2	0.50	1,000	46
Xylene	130	2.2	1	0.65**	204	15

*GLEAMS manual unless otherwise noted.
**Estimated relative to water solubility of pesticides listed in GLEAMS manual.
***Bioconcentration factor. Can be interpreted as low if <10, medium if 10 to 1,000, high if >1,000

3.1.3 Chlorpyrifos

The solubility of chlorpyrifos in water is 2 mg/L at 25 °C (Budavari et al.1989). Measured and estimated K_{oc}s range from 1,862 to 85,590 (EFDB 2001). A value of 31,000 was selected for use in the risk assessment, based on EPA (2000b), who concluded that chlorpyrifos was generally immobile in soil.

Chlorpyrifos degrades by aerobic and anaerobic metabolism, principally to 3,5,6-trichloro-2-pyridinol (EPA 2000b). The persistence of chlorpyrifos in soils varies from a few days to more than 180 days, depending on soil type and environmental conditions, although it is usually between 60 and 120 days (EPA 2000b, Extoxnet 2000). Residues remain on plant surfaces for 10 to 14 days (Extoxnet 2000). EPA (2000b) estimated the foliar half-life as 7 days.

A BCF of 2,729 was measured in whole rainbow trout (EPA 2000b).

3.1.4 Diazinon

Diazinon has a water solubility of 40 mg/L (Verschueren 1983). HSDB (2001) reported the K_{oc} in three soils to range from 40 to 432, with an average of 191. In addition, a value of 13.9 was measured in a clay loam (HSDB 2001). It has been shown to be moderately mobile in soils (EPA 2001a).

Diazinon degrades by hydrolysis, photolysis, and microbial metabolism. Its main degradate is diazoxon, which further degrades to oxypyrimidine (EPA 2001a). Soil half-lives were reported as 37 and 39 days (EPA 2001a). EPA (2001a) reported a foliar dissipation half-life of 5.3 days.

The BCF for diazinon in bluegill sunfish was 542 (EPA 2001a).

3.1.5 Dicamba

The water solubility of dicamba is 6,500 mg/L at 25 °C (Extoxnet 2000). The average K_{oc} measured in five soils was 2.2 (EFDB 2001). It is highly mobile in soil and may contaminate groundwater (Extoxnet 2000).

Microbial degradation is the principal environmental fate process for dicamba, forming the primary metabolite 3,6-dichlorosalicylic acid (HSDB 2001). The soil half-life was 16 days in clay loam and sandy loam (HSDB 2001). Knisel et al. (1993) listed a foliar half-life of 9 days.

BCFs for dicamba were estimated to be 28 and 8, based on a log octanol-water partition coefficient (K_{ow}) of 2.21 and a water solubility of 5,600 mg/L, respectively (HSDB 2001).

3.1.6 Dimethoate

The water solubility of dimethoate is 25,000 mg/L (Extoxnet 2000). Based on experimental K_{oc} values of 18, 36, 5.2, and 20 (average = 20), and an additional value in clay loam of 18, dimethoate is not expected to adsorb to soil (HSDB 2001).

Dimethoate degrades primarily to CO_2, with small amounts of desmethyl dimethoate and dimethylthiophosphoric acid. Dimethoxon, a toxicologically significant metabolite, was also identified in field dissipation studies, but it degraded rapidly to undetectable levels while the parent compound was still measurable (EPA 1999b). Soil half-lives ranging generally from 4 to 16 days, but as high as 122 days, have been reported; a representative value would be 20 days (Extoxnet 2000). EPA (1999b) reported a soil half-life of 2.4 days in moist aerobic soils. A foliar half-life of 3.6 days was measured on citrus leaves (Wu and Fan 1997).

A BCF of 2.3 (log BCF = 0.36) was calculated from dimethoate's K_{ow} (EFDB 2001).

3.1.7 Esfenvalerate

Esfenvalerate is the alpha (or S,S-) isomer of fenvalerate, which is a mixture of four optical isomers.

The low water solubility of fenvalerate, 0.002 mg/L, and reported K_{oc} of 5,300 indicate that it has low potential for mobility and a tendency to adsorb to various environmental media (Extoxnet 2000, WHO 1990a).

Fenvalerate had a half-life of 75 to 80 days in sandy loam and silty clay loam soils, degrading to CO_2, 4-chloro-α-(1-methylethyl)-benzeneacetic acid, 4'-OH-fenvalerate, and $CONH_2$-fenvalerate (Lee 1985). The World Health Organization summarized the degradation processes as ester cleavage, diphenyl ether cleavage, ring hydroxylation, hydration of the cyano group to amide, and further oxidation of the fragments formed to yield carbon dioxide (WHO1990a). Eisler (1992) reported soil half-lives ranging from 3 to 9 weeks, with transformed products not persisting longer than the parent compound. Reported foliar half-lives include 2.46 to 4.46 days on sugarcane leaves, 11 to 19 days on alfalfa (depending on weather), 22 hours in broccoli fields, and 40 hours in cauliflower fields ((Hill et al. 1982, Maddy et al. 1985, Southwick et al. 1995). WHO (1990a) reported the foliar half-life for fenvalerate as 14 days.

Esfenvalerate's BCF was measured to be 1,400 in fathead minnows, indicating a potential for bioconcentration in aquatic species (HSDB 2001).

3.1.8 Glyphosate

Glyphosate has a moderate to strong tendency to adsorb to soil particles, reflected in its high estimated K_{oc} of 24,000 and measured K_{oc} in a silt loam of 4,900 (Extoxnet 2000, HSDB 2001). Its high water solubility of 12,000 mg/L indicates that any free glyphosate in the soil column will exist as dissolved species (Budavari et al. 1989, Extoxnet 2000).

The half-life of glyphosate in the soil averages 60 days (Ghassemi et al. 1981, HSDB 2001). EPA (1993) reported laboratory-determined soil half-lives of 1.85 and 2.06 days in a sandy loam and a silt loam, respectively. Extoxnet (2000) reported soil half-lives ranging from 1 to 174 days, with an average of 47 days. The major metabolite of glyphosate is aminomethylphosphonic acid (AMPA), which is formed through biodegradation, and further degrades to CO_2, although at a slower rate. The median half-life of AMPA in eight sites was 240 days (EPA 1993). Reported foliar half-lives for glyphosate are 10.4 to 26.6 days, 2 days in sugar maple, and 8 days on alder (HSDB 2001, Newton et al. 1984, Pitt et al. 1994).

Glyphosate's BCF was measured as 0.52 in whole fish (EPA 1993).

3.1.9 Hexazinone

Hexazinone has a high water solubility of 33,000 mg/L, and a low K_{oc} of 43 (measured in a silt loam), giving it a tendency toward high mobility and low soil adsorption (Extoxnet 2000, HSDB 2001).

Measured field half-lives range from less than 30 to 180 days, with a representative value of 90 days (Extoxnet 2000). EPA (1994a) reported a field dissipation half-life of 154 days in a silty clay loam. Hexazinone is subject to photodegradation and biodegradation; major degradation products are 3-hydroxy-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5–triazine-2,4(1H,3H)-dione and 3-(ketocyclohexyl)-6-(dimethylamino)-1-methyl-1,3,5–triazine-2,4(1H,3H)-dione. EPA (1994a) stated that the available data suggest that the degradates are also persistent and mobile. Foliar half-lives of 19 to 59 days were measured for two hexazinone formulations (Michael et al. 1999). Knisel et al. (1993) recommended a value of 30 days.

The BCF for hexazinone is 2, measured in a study by Rhodes (1980).

3.1.10 Horticultural Oil

The horticultural oil proposed for use at the seed orchard consists of paraffinic hydrocarbon oil. Paraffinic oils are alkane organic compounds found in petroleum.

Knisel et al. (1993) listed a water solubility for petroleum oils of 100 mg/L and a K_{oc} of 1,000 for use in the GLEAMS model.

Paraffinic oils degraded in a laboratory test using an agricultural sandy loam soil with a half-life of approximately 6 weeks (Battersby and Morgan 1997). No data on metabolites were available. Knisel et al. (1993) recommended a foliar half-life of 2 days for petroleum oils.

A BCF of 46 was estimated based on its water solubility.

3.1.11 Permethrin

Permethrin's water solubility is 0.04 mg/L (Verschueren 1983). A K_{oc} of 63,096 was reported, indicating a strong tendency to bind to soil particles and low potential for mobility (EFDB 2001)..

Soil half-lives for permethrin were listed as 30 to 38 days (Extoxnet 2000). Permethrin degrades in soil by hydrolysis of the ester linkage, forming 3-phenoxybenzyl alcohol (which further degrades to 3-phenoxy-benzoic acid) and 3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylic acid, followed by further breakdown, producing CO_2 (Jordan et al. 1982). WHO (1990b) stated that permethrin degrades on plants with a half-life of approximately 10 days.

A BCF of 480 was measured in sheepshead minnows (HSDB 2001).

3.1.12 Picloram

The water solubility of the potassium acid salt of picloram, contained in the Tordon® 22K formulation, is 740,000 mg/L at 20 °C (EPA 1995). Averaged K_{oc}s of 17 and 26 were reported from two review sources (EFDB 2001). Extoxnet (2000) listed a K_{oc} of 16. Picloram's high solubility and low K_{oc} predict that it will be mobile in the soil, with a potential for groundwater contamination.

Data on aerobic soil metabolism show that picloram acid degraded with half-lives ranging from 167 to 513 days in seven soils. Carbon dioxide is the major degradate, and two minor degradates are 4-amino-3,5-dichloro-2-pyridinol and 4-amino-2,3,5-trichloropyridine (EPA 1995). Extoxnet (2000) reported half-lives in soil of 20 to 300 days, with an average of 90 days. Knisel et al. (1993) listed a foliar half-life of 8 days for picloram salt.

In bluegill sunfish, the measured BCF was less than 0.54 (Extoxnet 2000).

3.1.13 Propargite

Propargite's solubility in water is 0.63 mg/L at 25 °C, and its median K_{oc} is 31,061 (EPA 2000c). It is immobile in soils (EPA 2000c).

Propargite dissipated from a sandy clay loam with a half-life of 78 days (EPA 2000c). In another soil degradation study, the aerobic soil half-life was determined to be 67 days, with major degradates identified as *p*-tertiary butylphenoxycyclohexanol, 2-{4-(2-hydroxycyclohexoxy)phenyl]-2,2-dimethyl acetic acid, *p*-tertiary butylphenol, and 2-(*p*-tertiary butylphenoxy)cyclohexanol sulfuric acid (Comezoglu et al. 1996). The foliar half-life was measured as 7 to 14 days on orange trees, and a median value of 13 days on nectarine foliage (Smith 1991).

A BCF of 775 was measured in bluegill sunfish (EPA 2000c).

3.1.14 Triclopyr

Triclopyr may be formulated as either the butoxyethyl ester of triclopyr acid (Garlon® 4), or the triethylamine salt (Garlon® 3A) of triclopyr acid. Provolt proposes only use of the butoxyethyl ester. Triclopyr acid is the environmental degradate formed by both compounds, and is mobile in soil (EPA 1998). EPA (1998) reported that the K_{oc} for triclopyr acid ranges from 25 to 384. Triclopyr acid degrades to 3,5,6-trichloro-2-pyridinol and, ultimately, CO_2 (EPA 1998).

The BCF for triclopyr acid in whole bluegill sunfish was reported as 1.08 (Extoxnet 2000).

Additional information specific to the butoxyethyl ester form of triclopyr is provided in the following paragraphs.

Triclopyr Ester

Triclopyr ester has a water solubility of 6.8 mg/L (EPA 1998). Knisel et al. (1993) listed the K_{oc} as 780 for triclopyr ester.

Knisel et al. (1993) listed soil half-lives of 46 days for both the amine and ester forms. Triclopyr ester hydrolyzes to triclopyr acid and 2-butoxyethanol, which biodegrades to 2-butoxyacetic acid, then forms CO_2. The foliar half-life was 15 days when triclopyr ester was applied to clearcut timberland in southwest Washington (EPA 1998). Knisel et al. (1993) listed foliar half-lives of 15 days for both triclopyr amine and triclopyr ester.

3.1.15 Other Ingredients

The "other ingredients" (formerly referred to as "inert ingredients") in the pesticide formulations are chemicals other than the active ingredient. As described in Section 1.0, EPA has classified these other ingredients into four categories, based on the degree of toxicity posed by the chemical:

- List 1: Inerts of toxicological concern.
- List 2: Potentially toxic inerts, with high priority for testing
- List 3: Inerts of unknown toxicity
- List 4: Inerts of minimal concern

There are no List 1 ingredients in the proposed pesticide formulations. Four List 2 ingredients are present in certain formulations, as described in the following paragraphs.

Cyclohexanone

Cyclohexanone is present in the Digon® 400 formulation of dimethoate. It appears on EPA's List 2 (potentially toxic inerts with a high priority for testing).

Its water solubility is 23,000 mg/L at 20 °C (Verschueren 1983). A K_{oc} of 17 was estimated based on this water solubility (HSDB 2001). Cyclohexanone is likely to be mobile in soil.

A soil half-life was not available for cyclohexanone. However, it would be expected to readily volatilize and photodegrade from the surface layers of soils (HSDB 2001). In biological oxygen demand and chemical oxygen demand tests, 50% metabolism occurred in 20 hours in an adapted microbial culture, and in 5 days in a mixed microbial culture (HSDB 2001). An atmospheric half-life of 4.3 days was measured for photolysis of cyclohexanone (HSDB 2001). Based on these data points, a soil half-life of 5 days was selected for use in the risk assessment. Based on relationships described in Knisel et al. (1993) a foliar half-life of 2.5 days was estimated, based on the soil half-life estimation.

BCFs calculated for cyclohexanone are 1.4, 2.4, 2.5, and 3.6 (EFDB 2001, HSDB 2001).

Ethylbenzene

Ethylbenzene is present in the Asana® XL formulation of esfenvalerate and the Pounce® 3.2EC formulation of permethrin. It appears on EPA's List 2.

The water solubility of ethylbenzene is 161.2 mg/L at 25 °C (EFDB 2001). Its K_{oc} was measured in a silt loam to be 164 (EPA 2001b), indicating low affinity to bind to soils.

Mackay et al. (1992) suggested a soil half-life of 71 days for ethylbenzene. Based on the soil half-life, a foliar half-life of 35 days was estimated.

A BCF of 15 was reported (EFDB 2001).

Light Aromatic Solvent Naphtha

Light aromatic solvent naphtha is present in the Pounce® 3.2EC formulation of permethrin. It appears on EPA's List 2.

The term "light aromatic solvent naphtha" refers to a group of compounds, consisting mainly of C_8 through C_{10} aromatic hydrocarbons. Naphthalene is a representative member of this group. EPA (1994b) listed the solubility of naphthalene, 0.03 mg/L, as applicable to this class of compounds. EPA (1994b) also estimated the range of K_{oc}s for light aromatic solvent naphthas as 500 to 2,000; a value of 1,000 was selected for use in the risk assessment. It is expected to adsorb moderately to strongly to soil.

Soil half-lives of 17 to 48 days were reported for naphthalene (Howard et al. 1991). Based on a soil half-life of 48 days, a foliar half-life of 24 days was estimated.

BCFs of 40 to 1,000 were reported for naphthalene (HSDB 2001).

Petroleum Distillates

Petroleum distillates are an inert ingredient in the Digon® 400 formulation of dimethoate. The data presented in the discussion of horticultural oil (Section 3.1.11) are also appropriate to the environmental fate assessment of petroleum distillates.

Xylene

Xylene is present in the Asana® XL formulation of esfenvalerate and the Pounce® 3.2EC formulation of permethrin. It appears on EPA's List 2. Xylene may occur as *o*, *m*, and *p* isomers

The solubility of mixed xylenes in water is 130 mg/L (ATSDR 1995). K_{oc}s for the three xylene isomers were reported as 129 to 204 (ATSDR 1995). It is expected to have moderate to high mobility in soils (HSDB 2001).

On surface soils, the major fate process is volatilization; a soil half-life of 2.2 days was reported (ATSDR 1995). A foliar half-life of 1 day was estimated, based on the soil half-life.

A BCF of 15 was measured in goldfish (EFDB 2001).

3.1.16 Fertilizers

Four types of fertilizers are proposed for use at Provolt: ammonium sulfate, ammonium phosphate, ammonium nitrate, and potassium nitrate. Each of these fertilizers is very soluble in water. Therefore, the environmental behavior of the dissolved species is addressed in this section. Sources for the following information include Beegle (1999), the Food and Agriculture Organization (FAO 2000), and Oldham (2000).

- Ammonium sulfate, $(NH_4)_2SO_4$: produces two **ammonium** ions (NH_4^+) and one **sulfate** ion (SO_4^-).

- Monoammonium phosphate, $NH_4H_2PO_4$: releases one **ammonium** ion and one **phosphate** (PO_4^{3-}) ion.

- Ammonium nitrate, NH_4NO_3: dissolves to form the **ammonium** ion (NH_4^+) and the **nitrate** ion (NO_3^-).

- Potassium nitrate, $KNO_{3:}$ dissolves to release one **potassium** ion (K^+) and one **nitrate** ion.

The fate of the dissolved species is described in the following paragraphs.

Ammonium

The ammonium ion adsorbs to soil particles. It is converted by soil bacteria to the nitrate ion, starting within two or three days at temperatures of 50 °F and higher, and is completely converted within a month of application. Plants can take up nitrogen in both the ammonium and nitrate forms.

Nitrate

Nitrate leaches readily from soils. Its nitrogen can also be released to the air as N_2, N_2O, and NO, if soils are saturated (i.e., anaerobic), allowing denitrification to occur by way of microbial action. Nitrate is a form of nitrogen that plants can readily absorb.

Phosphate

Phosphate does not adsorb to soils, but can become bound to other soil species, such as iron and aluminum, at low or high pHs. It is most soluble, and therefore most available to plants, in soils with a neutral pH, where it maintains the form of orthophosphate, H_2PO4^-. Phosphates can be transported to surface waters if overland runoff and erosion occurs, where they may contribute to eutrophication of lakes and ponds.

Potassium

Potassium generally adsorbs to soil particles, but is released and becomes available for plant uptake and leaching slowly.

Sulfate

Sulfate does not bind to soil particles, and therefore is readily available for plant uptake and can leach. Sulfate is the form of sulfur that plants absorb.

3.2 Runoff and Leaching of Pesticides and Fertilizers

A number of models have been developed to estimate off-target transport of pesticides. Many models have been validated by studies across the country, and have been improved to more accurately predict the movement of water on the surface and through the soil profile. Predicting the estimated environmental concentrations of pesticides at Provolt relied primarily on mathematical modeling for the following reasons:

- Conducting site-specific monitoring studies at individual sites would be prohibitively expensive and time consuming, and

- Sophisticated models have been validated in field tests, and are appropriate for application to this problem.

The U.S. Environmental Protection Agency and other regulatory agencies recognize the value of modeling for predicting impacts.

Predicting environmental concentrations resulting from pesticide and fertilizer use at Provolt is complicated by the wide range of chemical, environmental, and operational variables. To simplify the task, the modeler chooses a limited number of scenarios based on anticipated operations and circumstances. While the scenarios chosen in this study are intended for use in predicting expected conditions, a conservative bias was incorporated when assumptions were required. This is useful in overcoming the limitations and uncertainties that accompany modeling. If a model predicts that the less favorable circumstances produce acceptable results, then one can predict with greater confidence that the normal or more favorable circumstances will also produce acceptable results.

The computer-based USDA Groundwater Loading Effects of Agricultural Management Systems model was used in this assessment to predict runoff and leaching. The U.S. Geological Survey's Method of Characteristics model was used in conjunction with published results of field studies to estimate the effectiveness of buffer areas in attenuating chemical concentrations during shallow subsurface lateral flow.

3.2.1 The GLEAMS Model

The insecticides and fungicide applied to the seed orchard trees, the herbicides applied in the orchards, and the fertilizers applied in the orchards were modeled to estimate their environmental fate and transport, using the Groundwater Loading Effects of Agricultural Management Systems (GLEAMS) model.

The GLEAMS model, developed by the USDA Agricultural Research Service (Leonard et al. 1987, Leonard et al. 1988), is a computerized mathematical model developed for field-sized areas to evaluate the movement and degradation of chemicals within the plant root zone under various crop management systems. Version 3.0 of GLEAMS, a Microsoft Windows-based program used for this analysis, has undergone a number of improvements including the improved handling of forested areas (Knisel and Davis 2000). The model has been tested and validated using a variety of data on pesticide and bromide movement (see, for example, Leonard et al. 1987, Crawford et al. 1990). The hydrology and erosion components of GLEAMS are essentially the same as those of the CREAMS (Chemicals, Runoff, and Erosion from Agricultural Management Systems) model (Knisel 1980). CREAMS is a physically based model that had been validated using data from diverse climatic and physiographic regions (Bush et al. 1989, Knisel 1980, Knisel et al. 1983, Lorber and Mulkey 1982, Nutter et al. 1984). Improvements made during the development of GLEAMS included a new emphasis on prediction of chemical losses through leaching to groundwater, and a more sophisticated handling of irrigation. The following paragraphs briefly discuss the structure and function of the model.

Components

GLEAMS has four main components: hydrology, erosion, nutrients, and pesticides. The hydrology component of GLEAMS subdivides the soil within the rooting zone into as many as 12 computational layers. Soils data describing porosity, water retention characteristics, and organic matter content for the site-specific soil layers (horizons) are collected for model initialization. During a simulation, GLEAMS computes a continuous accounting of the water balance for each layer, including percolation, evaporation, and transpiration. Evaporation of chemicals from the soil surface is not represented, but evaporation of water can cause chemicals to move upward through the soil.

The erosion component of GLEAMS accounts not only for the basic soil particle size categories (sand, silt, and clay), but also for small and large aggregates of soil particles. Furthermore, the program accounts for the unequal distribution of organic matter between soil fractions, and uses this information and surface-area relationships to calculate an enrichment ratio that describes the greater concentration of chemicals in eroding soil compared with the concentration in surface soil.

The pesticide component of GLEAMS can represent chemical deposition directly on the soil, the interception of chemicals by foliage, and subsequent washoff. Degradation rates are allowed to differ between plant surfaces and soil, and between soil horizons. Degradation calculations are performed on a daily time interval. Redistribution of chemicals because of hydrologic processes is also calculated on a daily time step. The distribution of a chemical between dissolved and sorbed states is described as a simple linear relationship, being directly proportional to the K_{oc} and the organic matter content of the soil. The extraction of chemicals from the soil surface into runoff is calculated accounting for sorption (assumed to be relatively rapid) and using a related parameter describing the depth of the interaction of surface runoff and surface soil. Percolation of chemicals is calculated through each of the soil layers, and the amount that passes through the last soil layer is accumulated as the potential loading to the vadose zone or groundwater. Input data required by the GLEAMS model consist of several separate files representing rainfall data, temperature data, hydrology parameters, erosion parameters, nutrient parameters, and chemical parameters.

Parameter Files

The rainfall data file contains the daily rainfall for the period of simulation. The temperature data file contains the daily or monthly mean temperature for the simulation period. The model determines rain and snow from the temperature data file.

The hydrology parameter file contains information on the size, shape, and topography of the field, hydraulic conductivity, soil water storage, leaf area indices, and irrigation practices. This file also contains the runoff curve number, which describes the tendency for water to run off the surface of the soil.

The erosion parameter file contains information needed to calculate erosion, sediment yield, and particle composition of the sediment on a storm-by-storm basis. The input data can represent a number of optional configurations of fields, channels, and impoundments, but the representative scenarios for analysis in this study represented a single field for each orchard unit.

Pesticide parameter files were prepared for all pesticides describing their characteristics and particular use pattern at the seed orchard. Information was included on water solubility, foliar and soil half-lives, K_{oc}s, the tendency for the pesticide to wash off plant surfaces, and the expected application rate and schedule. For modeling purposes, it was assumed that there were no residues of pesticide on the site at the beginning of the ten-year simulation; however, persistence of residues from year to year during the simulation was evaluated.

Nutrient parameter files were prepared containing some background information on the orchard soils and their typical mineral content, and detailed times, amounts, and dates for each fertilizer application.

GLEAMS Output Structure

Output from the GLEAMS model includes accounting of concentrations by soil layer for each chemical, and the movement of chemical residues in percolating soil waters, surface runoff waters, and those residues sorbed to eroded soil particles on a daily basis. Separate output files are produced describing hydrology, erosion, nutrients, and pesticides in more detail. Two selected variable output files were also produced by GLEAMS for each field/scenario combination. These selected variable output files enable the model user to obtain chemical masses or concentrations in runoff or leaching water, water runoff volumes, and mass or concentration of eroded sediment in tabular form useful for automated analyses.

Model Setup

The objective of this simulation was to estimate soil chemical concentrations, initial maximum runoff loadings, and long-term chemical loss in runoff, sediment, and soil below the root zone. The analysis focused on typical environmental characteristics and pesticide/fertilizer use patterns relevant to Provolt.

The environmental input parameters were selected to represent the conditions at the seed orchard as realistically as possible. Specific soil characteristics used in the model simulations are provided in Table 3-2. The soil characteristics are described to the modeled rooting depth of 28 inches, which can be interpreted as the depth from which water is actively taken up by the vegetation. The soil types in the northern portion of the seed orchard (north of Highway 238) and in a smaller area near Williams Creek are primarily sandy loams and fine sandy loams. For purposes of modeling, these sandy soils were represented using the characteristics of the dominant series, Central Point sandy loam. It is well-drained, with slow runoff and moderately high permeability. The dominant soil type in the southern portion of the seed orchard is a loam of the Kerby series. It is well-drained, with slow runoff and moderate permeability. The organic matter content near the surface of both soils at the seed orchard ranges from 2 to 4 percent. For purposes of modeling, the organic matter content was taken to be 3 percent in the first horizon, and 1.5 percent in the second horizon. The characteristics of the seed orchard soils place them in hydrologic soil groups A to B (USDA 1983). The Central Point soil type modeled is listed as 16, and the Kerby soil is listed as 52 in the USDA Soil Conservation Service (SCS) Soil Survey of Josephine County.

Table 3-2. Soil Characteristics within the Rooting Zone

Soil Characteristic	Units	Central Point Sandy Loam		Kerby Loam	
Horizon		1	2	1	2
Modeled soil horizon depth (from surface)	in	0-15	15-28	0-7	7-28
Effective saturated conductivity	in/hr	4	4	1.3	1.3
Soil porosity	cm^3/cm^3	0.40	0.40	0.40	0.40
Assumed field capacity	cm/cm	0.22	0.22	0.26	0.26

Reference: USDA 1983

The scenarios modeled are summarized in Table 3-3. The corresponding application rates and treatment dates are provided in Table 2-1 of Section 2.0. Additional assumptions and inputs to the simulations included the following:

- Some soil characteristics vary by layer for the soils at Provolt. Central Point sandy loam is underlain by gravelly sandy loam to a depth of 60 or more inches. Kerby loam is underlain by extremely gravelly sand to a depth of 60 inches or more.

- Daily rainfall data were obtained for a ten-year period (1991 to 2000) from records kept at Provolt. Simulations were run for all ten years with the same pesticide and fertilizer applications each year to determine the variability of runoff concentrations from year to year, and to be able to make statistical estimates of the frequency of occurrence of a given level of runoff. The long period of simulation also allowed an evaluation of the tendency for a chemical's environmental persistence, if residues remain after one year, to contribute to an increased concentration in runoff or leachate in later years.

Table 3-3. GLEAMS Modeling Scenarios

Chemical	Application Method	Fields Modeled
Insecticides		
Chlorpyrifos	Hydraulic sprayer with hand-held wand (typical) High-pressure hydraulic sprayer (maximum)	*typical:* 300 trees scattered in BU7 and BU9 *maximum:* 300 trees scattered in BU7 and BU9, plus half re-treated
Diazinon	Hydraulic sprayer with hand-held wand (typical) High-pressure hydraulic sprayer (maximum)	*typical:* 300 trees scattered in BU7 and BU9 *maximum:* 300 trees scattered in BU7 and BU9, plus half re-treated
Dimethoate	Hydraulic sprayer with hand-held wand	*typical:* half of the trees in BU4 *maximum:* half of the trees in BU4
Esfenvalerate	High-pressure hydraulic sprayer	*typical:* all trees in BU1, BU16, and BU17 *maximum:* all trees in BU1, BU16, and BU17
Esfenvalerate	Hydraulic sprayer with hand-held wand	*typical:* half of the trees in BU4 *maximum:* half of the trees in BU4
Horticultural Oil	High-pressure hydraulic sprayer	*typical:* all trees in BU1 *maximum:* all trees in BU1
Permethrin	High-pressure hydraulic sprayer	*typical:* all trees in BU1, BU16, and BU17 *maximum:* all trees in BU1, BU16, and BU17
Permethrin	Hydraulic sprayer with hand-held wand	*typical:* half of the trees in BU4 *maximum:* half of the trees in BU4
Propargite	Hydraulic sprayer with hand-held wand	*typical:* all trees in BU15 *maximum:* all trees in BU11 and BU15
Fungicide		
Chlorothalonil	High-pressure hydraulic sprayer	*typical:* all trees in BU15 *maximum:* all trees in BU11 and BU15
Herbicides		
Dicamba	Hydraulic sprayer with hand-held wand	*typical:* 9 ft^2 squares around sprinkler heads in BU6 *maximum:* 9 ft^2 squares around sprinkler heads in BU6
Glyphosate	Hydraulic sprayer with hand-held wand	*typical:* 9 ft^2 squares around sprinkler heads in BU6 *maximum:* 9 ft^2 squares around sprinkler heads in BU6
Hexazinone	Hydraulic sprayer with hand-held wand	*typical:* 9 ft^2 squares around sprinkler heads in BU6 *maximum:* 9 ft^2 squares around sprinkler heads in BU6
Picloram	Hydraulic sprayer with hand-held wand	*typical:* 9 ft^2 squares around sprinkler heads in BU6 *maximum:* 9 ft^2 squares around sprinkler heads in BU6
Triclopyr	Hydraulic sprayer with hand-held wand	*typical:* 9 ft^2 squares around sprinkler heads in BU6 *maximum:* 9 ft^2 squares around sprinkler heads in BU6

Table 3-3. GLEAMS Modeling Scenarios (continued)

Chemical	Application Method	Fields Modeled
Ammonium sulfate + ammonium phosphate + ammonium nitrate + potassium nitrate	Broadcast spreader	typical: All production units and arboretums *maximum:* All production units and arboretums

- Irrigation water was added to the rainfall file based on records kept for the past eight years. Orchard units 15, 16, and 17 are irrigated from Laurel Hill Ditch, and they receive more frequent irrigation than the other units irrigated from the spring well.

- Temperature data were input as monthly average minimum and maximum, based on records for Medford, OR.

- The runoff curve number was assumed to be 60, appropriate for forests in good condition and hydrologic soil groups A and B. The hydrologic soil group is a categorization (from A to D) developed by the U.S. Natural Resources Conservation Service describing the potential for runoff from a soil. The hydrologic soil group must be considered along with the vegetation type to determine the runoff curve number. The runoff curve number is used by GLEAMS (and other models) to determine the amount of water that runs off from the land after a given amount of rain.

- The maximum effective rooting depth was assumed to be 28 inches for both soils. Thus, the depth of horizon 2 (Table 3-2) was set at 28 inches for modeling purposes.

- The soil erodibility factors (Ks) were 0.10 and 0.32 for Central Point and Kerby soils, respectively, based on the soil survey.

- The vegetative cover factor for erosion calculations (C) was estimated to be 0.004, representing good cover, primarily with grasses.

- The average slope for the southern orchard units (typically with Kerby soil) was 0.94 percent, and for the northern units (typically with Central Point soil) it was 2.4 percent. The average slope was estimated along the major flow path using topographic maps.

A complete set of GLEAMS input and output tables was created for each combination of scenario (typical or maximum)/chemical/soil series.

Accuracy and Limitations of GLEAMS Modeling Predictions

For a detailed discussion of the validation of GLEAMS, its sensitivity to errors in input parameters, and its expected accuracy, the reader should refer to the model documentation referenced at the beginning of this section. In addition to these studies, Mueller et al. (1992) evaluated the ability of the GLEAMS model to simulate movement of three herbicides using site-specific soil, environmental, and pesticide data. Field studies were used to examine alachlor and metribuzin movement in sandy loam soil in which cotton was grown, and norflurazon movement in a loamy sand soil. During the course of the study, actual herbicide concentrations were always greatest near the soil surface. The total herbicide present in each profile less than 20 days after application was accurately predicted by the GLEAMS model simulations. Herbicide movement into the soil profile in later simulations was overestimated by the model. Predictions from the model generally agreed with the relative location of alachlor and metribuzin in simulations less than seven days after herbicide application; beyond seven days after herbicide application, simulations deviated from actual concentrations. GLEAMS inaccurately predicted that norflurazon would be located throughout the soil profile, although the predicted depth to the limit of detection by the model was accurate (Mueller et al. 1992).

Crawford et al. (1990) compared GLEAMS simulation results to those of a field monitoring study examining the movement of carbofuran applied in an Appalachian mountain pine seed orchard. The predicted movement of carbofuran by GLEAMS agreed with results measured in the field, including time of initial pesticide movement, peak residue time, and residue dissipation time. Nutter et al. (1984) compared CREAMS (precursor of the GLEAMS model) model predictions of hexazinone concentrations in stormflow for four forested watersheds with the results of concentrations measured in the field over a 13-month period. Hexazinone concentrations in the initial stormflow events were accurately predicted by CREAMS. However, concentrations in stormflow two months or longer after hexazinone applications were underestimated by the model.

The GLEAMS computer model can provide a large amount of information without having to conduct expensive field studies and the subsequent chemical analysis. However, the model is sensitive to input parameters. Any site-specific parameters that were not directly measured and had to be estimated based on available literature introduce potential sources of error into the model. These parameters include pesticide decay rates, foliar washoff, K_{oc}, and soil curve numbers. The decay rates and foliar washoff factors govern the quantity of the contaminant available for movement, whereas the sorption coefficients and the runoff curve numbers govern the actual movement of the contaminants. The areal coverage influences the mass of pesticide that reaches the ground from application. Uncertainty in these parameters causes the majority of model uncertainty.

3.2.2 Buffer Zone Attenuation

The GLEAMS model was used to predict runoff of chemicals and water as they might be measured at the edge of each orchard unit. The Provolt seed orchard units generally have significant areas of untreated field edges and well-vegetated buffers between treated acreage and receiving streams. These untreated intervening areas (collectively termed "buffer zones" here) are expected to have a very significant effect in reducing the amount of chemicals that actually reaches stream water. Buffer zones of various types are well-known controls in conventional agriculture, but their significance is even greater under the circumstances present at Provolt. The seed trees and well-managed surface vegetation present at the orchard makes it more similar to a well-forested watershed than an agricultural area. True overland flow is very rare in well-managed forests and, although runoff does reach streams, it is mostly via subsurface shallow flow, which can be quite rapid (for example, "macropore flow") (Bush et al. 1986, Crawford et al. 1990). This type of lateral flow to streams has also been termed "interflow." Deeper groundwater flow also occurs, especially to perennial streams, and contributes to the "base flow" that continues even long after local rains. During rainfall events, true surface runoff normally occurs first from stream banks, and then as the rain continues (and especially if it intensifies) from successively larger areas surrounding the streams. This phenomenon has been called the "variable source area concept" (Hewlett and Nutter 1970, Dowd and Nutter 1985). Lobbe et al. (1990) reported that surface runoff normally accounts for less than 0.14 percent of the total precipitation and, under very wet conditions, this can increase to as much as 1 percent of the total water budget. However, the climate at Provolt is characterized by fairly even precipitation with very few large, sudden rainfalls. This climate and the surface condition at the seed orchard are conducive to percolation rather than direct runoff of rainfall. Stream flow from the orchard area is primarily due to subsurface flow. Buffer zones between treated acreage and receiving streams at Provolt will typically be in the range of 25 to 100 feet or more.

To account for the attenuating affect of buffer zones, the Method of Characteristics (MOC) model developed by the U.S. Geological Survey was used (version 3.2 with extended array dimensions, 1996) (Konikow et al. 1994). MOC is a two-dimensional groundwater flow and chemical transport model, and it computes changes in concentration over time accounting for the processes of dispersion, adsorption, and degradation. The model was set up to represent steady saturated shallow subsurface flow across a minimum 30-foot buffer zone. The near-surface saturated zone was assumed to be 13 inches thick, to represent the very permeable upper horizon. Four percent organic matter was assumed. Hydraulic conductivity of the soil in the surface horizon was assumed to be relatively high to account for its greater porosity, including macropores. The slope was assumed to be 40 percent, so the results are considered conservative for Provolt, where slopes are less. For purposes of calculation, the field edge and buffer zone were divided into 10-foot square cells. The results were expressed in terms of the fraction of chemical passing through the buffer zone, calculated as the ratio of the concentration exiting the buffer zone to the concentration entering it from the treated area. The model predicts greater attenuation of those pesticides with greater K_{oc} values due to adsorption, and less attenuation of those with low K_{oc} values. Additionally, a minimum fraction of pesticide passing the buffer zones was assumed to be 1 percent for typical scenarios and 5 percent for maximum scenarios. These minimum values are intended to account for the limited precision of the modeling and possible exceptions to model assumptions in some areas, such as concentrated overland flow where fields are not uniform. The resulting fractions of chemicals passing the buffer zones range from 1.0 to 20.5 percent. For nutrients, literature values were used from a study of runoff from grasslands, where an average of 6 percent of nitrogen and 2 percent of phosphorus was observed to pass buffer strips (Heathwaite et al. 1998).

3.2.3 Statistical Treatment of Results and Stream System Routing

Runoff timing followed very similar patterns among the various orchard units treated with a given pesticide. Consequently, a representative unit with the dominant Kerby soil was used to determine the specific rainfall events (by Julian day) that represented frequencies of occurrence of once per year (for typical scenarios) and once per eight years (for maximum scenarios). The rainfall events (and corresponding days) differed among the chemicals because of differences in the specific dates on which a particular chemical chemical may be used. The representative storms were chosen as follows:

- A program was written (in Visual Basic for Applications within MS Excel, also used for subsequent programming described below) to read the appropriate typical scenario GLEAMS output files for each chemical and to create a spreadsheet of total chemical in runoff for each day (dissolved plus adsorbed) in terms of g/ha. The days were sorted, and the day representing the degree of runoff with a frequency of once per year was recorded for each chemical.

- The above process was repeated, reading maximum scenario GLEAMS output files, and sorting to find the event with the greatest chemical mass in runoff in the ten-year period.

- Linear regression analysis was also performed for each chemical using the spreadsheet files created as described above to look for trends in runoff of chemicals over time, which was thought to be possible due to build-up of pesticide over time. No statistically significant trends were found (at $\alpha = 0.05$), although the second and subsequent years did tend to have somewhat higher chemical concentrations in runoff than the first year. Thus, no significant build-up over time was seen on the surface to increase runoff after the second year. This is attributed to a combination of degradation and leaching into the soil profile.

Runoff of pesticides, nutrients, and water were distributed among the onsite irrigation ditches, Williams Creek, and ultimately the Applegate River using the following procedure:

- Topographic maps were used to determine toward which of the eight ditch or creek segments each orchard unit drained, and estimates were made of the percentage flowing to each segment.

- A spreadsheet was created with the appropriate daily runoff values (pesticide, nutrient, and water) calculated by GLEAMS for each orchard unit/chemical combination for the typical scenarios, and a similar spreadsheet was created for the maximum scenarios.

- For each unit treated with each chemical, the mass of chemical in runoff entering each ditch/creek segment was calculated, accounting for attenuation by buffers, and added to each succeeding downstream segment until reaching the Applegate River.

- Similarly, runoff water draining from treated units was apportioned to all downstream segments. However, the process also had to estimate and account for water from all untreated areas draining into each segment, and upstream of the study area. This was accomplished by assuming that the runoff per unit area was the same for treated and untreated areas. This runoff was on a daily basis, and specific for each day studied.

- A nominal base flow draining into the larger ditch and creek segments was also included. This base flow was estimated conservatively, that is, by using dry weather flow records.

- Flow to the pond on the orchard property is controlled at the entrance to an underground pipe originating from Bridge Point Ditch. Water was assumed to flow from this source on the day of treatment, so that 10 percent of the water in the pond was replenished per day.

- Concentrations were calculated from the above procedures for each chemical/stream segment combination for typical scenarios, and then similarly for maximum scenarios.

The resulting concentrations were calculated for segments of Williams Creek above and below Bridge Point Ditch, for Spencer Ditch, segments of Bridge Point Ditch above and below the confluence with Spencer Ditch, Laurel Hill Ditch, the pond, and the Applegate River.

For use in the risk assessment estimates, the highest concentration in an onsite irrigation ditch was identified, along with the concentrations in Williams Creek, the pond, and the Applegate River. These values are presented in Table 3-4, and can be considered to represent 24-hour average concentrations.

3.2.4 Potential Leaching to Groundwater

The GLEAMS simulations calculated estimates of the mass per unit area of each chemical leaching below the rooting zone. During the first year, no significant residues leached below the rooting zone. However, if pesticide and fertilizer applications are continued, small amounts of some of the chemicals are predicted to leach below the rooting zone by the end of the second year. A two-year period was considered sufficient for analysis of leaching rates to groundwater because leaching is much less variable than surface runoff and because the low predicted concentrations indicated that this is not a pathway of concern at the seed orchard. Simple dilution calculations were done to estimate the maximum concentrations that might occur in groundwater directly beneath treated units for each scenario. The following assumptions were used:

- No attenuation was assumed during leaching through the vadose (unsaturated) zone overlying the aquifer. (This will lead to an overestimation of concentrations in the aquifer to the extent that degradation, adsorption, or dispersion occur in the vadose zone.)

- All chemical residues leaching during a two-year period (1991 to 1992) were assumed to reach the aquifer.

- The aquifer is assumed to be 50 feet (15.2 m) thick, and completely saturated. The aquifer may be at different depths under the various orchard units, but the thickness is assumed to be the same, so that the same dilution factor applies to the aquifer under each orchard unit.

- Porosity was taken to be 40 percent.

In both typical and maximum scenarios, only three pesticides were seen to leach below the rooting zone: dimethoate, hexazinone, and picloram, as well as nitrate and phosphate from the application of fertilizer. Even in the maximum scenarios, the highest residues are not above approximately 0.0035 mg/L, in spite of the conservative assumptions. Table 3-5 lists the estimated concentrations in groundwater. Movement of groundwater away from the orchard units will lead to even lower concentrations due to dispersion, adsorption, and degradation.

3.2.5 Prediction of Concentrations Due To Accidental Spills

Concentrations in irrigation ditches and Williams Creek that could occur if accidental spills of pesticides or fertilizer entered the tributary streams were estimated using the Exposure Analysis

Table 3-4. Estimated Surface Water Concentrations from Runoff and Erosion (mg/L)

		Ditch Segments		Williams Creek		Applegate River		Pond	
Chemical	**App Method**	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**
Chlorpyrifos	HPHS & HHW	-0-	4.87E-008	-0-	6.20E-009	-0-	2.41E-009	-0-	4.87E-009
Diazinon	HPHS & HHW	-0-	2.92E-009	-0-	3.25E-009	-0-	2.12E-010	-0-	2.92E-010
Dimethoate	HHW & BP	-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	7.38E-009	-0-	8.00E-009	-0-	3.11E-009	-0-	-0-
Esfenvalerate	HPHS	-0-	2.48E-009	-0-	7.93E-010	-0-	1.22E-009	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	8.72E-013	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	HHW & BP	-0-	9.25E-010	-0-	1.00E-009	-0-	3.90E-010	-0-	-0-
Ethylbenzene		-0-	3.03E-011	-0-	5.37E-011	-0-	3.49E-012	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Horticultural Oil	HPHS	-0-	-0-	-0-	-0-	-0-	5.68E-010	-0-	-0-
Permethrin	HPHS	-0-	2.10E-009	-0-	6.69E-010	-0-	1.16E-009	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	3.87E-012	-0-	-0-
Light aromatic solvent naphtha		-0-	5.22E-009	-0-	1.67E-009	-0-	3.04E-009	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HHW & BP	-0-	1.62E-011	-0-	1.75E-011	-0-	6.82E-012	-0-	-0-
Ethylbenzene		-0-	1.81E-011	-0-	3.21E-011	-0-	2.09E-012	-0-	-0-
Light aromatic solvent naphtha		-0-	3.67E-010	-0-	3.98E-010	-0-	1.55E-010	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Propargite	HPHS, HHW, & BP	-0-	1.77E-008	-0-	4.46E-009	-0-	7.45E-009	-0-	-0-
Chlorothalonil	HPHS & HHW	-0-	8.17E-009	-0-	6.57E-009	-0-	3.45E-009	-0-	-0-
Dicamba	HHW, BP, Boom, Wick	-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW & BP	-0-	5.82E-010	-0-	6.31E-010	-0-	2.46E-010	-0-	-0-
Glyphosate	Boom & Wick	-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW & BP	-0-	8.93E-012	-0-	1.58E-011	-0-	1.03E-012	-0-	-0-
Picloram	HHW & BP	-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW & BP	-0-	3.30E-010	-0-	3.58E-010	-0-	1.39E-010	-0-	-0-
Total Fertilizer	Spreader								
NO3 (as N)		-0-	6.52E-004	-0-	7.03E-005	-0-	3.32E-005	-0-	6.52E-005
NH4 (as N)		-0-	4.08E-008	-0-	6.11E-008	-0-	2.47E-009	-0-	4.08E-009
PO4 (as P2O5)		-0-	5.24E-007	-0-	5.74E-008	-0-	2.91E-008	-0-	5.24E-008

*HPHS = high-pressure hydraulic sprayer; HHW = hydraulic sprayer with hand-held wand, BP = backpack sprayer

Note: 1 mg/L = 1 part per million (ppm) = 0.001 parts per billion (ppb)

Table 3-5. Estimated Groundwater Concentrations

		Estimated Groundwater Concentration (mg/L)	
Chemical	**Method**	**Typ**	**Max**
Chlorpyrifos	HPHS & HHW	-0-	-0-
Diazinon	HPHS & HHW	-0-	-0-
Dimethoate	HHW & BP	-0-	1.3E-008
Cyclohexanone		-0-	-0-
Petroleum distillate		-0-	-0-
Esfenvalerate	HPHS, HHW, & BP	-0-	-0-
Ethylbenzene		-0-	-0-
Xylene		-0-	-0-
Horticultural Oil	HPHS, HHW, & BP	-0-	-0-
Permethrin	HPHS, HHW, & BP	-0-	-0-
Ethylbenzene		-0-	-0-
Light aromatic solvent naphtha		-0-	-0-
Xylene		-0-	-0-
Propargite	HPHS, HHW, & BP	-0-	-0-
Chlorothalonil	HPHS	-0-	-0-
Dicamba	HHW, BP, Boom, & Wick	-0-	-0-
Glyphosate	HHW, BP, Boom, & Wick	-0-	-0-
Hexazinone	HHW & BP	-0-	5.9E-009
Picloram	HHW & BP	-0-	5.9E-013
Triclopyr butoxyethyl ester	HHW & BP	-0-	-0-
Total Fertilizer	Spreader		
NO3 (as N)		-0-	-0-
NH4 (as N)		-0-	-0-
PO4 (as P2O5)		1.20E-004	1.20E-004

*HPHS = high-pressure hydraulic sprayer; HHW = hydraulic sprayer with hand-held wand, BP = backpack sprayer
Note: 1 mg/L = 1 part per million (ppm) = 0.001 parts per billion (ppb)

Modeling System (EXAMS) model. EXAMS was developed at EPA's Center for Exposure Assessment Modeling at Athens, GA (Burns 2000). The current version of the EXAMS model is 2.98.01 (Jan. 2001). The network of irrigation ditches, Williams Creek, and the adjoining sections of the Applegate River were represented by seven segments, each composed of two compartments: one for the surface sediments and the other for the overlying water. Appropriate volumes and flows were utilized to represent typical conditions during times of application.

EXAMS is capable of representing many types of chemical and biological reactions, but reactions were not very significant during the short time periods that would be expected for a spilled chemical to travel to downstream receptors. Simple half-lives were entered as rate constants into the model. The most important parameter for influencing the rate and extent of transport of the organic chemicals was the adsorption coefficient (input as K_{oc}). Spills were input as instantaneous loads of concentrate or tank mix to the appropriate compartment representing each of the three potential spill sites considered:

Accidental spill of pesticide concentrate

- Mixing area near administrative buildings.

Spill of pesticide tank mix or fertilizer load

- The point where three orchard roads converge at a crossing of Bridge Point Ditch, near the southwest corner of OU9B.

- The point where Highway 238 crosses Williams Creek.

The volume spilled for concentrates was the size of a typical container as the chemical is sold. For tank mixes, the volume used in these estimates was 250 gallons for a high-pressure hydraulic sprayer, 100 gallons for a spray boom, 20 gallons for a hydraulic sprayer with hand-held wand, 5 gallons for a backpack sprayer, 0.5 gallons for a hand-held wick; and 2,000 pounds of fertilizer.

Chemical concentrations varied over time, but the hydrology was at a steady-state. A separate EXAMS simulation was performed for each chemical and spill site. Each simulation was run for several hours (usually 20), the time of maximum concentration in the Applegate River was determined, and the corresponding concentration was recorded. Inorganic fertilizers were treated as inert tracers, so that only hydrodynamic processes affected their transport and fate.

Results of the modeling show that maximum residues from spills would reach the Applegate River in less than one hour.

Concentrations that could potentially occur in groundwater in the vicinity of the domestic well near the orchard office were estimated as follows:

- Sixty percent of spilled material was assumed to be cleaned up, and forty percent was left in the soil. This assumption may overestimate potential residues because mixing will typically occur on relatively impervious surfaces, and some of the materials are solids which can easily be cleaned up to a greater degree.

- Two percent of spilled material left on surface soils was assumed to leach to the aquifer. This is the upper limit of fractions leaching below the rooting zone after the second year of application in the GLEAMS modeling of the pesticides.

- The leached residues were assumed to be dispersed in groundwater representing a 70-foot thick aquifer with an area of eight hectares. This represents approximately one-third of the total area of the three orchard units closest to the domestic well.

- Porosity was taken to be 0.4.

3.3 Off-Target Pesticide Drift

The AgDRIFT® (v. 2.0) computer model was used to estimate off-target drift deposition from ground boom applications of pesticides. Data from field studies were used to characterize drift from applications using high-pressure hydraulic sprayers, hand-held wands, and backpack sprayers.

3.3.1 Ground Boom Drift

The AgDRIFT model was developed as a cooperative effort among the EPA Office of Research and Development, the USDA Agricultural Research Service, the USDA Forest Service, and the Spray Drift Task Force (a consortium of chemical pesticide registrants) (Teske et al. 2001). The public use version of AgDRIFT offers the program's Tier I, or screening-level, approach for estimating drift from ground boom applications.

A tractor-pulled spray rig with a boom may be used to apply the herbicides dicamba or glyphosate. Table 3-6 presents the drift modeling results for these applications, based on the results obtained using the terrestrial and stream assessment tools in AgDRIFT.

Table 3-6. Estimated Drift Deposition from Boom Applications

	Concentration (mg/L)[a]				
	Ditch		Williams Creek		Deposition at 25 Feet (lb/acre)
Pesticide	Typ	Max	Typ	Max	Typ[b]
Dicamba	5.17×10^{-7}	1.11×10^{-6}	6.73×10^{-8}	1.73×10^{-7}	0.0111
Glyphosate	5.17×10^{-7}	2.22×10^{-6}	6.73×10^{-8}	3.47×10^{-7}	0.0111

[a]24-hour average concentrations.
[b]Drift at 25 feet is only required for typical applications in this assessment.

3.3.2 Drift from Hand-Held Ground Methods

Drift from high-pressure hydraulic sprayers was estimated based on a study by Haverty et al. (1983). This method may be used to apply chlorpyrifos, diazinon, esfenvalerate, horticultural oil, permethrin, propargite, or chlorothalonil to individual orchard trees. In the study, drift deposition was measured at five distances from treated trees. No drift was found at 12 m (39.3 ft), the farthest distance evaluated. At 1, 3, 5, and 8 m (3.3, 9.8, 16.4, and 26.2 ft) from the trees, average drift deposition corresponded to 383, 109, 17.5, and 2.3 lb/acre per lb applied to each tree. Since all surface water at Provolt is farther than 50 feet from the edge of orchard areas, no significant drift is expected to reach surface water from this application method. Drift deposition at 3 and 25 feet from a sprayed tree was calculated to correspond to the drift rate identified for a distance of 3.3 and 26.2 feet, respectively, in the field study.

Drift from hydraulic sprayers with hand-held wands and backpack sprayers was estimated based on a field study by Hatterman-Valenti et al. (1995). Drift from three different hand-held methods of applying herbicides was measured in this study. The mean values for all three methods were used to approximate the drift from a hydraulic sprayer with a hand-held wand. The drift values for a spray gun with a 4 gallon-per-minute tip (associated with the least drift of the three types of equipment) were used to estimate potential drift from backpack sprayer operations, since no studies of drift specific to backpack sprayers were identified in a review of the available literature. It is likely that use of this study overestimates potential drift from a backpack sprayer, due to the higher application spray rate, but it can be useful as an upper bound estimate of potential drift from this method. The mean drift deposition values for the three types of equipment in the study were 1.0%, 0.4%, and 0.2% of the nominal application rate at 0.9, 1.5, and 2.1 m (3, 5, and 7 ft) from the treated area. The spray gun with the 4 gallon-per-minute tip resulted in corresponding values of 0.08%, 0.04%, and 0.03% of the application rate at the same distances. Based on these results, very little drift is expected outside of the treated areas from either of these methods. For use in the risk assessment, a value of 0.01% and 0.001% of the application rate was estimated as the drift rate at 25 feet from the treated area. For fenceline applications of herbicides, the drift rates corresponding to a distance of 0.9 m (3 ft) were applied to determine drift deposition outside of the fenceline. No off-target drift is expected for any other scenarios.

3.4 References

Agency for Toxic Substances and Disease Registry. 1995. Toxicology profiles for xylenes (update). U.S. Department of Health and Human Services. Atlanta, GA.

ATSDR. See Agency for Toxic Substances and Disease Registry.

Battersby, N.S., and P. Morgan. 1997. A note on the use of the CEC L-33-A-93 test to predict the potential biodegradation of mineral oil based lubricants in soil. Chemosphere 35(8):1773-1779.

Beegle, D.B. 1999. Soil fertility management. In *Penn State Agronomy Guide.* University Park, PA.

Budavari, S., M.J. O'Neil, A. Smith, and P.E. Heckelman, eds. 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals*. Merck & Co., Inc. Rahway, NJ.

Burns, L.A. 2000. Exposure Analysis Modeling System (EXAMS): User manual and system documentation. Report No. EPA/600/R-00/081, Revision B, September, 2000. National Exposure Research Laboratory, USEPA/ORD. Research Triangle Park, NC.

Bush, P.B., D.G. Neary, J.F. Dowd, D.C. Allison, and W.L. Nutter. 1986. Role of models in environmental impact assessment. Proceedings Southern Weed Science Society 39:502-512.

Bush, P.B., W.L. Nutter, D.G. Neary, and J.W. Taylor. 1989. Pesticide movement from southern pine seed orchards: Use of CREAMS model to facilitate evaluation of off-site pesticide movement. CREAMS/GLEAMS Symposium. USDA Agricultural Research Service. Athens, GA.

Caux, P.-Y., R.A Kent, G.T. Fan, and G.L. Stephenson. 1996. Environmental fate and effects of chlorothalonil: A Canadian perspective. Critical Reviews in Environmental Science and Technology 26(1):45-93.

Comezoglu, S.N., V.T. Ly, J. Wu, W.H. Harned, D.G. Dzialo, and C.K. White. 1996. Degradation of [^{14}C]-propargite in soil. Abstracts of Papers of the American Chemical Society 211(1-2):Agro 52.

Crawford, L.A., J.F. Dowd, P.B. Bush, Y.C. Berisford, and J.W. Taylor. 1990. Using GLEAMS to estimate insecticide movement from an Appalachian mountain seed orchard. National Research Conference: Pesticides in the Next Decade–The Challenges Ahead. Virginia Water Resources Research Center. Blacksburg, VA.

Dowd, J.F., and W.L. Nutter. 1985. Physical factors in forest hydrology. Forestry and Water Quality: A Mid-South Symposium. May 8-9, 1985. Little Rock, AR.

EFDB. See Environmental Fate Database.

Eisler, R. 1992. Fenvalerate hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Environmental Fate Database. 2001. On-line database. Syracuse Research Corporation. http://esc.syrres.com/efdb.htm

EPA. See U.S. Environmental Protection Agency.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

FAO. See Food and Agriculture Organization of the United Nations.

Food and Agriculture Organization of the United Nations. 2000. Fertilizers and their use: A pocket guide for extension officers. 4th ed. Co-prepared by FAO and International Fertilizer Industry Association. Rome, Italy.

Ghassemi, M., L. Fargo, P. Painter, S. Quinlivan, R. Scofield, and A. Takata. 1981. Environmental fates and impacts of major forest use pesticides. TRW Inc. Redondo Beach, CA.

Hatterman-Valenti, H., M.D.K. Owen, and N.E. Christians. 1995. Comparison of spray drift during postemergence applications to turfgrass. Weed Technology 9:321-325.

Haverty, M.I., M. Page, P.J. Shea, J.B. Hoy, and R.W. Hall. 1983. Drift and worker exposure resulting from two methods of applying insecticides to pine bark. Bulletin of Environmental Contamination and Toxicology 30:223-228.

Hazardous Substances Databank. 2001. On-line database in Toxnet. National Library of Medicine. http://toxnet.nlm.nih.gov/

Heathwaite, A.L., P. Griffiths, and R.J. Parkinson. 1998. Nitrogen and phosphorus in runoff from grassland with buffer strips following application of fertilizers and manures. Soil Use and Management 14:142-148.

Hewlett, J.D., and W.L. Nutter. 1970. The varying source area of streamflow from upland basins. Paper presented at the Symposium on Interdisciplinary Aspects of Watershed Management, August 3-6, 1970. Montana State University. Bozeman, MT.

Hill, B.D., W.A. Charnetski, G.B. Schallje, and B.D. Schaber. 1982. Persistence of fenvalerate in alfalfa: Effect of growth dilution and heat units on residue half-life. Journal of Agricultural and Food Chemistry 30:653-657.

Howard, P.M., R.S. Boethling, W.F. Jarvis, W.M. Meylan, and E.M. Michalenko. 1991. *Handbook of Environmental Degradation Rates.* Lewis Publishers. Chelsea, MI.

HSDB. See Hazardous Substances Databank.

Jordan, E.G., D.D. Kaufman, and A.J. Kayser. 1982. The effect of soil temperature on the degradation of cis, trans-permethrin in soil. Journal of Environmental Science and Health, Part B 17(1):1-17.

Knisel, W.G., ed. 1980. CREAMS: A field-scale model for chemicals, runoff, and erosion from agricultural management systems. U.S. Department of Agriculture, Science and Education Administration, Conservation Research Report No. 26.

Knisel, W.G., and F.M. Davis. 2000. GLEAMS: Groundwater Loading Effects of Agricultural Management Systems. Version 3.0 User Manual, Publication No. SEWRL-WGK/FMD-050199, revised 8/15/00. USDA Agricultural Research Service. Southeast Watershed Research Laboratory. Tifton, GA.

Knisel, W.G., G.R. Foster, and R.A. Leonard. 1983. CREAMS: A system for evaluating management practices. In *Agricultural Management and Water Quality*. F.W. Schaller and G.W. Bailey, eds. Iowa State University Press. Ames, IA.

Knisel, W.G., F.M. Davis, R.A. Leonard, and A.D. Nicks. 1993. GLEAMS user manual. Version 2.10. November 1, 1993. USDA Agricultural Research Service. Southeast Watershed Research Laboratory. Tifton, GA.

Konikow, L.F., G.E. Granato, and G.Z. Hornberger. 1994. User's guide to revised method-of-characteristics solute-transport model (MOC--Version 3.1): U.S. Geological Survey. Water Resources Investigations Report 94-4115.

Lee, P.W. 1985. Fate of fenvalerate (Pydrin insecticide) in the soil environment. Journal of Agricultural and Food Chemistry 33:993-998.

Leonard, R.A., W.G. Knisel, and D.A. Still. 1987. GLEAMS: Groundwater loading effects of agricultural management systems. Transactions of the ASAE 30(5):1403-1418.

Leonard, R.A., W.G. Knisel, F.M. Davis, and A.W. Johnson. 1988. Modeling pesticide metabolite transport with GLEAMS. Proceedings: Planning Now For Irrigation and Drainage. IR Div/ASCE. Lincoln, NE.

Lobbe, B.R., J.F. Dowd, and P.B. Bush. 1990. The influence of climatic variation on pesticide fate predictions. Pesticides in the next decade–The Challenges Ahead. National Research Conference. Virginia Water Resources Research Center. Blacksburg, VA.

Lorber, M.N., and L.A. Mulkey. 1982. An evaluation of three runoff loading models. Journal of Environmental Quality 11:519-529.

Mackay, D., W.Y. Shiu, and K.C. Ma. 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volume I–Monoaromatic Hydrocarbons, Chlorobenzenes, and PCBs*. Lewis Publishers. Chelsea, MI.

Maddy, K.T., S.L. Kilgore, K. Jacobs, and C.R. Smith. 1985. A study of foliar residues of fenvalerate (Pydrin®) after field application in Santa Barbara and Ventura Counties, California in 1984. California Department of Food and Agriculture. Sacramento, CA.

Michael, J.L., E.C. Webber, Jr., D.R. Bayne, J.B. Fischer, H.L. Gibbs, and W.C. Seecock. 1999. Hexazinone dissipation in forest ecosystems and impacts on aquatic communities. Abstract: Canadian Journal of Forest Research 29(7):1170-1181.

Mueller, T.C., R.E. Jones, P.B. Bush, and P.A. Banks. 1992. Hazard assessment: Comparison of PRZM and GLEAMS computer model predictions with field data for alachlor, metribuzin and norflurazon leaching. Environmental Toxicology and Chemistry 11:427-436.

Newton, M., K.M. Howard, B.R. Kelpsas, R. Danhaus, C.M. Lottman, and S. Dubelman. 1984. Fate of glyphosate in an Oregon forest ecosystem. Journal of Agricultural and Food Chemistry 32:1144-1151.

Nutter, W.L., T. Thacs, P.B. Bush, and D.G. Neary. 1984. Simulation of herbicide concentrations in stormflow from forested watersheds. Water Resources Bulletin 20:851-857.

Oldham, L. 2000. Soil fertility and fertilizers for master gardeners. Mississippi State University Extension Service. Starkville, MS.

Pitt, D.G., T. Buscarini, B. Staznik, D.R. Thomas, and E.G. Kettela. 1994. Initial deposits and persistence of forest herbicide residues in sugar maple (Acer saccharum) foliage. Abstract: Canadian Journal of Forest Research 24 (11):2251-2262.

Rhodes, R.C. 1980. 14C-Labeled hexazinone in water and bluegill sunfish. Journal of Agricultural and Food Chemistry 28(2):306-310.

Smith, C.R. 1991. Dissipation of dislodgeable propargite residues on nectarine foliage. Bulletin of Environmental Contamination and Toxicology 46:507-511.

Southwick, L.M., G.H. Willis, T.E. Reagan, and L.M. Rodriguez. 1995. Residues in runoff and on leaves of azinphosmethyl and esfenvalerate applied to sugarcane. Environmental Entomology 24(5):1013-1017.

Teske, M.E., S.L. Bird, D.M. Esterly, S.L. Ray, and S.G. Perry. 2001. A user's guide for AgDRIFT® 2.0: A tiered approach for the assessment of spray drift of pesticides. Prepared for Spray Drift Task Force (D.R. Johnson, Project Manager). Macon, MO.

USDA. See U.S. Department of Agriculture.

U.S. Department of Agriculture. 1983. Soil survey of Josephine County. Soil Conservation Service. Washington, DC.

U.S. Environmental Protection Agency. 1984. Health and environmental effects profile for acephate. EPA/600/X-84/338. Environmental Criteria and Assessment Office. Cincinnati, OH.

U.S. Environmental Protection Agency. 1993. Reregistration eligibility decision (RED): Glyphosate. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1994a. Reregistration eligibility decision (RED): Hexazinone. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1994b. Draft cleaner technologies substitutes assessment (CTSA): Screen reclamation. EPA 744R-94-005a. Office of Pollution Prevention and Toxics. Washington, DC.

U.S. Environmental Protection Agency. 1995. Reregistration eligibility decision (RED): Picloram. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1998. Reregistration eligibility decision (RED): Triclopyr. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1999a. Reregistration eligibility decision (RED): Chlorothalonil. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1999b. Environmental Fate and Effects Division's revised chapter for the dimethoate RED. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000a. Revised environmental fate and effects assessment: Acephate. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000b. Reregistration eligibility science chapter for chlorpyrifos: Fate and environmental risk assessment chapter. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000c. Environmental Fate and Effects Division science chapter for reregistration eligibility document for propargite. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2001a. Environmental risk assessment for diazinon. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2001b. Technical fact sheet: Ethylbenzene. Office of Groundwater and Drinking Water. Washington, DC.

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals.* 2nd ed. Van Nostrand Reinhold. New York.

WHO. See World Health Organization

World Health Organization. 1990a. Environmental health criteria 95: Fenvalerate. Geneva, Switzerland.

World Health Organization. 1990b. Environmental health criteria 94: Permethrin. Geneva, Switzerland.

Wu, J., and D. Fan. 1997. Degradation of dimethoate in chrysanthemums and soil. Bulletin of Environmental Contamination and Toxicology 59:564-569.

4.0 HUMAN HEALTH HAZARD ASSESSMENT

4.1 Introduction

This section presents the results of the hazard assessment—a review of available toxicological information on the potential human health hazards associated with the pesticides, fertilizers, and other ingredients proposed for use at Provolt. Section 4.2 provides background information to familiarize the reader with the terminology and technical information in this hazard analysis. Section 4.3 describes the hazard analysis methodology. Section 4.4 summarizes each chemical's toxic properties and identifies the toxicity values used in this risk assessment. Section 4.5 lists hazard analysis data gaps that affect the ability to quantify risks from these pesticides, and Section 4.6 lists the references cited.

4.2 Background Information

Much of the data on pesticide toxicity have been generated to comply with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended (7 U.S.C. 136 et seq.), which establishes procedures for registering, classifying, and regulating all pesticides. Other significant sources of information include published literature and studies conducted by chemical manufacturers.

Because of the obvious limitations on testing in humans, information on effects in non-human test systems usually provides the basis for an informed judgment as to whether an adverse impact is correlated with a particular exposure. These animal toxicity test results may be supplemented by information on a pesticide's effects on humans, such as the results of dermatologic or exposure testing in humans, and occasional studies of low-level dosing of human volunteers by oral or other routes.

Toxicity tests in laboratory animals are designed to identify specific toxic endpoints (effects of concern), such as lethality or cancer, and the doses associated with such effects. Studies vary according to the test species used, the endpoint, test duration, route of administration, and dose levels. The dosing schedule, number of test groups, and number of animals per group also vary from one test to another, but the tests are generally designed to demonstrate whether a causal relationship exists between administered doses and any observed effects.

4.2.1 Duration of Tests

The duration of toxicity tests ranges from single-dose (acute) or short-term (subacute) tests, through longer subchronic studies, to chronic studies that may last up to the lifetime of an animal. Acute toxicity studies involve administering a chemical to each member of a test group, either in a single dose or in a series of doses over a period less than 24 hours. Subacute, subchronic, and chronic studies are used to determine the effects of multiple doses. Subacute toxicity studies involve repeated exposure to a chemical for one month or less. Subchronic toxicity studies generally last from one to three months, and chronic studies last for more than three months.

Acute studies are used primarily to determine doses that are immediately lethal, which results in limited utility in an assessment of long-term or repeated low-level human exposures. Acute and subacute toxicity studies include dermal irritation tests, dermal sensitization tests, eye irritation tests, and inhalation exposure or daily oral dosing of laboratory animals for up to one month to further define effects from limited exposures.

Longer term studies are designed to characterize the dose-response relationship resulting from repeated exposure to a compound. All other things being equal, the greater the duration of the study, the more reliable will be the resulting value for estimating the effects of subchronic or chronic exposures in humans. Adverse effects in laboratory tests may include overt clinical signs of toxicity, reduced food consumption, abnormal body weight change, abnormal clinical hematology or chemistry, or visible or microscopic abnormalities in the tissue of the test organism. Chronic studies in rats or mice that continue for longer periods of time, usually about two years, may also be used to assess the carcinogenic potential of a chemical.

4.2.2 Routes of Exposure

For assessing hazards from chemicals proposed for use at Provolt, the routes of administration in laboratory tests that reflect the likely types of exposures to humans include dermal (applied to the skin), inhalation (through exposure to vapors or aerosol particles), and oral by dietary (in food or water) or gavage (forced into the stomach through tubing). Selection of the route of administration of a particular test material is based on the probable route of human exposure. Oral, dermal, and inhalation doses most nearly duplicate the likely routes of pesticide exposure for humans.

4.2.3 Units

A dose is expressed as milligrams of a chemical per kilogram of body weight of the test animal (mg/kg), in parts per million (ppm) in the animal's diet, in milligrams per liter in the water that it drinks, or mg/L or milligrams per cubic meter (mg/m^3) in the air that the animal breathes. In chronic studies, the test substance is generally administered in the diet at specified amounts in parts per million. The known weight of the animal over the test period is used to convert parts per million in the diet to milligrams of a chemical per kilogram of body weight per day (mg/kg/day) for extrapolation to humans. In most chronic toxicity studies, at least two dosing levels are used, in addition to a zero-dose, or control group. In general, the control group receives only the vehicle (for example, water or saline) used in administering the test material. In a dietary study, the animal’s feed would serve as the vehicle.

4.2.4 Toxicity Background Information

The following paragraphs provide information on specific topics to assist the reader in understanding the summary data provided in the hazard analyses of the chemicals (Section 4). The information in these paragraphs is drawn primarily from Amdur et al. (1991), EPA (1989), and Lu (1985).

NOEL and LOEL

For examination of noncarcinogenic endpoints, toxicity testing can be used to estimate threshold exposure levels. The threshold level is the dose level at which a significant proportion of the test animals first exhibit the toxic effect. The threshold dose will vary among tested species and among individuals within species. Examples of toxic effects include pathologic injury to body tissue; a body dysfunction, such as respiratory failure; or another toxic endpoint, such as developmental defects in an embryo. It is not possible to determine threshold dose levels precisely; however, the no-observed-effect level (NOEL) indicates the dose at which there is no statistically or biologically significant increase in the frequency or severity of an adverse effect in individuals in an exposed group, when compared with individuals in an appropriate control group. The next higher dose level in the study is the lowest-observed-effect level (LOEL), at which adverse effects are observed. The true threshold dose level for the particular animal species in a study lies between the NOEL and the LOEL. If a chemical produces effects at the lowest dose tested in a study, the NOEL must be at some lower dose. If the chemical produces no effects, even at the highest dose tested, the NOEL is equal to or greater than the highest dose.

Cholinesterase Inhibition

Exposure to organophosphate insecticides (such as acephate, chlorpyrifos, diazinon, and dimethoate) results in the inhibition of cholinesterase enzyme activity, and specifically of acetylcholinesterase (AChE). AChE is responsible for the breakdown of acetylcholine, a neurotransmitter that permits the transmission of nerve impulses across the nerve synapse. Inhibition of AChE results in accumulation of acetylcholine and the continual transmission of nerve impulses. The extent of AChE inhibition caused by a given dose of pesticide is usually expressed as a percentage—either a percentage of normal activity or a percentage reduction compared with normal activity. At low doses, AChE inhibitors may cause localized effects in humans such as salivation, tearing, nasal discharge, blurred vision, or bronchial constriction; and systemic effects, such as nausea, sweating, dizziness, and muscular weakness. Effects of higher doses include irregular heartbeat, elevated blood pressure, cramps, vomiting, diarrhea, frequency of urination, convulsions, or fatality. Clinically significant inhibition is considered AChE depression of 20 percent or more compared with pretreatment values for plasma, erythrocyte, and brain AChE activities.

Neurotoxicity

Some chemicals may have adverse effects on the nervous system. Several procedures have been developed to detect neurotoxic effects. Neurologic examinations to help identify the site of adverse effects are performed in both animals and humans. Morphologic examinations are pathologic observations of abnormalities or lesions. Delayed neurotoxicity testing involves a single administration of a chemical to hens (which are readily susceptible to this type of neurotoxicity) followed by examination eight to 10 days later for signs of toxicity to the long axon of the neuron. Other types of studies that may be used to evaluate neurotoxicity include electrophysiologic examinations of neurons, muscles, and the brain; biochemical examinations of enzyme systems, ion transport systems, protein synthesis, neuronal biochemical composition, and neurotransmitter levels and binding sites; behavioral studies; and *in vitro* testing on cultured nerve cells.

Immunotoxicity

In general, four interrelated types of effects on the immune system are possible as a result of exposures to chemicals: immunosuppression, immune cell proliferation, alterations of host defense mechanisms against pathogens and tumors, and allergy or autoimmunity. Many tests are available that incorporate or are targeted primarily at an assessment of the effects of chemicals on the immune system. Allergic hypersensitivity is a particular form of immune system response to a foreign substance. Allergic hypersensitive reactions may be immediate, such as in anaphylactic (i.e., severe, potentially life-threatening allergic) reactions to insect bites or penicillin injections; or they may be delayed, as in the case of positive responses to tuberculin tests or contact dermatitis caused by poison ivy.

Reproductive Toxicity

Reproduction studies are conducted to determine the effect of a chemical on reproductive success, as indicated by fertility, fetotoxicity (direct toxicity to the developing fetus), maternal toxicity, and survival and weight of offspring. Some reproductive toxicity studies may involve administering the test compound during only one breeding and gestational cycle to evaluate perinatal and postnatal toxicity. Other reproduction studies continue through two or three generations of treated animals. Both male and female animals (usually rats) are exposed to the chemical beginning shortly after weaning (30 to 40 days of age) and continuing through breeding, gestation, and lactation. The offspring then receive the chemical in their feed until they are about 140 days old, at which time they are bred to produce another generation. The laboratory tabulates the percentage of females that conceive, number of full-term pregnancies, litter size, number of stillbirths, and number of live births. Viability counts and pup weights are noted. Indexes are scored for gestation, viability, and survival through lactation. During necropsy and histopathology examinations, special attention is given to effects on reproductive organs.

Developmental Toxicity

Developmental studies (also called teratogenicity studies) are used to determine the potential of a chemical to cause malformations in an embryo or a developing fetus between the time of conception and birth. For these tests, a compound is administered to gestational female animals (usually rats or rabbits) during the first trimester; and the fetuses are delivered by cesarean section one day before the estimated delivery date. The numbers of live, dead, and resorbed fetuses are determined, and skeletal and tissue abnormalities are observed.

Carcinogenicity

Carcinogenicity studies are used to determine the potential for a compound to cause malignant (cancerous) or benign (noncancerous) tumors when administered over an animal's lifetime. Several dose levels are used, with the highest set at the maximum tolerated dose, as established from preliminary studies. A control group is administered the vehicle (the liquid or food with which the test chemical is given) alone. Because tumors may arise in test animals for reasons unrelated to administration of the test compound, statistical analyses are applied to the tumor incidence results to determine the significance of observed results. Amdur et al. (1991) listed four types of responses that have generally been accepted as evidence of compound-induced tumors:

- The presence of types of tumors not seen in controls
- An increase in the incidence of the tumor types occurring in controls
- The development of tumors earlier than in controls
- An increased multiplicity of tumors

Some chemicals that elicit one or more of these responses may not be primary carcinogens (that is, tumor-inducers on their own), but may be enhancers or promoters. However, a carcinogenicity evaluation remains appropriate, because they may contribute to an increase in cancer incidence.

Cancer Slope Factor

In a carcinogenicity assay, the dose-specific tumor incidence data are used to calculate a cancer slope factor, which represents the probability that a 1-mg/kg/day chronic dose of the agent will result in formation of a tumor, and is expressed as a probability, in units of "per mg/kg/day" or $(mg/kg/day)^{-1}$. The curve relating dose to cancer probability, obtained using high doses of a chemical in laboratory animals, is assumed to approximate a straight line in the low-dose region. The slope of this line represents the cancer potency. The methodology used in calculating the cancer slope factors in this hazard analysis incorporates two conservative assumptions:

- It is assumed that the amount of carcinogen delivered to the target organ is proportional to the amount of carcinogen entering the body. However, some compounds may not exhibit linear pharmacokinetics. That is, for some compounds, the target organ dose may be lower than the entire dose received.

- It is assumed that there is no threshold for carcinogenic effects. That is, any dose, no matter how small, has some quantifiable probability of resulting in tumor formation. Again, chemical-specific research has indicated that this is not always a reliable assumption, and that some carcinogens may indeed have no-effect levels.

These assumptions may lead to an overestimate of cancer risk. However, there is no generally accepted methodology in use at this time to reflect such chemical-specific information. EPA’s 1996 proposed Guidelines for Carcinogen Risk Assessment reflect an effort to address these, and other, issues. These guidelines have not yet been finalized and are still undergoing internal EPA, interagency, and public review and comment.

Mutagenicity

Mutagenicity assays are used to determine a chemical's ability to cause physical changes (mutations) in the basic genetic material deoxyribonucleic acid (DNA), especially changes in the germ cells that could affect an embryo's viability or lead to genetic diseases or congenital anomalies. Mutagenicity data for a chemical might help in evaluating its carcinogenic mechanism of action, because many mutagens have been found to be carcinogens in laboratory animals and the sequence of cellular events that leads to carcinogenesis appears to be initiated by a mutation (Lu 1985). Tests used to detect gene mutations include assays in microorganisms such as bacteria, as well as in higher organisms such as yeasts, other fungi, and mammals. Effects on genetic material can also result in chromosomal aberrations, which are structural changes in chromosomes or changes in the number of chromosomes. In some cases, the existence of DNA damage caused by mutagens can be detected by biologic processes, such as DNA repair and recombination, that occur in association with DNA binding.

4.3 Hazard Analysis Methodology

The goal of the hazard analysis is to determine toxicity levels for quantification of risk. There are two types of toxicity endpoints: noncarcinogenic effects and carcinogenic effects.

For noncarcinogenic effects, it is generally assumed that there is a threshold level, and that doses lower than this threshold can be tolerated with little potential for adverse health effects. The U.S. EPA has determined threshold doses for many chemicals; these are referred to as reference doses (RfDs). The oral RfD is an estimate of the highest possible daily oral dose of a chemical that will pose no appreciable risk of deleterious effects to a human during his or her lifetime. The uncertainty of the estimate usually spans about one order of magnitude.

EPA selects the RfD using the lowest NOEL from the species and study most relevant to humans. In the absence of data from the most clearly relevant species, a study using the most sensitive species (the species that exhibited the lowest NOEL) is selected for use in RfD determination. This NOEL is divided by an uncertainty factor (usually 100) consisting of a factor of 10 to allow for the variation of response within the human population and a factor of 10 to allow for extrapolation to humans. Additional uncertainty factors may be applied to account for extrapolation from a shorter term study, overall inadequacy of data, or failure to determine a no-effect level. RfDs are expressed in units of mg/kg/day. EPA lists RfDs in its Integrated Risk Information System, a chemical risk database (EPA 2001). EPA's Office of Pesticide Programs also recommends RfDs for pesticides.

In many cases, exposures to the chemicals proposed for use at Provolt will not occur every day for a person's lifetime, but over a shorter duration. EPA's Risk Assessment Guidance for Superfund (EPA 1989) discusses the use of subchronic RfDs when exposures may range from two weeks to seven years in duration, instead of an individual's entire lifetime. These subchronic RfDs are not used in the assessment of risks from seed orchard chemicals, for the following reasons:

- The seed orchard pesticide and fertilizer use programs are anticipated to be in effect for more than seven years, exceeding the upper time limit for exposure in EPA's discussion of appropriate use of subchronic RfDs. It is safe to assume that length of employment and length of residence may make the exposure scenarios applicable to an individual worker or nearby resident for longer than a seven-year period.

- EPA (2000) stated that subchronic RfDs should not be used to evaluate risks to children, as they may not be sufficiently protective. Children are a subset of the general public whose risks are assessed in the analysis.

Additionally, the use of chronic RfDs provides a more conservative estimate of the dose-response relationship in all cases, decreasing the likelihood of underestimating any potential risks to any worker or member of the public.

For compounds that are known, probable, or possible human carcinogens, cancer slope factors that have been calculated by EPA or other appropriate sources are identified for use in this risk assessment.

Sources of information reviewed in this hazard analysis include EPA's IRIS database (EPA 2001), the National Library of Medicine's Hazardous Substance Databank (HSDB 2001), other databases, published literature, and data submitted to EPA by the chemical manufacturers to support pesticide registration under FIFRA.

4.4 Hazard Analyses

The following sections describe the toxicity of the pesticides, other ingredients, and fertilizers proposed for use at Provolt.

Table 4-1 summarizes the endpoints used in quantitative risk assessment. The data summarized in this table are extracted from the detailed toxicity reviews in the following sections.

4.4.1 Acephate

Acephate is an organophosphate insecticide. Provolt proposes to use Acecap® 97, which is 97% acephate in an implant capsule in the orchard units.

Noncarcinogenic Effects

EPA (2000) set an oral RfD for acephate of 0.004 mg/kg/day, based on a 90-day feeding study in rats. The toxic endpoint at the LOEL in this study was brain AChE inhibition. An acute delayed neurotoxicity study in chickens was negative, and a later study of effects on neuropathy target esterase supported the conclusion that acephate is not expected to produce organophosphate-induced delayed neurotoxicity (Chevron 1985, Wilson et al. 1990).

Table 4-1. Toxicity Endpoints

Chemical	RfD (mg/kg/day)	Dermal Absorption (%)	Cancer Slope Factor (per mg/kg/day)
Acephate	0.004	0.4 (1-hr)	0.0087
Chlorothalonil	0.015	0.15	0.00766
Chlorpyrifos	0.0003	1.78 (4-hr)	NA[a]
Diazinon	0.0002	2	NA
Dicamba	0.045	10	NA
Dimethoate	0.0005	11	NA
Esfenvalerate	0.02	3 (8-hr)	NA
Glyphosate	2	1.42 (24-hr)	NA
Hexazinone	0.05	1	NA
Horticultural oil	1	1	NA
Permethrin	0.05	1.7	0.016
Picloram	0.2	0.2	NA
Hexachlorobenzene	NA	23	1.7
Propargite	0.04	14.5 (8-hr)	0.201
Triclopyr	0.5	1.65 (8-hr)	NA
Inert Ingredients			
Cyclohexanone	5	10	NA
Ethylbenzene	0.1	3.4 (4-hr)	NA
Light aromatic solvent naphtha	0.02	10	NA
Xylene	2	3.9 (4-hr)	NA
Nitrate	1.6	NA	NA

[a]NA = Not applicable

In rat and rabbit teratology studies, no developmental defects were noted (EPA 1987, EPA 2000). In a three-generation reproduction study in rats, the reproductive LOEL was 25 mg/kg/day, due to decreased viability of offspring (EPA 2000). In a two-generation reproduction study in rats, fetal losses and decreased litter weights were found at the lowest dose tested of 2.5 mg/kg/day (EPA 2000). In a study in rats, Salama et al. (1993) demonstrated that acephate can cross the placental barrier; it was detected in fetal tissue within 10 minutes of oral administration.

Acephate was reported to be non-irritating and non-sensitizing to the skin, based on patch tests of nursery workers (O'Malley et al. 1995) and studies in guinea pigs (Agrochemicals Handbook 1994).

It is a minimal eye irritant (Valent 1994). A dermal penetration rate of 0.4 percent per hour was reported by Chevron (1987).

Carcinogenic and Mutagenic Effects

EPA (2000) considers acephate to be a possible human carcinogen, based on an increased incidence of hepatocellular carcinomas and adenomas in female mice. The 105-week study showed a statistically significant increase in tumors in females fed 1,000 ppm in the diet (13.3 mg/kg/day human equivalent dose). An oral cancer slope factor of 0.0087 $(mg/kg/day)^{-1}$ was calculated using the linearized multistage procedure (EPA 2000). Positive gene mutation and chromosomal aberration studies support the conclusion that acephate may affect DNA (Behera and Bhunya 1989, EPA 2000, Hour et al. 1998, Waters et al. 1982). However, other researchers have reported inconsistent results in studies for gene mutation, chromosomal aberration, and primary DNA damage; they attribute this to a weak mutagenic potential (Carver et al. 1985).

4.4.2 Chlorothalonil

Chlorothalonil is a fungicide. Provolt proposes to use Bravo® 500, which is 40.4% chlorothalonil as a liquid concentrate.

Noncarcinogenic Effects

EPA (2000) has set a chronic oral RfD of 0.015 mg/kg/day for chlorothalonil, based on a two-year feeding study in dogs in which effects on kidney cells were observed at the LOEL of 3 mg/kg/day; the NOEL was 1.5 mg/kg/day.

In a teratology study in rabbits, a NOEL of 10 mg/kg/day was determined, with reductions in maternal body weight and food consumption observed at the LOEL of 20 mg/kg/day (EPA 1999). In rats, maternal effects were noted at the LOEL of 400 mg/kg/day, with a NOEL of 100 mg/kg/day (EPA 2000). In a three-generation reproduction study in rabbits, decreased weight and adverse effects in offspring were observed at the lowest dose tested of 75 mg/kg/day (EPA 2000). In a two-generation reproduction study in rabbits, the maternal NOEL was <38 mg/kg/day (the lowest dose tested), and the NOEL for offspring was 115 mg/kg/day, based on lower body weights in offspring in the 234-mg/kg/day dose group (EPA 1999).

Zeneca (1998) stated that chlorothalonil may cause skin irritation and may be a potential skin sensitizer. A study by Boman et al. (2000) concluded that chlorothalonil is a potent contact allergen. EPA (1999) determined that chlorothalonil was a severe eye irritant. EPA (1999) calculated an upper limit for dermal absorption of chlorothalonil of 0.15%.

Carcinogenic and Mutagenic Effects

Zeneca (1998) stated that chlorothalonil may have oncogenic potential based on studies in rats and mice, although it does not appear to have any mutagenic potential or interact with DNA (Mizens et al. 1998). EPA (1999) reported the results of six carcinogenicity studies, three in rats and three in mice. Tumors of the stomach and kidneys were observed in five of the six studies. Based on one of the studies in rats in which renal adenomas and carcinomas and stomach papillomas were produced, a cancer slope factor of 0.00766 $(mg/kg/day)^{-1}$ was calculated. EPA (1999) classifies chlorothalonil as a probable human carcinogen. EPA (1999) concurred that chlorothalonil does not appear to be mutagenic.

4.4.3 Chlorpyrifos

Chlorpyrifos is an organophosphate insecticide. Provolt proposes to use Dursban 50W, which is 50% chlorpyrifos as a wettable powder in water-soluble packets.

Noncarcinogenic Effects

EPA (2000a) has established a chronic oral RfD of 0.003 mg/kg/day for chlorpyrifos, based on a 20-day study in humans in which decreased plasma cholinesterase was observed at the LOEL of 0.10 mg/kg/day. The NOEL in this study was 0.03 mg/kg/day. However, EPA's Office of Pesticide Programs recently reviewed the appropriateness of this study (both in terms of ethics and scientific validity) for use in setting a chronic RfD, and concluded that a more relevant value would be 0.0003 mg/kg/day, based on the weight of evidence from several studies in animals (EPA 2000b). In a chronic feeding study in beagle dogs, cholinesterase inhibition was observed at 0.03 mg/kg/day, with a NOEL of 0.01 mg/kg/day (EPA 2000c). In a chronic feeding study in rats, a NOEL of 0.0132 mg/kg/day was identified, with cholinesterase inhibition observed at the LOEL of 0.33 mg/kg/day (EPA 2000c).

An acute delayed neurotoxicity test in hens was negative at doses up to 110 mg/kg (EPA 2000c). In a developmental neurotoxicity study in rats, alterations in brain development were observed in the offspring of dams dosed with 1 mg/kg/day chlorpyrifos (EPA 2000c). Based on this study and a literature review, EPA (2000c) concluded that chlorpyrifos exposure may affect early nervous system development through mechanisms unrelated to cholinesterase inhibition.

Developmental toxicity, not including maternal toxic effects, was observed in studies in rats, mice, and rabbits at LOELs of 15, 25, and 140 mg/kg/day, respectively (EPA 2000c). In a two-generation reproduction study, reduced pup weight and mortality were observed in rats at a dose level of 5 mg/kg/day (EPA 2000c). In a three-generation reproduction study, no effects on offspring were observed at the highest dose tested of 1 mg/kg/day (EPA 2000c).

Blakley et al. (1999) reported that twice-weekly dosing of rats for 4 weeks with 5 mg/kg chlorpyrifos impaired T-lymphocyte blastogenesis and affected humoral immunity, although antibody and phagocytic responses remained normal, indicating changes in lymphocyte subpopulations. Thrasher et al. (1993) had previously linked chlorpyrifos exposure to immunologic abnormalities in humans, including antibiotic sensitivity, increased CD26 cells (a type of lymphocyte), and a higher rate of autoimmunity.

In vitro studies of the ability of chlorpyrifos to activate an estrogen receptor in human MCF7 breast cancer cells and in yeast cells into which the estrogen receptor alpha had been inserted were both negative (Vinggaard et al. 1999).

EPA (2000c) reported that chlorpyrifos was a slight eye irritant and a mild skin irritant in tests in rabbits, and did not cause dermal sensitization in a test in guinea pigs. Extoxnet (2000) stated that studies in humans suggest that skin absorption of chlorpyrifos in humans is limited. EPA (2000c) estimated that chlorpyrifos is dermally absorbed at a rate of 1 to 3% of the applied dose, based on measurement of urinary metabolites. A dermal absorption rate of 1.78% over 4 hours was measured in a study in rats (Tos-Luty et al. 1994).

Carcinogenic and Mutagenic Effects

No treatment-related tumors were observed in two chronic studies in rats and two chronic studies in mice (EPA 2000c). Chlorpyrifos has not demonstrated any mutagenicity in bacterial or mammalian cells, but resulted in slight genetic alterations in yeast cells and caused DNA damage to bacterial cells. Tests for evidence of DNA damage and repair in mammalian cells were negative (EPA 2000c).

4.4.4 Diazinon

Diazinon is an organophosphate insecticide. Provolt proposes to use Diazinon 50W, which is a wettable powder containing 50% diazinon.

Noncarcinogenic Effects

EPA (2000) recommended an RfD of 0.0002 mg/kg/day for diazinon, based on a review of seven feeding studies in dogs and rats. Three of the studies in rats produced NOELs of 0.02 mg/kg/day, with cholinesterase inhibition observed at the LOEL in each case. The LOEL in the fourth rat study exceeded 0.02 mg/kg/day. Although each of the three dog studies showed some plasma cholinesterase inhibition at a dose of 0.02 mg/kg/day, EPA considered it to be a minimal or borderline effect in the dog.

In a two-generation reproduction study in rats, a NOEL of 0.67 mg/kg/day was determined, based on increased pup mortality and decreased weight gain at that level (EPA 2000). No developmental toxicity was reported in teratogenicity studies in rats and rabbits at doses up to 100 mg/kg/day, the highest dose tested in both studies (EPA 2000). Abu-Qare et al. (1999) reported that a single dermal diazinon dose of 65 mg/kg to pregnant rats did not cause significant maternal or fetal cholinesterase inhibition.

There was no evidence of delayed neurotoxicity in a study in hens (EPA 2000).

Diazinon was a slight eye and skin irritant in studies in rabbits (EPA 2000). Although it was not a sensitizer in a test in guinea pigs, it caused dermal sensitization in about 10 percent of human volunteers tested (Platte Chemical Co. 1994, EPA 2000). EPA (2000) reviewed the existing data on dermal absorption of diazinon and concluded that there was no consistency across species. Although one study was conducted in humans, it failed to account for 97% of the applied dose. EPA determined that an absorption factor was not required, since a dermal NOEL was available, equal to 1 mg/kg/day. Applying a safety factor of 100, an RfD of 0.01 mg/kg/day was recommended for evaluating risks from dermal exposures to diazinon. This value would be equivalent to use of a 2% factor for dermal absorption in this risk assessment.

Carcinogenic Effects

Diazinon was not carcinogenic in lifetime feeding studies in rats (high dose = 40 mg/kg/day) and mice (high dose = 29 mg/kg/day), and is considered not likely to be a human carcinogen (EPA 2000).

EPA (2000) reported that diazinon was negative in two studies for gene mutation and one study of chromosomal aberrations. In studies for effects on DNA, diazinon was negative for causing unscheduled DNA synthesis, and was negative in two studies for sister chromatid exchange. Diazinon was weakly positive in one additional study of sister chromatid exchange, but the effect was not dose-related; that is, it did not increase consistently with increasing dose (EPA 2000).

4.4.5 Dicamba

Dicamba is an herbicide. The Banvel® formulation, containing 48.2% dicamba as a water-soluble liquid, is proposed for use by Provolt.

Noncarcinogenic Effects

In a three-generation reproduction study in rats, no effects were observed at the highest dose tested of 25 mg/kg/day (EPA 2001). In a developmental study in rats, no fetotoxic effects were observed, but the NOEL for maternal effects was 160 mg/kg/day (EPA 2001). EPA reviewed the available data in 1992, and set a chronic oral RfD of 0.03 mg/kg/day for dicamba, based on observations of maternal and fetal toxicity in a teratology study in rabbits at a dose level of 10 mg/kg/day; the NOEL in this study was 3 mg/kg/day (EPA 2001). The Office of Pesticide Programs reviewed an additional two-generation reproduction study in rabbits, in which the NOEL was 45 mg/kg/day, where observations at the LOEL of 136 mg/kg/day included decreased pup growth. Based on this study, a chronic oral RfD of 0.045 mg/kg/day was more recently recommended for dicamba (EPA 1999).

A one-year feeding study in dogs did not result in any adverse effects at the highest dose tested of 52 mg/kg/day (EPA 2001). Similarly, no effects were noted in two 2-year feeding studies in rats at the high doses of 25 and 125 mg/kg/day (EPA 2001). In a two-year feeding study in dogs, decreased body weights were observed at the LOEL of 0.625 mg/kg/day, with a NOEL of 0.125 mg/kg/day (EPA 2001). A two-year study in mice resulted in a NOEL of 115 mg/kg/day, based on increased mortality in males and decreased body weight gain in females at the LOEL of 360 mg/kg/day (EPA 1999). Slightly decreased body weight and food consumption, and histopathologic changes in liver cells, were observed at the LOEL of 500 mg/kg/day in a 90-day feeding study in rats with a NOEL of 250 mg/kg/day (EPA 2001).

In a 13-week neurotoxicity test in rats, rigid body tone, slightly impaired righting reflex, and impaired gait were observed at a dose of 767.9 mg/kg/day in males and 1,028.9 mg/kg/day in females (EPA 1999).

Dicamba produced mild to moderate eye irritation and skin irritation in studies in rabbits (EPA 1999). Micro Flo (1999) stated that the Banvel® formulation was extremely irritating to eyes and may be corrosive. It was negative for dermal sensitization in a study in guinea pigs (EPA 1999). No quantitative data were available for dermal absorption of dicamba. USDA (1984) assumed a conservative dermal absorption value of 10%.

Carcinogenic and Mutagenic Effects

No evidence of oncogenic effects was found in two-year feeding studies in mice and rats (EPA 1999). Dicamba was negative for mutagenic effects in eight studies of gene mutation and chromosomal aberrations (EPA 1999).

4.4.6 Dimethoate

Dimethoate is an organophosphate insecticide. Provolt proposes use of Digon® 400, containing 43.5% dimethoate as a liquid concentrate. Digon® 400 contains the other ingredient cyclohexanone, at a concentration of 35%, which appears on EPA's List 2 (potentially toxic inerts with a high priority for testing) (Wilbur-Ellis 1995).

Noncarcinogenic Effects

EPA's Office of Pesticide Programs recommended a chronic oral RfD of 0.0005 mg/kg/day for dimethoate, based on inhibition of brain and red blood cell cholinesterase at a dose level of 0.25 mg/kg/day in a 2-year study in rats (EPA 1999). The NOEL in this study was 0.05 mg/kg/day.

In a developmental study in rabbits, dimethoate caused a reduction in fetal weight at a dose of 40 mg/kg/day, with a NOEL of 20 mg/kg/day (EPA 1999). No developmental effects were observed in two studies in rats at the highest doses tested of 18 and 30 mg/kg/day (Srivastava and Raizada 1996, EPA 1999). A reproductive NOEL of 1.25 mg/kg/day was determined in a three-generation study in rats, based on decreases in number of live births, pup weight, and fertility at a dose of 6.5 mg/kg/day (EPA 1999). In a study in rabbits, ten doses of 30 mg/kg every other day resulted in a statistically significant increase in sperm abnormalities, persisting until the study ended at 50 days (Wlodarczyk et al. 1992).

No signs of acute delayed neurotoxicity were found in a study in hens (EPA 1999). However, an acute screening test in rats produced absence of pupil response at a dose of 20 mg/kg, with a NOEL of 2 mg/kg. At a dose of 200 mg/kg, reactions included tremors, decreased motor activity, and other symptoms indicating that coordination, sensory, and motor systems were affected. These effects were reversed by day 7 after treatment. There were no neuro-histopathological effects in either the central or peripheral nervous systems.

The potential for dimethoate to affect the immune system was studied in a three-generation test in rats (Institóris et al.1995). At a dose of 9.39 mg/kg/day, the number of spleen cells decreased in the first generation only. In another study cited by the authors, a single intraperitoneal dose of 75 mg/kg in mice caused reduced spleen and thymus weight. In a study in mice, a single oral dose of

16 mg/kg caused decreased spleen weights and hematological effects, including a decrease in total serum immunoglobulins (Aly and El-Gendy 2000).

Dimethoate was found to be neither a dermal irritant nor a skin sensitizer in tests in rabbits and guinea pigs (EPA 1999). However, dermatitis has been reported following high exposures in an occupational setting (Schena and Barba 1992). Severe eye irritation has occurred in workers manufacturing dimethoate, although this may be due to impurities (Extoxnet 2000). EPA (1999) reported a dermal absorption value of 11% for dimethoate, based on a study in rats.

Carcinogenic and Mutagenic Effects

Dimethoate has been classified as a potential human carcinogen by EPA's Office of Pesticide Programs (EPA 1999). Dimethoate produced equivocal hemolymphoreticular tumors in mice; had a weak and not dose-related association with combined spleen, skin, and lymph tumors in rats; and was positive for inducing unscheduled DNA synthesis in two studies, although it was negative or equivocal in tests for gene mutation and chromosomal effects. EPA recommended against using a cancer slope factor approach to quantify carcinogenic risks from dimethoate, and considers it more appropriate to use the RfD approach, due to the equivocal nature of the tumor results in laboratory animals. The chronic RfD approach is considered protective of any potential cancer risk.

4.4.7 Esfenvalerate

Esfenvalerate is a pyrethroid insecticide. Provolt proposes to use the Asana® XL formulation of esfenvalerate, which is 8.4% percent esfenvalerate as an emulsifiable concentrate. Asana® XL contains the other ingredients ethylbenzene (<1%) and xylene (<3%), both of which are listed on EPA's List 2 (potentially toxic inerts, with high priority for testing) (Du Pont 1999).

Esfenvalerate is the A-alpha-isomer of the pesticide fenvalerate. Fenvalerate is composed of four isomers, the A-alpha, B-alpha, A-beta, and B-beta. Because efficacy studies determined that the A-alpha isomer was the only isomer with significant insecticidal properties, it was developed to replace fenvalerate (EPA 2000). Data on fenvalerate are included in this hazard analysis when specific information on the toxicological properties of the esfenvalerate isomer is unavailable.

Noncarcinogenic Effects

EPA has established an oral RfD of 0.02 mg/kg/day for esfenvalerate, based on neurological dysfunction observed at the LOEL of 18.7 mg/kg/day in a 90-day rat-feeding study (EPA 1997). The NOEL in this study was 7.8 mg/kg/day. Esfenvalerate is classified as a Type II pyrethroid. Type II pyrethroids are associated with nerve discharges of long duration and nerve membrane depolarization. Symptoms in mammals include tremors, involuntary jerky or writhing movements, and clonic seizures (Eells and Dubocovich 1988).

In a two-generation reproduction study in rats, decreased pup weight was observed at the reproductive LOEL of 5 mg/kg/day, with a reproductive NOEL of 3.75 mg/kg/day. Systemic effects, including skin lesions and decreased body weight, were observed at the lowest dose tested of 3.75 mg/kg/day (EPA 1997). Developmental studies using esfenvalerate in rats and rabbits resulted in NOELs for systemic effects less than 2.5 and 3 mg/kg/day, respectively, based on behavioral and central nervous system symptoms at the lowest doses tested; the maternal toxicity NOELs for these studies were both identified as 2 mg/kg/day, based on dosing in an associated pilot study. No developmental effects were observed in either study, at the highest doses tested of 20 mg/kg/day in both species (EPA 1997). Effects on neurochemistry in offspring were reported at a fenvalerate dose of 10 mg/kg/day in a developmental study in rats, the only dose tested (Malaviya et al. 1993). Du Pont (1999) reported laboratory animal NOELs for reproductive effects of 4.2 to 7.3 mg/kg/day.

Fenvalerate gave results suggestive of a potential to disrupt hormone functions in an assay of its estrogenic potential in human breast cancer cells; the authors called for further study of this possibility for effects in both humans and wildlife (Go et al. 1999).

In humans, dermal overexposure to esfenvalerate can cause paraesthesia (an abnormal sensation such as burning or prickling) that may last up to 24 hours (Morgan 1996). It is slightly irritating to the eyes (Du Pont 1999). Esfenvalerate is not a skin sensitizer in animals (Du Pont 1999). The dermal penetration of fenvalerate in newborn human foreskin was found to be 9.32 percent after 48 hours (Shehata-Karam et al. 1988). This result is consistent with the results of a previous study by Grissom et al. (1985), who measured dermal penetration rates in mice of 1.9, 2.2, and 9.1 percent after one, six, and 24 hours, respectively. A dermal penetration rate of 3% for eight hours exposure was used in this risk assessment, based on the measured value of 9.1% for 24 hours.

Carcinogenic and Mutagenic Effects

No carcinogenicity studies have been performed with the esfenvalerate isomer. Although EPA has not formally classified fenvalerate's status, there is no indication that it is carcinogenic: five negative carcinogenicity studies have been conducted for fenvalerate in mice and rats (Cabral and Galendo 1990, WHO 1990, EPA 1997). No fenvalerate-related oncogenicity was reported in two studies in rats at doses up to 12.5 and 50 mg/kg/day; and in three studies in mice at doses up to 187.5 mg/kg/day (see next paragraph), 45 mg/kg/day, and 37.5 mg/kg/day (EPA 1997, EPA 2000). EPA (1997) concluded that there is no evidence of carcinogenicity for esfenvalerate in rats or mice.

In one mouse study, neoplastic pathological changes, diagnosed as multifocal microgranulomas, were observed in the lymph nodes, liver, and spleen of both sexes (Parker et al. 1983). Okuno et al. (1986) conducted a follow-up study to examine these changes, by feeding groups of male mice a diet containing only one of the four optical isomers of fenvalerate. No microgranulomatous changes were observed in mice fed the esfenvalerate isomer for one year at doses of 500 or 1,000 ppm (estimated to be 75 and 150 mg/kg/day, respectively). Microgranulomatous changes were observed only in mice treated with the B-alpha isomer; therefore, the authors concluded that the B-alpha isomer is the causative agent of microgranulomatous changes, rather than the esfenvalerate isomer.

No mutagenicity studies were available on the esfenvalerate isomer of fenvalerate. Fenvalerate was negative for DNA damage in *Bacillus subtilis*, gene mutation in Ames tests and two host-mediated assays, induction of sex-linked recessive lethal mutations in the fruitfly, and dominant lethal effects in mice (WHO 1990). EPA (1997) concluded that fenvalerate has not indicated mutagenicity in bacterial or two mammalian studies. However, fenvalerate gave uncertain results in a study for chromosomal aberrations in rats (WHO 1990), and researchers in Spain and India have reported mitotic disturbances, chromosome structural aberrations, and sister chromatid exchanges in assays with fenvalerate (Carbonell et al. 1989, Pati and Bhunya 1989, Puig et al. 1989). Therefore, it appears possible that one or more isomers of fenvalerate have some genotoxic potential.

4.4.8 Glyphosate

Glyphosate is an herbicide. Provolt proposes to use the Rodeo® (53.8% glyphosate) formulation.

Noncarcinogenic Effects

In 1992, EPA set an oral RfD for glyphosate of 0.1 mg/kg/day, based on increased incidence of renal tubular dilation at 30 mg/kg/day in offspring in a three-generation reproduction study in rats (EPA 2000). EPA's Office of Pesticide Programs has since concluded that this effect was not related to glyphosate dosing, and has recommended a new RfD for glyphosate of 2 mg/kg/day, based on a developmental study in rabbits (EPA 1993). In this study, maternal toxicity was present at a dose of 350 mg/kg/day, with a NOEL of 175 mg/kg/day; no development effects were found. In a developmental study in rats, increased numbers of litters and fetuses with unossified sternebrae, and decreased fetal body weights, were reported at a dose of 3,500 mg/kg/day, with a NOEL of 1,000 mg/kg/day (EPA 1993). The RfD of 2 mg/kg/day recommended by the Office of Pesticide Programs (EPA 1993) is used in this risk assessment.

In a one-year dog-feeding study, a decrease in absolute and relative pituitary weights was found at a dose of 100 mg/kg/day (EPA 2000). In a two-year feeding study in rats, no adverse effects were observed at the highest dietary level tested of 300 ppm (31 mg/kg/day in males, 34 mg/kg/day in females) (EPA 2000). In a subchronic study, rats fed 1,000 ppm (63 mg/kg/day–males, and 84 mg/kg/day–females) showed hematology effects that were possibly treatment-related, and those receiving 20,000 ppm had pancreatic lesions (EPA 1993).

The author of a study in which mice received the Roundup® formulation in their drinking water concluded that antibody production was unaffected, suggesting that the formulation is unlikely to cause immune dysfunction under normal application conditions (Blakley 1997). The highest concentration tested was 1.05% formulated product (equivalent to 4,305 mg glyphosate/L), estimated to be a dosing level of 21.5 mg glyphosate/day.

Glyphosate was reported to be a mild eye irritant and non-irritating to skin (Monsanto 2001). No irritation or sensitization was found in a study with human volunteers (Maibach 1986). Wester et al. (1991) measured the *in vivo* dermal absorption of glyphosate in the rhesus monkey to be 1.5% for 12 hours exposure. A value of 1.42 % over 24 hours was subsequently identified in an *in vitro* study in human skin (Wester et al. 1996).

Carcinogenic and Mutagenic Effects

Four carcinogenicity studies—in rats (two studies), mice, and beagles—have been conducted with glyphosate, each showing no evidence of any statistically significant glyphosate-related tumors (EPA 1993). The authors of a review study and EPA's Office of Pesticide Programs have concluded that glyphosate is not mutagenic and is not expected to pose any genotoxic hazard to humans (Li and Long 1988, EPA 1993). In later studies, some results reporting genotoxicity due to glyphosate have been reported (Williams et al. 2000). Williams et al. (2000) reviewed all reported information to date, and concluded that the more recent studies showing positive genotoxic results "used toxic dose levels, irrelevant endpoints/test systems, and/or deficient testing methodology." Based on a weight-of-evidence review of the entire database, they concluded that glyphosate does not pose a risk of heritable (leading to birth defects) or somatic (causing cancer) mutations in humans.

4.4.9 Hexazinone

Hexazinone is an herbicide. Provolt proposes the use of the 90% water-soluble powder formulation, Velpar®.

Noncarcinogenic Effects

In 1990, EPA set an RfD of 0.033 mg/kg/day for hexazinone, based on a NOEL of 10 mg/kg/day observed in a two-year rat-feeding study, with decreased body weight observed at the LOEL of 50 mg/kg/day (EPA 2000). This RfD incorporated an additional uncertainty factor of 3 (in addition to the standard factor of 100) in deriving the RfD from the NOEL, since a chronic study in dogs was not available and dogs appeared to be a more sensitive test species. In 1994, EPA's Office of Pesticide Programs reviewed a new one-year feeding study in dogs, and recommended an RfD of 0.05 mg/kg/day, based on a NOEL of 5 mg/kg/day, with changes in clinical chemistry and histopathology observed at the LOEL of 37.57 mg/kg/day (EPA 1994). The RfD of 0.05 mg/kg/day is used in the risk assessment.

In a teratology study in rats, the developmental NOEL was 100 mg/kg/day, with decreased fetal weights, and increased incidences of fetuses with no kidney papillae and unossified sternebrae, observed at a dose level of 400 mg/kg/day (EPA 1994). In a study in rabbits, the developmental NOEL was 50 mg/kg/day, with decreased fetal body weight and delayed ossification of extremities observed at the LOEL of 125 mg/kg/day (EPA 1994). In a two-generation reproduction study in rats, observations at a dose of 100 mg/kg/day included decreased maternal and pup weight, decreased maternal food consumption, and decreased pup survival; the NOEL was 10 mg/kg/day (EPA 1994). A three-generation reproduction study in rats showed decreased parental weights at a dietary level of 1,000 ppm (50 mg/kg/day) and decreased body weight gain in offspring at a dietary level of 2,500 ppm (125 mg/kg/day) (Kennedy and Kaplan 1984).

Hexazinone caused severe eye irritation and mild skin irritation in studies in rabbits (EPA 1994). It did not cause dermal sensitization in a study in guinea pigs (EPA 1994). When EPA (1994) compared the results of oral and dermal studies with hexazinone, they concluded that little or no dermal absorption was expected. No quantitative dermal absorption rate was available for hexazinone. However, EPA (1994) stated that comparison of dermal and oral toxicity study results indicated that little or no dermal absorption would occur. Therefore, a skin penetration of 1% was selected for use in the risk assessment.

Carcinogenic and Mutagenic Effects

EPA (1994) stated that hexazinone was not classifiable as to its carcinogenicity based on the results of existing studies, and that the RfD approach should be used to assess its risk to humans. A two-year feeding study in rats did not produce any evidence of oncogenicity at doses up to 2,500 ppm in diet (125 mg/kg/day) (EPA 1994). Equivocal evidence of liver tumors at a dose of 1,915 mg/kg/day in females was concluded from a two-year feeding study in mice; EPA (1994) stated the results were not entirely negative, but were not convincing.

In studies reviewed by EPA (1994), hexazinone was positive in one study for chromosomal aberrations, and was negative in two gene mutation studies in bacterial and mammalian cells and in a study for unscheduled DNA synthesis in rat hepatocytes. These results indicate that hexazinone may have a slight potential for mutagenic activity.

4.4.10 Horticultural Oil

Horticultural oil, consisting of paraffinic (alkane) hydrocarbon oil, also called mineral oil, is used as an insecticide. Provolt proposes to use Dormant Oil 435. The group of compounds regulated as mineral oil by EPA's Office of Pesticide Programs also includes petroleum compounds with CAS registry number 8002-05-9, which is an other ingredient on EPA's List 2 that comprises 8.5% of the Digon® 400 formulation of dimethoate.

Noncarcinogenic Effects

Mineral oil is approved for use as a food additive by the Food and Drug Administration, and is widely used in soaps and cosmetics (HSDB 2001). Subchronic feeding studies using mineral oil in rats and dogs resulted in dietary NOELs of 1,500 ppm in both species (Smith et al. 1995). In another study, oral administration of mineral oil to male rats three times a week for three months at a dose of 2 mL/kg did not produce any adverse effects (HSDB 2001). In a third 90-day study in rats, multifocal lipogranulomata in the mesenteric lymph nodes and liver of rats dosed at 6.4 mg/kg/day (Baldwin et al.1992). A later review of this observation reported that lipogranulomata in human mesenteric lymph nodes, spleen, and liver are common and generally considered to be clinically unimportant. Nash et al. (1996) reviewed the results of studies of dermal exposure to mineral oils, and concluded that there is no evidence of any hazard from topical exposure to mineral oils at any dose in several species tested, and that their conclusion is supported by the long and uneventful human use of white mineral oils in drug and non-drug topically applied products. The Food and Agriculture Organization of the United Nations (FAO 1998) and the World Health Organization (Margoni 1999) have set a temporary acceptable daily intake of 1 mg/kg/day for high viscosity mineral oils (which corresponds to the U.S. EPA definition of insecticidal mineral oils at 40 CFR 180.149); this value is used as the RfD in this risk assessment.

Amdur et al. (1991) stated that mineral oil is considered to be relatively nontoxic. Ingestion of mineral oil may interfere with the absorption of fat-soluble nutrients (HSDB 2001).

Paraffinic hydrocarbon oil caused slight skin irritation and mild eye irritation in tests in rabbits (Riverside/Terra 1995). It may cause allergic skin reactions in some individuals (Riverside/Terra

1995). Although no data on skin absorption were available, HSDB (2001) stated that mineral oil is poorly absorbed from the intestinal tract; therefore, a default value of 1% is considered acceptable for use in the risk assessment.

Carcinogenic and Mutagenic Effects

IARC (1987) concluded that highly refined mineral oils, such as those used in horticultural oil, are not classifiable as to their carcinogenicity in humans. They did not produce skin tumors in mice, as had been demonstrated with lesser or unrefined mineral oils. Both positive and negative mutagenicity test results have been reported for refined mineral oils (IARC 1987).

4.4.11 Permethrin

Permethrin is a synthetic pyrethroid insecticide. Provolt proposes to use the Pounce® 3.2EC formulation, which is an emulsifiable concentrate containing 38.4% permethrin. Pounce® 3.2EC also contains the other ingredients ethylbenzene (<2%), light aromatic solvent naphtha (<32.2%), and xylene (<10.2%), all of which are listed on EPA's List 2 (potentially toxic inerts, with high priority for testing) (FMC 1995).

Noncarcinogenic Effects

EPA (2000) set an oral RfD for permethrin of 0.05 mg/kg/day, based on a two-year rat-feeding study with a NOEL of 5 mg/kg/day, in which increased liver weights were observed at the LOEL of 25 mg/kg/day. In a one-year feeding study in dogs, effects at the LOEL of 100 mg/kg/day included increased alkaline phosphatase, increased liver weight, and hepatocellular swelling; the NOEL in this study was also 5 mg/kg/day (EPA 2000). Like esfenvalerate, permethrin is a Type II pyrethroid and is neurotoxic.

No developmental effects were found in a study in rats at the highest dose tested of 200 mg/kg, or in a study in rabbits at the highest dose tested of 400 mg/kg (EPA 2000). In a three-generation reproduction study in rats, the following effects were noted in offspring at the lowest dose tested of 25 mg/kg/day: centrilobular hepatocyte hypertrophy, cytoplasmic eosinophilia, and buphthalmos with persistent pupillary membranes (EPA 2000).

Permethrin did not affect humoral or cell-mediated immunity in a 28-day test in male rats at doses up to 125.7 mg/kg/day (Institóris et al. 1999).

Garey and Wolff (1997) reported that permethrin did not give any indication of causing endocrine disruption, as indicated by estrogenic activity, in tests using human endometrial cancer cells and breast cancer cells. However, Go et al. (1999) found that permethrin affected cell proliferation in a study using human breast cancer cells. In a third report, Saito et al. (2000) concluded that permethrin did not cause any significant estrogenic or anti-estrogenic effects based on three *in vitro* assays.

Dermal contact with permethrin can cause skin sensations such as numbing, burning, and tingling (FMC 1995). Permethrin is irritating to the skin and eyes (FMC 1995). Based on tests in rabbits and dogs, Snodgrass and Nelson (1982) predicted a dermal penetration rate of <8 percent for humans. Baynes et al. (1997) measured a dermal absorption rate of 1.2 to 1.7% for permethrin applied to mouse skin.

Carcinogenic and Mutagenic Effects

The National Research Council (1994) evaluated seven studies conducted to assess the carcinogenic potential of permethrin. Three studies in rats were negative, but the doses may not have been high enough to draw firm conclusions. In four studies in mice, two showed evidence of carcinogenicity, with increases in liver tumors in one study and lung adenomas and carcinomas in the second study. The Council concluded that permethrin was a possible human carcinogen, and recommended use of a cancer slope factor of 0.016 $(mg/kg/day)^{-1}$, based on the tumor incidence in the second mouse study. Permethrin was not mutagenic in several assays (EPA 1988, Djelic and Djelic 2000), but increased the number of chromosomal aberrations in one study in mice, and induced sister chromatid exchange and micronuclei in human lymphocytes *in vitro* (Institóris et al. 1999, Herrera et al. 1992).

4.4.12 Picloram

Picloram is an herbicide. Provolt proposes to use the Tordon® 22K formulation, containing 24.4% potassium salt of picloram as a liquid concentrate.

Noncarcinogenic Effects

In 1992, EPA set a chronic oral RfD of 0.07 mg/kg/day for picloram, based on a six-month feeding study in dogs with a NOEL of 7 mg/kg/day, in which increased liver weights were observed at the LOEL of 35 mg/kg/day (EPA 2000). In 1995, EPA's Office of Pesticide Programs reviewed this study again, and concluded that the NOEL should be 35 mg/kg/day, since increased liver weights were only observed in two males at that dose level (EPA 1995). They also recommended that the RfD should be based on the results of a chronic study in rats with a NOEL of 20 mg/kg/day, with observations at the LOEL of 60 mg/kg/day including increased liver weights in males and females and alterations in liver cells. The recommended RfD of 0.2 mg/kg/day is used in this risk assessment. Other chronic studies include a one-year feeding study in dogs with increased liver weights at 175 mg/kg/day, a two-year rat study with kidney and liver effects at 250 mg/kg/day, and a two-year mouse study with increased kidney weights at 1,000 mg/kg/day (EPA 1995).

No developmental toxicity was reported in two studies of picloram potassium salt at doses up to 400 mg/kg/day (picloram acid equivalent) in rabbits and 298 mg/kg/day (picloram acid equivalent) in rats (EPA 1995). In a two-generation reproduction study in rats, no reproductive effects were produced at the highest dose tested of 1,000 mg/kg/day, although effects on the kidneys were observed at the high dose, resulting in a parental NOEL of 200 mg/kg/day (EPA 1995). In a three-generation reproduction study in rats, reduced fertility was observed at a dose of 150 mg/kg/day; the NOEL was 50 mg/kg/day (EPA 2000).

Picloram was a moderate eye irritant, and was not a skin irritant or sensitizer in tests in laboratory animals (EPA 1995). However, the potassium salt of picloram and Tordon® 22K formulation have been shown to cause dermal sensitization in studies in guinea pigs, although this effect has not been demonstrated in humans (EPA 1995, Dow 2000). Dow (2000) stated that the Tordon® 22K formulation was a severe eye irritant. Extoxnet (2000) stated that skin absorption of picloram is minimal. A study in human volunteers concluded that only 0.2% of a dermally applied picloram dose was absorbed (HSDB 2001).

Carcinogenic and Mutagenic Effects

Based on negative results in chronic studies in rats and mice, EPA (1995) concluded that picloram acid and picloram potassium salt were not carcinogenic in humans. However, because of its metabolism to a compound thought to play a role in the carcinogenicity of the chemical di-(2-ethylhexyl)phthalate, EPA concluded that it was appropriate to quantitatively assess the cancer risk of the ethylhexyl ester of picloram. Provolt does not plan to use this ester, and proposes to use only a formulation containing the potassium salt of picloram.

All picloram compounds contain the manufacturing impurity hexachlorobenzene, which is a probable human carcinogen with a cancer slope factor of 1.7 $(mg/kg/day)^{-1}$ (EPA 1995). Hexachlorobenzene can be present in picloram at levels up to 100 ppm (0.01%) (EPA 1995). Dermal absorption of hexachlorobenzene is expected to be less than 23% (EPA 1995).

No evidence of mutagenicity was reported in 11 assays reviewed by EPA (1995) of picloram acid, isooctyl ester, and triisopropanolamine salt for gene mutation, chromosomal aberrations, or DNA damage. Extoxnet (2000) and HSDB (2001) reported two positive bacterial mutagenicity tests. These results suggest that picloram is either nonmutagenic or very weakly mutagenic.

4.4.13 Propargite

Propargite is an organosulfite miticide/acaricide. Provolt proposes to use Omite® CR, which contains 32% propargite as a wettable powder in water soluble bags.

Noncarcinogenic Effects

In 1990, EPA set a chronic oral RfD of 0.02 mg/kg/day, based on two studies: a two-year study in dogs in which no adverse effects were observed at the highest dose tested of 22.5 mg/kg/day; and a developmental study in rabbits in which maternal and fetotoxic effects were observed at the LOEL of 6 mg/kg/day and the NOEL was 2 mg/kg/day (EPA 2001). EPA's Office of Pesticide Programs recommended a chronic oral RfD of 0.04 mg/kg/day, based on a NOEL of 4 mg/kg/day where decreased body weight, decreased weight gain, and increased mortality were observed in males at a dose of 19 mg/kg/day in a two-year study in rats (EPA 2000a). The NOEL for females was 24 mg/kg/day. In a one-year study in beagle dogs, the NOEL was 5 mg/kg/day, and the LOEL was 38 mg/kg/day (EPA 2000b). The recommended RfD of 0.04 mg/kg/day is used in the risk assessment, since it is based on a more complete review of the currently available literature than the 1990 RfD, and is based on a NOEL lower than the lowest dose at which effects were observed in the study reviewed for the 1990 value.

In a developmental study in rabbits, an increased incidence of fused sternebrae in offspring was observed at a dose of 10 mg/kg/day, with a NOEL of 8 mg/kg/day for developmental effects (EPA 2000b). No developmental effects were reported at the highest dose tested of 105 mg/kg/day in a study in rats (EPA 2000b). In a two-generation reproduction study in rats, NOELs of 4 and 20 mg/kg/day were reported, based on systemic effects to parents and offspring, respectively, at the next higher doses of 20 and 40 mg/kg/day. No effects on reproduction were observed at the highest dose tested of 40 mg/kg/day (EPA 2000b).

EPA (2000b) reported the results of studies demonstrating that propargite is corrosive to the eyes and skin, and caused dermal sensitization in guinea pigs. The Omite® CR formulation was associated with an outbreak of dermatitis among orange pickers in 1986 (Hayes and Laws 1991). In a study in rats, a dermal absorption factor of 14.5% was measured for 8 hours exposure to propargite (EPA 2000b).

Carcinogenic and Mutagenic Effects

Propargite is considered a possible human carcinogen, based on findings of tumors of the gastrointestinal tract in two studies in rats. A cancer slope factor of 0.201 $(mg/kg/day)^{-1}$ was calculated (EPA 2000b). Two additional studies, one in rats and one in mice, were negative for carcinogenicity (EPA 2000b).

Propargite was negative in five studies reviewed by EPA (2000b) for gene mutation, chromosomal aberrations, and DNA damage and repair, and in studies for gene mutation in bacteria reviewed by Hayes and Laws (1991).

4.4.14 Triclopyr

Triclopyr is an herbicide. Provolt proposes to use Garlon® 4, containing 61.6% triclopyr butoxyethyl ester.

Noncarcinogenic Effects

EPA's Office of Pesticide Programs has recommended an oral RfD of 0.5 mg/kg/day for triclopyr, based on a two-generation reproduction study in rats with a NOEL of 5 mg/kg/day, in which kidney effects were observed in parental rats at a dose of 25 mg/kg/day (EPA 1998). In a two-year rat-feeding study, decreased hemoglobin and erythrocytes, and increased absolute and relative kidney weights, were observed in males at the LOEL of 36 mg/kg/day; the NOEL was 12 mg/kg/day (EPA 1998). In a two-year study in mice, NOELs for male and female mice were 143 and 135 mg/kg/day, respectively, with decreased weight gain observed at higher doses (EPA 1998). A NOEL of 10 mg/kg/day was found in a chronic study in dogs, with decreased weight gain, hematological and clinical chemistry findings, and liver effects at a dose of 20 mg/kg/day (EPA 1998).

Developmental studies have been conducted with both the acid and ester forms of triclopyr. In rats, the amine salt produced skeletal anomalies at a dose of 300 mg/kg/day, with a developmental NOEL of 100 mg/kg/day (EPA 1998). In rabbits, the developmental NOEL for the amine salt was

30 mg/kg/day, with decreased viable offspring at a dose of 100 mg/kg/day (EPA 1998). The ester was associated with skeletal abnormalities and effects on survival in rabbits at a dose of 100 mg/kg/day; the NOEL for these effects was 30 mg/kg/day (EPA 1998). In a two-generation reproduction study in rats using triclopyr acid, the reproductive NOEL was 25 mg/kg/day, based on decreases in litter size, body weight, weight gain, and survival at a dose of 250 mg/kg/day (EPA 1998).

A dermal absorption study in humans showed that approximately 1.65% of applied triclopyr was absorbed in eight hours (EPA 1998). Dermal irritation, skin sensitization, and eye irritation may result from exposure to the Garlon® 3A and Garlon® 4 formulations (Dow AgroSciences 1999, Dow AgroSciences 2001). The triethylamine salt was corrosive in an eye irritation study in rabbits, while the butoxyethyl ester caused only minimal irritation (EPA 1998). Both the triethylamine salt and the butoxyethyl ester were non-irritating to the skin of rabbits (EPA 1998). Both forms caused dermal sensitization in guinea pigs (EPA 1998).

Carcinogenic and Mutagenic Effects

In a rat oncogenicity study, a statistically significant increase in mammary tumors was observed when the number of adenomas (one) and adenocarcinomas (four) were combined for the high-dose females (36 mg/kg/day). However, the researchers reported that the incidence was within the range of historical controls, and that the statistically significant result was due in part to the low incidence (zero) of mammary tumors in control rats (Dow 1987). In a study in mice, females had a trend toward increased mammary gland tumors, but there was no statistical significance when compared with controls (EPA 1998). EPA (1998) stated that triclopyr was not classifiable as to its carcinogenicity, and that the overall evidence was marginal: not entirely negative, yet not convincing.

Triclopyr has been non-mutagenic in all of the various systems in which it has been tested, except for one very weak positive response with questionable statistical significance in a rat dominant lethal study. However, negative data were obtained in a dominant lethal study in mice with the same high dose level (EPA 1998).

4.4.15 Other Ingredients

The Bureau of Land Management decided that any pesticide formulations containing ingredients on EPA's List 1 (inerts of toxicologic concern) or List 2 (potentially toxic inerts, with high priority for testing) would be further evaluated. Specifically, the risks from those other ingredients would be included in the risk assessment, along with the active ingredient.

The Digon® 400 formulation of dimethoate contains cyclohexanone and petroleum distillates, which are on List 2. The Asana® XL formulation of esfenvalerate and the Pounce® 3.2EC formulation of permethrin contain ethylbenzene and xylene, which are on List 2. Pounce® 3.2EC also contains light aromatic solvent naphtha, which is on List 2.

The following paragraphs present the hazard analysis for these other ingredients.

Cyclohexanone

Cyclohexanone is found as an other ingredient in the Digon® 400 formulation of dimethoate (35%). Cyclohexanone is considered by EPA to be a potentially toxic inert ingredient with a high priority for testing.

Chronic administration of cyclohexanone to mice and rats in drinking water led to increased mortality in mice at a concentration of 13,000 mg/L (IARC 1989). Rats who ingested drinking water with cyclohexanone concentrations higher than 3,300 mg/L had dose-related body weight loss (EPA 2001). This NOEL corresponds to a dose level of 462 mg/kg/day. The LOEL was 6,500 mg/L, or 910 mg/kg/day. Based on this study, EPA (2001) set a chronic oral RfD of 5 mg/kg/day for cyclohexanone Gosselin et al. (1984) characterized cyclohexanone as a weak central nervous system depressant.

A developmental study in mice showed no maternal or developmental effects at a high dose of 50 mg/kg/day (IARC 1989). Another mouse developmental study reported reduced maternal weight gain, decreased pup weights, and maternal mortality at a dose of 2,200 mg/kg/day (IARC 1989). A multi-generation reproduction study in mice reported that cyclohexanone affected the viability and growth of offspring at a dietary concentration of 1% (10,000 ppm) (IARC 1989). This level is estimated to correspond to a dose of 1,500 mg/kg/day.

Dermal and eye irritation in rabbits were observed after exposure to cyclohexanone (Gupta et al. 1979). One report was made of a case in which dermal sensitization in a human was attributed to cyclohexanone (IARC 1999). However, a test for dermal sensitization in guinea pigs was negative (IARC 1989). IARC (1999) reported that the permeation rate of cyclohexanone liquid through human skin was 37 to 69 mg/cm^2 per hour, indicating little dermal absorption.

Cyclohexanone administered to mice and rats in their drinking water for two years caused slight increases in tumors in both species, but only at the lowest dose tested in each case, which is an unusual finding in a carcinogenicity assay (IARC 1999). Drinking water concentrations were 6,500, 13,000, and 25,000 mg/L for mice; and 3,300 and 6,500 mg/L for rats. IARC has concluded that cyclohexanone is not classifiable as to its carcinogenicity for humans.

Cyclohexanone did not cause gene mutations in bacterial cells, but was positive for inducing chromosomal aberrations in cultured human cells and in treated rats (IARC 1999). In a test in Chinese hamster ovary cells, cyclohexanone induced sister chromatid exchange and gene mutation, but only in the absence of metabolic activation. It was negative for chromosomal aberrations (Aaron et al. 1985). A test for induction of sex-linked recessive lethal mutations in the fruit fly was negative (EPA 1986).

Ethylbenzene

Ethylbenzene is found as an other ingredient in the Asana® XL formulation of esfenvalerate (<1%) and the Pounce® 3.2EC formulation of permethrin (<2%). Ethylbenzene is considered by EPA to be a potentially toxic inert ingredient with a high priority for testing.

EPA (2001) set an oral RfD of 0.1 mg/kg/day for ethylbenzene, based on liver and kidney toxicity in a rat study at a dose of 291 mg/kg/day. The NOEL was 97.1 mg/kg/day.

In rats, oral exposure to 500 mg/kg affected the reproductive cycle (Von Burg 1992).

According to Von Burg (1992), studies of inhalation exposure suggest that ethylbenzene may cause central nervous system effects.

Skin irritation and slight eye irritation resulted from ethylbenzene application to the skin and eyes of rabbits (Von Burg 1992). A dermal absorption rate in mice of 3.4% of the applied dose from a 4-hour exposure was measured by Susten et al. (1990).

EPA (2001) lists ethylbenzene as not classifiable as to human carcinogenicity. Two-year inhalation studies were conducted in rats and mice. In the high-exposure groups (750 ppm, 6 hours per day, 5 days per week), ethylbenzene inhalation induced neoplasms in the kidneys and testes of rats, in the lungs of male mice, and in the liver of female mice (Chan et al. 1998).

Ethylbenzene showed no mutagenic activity in Ames assays, other bacterial mutation assays, and a mitotic gene conversion test in *Saccharomyces cerevisiae*. However, one test showed increased sister chromatid exchanges in human lymphocyte culture (EPA 2001).

Light Aromatic Solvent Naphtha

Light aromatic solvent naphtha is found as an other ingredient in the Pounce® 3.2EC formulation of permethrin (<32.2%). Light aromatic solvent naphtha is considered by EPA to be a potentially toxic inert ingredient with a high priority for testing. The term “light aromatic solvent naphtha” refers to a group of compounds, consisting mainly of C_8 through C_{10} aromatic hydrocarbons. Naphthalene is a representative member of this group.

EPA (2001) has set a chronic oral RfD of 0.02 mg/kg/day for naphthalene, based on decreased body weight in male rats at a dose of 142 mg/kg/day in a subchronic study; the NOEL was 71 mg/kg/day. Immune system effects were observed at higher doses in this study. Human experience in accidental overexposures suggests that the development of hemolytic anemia and cataracts may be associated with naphthalene, but available data do not provide sufficient information to characterize the dose-response relationship for these endpoints (EPA 2001).

An inhalation teratology study in mice using light aromatic solvent naphtha resulted in a NOEL of 100 ppm in air, decreased maternal and fetal weight gain at a level of 500 ppm, and fetal mortality, skeletal effects, and cleft palate at a level of 1,500 ppm (McKee et al. 1990). Single oral doses of 16 mg/kg naphthalene in pregnant rabbits produced cataracts and retinal damage in the offspring (HSDB 2001).

No evidence of neurotoxicity was found in six-month inhalation study in rats using light aromatic solvent naphtha at concentrations up to 1,500 ppm (Douglas et al. 1993).

Dermal irritation, eye irritation, cataracts, and skin sensitization have been linked to naphthalene exposure (HSDB 2001).

EPA (2001) has classified naphthalene as a possible human carcinogen, based on evidence that suggests it may produce tumors when inhaled. In a study in mice, mostly benign tumors and one adenocarcinoma were produced at the highest dose tested (EPA 2001). Insufficient data preclude development of a cancer slope factor for use in human risk assessment (EPA 2001). Naphthalene has produced a mix of results in mutagenicity assays in bacterial, insect, and mammalian systems, although most of the studies were negative (EPA 2001).

Petroleum Distillates

Petroleum distillates are an other ingredient in the Digon® 400 formulation of dimethoate (8.5%). The toxicity data presented in the discussion of horticultural oil (Section 4.4.11) are also appropriate to the hazard assessment of petroleum distillates.

Xylene

Xylene is an other ingredient in the Asana® XL formulation of esfenvalerate (<3%) and the Pounce® 3.2EC formulation of permethrin (<10.2%). EPA considers xylene to be a potentially toxic inert ingredient, with a high priority for testing.

EPA (2001) set an oral RfD of 2 mg/kg/day for xylene, based on a study in rats in which hyperactivity, decreased body weight, and increased mortality were observed at a dose of 357 mg/kg/day. The NOEL was 179 mg/kg/day. Exposure to xylene has been associated with central nervous system effects (IARC 1989).

Xylene is fetotoxic and teratogenic in mice at high oral doses, but EPA (2001) stated that the calculated RfD should be protective of these effects.

Skin and eye irritation have been reported in studies of human volunteers exposed to xylene (IARC 1989). McDougal et al. (1990) found that 3.9% of the received dose of xylene was absorbed through the skin from a combined dermal and inhalation 4-hour exposure.

EPA (2001) considers xylene to be not classifiable as to its carcinogenicity. In an oral study in mice, xylene did not result in significant increases in tumor response incidence. However, a study in rats produced equivocal findings. Limited dermal studies have indicated that xylene may be a promoter or co-carcinogen for skin cancer, but not a primary carcinogen. Insufficient data have been generated to reach a conclusion regarding xylene's potential for carcinogenicity (EPA 2001, ATSDR 1995).

4.4.16 Fertilizers

Four types of fertilizer compounds are proposed for use by Provolt: ammonium sulfate, ammonium phosphate, ammonium nitrate, and potassium nitrate.

With the exception of possible nitrate ingestion exposure, no exposures associated with systemic health impacts to humans would be expected from the proposed fertilizers or their degradation products. This is consistent with the nature of the chemicals (see following discussions) and statements such as EPA's position on the use of fertilizers as other ingredients in pesticide products: "The fertilizer components of these [granular pesticide] products are considered analogous to the innocuous inert ingredients described above with the exception of eye irritation" (EPA 2001a). Fertilizer salts are associated with a potential for skin, eye, and respiratory tract irritation, warranting the use of personal protective equipment to minimize direct contact with granules and dusts.

Ammonium Sulfate

Sax and Lewis (1989) reported an oral toxic dose in humans of 1,500 mg/kg for ammonium sulfate. It is regulated by the Food and Drug Administration (21 CFR 184.1143) for use in food, and is generally recognized as safe with a limitation of 0.15% in baked goods and 0.1% in gelatins and puddings when used in accordance with good manufacturing practices (Lewis 1989). Ammonium sulfate may be a slight eye, skin, and inhalation irritant (J.R. Simplot 1985).

EPA has set a secondary maximum contaminant level for sulfate in drinking water of 250 mg/L, based on taste and odor. This is not an enforceable standard, but a recommendation for state and local water systems. Sulfate occurs naturally in drinking water. Ingesting high levels of sulfate from drinking water ($\geq$1,200 mg/L) or other sources may be associated with diarrhea (EPA 1999).

Monoammonium Phosphate (MAP)

MAP is used as a general purpose food additive and is generally recognized as safe by the Food and Drug Administration (HSDB 2001).

The Food and Agriculture Organization stated that a total dietary phosphorus level of 30 mg/kg/day is considered safe (HSDB 2001).

Ammonium salts such as MAP can cause irritation and swelling from contact with the eye (HSDB 2001). These compounds are also irritating to the skin and respiratory tract (HSDB 2001).

Ammonium Nitrate

No evidence of chromosomal aberrations was identified in a study in which mice were dosed with up to 417 mg/kg ammonium nitrate (Nechkina 1992).

In the environment, ammonium nitrate degrades to form ammonium and nitrate ions.

EPA (2001b) has set a chronic oral RfD of 1.6 mg/kg/day for nitrate, based on human epidemiological surveys that demonstrated a drinking water NOEL of 1.6 mg/L and a LOEL of 1.8 mg/L for early clinical signs of methemoglobinemia in infants. Methemoglobinemia results in decreased oxygen transport from lungs to the body's tissues. EPA regulates the amount of nitrate in drinking water. The maximum contaminant level (MCL) for total nitrate and nitrite in drinking water is 10 mg nitrogen (N, from nitrate and nitrite) per liter (10 mg N/L × 4.45 = 44.5 mg nitrate/L).

Nitrate is a normal component of the human diet (EPA 2001b). A typical daily intake by an adult in the U.S. is about 75 mg/day. Nitrate has not been shown to be carcinogenic in laboratory animals except when the animal simultaneously receives nitrosable amines (Fan and Steinberg 1996).

Ammonium nitrate can be irritating to the skin and respiratory tract (HSDB 2001).

Potassium Nitrate

Oral administration studies in laboratory animals concluded that potassium nitrate was associated with reproductive and developmental effects at levels of 25,000 ppm in the diet during the second trimester in rats, and at 30,000 mg/L in drinking water in a study in guinea pigs (HSDB 2001).

Potassium nitrate dissolves to form potassium and nitrate. Potassium compounds are ubiquitous in the earth's crust, and the element is naturally found in the human bloodstream. Acute oral potassium poisoning is rare, since large doses usually induce vomiting (HSDB 2001). The toxic properties of nitrate to humans are summarized under "Ammonium Nitrate," above.

4.5 Data Gaps

For the endpoints evaluated in this quantitative risk assessment, there are no data gaps in the information available for acephate, chlorothalonil, chlorpyrifos, diazinon, dimethoate, esfenvalerate, glyphosate, permethrin, picloram, and propargite.

No studies of dermal absorption were available for dicamba. USDA (1984) recommended a value of 10% as a conservative assumption. This value is used in the risk assessment.

Hexazinone's carcinogenic potential is unknown, with equivocal results from one study in mice and negative results from a study in rats. Cancer risks are not quantified for this pesticide.

Conclusive information was not available on triclopyr's potential for carcinogenicity. Therefore, no judgment was made as to whether it is potentially carcinogenic, and no quantitative cancer risk analysis was conducted.

No dermal absorption factor was identified for cyclohexanone. A value of 10% was selected for use in the risk assessment. Carcinogenicity findings for cyclohexanone were inconclusive. No quantitative analysis of the compound's cancer risk is conducted.

Inhalation studies of ethylbenzene in rats and mice resulted in some tumors in the high-exposure groups, although EPA lists it as not classifiable as to human carcinogenicity. No cancer risk assessment is conducted for this chemical.

Although naphthalene is considered a possible human carcinogen, the available data do not allow calculation of a cancer slope factor; therefore, no quantitative estimate of cancer risk from light aromatic solvent naphtha compounds is made. No dermal absorption data were available, so a default value of 10% was selected for use in the risk assessment.

For xylene, one negative and one equivocal carcinogenicity study were reported, and dermal studies have indicated a potential for xylene to be a promoter or co-carcinogen for skin cancer. Due to the lack of conclusive information, no judgment was made in this risk assessment as to whether xylene is potentially carcinogenic, and no quantitative cancer risk analysis was conducted for it.

No dermal absorption data were available for the fertilizers. A value of 1% was used in the risk assessment.

4.6 References

ATSDR. See Agency for Toxic Substances and Disease Registry.

EPA. See U.S. Environmental Protection Agency.

FAO. See Food and Agriculture Organization.

HSDB. See Hazardous Substances Databank.

IARC. See International Agency for Research on Cancer.

USDA. See U.S. Department of Agriculture.

WHO. See World Health Organization.

Section 4.2

Amdur, M.O., J. Doull, and C.D. Klaassen. 1991. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 4th ed. Pergamon Press. New York.

Lu, F.C. 1985. *Basic Toxicology: Fundamentals, Target Organs, and Risk Assessment.* Hemisphere Publishing Corporation. New York.

U.S. Environmental Protection Agency. 1989. Risk assessment guidance for Superfund, Volume I: Human health evaluation manual (Part A). EPA/540/1-89/002. Office of Emergency and Remedial Response. Washington, DC.

Section 4.3

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

U.S. Environmental Protection Agency. 1989. Risk assessment guidance for Superfund, Volume I: Human health evaluation manual (Part A). EPA/540/1-89/002. Office of Emergency and Remedial Response. Washington, DC.

U.S. Environmental Protection Agency. 2000. Region 4 human health risk assessment bulletins–Supplement to RAGS. Waste Management Division. Atlanta, GA. www.epa.gov/region04/waste/oftecser/healtbul.htm

U.S. Environmental Protection Agency. 2001. Integrated risk information system: On-line database. Office of Research and Development. Cincinnati, OH.

Section 4.4

Acephate (4.4.1)

Agrochemicals Handbook. 1994. On-line database. Dialog Information Systems. Palo Alto, CA.

Behera, B.C., and S.P. Bhunya. 1989. Studies on the genotoxicity of asataf (acephate), an organophosphate insecticide, in a mammalian in vivo system. Mutation Research 223:287-293.

Carver, J.H., J. Bootman, M.C. Cimino, H.J. Esber, P. Kirby, B. Kirkhart, Z.A. Wong, and J.A. MacGregor. 1985. Genotoxic potential of acephate technical: In vitro and in vivo effects. Toxicology 35:125-142.

Chevron Chemical Co. 1985. Acute delayed neurotoxic study in chickens with Chevron acephate technical: Final report. (Prepared by Wildlife International Ltd., St. Michaels, MD.) Ortho Agricultural Chemicals Division. Richmond, CA.

Chevron Chemical Co. 1987. Orthene: Greenhouse worker exposure. Ortho Agricultural Chemicals Division. Richmond, CA.

Hour, T., L. Chen, and J. Lin. 1998. Comparative investigation on the mutagenicities of organophosphate, phthalimide, pyrethroid and carbamate insecticides by the Ames and lactam tests. Mutagenesis 13(2):157-166.

O'Malley, M., P. Rodriguez, and H.I. Maibach. 1995. Pesticide patch testing: California nursery workers and controls. Contact Dermatitis 32:61-63.

Salama, A.K., N.M. Bakry, and M.B. Abou-Donia. 1993. A review article on placental transfer of pesticides. Journal of Occupational Medicine and Toxicology 2(4):383-397.

U.S. Environmental Protection Agency. 1987. Guidance for the reregistration of pesticide products containing acephate as the active ingredient. Office of Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system: On-line database. Office of Research and Development. Cincinnati, OH.

Valent USA Corporation. 1994. Material safety data sheet: ORTHENE® Turf, Tree, and Ornamental WSP. Walnut Creek, CA.

Waters, M.D., S.S. Sandhu, V.F. Simmon, K.E. Mortelmans, A.D. Mitchell, T.A. Jorgenson, D.C.L. Jones, R. Valencia, and N.E. Garrett. 1982. Study of pesticide genotoxicity. In *Genetic Toxicology*, R.A. Fleck and A. Hollaender, eds. Plenum Publishing Corp.

Wilson, B.W., J.D. Henderson, T.P. Kellner, S.F. McEuen, L.C. Griffis, and J.C. Lai. 1990. Acetylcholinesterase and neuropathy target esterase in chickens treated with acephate. NeuroToxicology 11:483-492.

Chlorothalonil (4.4.2)

Boman, A., J. Montelius, R.L. Rissanen, and C. Liden. 2000. Sensitizing potential of chlorothalonil in the guinea pig and the mouse. Abstract: Contact Dermatitis 43(5):273-9.

Mizens, M., J.C. Killeen, Jr., and G.L. Eilrich. 1998. The mutagenic potential of chlorothalonil: In vivo chromosome aberration studies. Mutation Research 403:269-272.

U.S. Environmental Protection Agency. 1999. Reregistration eligibility decision (RED): Chlorothalonil. EPA 738-R-99-004. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. On-line database. Cincinnati, OH.

Zeneca Ag Products. 1998. Material safety data sheet: Bravo 500. Wilmington, DE.

Chlorpyrifos (4.4.3)

Blakley, B.R., M.J. Yole, P. Brousseau, H. Boermans, and M. Fournier. 1999. Effect of chlorpyrifos on immune function in rats. Abstract: Veterinary and Human Toxicology 41(3):140-144.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Thrasher, J.D., R. Madison, and A. Broughton. 1993. Immunologic abnormalities in humans exposed to chlorpyrifos: Preliminary observations. Archives of Environmental Health 48(2):89-92.

Tos-Luty, S., J. Latuszynska, J. Halliop, A. Tochman, E. Przylepa, E. Bychawski, and D. Obuchowska. 1994. Skin penetration of selected pesticides. Abstract: Annals of Agricultural and Environmental Medicine 1(1):57-67.

U.S. Environmental Protection Agency. 2000a. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

U.S. Environmental Protection Agency. 2000b. Human health risk assessment: Chlorpyrifos. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000c. Toxicology chapter for chlorpyrifos. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Vinggaard, A.M., V. Breinholt, and J.C. Larsen. 1999. Screening of selected pesticides for oestrogen receptor activation in vitro. Abstract: Food Additives and Contaminants: Analysis, Surveillance, Evaluation, Control 16(12):533-542.

Diazinon (4.4.4)

Abu-Qare, A.W., C.F. Brownie, and M.B. Abou-Donia. 1999. Inhibition and recovery of maternal and fetal cholinesterase enzymes following a single dermal dose of diazinon alone, or in combination with methyl parathion in Sprague-Dawley rats. Abstract: Toxicologist 48(1-S):150.

Platte Chemical Co. 1994. Material safety data sheet: Clean Crop Diazinon 50W Insecticide. Fremont, NE.

U.S. Environmental Protection Agency. 2000. Diazinon: Revised HED human health risk assessment for the Reregistration Eligibility Decision (RED). Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Dicamba (4.4.5)

Micro Flo Company. 1999. Material safety data sheet: Banvel®. Memphis, TN.

U.S. Department of Agriculture. 1984. Pesticide background statements, Volume I: Herbicides. Agriculture Handbook Number 633. U.S. Forest Service. Washington, DC.

U.S. Environmental Protection Agency. 1999. Dicamba (3,6-dichloro-o-anisic acid): Pesticide tolerance. Federal Register 64(3):759-769. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Dimethoate (4.4.6)

Aly, N.M., and K.S. El-Gendy. 2000. Effect of dimethoate on the immune system of female mice. Journal of Environmental Science and Health B35(1):77-86.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Institóris, L., O. Siroki, and I. Dési. 1995. Immunotoxicity study of repeated small doses of dimethoate and methylparathion administered to rats over three generations. Human and Experimental Toxicology 14:879-883.

Schena, D., and A. Barba. 1992. Erythema-multiforme-like contact dermatitis from dimethoate. Abstract: Contact Dermatitis 27(2):116-117.

Srivastava, M.K., and R.B. Raizada. 1996. Development effect of technical dimethoate in rats: Maternal and fetal toxicity evaluation. Abstract: Indian Journal of Experimental Biology 34(4):329-333.

U.S. Environmental Protection Agency. 1999. Dimethoate: The updated, revised HED chapter of the Reregistration Eligibility Decision Document (RED). Office of Pesticide Programs. Washington, DC.

Wilbur-Ellis Company. 1995. Material safety data sheet: Digon 400 systemic insecticide - miticide. Fresno, CA.

Wlodarczyk, B., B. Biernacki, and M. Minta. 1992. Spermatotoxicity of dimethoate in rabbits. Abstract: Med Weter 48(11):512-514.

Esfenvalerate (4.4.7)

Cabral, J.R.P., and D. Galendo. 1990. Carcinogenicity study of the pesticide fenvalerate in mice. Cancer Letters 49:13-18.

Carbonell, E., M. Puig, N. Xamena, A. Creus, and R. Marcos. 1989. Mitotic arrest induced by fenvalerate in human lymphocyte cultures. Toxicology Letters 48:45-48.

Du Pont Agricultural Products. 1999. Material safety data sheet: "Asana" XL insecticide. Wilmington, DE.

Eells, J.T., and M.L. Dubocovich. 1988. Pyrethroid insecticides evoke neurotransmitter release from rabbit striatal slices. The Journal of Pharmacology and Experimental Therapeutics 246(2):514-521.

Go, V., J. Garey, M.S. Wolff, and B.G. Pogo. 1999. Estrogenic potential of certain pyrethroid compounds in the MCF-7 human breast carcinoma cell line. Abstract: Environmental Health Perspectives 107(3):173-177.

Grissom, R.E., Jr., C. Brownie, and F.E. Guthrie. 1985. Dermal absorption of pesticides in mice. Pesticide Biochemistry and Physiology 24:119-123.

Malaviya, M., R. Husain, P.K. Seth, and R. Husain. 1993. Perinatal effects of two pyrethroid insecticides on brain neurotransmitter function in the neonatal rat. Abstract: Veterinary and Human Toxicology 35(2):119-122.

Morgan, R.P. 1996. Toxicology of pyrethroids. In *Pesticide Poisoning Handbook.* http://hammock.ifas.ufl.edu/txt/fairs/19729.

Okuno, Y., T. Seki, S. Ito, H. Kaneko, T. Watanabe, T. Yamada, and J. Miyamoto. 1986. Differential metabolism of fenvalerate and granuloma formation: II—Toxicological significance of a lipophilic conjugate from fenvalerate. Toxicology and Applied Pharmacology 83:157-169.

Parker, C.M., C.B. McCullough, J.B.M. Gellatly, and C.D. Johnston. 1983. Toxicologic and carcinogenic evaluation of fenvalerate in the B6C3F1 mouse. Fundamental and Applied Toxicology 3:114-120.

Pati, P.C., and S.P. Bhunya. 1989. Cytogenetic effects of fenvalerate in mammalian in vivo test system. Mutation Research 222:149-154.

Puig, M., E. Carbonell, N. Xamena, A. Creus, and R. Marcos. 1989. Analysis of cytogenetic damage induced in cultured human lymphocytes by the pyrethroid insecticides cypermethrin and fenvalerate. Mutagenesis 4(1):72-74.

Shehata-Karam, H., N.A. Monteiro-Riviere, and F.E. Guthrie. 1988. In vitro penetration of pesticides through human newborn foreskin. Toxicology Letters 40:233-239.

U.S. Environmental Protection Agency. 1997. Fenvalerate; Pesticide tolerances. Federal Register 62(228):63019-63027. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

World Health Organization. 1990. Environmental health criteria 95: Fenvalerate. International Program on Chemical Safety, WHO. Geneva.

Glyphosate (4.4.8)

Blakley, B.R. 1997. Effect of Roundup and Tordon 202C herbicides on antibody production in mice. Abstract: Veterinary and Human Toxicology 39(4):204-206.

Li, A.P., and T.J. Long. 1988. An evaluation of the genotoxic potential of glyphosate. Fundamental and Applied Toxicology 10:537-546.

Maibach, H.I. 1986. Irritation, sensitization, photoirritation and photosensitization assays with a glyphosate herbicide. Contact Dermatitis 15:152-156.

Monsanto Company. 2001. Material safety data sheet: Roundup Original [TM] Herbicide. St. Louis, MO.

U.S. Environmental Protection Agency. 1993. Reregistration eligibility decision: Glyphosate. EPA 738-R-93-014. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Wester, R.C., J. Melendres, R. Sarason, J. McMaster, and H.I. Maibach. 1991. Glyphosate skin binding, absorption, residual tissue distribution, and skin decontamination. Fundamental and Applied Toxicology 16:725-732.

Wester, R., D. Quan, and H.I. Maibach. 1996. In vitro percutaneous absorption of model compounds glyphosate and malathion from cotton fabric into and through human skin. Abstract: Food and Chemical Toxicology 34(8):731-735.

Williams, G.M., R. Kroes, and I.C. Munro. 2000. Safety evaluation and risk assessment of the herbicide Roundup and its active ingredient, glyphosate, for humans. Regulatory Toxicology and Pharmacology 31:117-165.

Hexazinone (4.4.9)

Kennedy, G.L., and A.M. Kaplan. 1984. Chronic toxicity, reproductive, and teratogenicity studies of hexazinone. Fundamental and Applied Toxicology 4:960-971.

U.S. Environmental Protection Agency. 1994. Reregistration eligibility decision (RED): Hexazinone. EPA 738-R-94-022. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. Office of Research and Development. Cincinnati, OH.

Horticultural Oil (4.4.10)

Amdur, M.O., J.D. Doull, and C.D. Klaassen. 1991. *Casarett and Doull's Toxicology: The Basic Science of Poisons.* 4th ed. Pergamon Press. New York.

Baldwin, M.K., P.H. Berry, D.J. Esdaile, S.L. Linnett, J.G. Martin, G.C. Peristianis, R.A. Priston, B.J. Simpson, and J.D. Smith. 1992. Feeding studies in rats with mineral hydrocarbon food grade white oils. Abstract: Toxicologic Pathology 20(3 Pt 1):426-435.

Food and Agriculture Organization. 1998. Joint FAO/WHO Expert Committee on Food Additives: Fifty-first meeting. Geneva. http://www.who.int/pcs/jecfa/summary_51.htm

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

International Agency for Research on Cancer. 1987. Mineral oils: Untreated and mildly treated oils (Group 1), highly refined oils (Group 3). IARC Monographs, Supplement 7:252.

Margoni, D. 1999. White oils and the food industry. British Food Journal 101(3):229-237.

Nash, J.F., S.D. Gettings, W. Diembeck, M. Chudowski, and A.L. Kraus. 1996. A toxicological review of topical exposure to white mineral oils. Abstract: Food Chemistry and Toxicology 34(2):213-225.

Riverside/Terra Corp. 1995. Material safety data sheet: Dormant Oil 435. Sioux City, IA.

Smith, J.H., M.G. Bird, S.C. Lewis, J.J. Freeman, G.K. Hogan, and R.A. Scala. 1995 Subchronic feeding study of four white mineral oils in dogs and rats. Abstract: Drug Chemistry and Toxicology 18(1):83-103.

Permethrin (4.4.11)

Baynes, R.E., K.B. Halling, and J.E. Riviere. 1997. The influence of diethyl-m-toluamide (DEET) on the percutaneous absorption of permethrin and carbaryl. Abstract: Toxicology and Applied Pharmacology 144(2):332-339.

Djelic, N., and D. Djelic. 2000. Evaluation of cytotoxic and genotoxic effects of permethrin using in vitro micronucleus test. Abstract: Acta Veterinaria-Beograd 50(4):263-269.

FMC Corporation. 1995. Material safety data sheet: Pounce® 3.2 EC Insecticide. Agricultural Products Group. Philadelphia, PA.

Garey, J., and M. Wolff. 1997. Estrogenic and antiprogestagenic activities of pyrethroid insecticides. Abstract: Biochemical & Biophysical Research Communications 25(3):855-859.

Go, V., J. Garey, M.S. Wolff, and B.G. Pogo. 1999. Estrogenic potential of certain pyrethroid compounds in the MCF-7 human breast carcinoma cell line. Abstract: Environmental Health Perspectives 107(3):173-177.

Herrera, A., C. Barrueco, C. Caballo, and E. de la Peña. 1992. Effect of permethrin on the induction of sister chromatid exchanges and micronuclei in cultured human lymphocytes. Environmental and Molecular Mutagenesis 20:218-222.

Institóris, L., U. Undeger, O. Siroki, M. Nehéz, and I. Dési. 1999. Comparison of detection sensitivity of immuno- and genotoxicological effects of subacute cypermethrin and permethrin exposure in rats. Abstract: Toxicology137(1):47-55.

National Research Council. 1994. *Health Effects of Permethrin-Impregnated Army Battle-Dress Uniforms.* National Academy Press. Washington, DC.

Saito, K., Y. Tomigahara, N. Ohe, N. Isobe, I. Nakatsuka, and H. Kaneko. 2000. Lack of significant estrogenic or antiestrogenic activity of pyrethroid insecticides in three in vitro assays based on classic estrogen receptor alpha-mediated mechanisms. Abstract: Toxicological Sciences 57(1):54-60.

Snodgrass, H.L., and D.C. Nelson. 1982. Dermal penetration and distribution of 14C-labeled permethrin isomers, study no. 75-51-0351-83. U.S. Army Environmental Hygiene Agency. Aberdeen Proving Ground, MD.

U.S. Environmental Protection Agency. 1988. Peer review of permethrin. Office of Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Picloram (4.4.12)

Dow AgroSciences. 2000. Material safety data sheet: Tordon* 22K herbicide. Indianapolis, IN.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

U.S. Environmental Protection Agency. 1995. Reregistration eligibility decision (RED): Picloram. EPA 738-R95-019. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Propargite (4.4.13)

Hayes, W.H., Jr., and E.R. Laws, Jr., eds. 1991. *Handbook of Pesticide Toxicology.* Academic Press, Inc. San Diego, CA.

U.S. Environmental Protection Agency. 2000a. Propargite; Chemical No. 097601. HED’s revised human health risk assessment for propargite, case # 0243. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000b. The revised HED toxicology chapter for the risk assessment for the reregistration eligibility decision document (RED), Case # 0243. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Triclopyr (4.4.14)

Dow Chemical Co. 1987. Supplemental information concerning triclopyr and picloram (with additional 2,4-D comments) for use in development of a vegetation management environmental impact statement for the Southern Region. Submitted to U.S. Forest Service and LABAT-ANDERSON Incorporated. Midland, MI.

Dow AgroSciences. 1999. Material safety data sheet: Garlon* 4 herbicide. Indianapolis, IN.

Dow AgroSciences. 2001. Material safety data sheet: Garlon* 3A herbicide. Indianapolis, IN.

U.S. Environmental Protection Agency. 1998. Reregistration eligibility decision (RED): Triclopyr. EPA 738-R-98-011. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Other Ingredients (4.4.15)

Cyclohexanone

Aaron, C.S., J.G. Brewen, D.G. Stetka, W.T. Bleicher, and M.C. Spahn. 1985. Comparative mutagenesis in mammalian cells (CHO) in culture: Multiple genetic endpoint analysis of cyclohexanone in vitro. Environmental Mutagenesis 7(Suppl. 3):60-61.

Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. *Clinical Toxicology of Commercial Products*. 5th ed. Williams & Wilkins. Baltimore, MD.

Gupta, P.K., W.H. Lawrence, J.E. Turner, and J. Autian. 1979. Toxicological aspects of cyclohexanone. Toxicology and Applied Pharmacology 49:525-533.

International Agency for Research on Cancer. 1989. Cyclohexanone. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 47:157-169.

International Agency for Research on Cancer. 1999. Cyclohexanone. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 71(3):1359-1364.

U.S. Environmental Protection Agency. 1986. *Drosophila melanogaster* sex linked recessive lethal test of cyclohexanone with cover letter dated 101586 (final report). Doc #40-8666139. Abstract. Office of Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Ethylbenzene

Chan, P.C., J.K. Hasemani, J. Mahleri, and C. Aranyi. 1998. Tumor induction in F344/N rats and B6C3F1 mice following inhalation exposure to ethylbenzene. Toxicology Letters 99:23-32.

Susten, A.S., R.W. Niemeier, and S.D. Simon. 1990. In vivo percutaneous absorption studies of volatile organic solvents in hairless mice II. Toluene, ethylbenzene and aniline. Journal of Applied Toxicology 10(3):217-225.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Von Burg, R. 1992. Toxicology update: Ethylbenzene. Journal of Applied Toxicology 12(1):69-71.

Light aromatic solvent naphtha

Douglas, J.F., R.H. McKee, S.Z. Cagen, S.L. Schmitt, P.W. Beatty, M.S. Swanson, C.A. Schreiner, C.E. Ulrich, and B.Y. Cockrell. 1993. A neurotoxicity assessment of high flash aromatic naphtha. Abstract: Toxicology and Industrial Health 9(6):1047-1058.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

McKee, R.H., Z.A. Wong, S. Schmitt, P. Beatty, M. Swanson, C.A. Schreiner, and J.L. Schardein. 1990. The reproductive and developmental toxicity of high flash aromatic naphtha. Abstract: Toxicology and Industrial Health 6(3-4):441-460.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Xylene

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for xylenes (update). Division of Toxicology/Toxicology Information Branch. Atlanta, GA.

International Agency for Research on Cancer. 1989. Xylene. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans 47:125-156.

McDougal, J.N., G.W. Jepson, H.J. Clewell III, M.L. Gargas, and M.E. Andersen. 1990. Dermal absorption of organic chemical vapors in rats and humans. Fundamental and Applied Toxicology 14:299-308.

U.S. Environmental Protection Agency. 2001. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

<u>Fertilizers (4.4.16)</u>

Fan, A.M., and V.E. Steinberg. 1996. Health implications of nitrate and nitrite in drinking water: An update on methemoglobinemia occurrence and reproductive and developmental toxicity. Regulatory Toxicology and Pharmacology 23:35-43.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

J.R. Simplot Co. 1985. Material safety data sheet: Ammonium sulfate. Pocatello, ID.

Lewis, R.L., Sr. 1989. *Food Additives Handbook.* Van Nostrand Reinhold. New York.

Nechkina, M.A. 1992. Assessment of mutagenic effect of ammonium nitrate and 2,4-D acid. Gig Sanit 0(2):66-67.

Sax, N.I., and R.J. Lewis, Sr. 1989. *Dangerous Properties of Industrial Materials.* 7th ed. Van Nostrand Reinhold. New York.

U.S. Environmental Protection Agency. 1999. Health effects of exposure to high levels of sulfate in drinking water study. EPA 815-R-99-01. Office of Water. Washington, DC.

U.S. Environmental Protection Agency. 2001a. Pesticide registration notice 2001-2: Acute toxicity data requirements for granular pesticide products, including those with granular fertilizers in the product. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2001b. Integrated risk information system. On-line database. Office of Research and Development. Cincinnati, OH.

Section 4.5

U.S. Department of Agriculture. 1984. Pesticide background statements, Volume I: Herbicides. Agriculture Handbook Number 633. U.S. Forest Service. Washington, DC.

5.0 HUMAN HEALTH EXPOSURE ASSESSMENT

5.1 Introduction

This section describes the human populations potentially exposed to pesticides at Provolt and the scenarios for which doses were estimated. There are two populations potentially at risk—members of the public and seed orchard workers. The public near the seed orchard includes adults and children. In this analysis, it was assumed that an adult member of the public weighs 71.8 kg (158 lb) and a six-year old child weighs 22.6 kg (49.8 lb) (EPA 1999). Workers include both employees of the seed orchard and contracted workers. Their job functions include mixing concentrated pesticides with water, loading pesticide mixtures and fertilizers into application equipment, applying pesticides and fertilizers, and job functions requiring re-entry to treated areas.

5.2 Exposure and Dose

Two primary conditions are necessary for a human to receive a chemical dose that may result in a toxic effect. First, the chemical must be present in the person's immediate environment—in the air, on a surface such as vegetation that may contact the skin, or in food or water—so that it is available for intake. The amount of the chemical present in the person's immediate environment is the exposure level. Second, the chemical must enter the person's body by some route. Chemicals in the air may be inhaled into the air passages and lungs, or may form deposits on the skin as they settle out of the air. Chemicals on vegetation, on clothing that is in contact with the skin, or on the skin itself, may penetrate the skin. Chemicals in food or water may be ingested. The amount of a chemical that moves into the body by any of those routes constitutes the dose.

While two people may be subjected to the same level of exposure (for example, two workers applying pesticide with backpack sprayers), one may get a much lower dose than the other by wearing protective clothing, using a respirator, or washing immediately after spraying. Exposure, then, is the amount of a chemical available for intake into the body; dose is the amount of the substance that actually enters the body.

5.3 Potential Exposures

This subsection describes the populations that may be exposed to the pesticides and fertilizers as a result of their use at Provolt. This subsection also lists the representative human health exposure scenarios analyzed in this risk assessment.

5.3.1 Affected Populations and Exposure Scenarios

The human population that could be exposed to pesticides and fertilizers used at Provolt falls into two groups. The first group is the public who may be subject to nonoccupational exposure. This group includes official or unscheduled visitors to the orchard, residents living near the site, and members of the public engaging in recreational activities, such as hiking in or near treated areas. The second group is the workers directly involved in the application of pesticides and fertilizers, including mixer/loaders, applicators, and irrigation system maintenance workers. Workers may also be exposed to the chemicals under the conditions described for public exposures.

For members of the public, the exposure scenarios analyzed in this risk assessment consist of the following:

- Ingestion of groundwater
- Ingestion of water from Applegate River or Williams Creek (not known sources of drinking water).
- Ingestion of fish from Applegate River, Williams Creek, or the pond near the seed orchard office.
- Ingestion of deer hunted near grounds.
- Ingestion of quail and Canada goose hunted near grounds.
- Ingestion of blackberries.
- Dermal exposure to insecticide/fungicide drift residues on vegetation, or herbicide treatment residues on vegetation, during recreational hiking on orchard grounds.
- Dermal exposure to residues on dogs following recreational use of site.

The categories of workers evaluated in this risk assessment for occupational exposure to pesticides are as follows:

- High-pressure hydraulic sprayer mixer/loader/applicator
- Hydraulic sprayer with hand-held wand mixer/loader/applicator
- Tractor-pulled spray rig with boom mixer/loader/applicator
- Backpack sprayer mixer/loader/applicator
- Hand-held wick mixer/loader/applicator
- Broadcast fertilizer spreader loader/applicator
- Irrigation system maintenance personnel

Several accidental exposure scenarios were also evaluated:

- Ingestion of groundwater after a spill of concentrate.
- Ingestion of fish and water containing runoff from a spill of concentrate.
- Ingestion of fish and water downstream of a spill of tank mix directly into a stream.
- Spill of pesticide concentrate onto worker's skin.
- Spill of pesticide mixture onto worker's skin.
- Spray of worker with tank mix of pesticide.

5.3.2 Levels of Exposure

To allow for some of the uncertainty inherent in any quantitative risk assessment, two levels of human exposure were evaluated.

Typical Exposures. Typical exposure assumptions attempt to target the average dose an individual may receive if all exposure conditions are met. These assumptions include the application rate usually used at Provolt, typical number of applications per year, and other similar assumptions.

Maximum Exposures. Maximum exposure assumptions attempt to define the upper bound of credible doses that an individual may receive if all exposure conditions are met. These assumptions include the maximum application rate according to the label, maximum number of applications per year, and other similar assumptions.

5.4 Potential Exposures to Members of the Public

The doses to members of the public from Provolt's proposed pesticides were estimated for eight types of exposure scenarios. In this analysis, doses from two routes of exposure were estimated—dietary and dermal. The following sections describe the parameters used in calculating these doses.

5.4.1 Ingestion of Groundwater

This scenario investigates the risk from drinking well water contaminated by leachate of pesticides or fertilizers proposed for use at the seed orchard. For this scenario, it was assumed that a 71.8-kg adult drinks 1.51 L (0.4 gal) of water per day, and a six-year-old 22.6-kg child drinks 0.74 L (0.20 gal) per day, based on statistics presented in EPA (1999). Concentrations of chemicals in groundwater were estimated as described in Section 3.2. The following equation was used to calculate the dose to adults and children:

$$DOSE = CONC \times AMT / BW$$

where:

DOSE	=	dietary dose from drinking contaminated water (mg/kg)
CONC	=	concentration of chemical in groundwater (mg/L)
AMT	=	water consumption amount (L)
BW	=	body weight (kg)

5.4.2 Ingestion of Surface Water

These scenarios estimate the dose from drinking water from Williams Creek or the Applegate River, which are not known sources of drinking water, after the respective stream receives contaminated runoff. Concentrations of chemicals in surface water were estimated as described in Section 3.2. Body weights and daily water ingestion amounts were the same as in the groundwater ingestion scenario. The same equation was used to calculate the dose to adults and children:

$$DOSE = CONC \times AMT / BW$$

where:

DOSE	=	dietary dose from drinking contaminated water (mg/kg)
CONC	=	concentration of pesticide in creek or river (mg/L)
AMT	=	water consumption amount (L)
BW	=	body weight (kg)

5.4.3 Ingestion of Fish from Creek, River, or Pond

In these scenarios, it was assumed that an adult or child ingests fish caught in Williams Creek or the Applegate River downstream of orchard drainages after they receive stream water containing runoff from treated areas, or fish caught in the onsite pond. It was assumed that 0.129 kg of fish per day are ingested by both adults and children (EPA 1999). This dietary dose to members of the public was calculated using the following equation:

$$DOSE = CONC \times BCF \times AMT / BW$$

where:

DOSE	=	pesticide dose from ingesting fish from creek, river, or pond (mg/kg)
CONC	=	concentration of pesticide in creek, river, or pond (mg/L)
BCF	=	bioconcentration factor (mg/kg per mg/L)
AMT	=	fish consumption amount (kg)
BW	=	body weight (kg)

5.4.4 Ingestion of Venison

This scenario estimated the dietary dose to a person who consumes venison from deer that have been exposed to pesticides. The concentration of pesticide in the meat of the animal was calculated based on the total dose to these animals determined in the non-target species risk assessment. It was assumed that 4 and 2 oz. (0.113 and 0.0567 kg) of venison are ingested by adults and children, respectively. The dietary dose was computed as follows:

$$DOSE = DEER \times BTF \times AMT / BW$$

where:

DOSE	=	dietary dose from consumption of contaminated meat (mg/kg)
DEER	=	dose to animal (mg/kg)
BTF	=	biotransfer factor (unitless)
AMT	=	venison consumption amount (kg)
BW	=	body weight (kg)

5.4.5 Ingestion of Quail or Goose

These scenarios estimated the dietary dose to a person who consumes quail or geese that have been exposed to pesticides. The concentrations of pesticide in the flesh of a California quail (valley quail) and a Canada goose were calculated based on the total doses to these species, using the same assumptions applied to other avian species in the non-target species risk assessment. It was assumed that 0.556 and 0.332 g/kg/day are consumed by a six-year-old child or a 20 to 39-year-old adult, respectively, based on the 50th percentile poultry ingestion rates in EPA (1999). The dietary dose was computed as follows:

$$DOSE = POULTRY \times BTF \times AMT / BW$$

where:

DOSE	=	dietary dose from consumption of contaminated meat or poultry (mg/kg)
POULTRY	=	dose to bird (mg/kg)
BTF	=	biotransfer factor (unitless)
AMT	=	poultry consumption amount (kg)
BW	=	body weight (kg)

5.4.6 Ingestion of Blackberries

This scenario estimated the dietary dose to a person who consumes blackberries that received spray drift at 25 feet from a treated area (all typical scenarios and maximum scenario for non-herbicides) or a direct spray (maximum scenario for herbicides). The concentration of pesticide on the fruit was calculated based on the mean of the residue levels measured on blackberries as reported by Hoerger and Kenaga (1972): 1.85 mg residue per kilogram berry per lb/acre applied. It was assumed that 0.001723 and 0.0001364 kg/day of blackberries was consumed by adults and children, respectively, which represent the mean intake of “other berries” (adjusted for body weight) presented in EPA (1999). The dietary dose was computed as follows:

$$DOSE = RES \times AMT \times DEP / BW$$

where:

DOSE	=	dietary dose from consumption of contaminated blackberries (mg/kg)
RES	=	mg pesticide per kg of berries per lb/acre pesticide drift or applied (mg/kg)
DEP	=	deposition of pesticide on blackberries due to drift or direct application (lb/acre)
AMT	=	blackberry consumption amount (kg)
BW	=	body weight (kg)

5.4.7 Recreational Hiking

The dermal dose to recreational hikers on seed orchard grounds was investigated in this scenario. Lavy et al. (1980) conducted a study to estimate the vegetation contacted by persons walking through a forest area treated at a rate of 2 lb/A. Although the study found that residues were below the detectable limit, this risk assessment assumed that one-half of the detectable limit was available for contact. The limit of detection in the study was 0.5 mg/m^2. For calculating exposed skin area, the typical scenario assumed that 25 percent of total skin area is exposed, while the maximum scenario assumed that 50 percent of total skin area is exposed. The calculation for the dose was as follows:

$$DOSE = (DEP \times RATE \times SA \times SAF \times DPR) / BW$$

where:

DOSE	=	dose from recreational site use (mg/kg)
DEP	=	typical or maximum drift deposition at 25 feet from treated area for insecticides/fungicide, application rate for herbicides (lb/acre)
RATE	=	dermal dose rate from Lavy et al. study (0.5 mg/m^2 per 2 lb/acre)
SA	=	total skin surface area (1.94 m^2 adult and 0.79 m^2 child)
SAF	=	fraction of total skin surface area that actually contacts the vegetation (unitless)
DPR	=	dermal penetration rate (unitless)
BW	=	body weight (kg)

5.4.8 Petting Dog with Residues

The dermal dose from petting a dog with residues on its fur was estimated assuming that a dog is exposed to pesticides while walking with a hiker through the seed orchard grounds. The dog is assumed to travel through areas that contain drift from applications to orchard trees, or through areas sprayed with herbicides. The dog is assumed to weigh 40 pounds, with a surface area of 0.72 m^2; half and three-quarters of this surface area are assumed to have pesticide residues in the typical and maximum scenarios, respectively. Half of the residue level on the animal's fur is assumed to be transferred to a person's hand, and a fraction of that is subsequently absorbed, based on each pesticide's dermal penetration rate. The dose was calculated as follows:

$$DOSE = DDE \times 0.5 \times DPR / BW$$

where:

DOSE	=	dose from petting a dog with residues on fur (mg/kg)
DDE	=	dog's dermal exposure (see below) (mg)
0.5	=	fraction of residues transferred to human hand
DPR	=	dermal penetration rate (unitless)
BW	=	human body weight (kg)

and:

$$DDE\ (mg) = DEP \times RATE \times SA \times FRAC$$

where:

DEP	=	drift deposition at 25 feet from treated area for insecticides/fungicide, application rate for herbicides (lb/acre)
RATE	=	dermal dose rate from Lavy et al. study (see Section 5.4.5) (0.5 mg/m^2 per 2 lb/acre)
SA	=	total surface area (0.72 m^2 for a 40-lb dog)
FRAC	=	fraction of surface area receiving pesticide residues (0.5 typical, 0.75 maximum)

5.4.9 Lifetime Doses to the Public

Lifetime doses to members of the public were calculated for the potential carcinogens evaluated in this risk assessment: acephate, chlorothalonil, permethrin, the hexachlorobenzene contaminant in picloram, and propargite. The lifetime dose was estimated by assuming that 95 percent of the time the person is exposed to the typical dose, and five percent of the time the person is exposed to the maximum dose. The annual frequency of exposure was calculated as 0.95 x the annual number of applications in the typical case plus 0.05 x the annual number of applications in the maximum case. This annual dose was assumed to occur repeatedly over a nine-year period in an individual's life, the typical length of residency at one address, and was averaged over a 75-year lifetime (EPA 1999).

5.5 Potential Exposures to Provolt Seed Orchard Workers

The doses to workers from pesticides and fertilizers were estimated for all workers applying the chemicals or who may be exposed while working in a treated area.

The use of protective clothing can substantially reduce worker doses. The manufacturer of each of the pesticides used at Provolt provides product labeling recommending the protective clothing to be worn while handling or applying the pesticides. Table 5-1 presents these recommendations, which are followed by Provolt seed orchard workers. Workers re-entering treated areas abide by the restricted entry intervals specified on each pesticide’s label; these restricted entry intervals are summarized in Table 5-2. Re-entry before the interval is concluded requires the use of the protective clothing for early re-entry specified on each pesticide’s label.

Five of the pesticides proposed for use at the seed orchard are classified as restricted use: chlorpyrifos, diazinon, esfenvalerate, permethrin, and picloram. Restricted use pesticides must be applied by, or under the direction of, a certified applicator.

5.5.1 Ground Equipment Mixer/Loader

Estimating total doses to workers operating application equipment required combining the dose from the mixing/loading operation with the dose from the application operation, since (1) except for aerial applications, these workers may perform both functions, and (2) in some cases, the studies that form the basis for these calculations only monitored the doses received from application. Doses to mixer/loaders using liquid concentrates or powders not contained in water-soluble packets were based on an exposure study conducted by Nash et al. (1982) in which the mean 2,4-D dose was 1.74×10^{-4} mg/kg per lb. The dose was calculated as follows:

$$DOSE_{m/l} = STUDY \times LB \times PCF \times DPR / 24D$$

where:

$DOSE_{m/l}$	=	pesticide dose to mixer/loaders (mg/kg)
STUDY	=	dose to mixer/loaders from 2,4-D study by Nash et al. (1982) (mg/kg per lb a.i.)
LB	=	total pesticide mixed/loaded (lb a.i.)
PCF	=	protective clothing factor (0.1) (Spencer et al. 1991)
DPR	=	4-hr dermal penetration rate (unitless)
24D	=	dermal penetration rate for 2,4-D (0.42% per hour x 4 hours)

Table 5-1. Summary of Personal Protective Equipment for Workers

Pesticide	Label-Required PPE
Acephate: Acecap 97	None specified
Chlorpyrifos: Dursban 50W	Long-sleeved shirt and long pants, eye protection, waterproof gloves, chemical-resistant headgear
Diazinon: Diazinon 50W	Long-sleeved shirt and long pants, shoes plus socks, waterproof gloves
Dimethoate: Digon 400	Long-sleeved shirt and long pants; chemical-resistant gloves such as barrier laminate, butyl rubber, nitrile rubber, or viton; chemical-resistant footwear plus socks; protective eyewear; chemical- resistant headgear
Esfenvalerate: Asana XL	Long-sleeved shirt and long pants; chemical-resistant gloves such as barrier laminate or neoprene rubber or nitrile rubber or viton; shoes plus socks; protective eyewear
Horticultural Oil: Dormant Oil 435	Long-sleeved shirt and long pants, chemical resistant gloves (such as barrier laminate or nitrile rubber or neoprene rubber or viton), shoes plus socks, and protective eyewear
Permethrin: Pounce 3.2 EC	Long-sleeved shirt and long pants, chemical-resistant gloves such as barrier laminate or viton, shoes plus socks
Propargite: Omite CR	Long-sleeved shirt and long pants, waterproof gloves, shoes plus socks, protective eyewear
Chlorothalonil: Bravo 500	Long-sleeved shirt and long pants, waterproof gloves, shoes plus socks, protective eyewear
Dicamba: Banvel	Long-sleeved shirt and long pants, waterproof gloves, shoes plus socks, protective eyewear
Glyphosate: Rodeo	None specified.
Hexazinone: Velpar	Long-sleeved shirt and long pants, shoes plus socks, protective eyewear
Picloram: Tordon 22K	Long-sleeved shirt and long pants, waterproof gloves, shoes plus socks
Triclopyr: Garlon 4	Long-sleeved shirt and long pants; chemical-resistant gloves such as barrier laminate, nitrile rubber, neoprene rubber, or viton; shoes plus socks

Table 5-2. Restricted Entry Intervals

Pesticide	Restricted Entry Interval
Acephate: Acecap 97	None
Chlorpyrifos: Dursban 50W	12 hours
Diazinon: Diazinon 50W	12 hours
Dimethoate: Digon 400	48 hours
Esfenvalerate: Asana XL	12 hours
Horticultural Oil: Dormant Oil 435	12 hours
Permethrin: Pounce 3.2 EC	12 hours
Propargite: Omite CR	7 days
Chlorothalonil: Bravo 500	48 hours
Dicamba: Banvel	24 hours
Glyphosate: Rodeo	None
Hexazinone: Velpar	24 hours
Picloram: Tordon 22K	12 hours
Triclopyr: Garlon 4	12 hours

5.5.2 High-Pressure Hydraulic Sprayer Mixer/Loader/Applicator

Doses to high-pressure hydraulic sprayer mixer/loader/applicators were estimated based on mixer/loader doses from Nash et al. (1982) and high-pressure hydraulic sprayer doses measured by Haverty et al. (1983). In the applicator exposure study, 1.12 mg of carbaryl were deposited on the worker for each lb a.i. applied. Doses were calculated as follows:

$$DOSE = DOSE_{m/l} + STUDY \times APP \times TREES \times PCF \times DPR / BW$$

where:

DOSE = dose to high-pressure hydraulic sprayer mixer/loader/applicator (mg/kg/day)
$DOSE_{m/l}$ = dose from mixing/loading part of operation (see Section 5.5.3) (mg/kg/day) (where appropriate; not used if pesticide is formulated in water soluble bags)
STUDY = exposure of applicator in study by Haverty et al. (mg per lb a.i. applied)
APP = application rate (lb/tree)
TREES = area treated (trees/day)
PCF = protective clothing factor (0.1)
DPR = 4-hr dermal penetration rate (unitless)
BW = body weight (71.8 kg)

5.5.3 Hydraulic Sprayer with Hand-Held Wand Mixer/Loader/Applicator

Doses to mixer/loader/applicators using a low-pressure hydraulic sprayer with a hand-held wand were estimated based on mixer/loader doses from Nash et al. (1982) and hand-held spray gun doses to applicators measured by Engelhard et al. (1980). In the applicator exposure study, 0.00284 mg of cadmium fungicide were deposited on the operator's clothing following application of 0.101 lb a.i., for an exposure rate of 0.028 mg/lb a.i. Doses were calculated as follows:

$$DOSE = DOSE_{m/l} + STUDY \times APP \times AREA / DAYS \times PCF \times DPR / BW$$

where:

DOSE	=	dose to high-pressure hydraulic sprayer mixer/loader/applicator (mg/kg/day)
$DOSE_{m/l}$	=	dose from mixing/loading part of operation (see Section 5.5.3) (mg/kg/day) (where appropriate; not used if pesticide is formulated in water soluble bags)
STUDY	=	exposure of applicator in study by Engelhard et al. (mg per lb a.i. applied)
APP	=	application rate (lb/tree)
AREA	=	area treated (acres)
DAYS	=	length of time required to complete one treatment (days)
PCF	=	protective clothing factor (0.1)
DPR	=	4-hr dermal penetration rate (unitless)
BW	=	body weight (71.8 kg)

5.5.4 Tractor-Pulled Spray Boom Mixer/Loader/Applicator

Doses to applicators using a tractor-drawn spray boom were estimated from the exposure data presented by Nash et al. (1982) for ground operations using 2,4-D. The mean dose was 0.000171 mg/kg per lb applied. The dose estimated for the applicator was added to the dose estimated for the mixer/loader to determine the total dose to the mixer/loader/applicator.

$$DOSE = DOSE_{m/l} + STUDY \times APP \times AREA / DAYS \times PCF \times DPR / 24D$$

where:

DOSE	=	pesticide dose to mixer/loader/applicators (mg/kg)
$DOSE_{m/l}$	=	pesticide dose from mixing/loading (mg/kg)
STUDY	=	2,4-D dose to applicator in Nash et al. study (mg/kg per lb a.i.)
APP	=	application rate (lb/acre)
AREA	=	area treated (acres)
DAYS	=	length of time to complete treatment (days)
PCF	=	clothing protection factor of 0.1 (unitless)
DPR	=	dermal penetration rate (unitless)
24D	=	dermal penetration rate for 2,4-D (0.42% per hour x 4 hours)

5.5.5 Backpack Sprayer

Doses from applications using a hand-carried backpack sprayer were based on a study by Middendorf (undated), in which mixer/loader/applicators received a mean dose (estimated by urinary metabolite measurement) of 0.0122 mg/kg per lb a.i. from mixing and application of triclopyr butoxyethyl ester. The dose to seed orchard workers was calculated as follows:

$$DOSE = STUDY \times APP \times AREA / DAYS \times DPR / TRI$$

where:

DOSE	=	dose to mixer/applicator (mg/kg/day)
STUDY	=	dose from Middendorf study (0.0122 mg/kg per lb a.i.)
APP	=	application rate (lb/acre)
AREA	=	area treated (acres)
DAYS	=	length of time required for one treatment (days)
DPR	=	dermal penetration factor (unitless)
TRI	=	dermal penetration rate for triclopyr (1.65% over 4 hours)

5.5.6 Hand-Held Wick Mixer/Loader/Applicator

Doses from applications using a hand-held wick were based on a the results of studies reported in EPA (1998), in which mixer/loader/applicators received mean dermal exposures of 2.46, 3.81 and 175 mg to the head and neck; upper arm, lower arm, chest, back, thigh, and lower leg; and hands, respectively, per lb a.i. applied. The doses to hands were adjusted by a protective clothing factor of 0.1 to represent that gloves are worn. The dose to seed orchard workers was calculated as follows:

$$DOSE = APP \times (AREA / DAYS) \times [R1 + R2 + (PCF \times R3)] \times DPR / BW$$

where:

DOSE	=	dose to mixer/loader/applicator (mg/kg/day)
APP	=	application rate (lb/acre)
AREA	=	area treated (acres)
DAYS	=	length of time required for one treatment (days)
R1	=	dermal exposure to the head and neck (2.46 mg/lb a.i.)
R2	=	dermal exposure to the upper arm, lower arm, chest, back, thigh, and lower leg (3.81 mg/lb a.i.)
PCF	=	protective clothing factor to represent use of gloves (0.1)
R3	=	dermal exposure to the hands (175 mg/lb a.i.)
DPR	=	dermal penetration factor (unitless)
BW	=	body weight (71.8 kg)

5.5.7 Broadcast Fertilizer Spreader

The dose to an applicator using a broadcast fertilizer spreader was not calculated for applicators applying fertilizers, since negligible toxicity is expected from any exposure pathways other than ingestion of nitrate in drinking water (see Section 4.4.16). No risk is expected from dermal exposure to fertilizer applicators at the seed orchard.

5.5.8 Irrigation System Maintenance Personnel

The dermal dose to irrigation system maintenance workers was investigated in this scenario. Lavy et al. (1980) conducted a study to estimate the vegetation contacted by persons walking through a forest area treated at a rate of 2 lb/A. Although the study found that residues were below the detectable limit, this risk assessment assumed that one-half of the detectable limit was available for contact. The limit of detection in the study was 0.5 mg/m^2. For calculating exposed skin area, the typical scenario assumed that 25 percent of total skin area is exposed, while the maximum scenario assumed that 50 percent of total skin area is exposed. The calculation for the dose was as follows:

$$DOSE = (DEP \times RATE \times e^{-kt} \times SA \times SAF \times DPR) / BW$$

where:

DOSE	=	dose from irrigation system maintenance (mg/kg)
DEP	=	insecticide/fungicide drift deposition at 3 feet from treated area or herbicide application rate (lb/acre)
RATE	=	dermal dose rate from Lavy et al. study (0.5 mg/m^2 per 2 lb/acre)
e^{-kt}	=	factor accounting for degradation at time *t* (7 days) at the pesticide specific degradation coefficient *k*
SA	=	total skin surface area (1.94 m^2)
SAF	=	fraction of total skin surface area that actually contacts the vegetation (unitless)
DPR	=	dermal penetration rate (unitless)
BW	=	body weight (kg)

5.5.9 Lifetime Doses to Workers

The lifetime doses for workers handling potential carcinogens (acephate, chlorothalonil, permethrin, the hexachlorobenzene contaminant in picloram, and propargite) were estimated assuming that a single worker applies the total amount of a given pesticide used annually. The number of days the worker is exposed to the pesticide was assumed to be the same as the typical number of applications of that pesticide annually. Daily doses were estimated assuming that 95 percent of the time the worker is exposed to the typical dose, and five percent of the time the worker is exposed to the maximum dose. Annual doses were multiplied by 7 years, the average employment tenure reported in EPA (1999), to indicate cumulative exposure, which was then averaged over a 75-year lifetime.

5.6 Potential Exposures From Accidents

In the event of an accident, members of the public and workers may be exposed to greater amounts of a pesticide or fertilizer than under normal exposure circumstances. An individual may ingest contaminated water or fish following a spill at the mixing area or into a ditch or creek. However, direct onsite exposure to the public during pesticide applications will be prevented by restricting access to the seed orchard facility during and after pesticide use. Workers may spill the pesticide concentrate or diluted pesticide mixture on their skin, or may be accidentally sprayed during an application.

5.6.1 Ingestion of Fish and Water after Spill

Three variations of this scenario were evaluated in the risk assessment, as follows:

Accidental spill of pesticide concentrate

- Mixing area near office on seed orchard grounds (groundwater ingestion of leached chemical, surface water and fish ingestion of chemical in runoff from spill area).

Spill of pesticide tank mix or fertilizer load into stream

- The point where three orchard roads converge at a crossing of Bridge Point Ditch, near the southwest corner of OU9B (surface water and fish ingestion).
- The point where Highway 238 crosses Williams Creek (surface water and fish ingestion).

As in the non-accident scenarios, it was assumed that adults and children drink 1.51 and 0.74 L of water per day, respectively, and that both eat 0.129 kg of fish. In the scenarios involving contaminated surface water, the fish and water were assumed to be taken from the Applegate River. This dietary dose to members of the public was calculated using the following equation:

$$DOSE = [(CONC \times H2O) + (CONC \times BCF \times FISH)] / BW$$

where:

DOSE	=	dose from ingesting fish and water contaminated by spill (mg/kg)
CONC	=	concentration of chemical in river (mg/L)
H2O	=	amount of water ingested (L)
BCF	=	bioconcentration factor (mg/kg per mg/L)
FISH	=	fish consumption amount (kg)
BW	=	body weight (kg)

Groundwater was assumed to be drawn from the domestic well near the office. Doses were calculated as follows:

$$DOSE = CONC \times AMT / BW$$

where:

DOSE	=	dietary dose from drinking contaminated water (mg/kg)
CONC	=	concentration of chemical in groundwater (mg/L)
AMT	=	water consumption amount (L)
BW	=	body weight (kg)

5.6.2 Spill of Pesticide Concentrate onto Worker

All liquid concentrate pesticide formulations used at the seed orchard were evaluated for the risks associated with a direct spill on a worker. Direct dermal exposure of workers to pesticides was calculated for spills of 0.5 L (approximately one pint) of pesticide concentrate. This exposure might result if a container of concentrate were spilled. It was further assumed that 50 percent of the saturated skin surface is covered with clothing, which allows 10 percent of the liquid to penetrate to the skin surface, and that one percent of the amount spilled directly on the skin remains after any dripping, shaking, or rough wiping to remove the majority of it. One hour was assumed to elapse before the worker is able to wash it off thoroughly. The dose from the spill of a pesticide concentrate was determined as follows:

$$DOSE = CONC \times SP \times 1\ gal/3.785\ L \times 1{\times}10^{6}\ mg/2.205\ lb \times (CL \times CP + SK \times ST) \times DPR\ /\ BW$$

where:

DOSE	=	dermal dose from spill of concentrate (mg/kg)
CONC	=	concentration of pesticide concentrate (lb a.i./gal)
SP	=	size of spill (0.5 L)
CL	=	portion of the pesticide that spills on clothing (0.5)
CP	=	portion of the pesticide on clothing that penetrates through the clothing (0.1)
SK	=	portion of the pesticide that spills on bare skin (0.5)
ST	=	portion of the pesticide on skin that remains (0.01)
DPR	=	dermal penetration rate for one hour (unitless)
BW	=	body weight (kg)

In an additional accident scenario, it was assumed that one acephate implant capsule within the carton has opened, distributing 25 percent of its contents over the other intact capsules (with the rest falling to the bottom of the box or remaining in the broken capsule). The applicator then would have some dermal exposure from handling the capsules with residues on them. The dose was calculated as follows:

$$DOSE = CAP \times RES \times DPR\ /\ BW$$

where:

DOSE	=	maximum dose to implant applicator (mg/kg/day)
CAP	=	number of capsules implanted per day
RES	=	residue level on each capsule (mg)
DPR	=	dermal penetration rate (unitless)
BW	=	body weight (kg)

5.6.3 Spill of Pesticide Mixture onto Worker

In this scenario, all assumptions are the same as for the spill of a pesticide concentrate, except that the diluted form of all chemicals applied as liquids was used as the input to the risk estimate. The equation is the same as in the previous scenario, except that the parameter CONC is defined as the concentration of pesticide in the diluted tank mix (lb a.i./gal).

5.6.4 Accidental Spray of Worker

In this scenario, a worker involved in a spraying operation is accidentally sprayed and receives a dermal dose at the application rate over half the skin surface area. It is assumed that clothing, dripping, and wiping prevents 90% of the spray from reaching or remaining on the skin, and that the individual is able to shower within one hour to remove the residues. The dose was calculated as follows:

$$DOSE = RATE \times 2.471\ acres/10{,}000\ m^2 \times 453{,}600\ mg/lb \times SA \times SAS \times REM \times DPR\ /\ BW$$

where:

DOSE	=	dose from accidental spray (mg/kg)
RATE	=	application rate (lb/acre)
SA	=	body surface area (m^2)
SAS	=	fraction of body surface area receiving spray (0.5)
REM	=	spray amount remaining in contact with skin (0.1)
DPR	=	dermal penetration rate for one hour (unitless)
BW	=	body weight (kg)

5.6.5 Lifetime Doses From Accidents

Lifetime doses to members of the public and workers from accidents were calculated assuming that only one accident of the magnitude described above would occur involving any individual. Lifetime doses were calculated for those chemicals considered to be potential carcinogens in this risk assessment: acephate, chlorothalonil, permethrin, the hexachlorobenzene contaminant in picloram, and propargite.

5.7 References

Engelhard, A.W., R.C. Ploetz, and A.J. Overman. 1980. Human exposure to pesticides: Measure and evaluation of the risk to the applicator. Foliage Digest 2(12):10-13.

Haverty, M.I., M. Page, P.J. Shea, J.B. Hoy, and R.W. Hall. 1983. Drift and worker exposure resulting from two methods of applying insecticides to pine bark. Bulletin of Environmental Contamination and Toxicology 30:223-228.

Hoerger, F., and E.E. Kenaga. 1972. Pesticide residues on plants: Correlation of representative data as a basis for estimation of their magnitude in the environment. In *Environmental Quality and Safety: V1–Global Aspects of Toxicology and Technology as Applied to the Environment.* F. Coulston, ed. Academica Press. New York, NY.

Lavy, T.L., J.S. Shepard, and D.C. Bouchard. 1980. Field worker exposure and helicopter spray pattern of 2,4,5-T. Bulletin of Environmental Contamination and Toxicology 24:90-96.

Middendorf, P.J. Undated. Forest worker exposures to triclopyr (3,5,6-trichloro-2-pyridinyloxyaceticacid), butoxyethyl ester during directed foliar applications of Garlon™4 herbicide. Georgia Institute of Technology. Atlanta, GA.

Nash, R.G., P.C. Kearney, J.C. Maitlen, C.R. Sell, and S.N. Fertig. 1982. Agricultural applicators exposure to 2,4-dichlorophenoxyacetic acid. In *Pesticide Residues and Exposures.* J.R. Plimmer (ed.). American Chemical Society Symposium Series No. 182. Washington, DC.

Spencer, J.R., S.R. Bissell, J.R. Sanborn, F.A. Schneider, S.S. Margetich, and R.I. Krieger. 1991. Chlorothalonil exposure of workers on mechanical tomato harvesters. Toxicology Letters 55:99-107.

U.S. Environmental Protection Agency. 1998. PHED surrogate exposure guide: Estimates of worker exposure from the Pesticide Handler Exposure Database version 1.1. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1999. Exposure factors handbook. CD-ROM version. EPA/600/C-99/001. Office of Research and Development. Washington, DC.

6.0 HUMAN HEALTH RISK CHARACTERIZATION

6.1 Introduction

This section characterizes the estimated risks to the health of workers and members of the public that may result from any of the pesticides or fertilizers proposed for use at Provolt. In the risk characterization, the human doses estimated in the exposure assessment (Section 5.0) are compared with the toxicity characteristics described in the hazard assessment (Section 4.0), to arrive at estimates of risk.

Section 6.2 describes the methods used to evaluate human health risks, including both noncarcinogenic and carcinogenic risks. Section 6.3 contains the results of the quantitative risk characterization for the pesticides and fertilizers proposed for use at Provolt. Section 6.4 addresses cumulative human health risks, and Section 6.5 discusses the uncertainties in this risk assessment.

6.2 Methodology for Assessing Human Health Risks

6.2.1 Noncarcinogenic Risk Estimation

In this risk assessment, the potential risks were evaluated by comparing the representative doses (estimated in the exposure assessment) with the RfDs (identified in the hazard assessment). All the RfDs used in this risk analysis take into account multiple exposures over several years and represent acceptable dose levels. The comparison of dose to RfD consists of a simple ratio, called the Hazard Index:

$$\textit{Hazard Index} = \textit{Estimated Dose (mg/kg/day)} \div \textit{RfD (mg/kg/day)}$$

If the estimated dose does not exceed the RfD, the hazard index will be one or less, indicating a negligible risk of noncarcinogenic human health effects. It is important to note two characteristics of the hazard index: (1) the greater the value of the hazard above one, the greater the level of concern; but (2) the level of concern does not increase linearly as the hazard index increases, because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects. Thus, the interpretation of the potential toxic response associated with a particular hazard index can range widely depending on the chemical (EPA 1989).

A dose estimate that exceeds the RfD, although not necessarily leading to the conclusion that there will be toxic effects, clearly indicates a potential risk for adverse health effects. Risk is presumed to exist if the hazard index is greater than one. However, comparing one-time or once-a-year doses (such as those experienced by the public or in an accident) to RfDs derived from long-term studies with daily dosing tends to exaggerate the risk from those infrequent events.

For workers and the public, hazard indices were computed for each chemical, application, and scenario for typical, maximum, and accident situations. For pesticide formulations containing other ingredients on EPA's List 1 or 2 of Inert Ingredients, the hazard indices for each component of the formulation are added together, to indicate the total risk to the exposed individual from that pesticide.

If the hazard index exceeds one, the risk may require mitigation, depending on the circumstances of exposure. For workers, this may mean reducing the quantity of pesticide to which the worker is exposed or increasing the level of protective clothing. For members of the public, it may mean decreasing the application rate or using measures to reduce the potential for runoff to reach streams. In some cases, the simple mitigation procedures will not reduce exposures (and thereby decrease the hazard index) to an acceptable level. In these cases, the seed orchard manager may consider use of a different pesticide or use a non-pesticide method to control the target pest.

6.2.2 Cancer Risk Estimation

As a result of the review of cancer studies presented in the Human Health Hazard Assessment (Section 4.0), a risk analysis for cancer was conducted for five of the chemicals analyzed in this document—acephate, chlorothalonil, permethrin, the hexachlorobenzene contaminant in picloram, and propargite.

The mechanism for cancer dose-response can be complex, and EPA is currently developing updated guidance for deriving cancer slope factors that are applicable to human health risk assessment from the results of studies in laboratory animals. In laboratory studies, high doses are used to elicit an observable cancer incidence in a finite group of test animals. Historically, carcinogenic effects were assumed to have no threshold, requiring extrapolation to compare exposures from the much lower doses associated with environmental exposure to chemicals. EPA's current guidance in force, the 1986 *Guidelines for Carcinogen Risk Assessment*, provided a basic rationale for linear dose-response assumptions in cancer risk assessment (EPA 1986a). However, new perspectives on methods to assess risks of cancer are gaining wider acceptance, such as consideration of mode of action, thresholds for carcinogenicity, and incorporating other types of biological data. In 1996, EPA proposed revised guidelines for carcinogen risk assessment which address these (and other) issues, but they have not yet been finalized. Estimation of cancer slope factors using updated methods is occurring on a chemical-by-chemical basis, as new laboratory studies are completed and new risk assessments are conducted. For all of the chemicals determined to be possible or probable human carcinogens in this risk assessment, a linear (no-threshold) approach was used in calculating the cancer slope factors, in accordance with the guidance that has been in effect.

Cancer risk from a chemical is expressed as the probability that cancer will occur over the course of a person's lifetime, as a result of the stated exposure. This risk probability is calculated as follows:

$$RISK = DOSE \times CSF \times OCC / LIFE$$

where:

RISK	=	the lifetime probability of cancer as a result of the specified exposure
DOSE	=	estimated dose (mg/kg/day)
CSF	=	cancer slope factor (per mg/kg/day)
OCC	=	number of occurrences of the daily dose during an individual's lifetime
LIFE	=	the number of days in a 75-year lifetime (27,375 days)

The resulting cancer probabilities are compared to a benchmark value of 1×10^{-6} (or 1 in 1 million), a value commonly accepted in the scientific community as representing a cancer risk that would result in a negligible addition to the background cancer risk of approximately one in four in the United States. In some occupational health risk assessments, cancer risks as high as 1×10^{-4} (1 in 10,000) can be considered acceptable. However, the benchmark of 1 in 1 million is used for both workers and the public in this risk assessment.

6.3 Potential Risks to Human Health from the Proposed Chemicals

This subsection presents the results of the quantitative risk analysis for the pesticides and fertilizers proposed for use at Provolt. Section 6.3.1 summarizes the estimated risks from public exposures, Section 6.3.2 describes estimated risks from worker exposures, and Section 6.3.3 presents the estimated risks for public and worker exposures from accidents. In each section, the discussion summarizes the scenarios for which the estimated hazard index is greater than one, which indicates that there is a risk of noncancer health effects from that type of exposure, or for which the estimated cancer risk is greater than 1 in 1 million. Hazard indices and cancer risks from scenarios that are not discussed in the following sections are all associated with negligible risks. Tables 6-1 through 6-28 at the end of this chapter (following Section 6.5) present the estimated hazard indices and cancer risks for all chemicals in all the scenarios evaluated.

The risk tables in this section use scientific notation, since many of the values are very small. For example, the notation 3.63E-001 represents 3.63×10^{-1}, or 0.363. Similarly, 4.65E-009 represents 4.65×10^{-9}, or 0.00000000465.

6.3.1 Risks to the Public

The hazard indices and cancer risks calculated for typical and maximum exposures to the public are summarized in Tables 6-1 to 6-12.

For members of the public, hazard indices were less than one for all typical and maximum exposure scenarios, and cancer risks were all less than 1×10^{-6} (one in one million), ranging up to 7.54×10^{-20} to 8.98×10^{-10} (8.98 in ten billion).

There is a block of private property outside the eastern border of the seed orchard grounds. Risks from seed orchard pesticide drift to users of these properties would be no greater than risks from the drift calculations that were applied to recreational hikers or blackberry harvesters, which do not exceed the levels of concern. That is, all hazard indices are less than one and all cancer risks are less than one in one million for these scenarios.

6.3.2 Risks to Workers

The hazard indices and cancer risks that were estimated for worker exposures are presented in Tables 6-13 to 6-18.

For typical scenarios, all hazard indices are less than one, except for a hand-held hydraulic wand mixer/loader/applicator applying dimethoate (HI = 6.13), and a backpack sprayer applying dimethoate (HI = 4,220), permethrin (HI = 1.34), propargite (HI = 8.56), or dicamba (HI = 1.64). In the maximum scenarios, the hazard indices exceed one for a high-pressure hydraulic sprayer mixer/loader/applicator applying diazinon; a hand-held hydraulic wand mixer/loader/applicator applying diazinon or dimethoate; a backpack sprayer applying dimethoate, permethrin, propargite, dicamba, or hexazinone; and an irrigation system maintenance worker encountering residues of chlorpyrifos or diazinon. The estimated cancer risk to backpack sprayers applying propargite is 2.54 in 100 thousand, exceeding the standard point of departure of one in one million. All other worker cancer risks were less than one in one million. If applications of these pesticides were prescribed, risks to mixer/loader/applicators could be mitigated by decreasing the application rate, using water soluble bags (if available), spreading the work over a longer time period, increasing the use of personal protective equipment, and dividing the work between two or more workers. Risks to irrigation system maintenance workers could be mitigated by increasing the time period between applications and maintenance activities to allow additional degradation, decreasing the application rate, increasing the use of personal protective equipment, and dividing the work between two or more workers.

6.3.3 Risks from Accidents

Risks to members of the public from accidents are presented in Tables 6-19 through 6-22. Risks for accidents involving workers are presented in Tables 6-23 through 6-25.

Risks from Accidents to Members of the Public

For a spill of a container of pesticide concentrate or fertilizer at the mixing area, no risks to the public from drinking groundwater contaminated by leached chemical were predicted. If precipitation caused runoff of spill residues to surface water from the spill site, risks were predicted from chlorpyrifos and diazinon to adults and children consuming fish or surface water from the Applegate River. All estimated cancer risks were less than one in one million.

For a spill of an application tankload of mixed pesticide into Bridge Point Ditch, risks to the public from drinking water and eating fish from the Applegate River are predicted for chlorpyrifos, diazinon, propargite, and chlorothalonil. All cancer risks are less than one in one million.

For a spill of an application tankload of mixed pesticide into Williams Creek, risks to the public from drinking water and eating fish from the Applegate River are predicted for chlorpyrifos, diazinon, propargite, and chlorothalonil. All cancer risks are less than one in one million.

Risks from Accidents to Workers

In the scenario in which a worker spills liquid pesticide concentrate on the skin, hazard indices exceed one (ranging up to 10,100 for dimethoate) for handling acephate implants that were spilled on, and for dimethoate, esfenvalerate, permethrin, chlorothalonil, and dicamba. Estimated cancer risks were all less than one in one million.

In the scenario in which a worker spills tank-mixed diluted pesticide on the skin, hazard indices are greater than one for chlorpyrifos, diazinon, dimethoate, and dicamba. All estimated cancer risks are less than one in one million.

Hazard indices for the accident scenario in which a worker was directly sprayed exceed one for dimethoate. Estimated cancer risks are all less than one in one million.

6.4 Cumulative Human Health Risks

When humans are exposed to more than one chemical at a time, the potential risk may be a result of additive, antagonistic, or synergistic toxicity among the chemicals. Synergistic toxicity occurs when two chemicals interact to create a toxic effect greater than the sum of the toxic effects of each chemical individually. Antagonism occurs if one chemical decreases the adverse effects of another, such as the action that a drug has when it is administered to counteract the effect of a chemical toxin. The risks from simultaneous exposure to two or more chemicals are assumed to be additive in the absence of specific information on synergism or antagonism (EPA 1986b).

The literature review undertaken for this risk assessment included chemical interactions among the evaluated chemicals. No specific data on synergistic or antagonistic toxicity among the chemicals evaluated in this risk assessment were identified. Additive risks were therefore assumed for all scenarios that could overlap in time for one individual, leading to simultaneous doses of more than one pesticide in any exposure scenario. Since the maximum scenarios evaluated in this risk assessment involve upper-bound assumptions for exposure parameters, aggregation of risks by multiple exposure routes and for combinations of pesticides was limited to the results of the typical exposure scenarios, to avoid an unrealistic overestimation of the total cumulative risk from proposed chemical use at the seed orchard.

The results of the cumulative risk assessment for members of the public are presented in Table 6-26. The chemical-specific values in these tables represent the aggregated risks from all routes of exposure for each chemical, as estimated for the typical scenarios. These aggregated risks are added together to provide an upper bound estimate of the cumulative risk for adults and children. Actual cumulative risk values are likely to be far less than the results estimated here, since (1) it is highly unlikely that one individual would be exposed to every chemical in all of the scenarios evaluated in the risk assessment; (2) several pesticides are proposed for use as alternatives for certain groups of target pests or weeds, and if one was selected for use in a given season, the alternatives would not also be used; (3) where multiple application methods are possible for a proposed pesticide treatment scenario, the method with the highest associated risk was included in the cumulative assessment; and (4) the temporal spacing of the potential chemical applications would correspond to a timeline in which some exposure routes were no longer active due to dissipation and degradation, prior to application of other chemicals. The upper bound cumulative risk estimates are as follows:

- Cumulative hazard indices are 0.0503 and 0.0718 for adult and child members of the public, respectively.

- Cumulative cancer risks are 1.33×10^{-9} and 2.20×10^{-9} for adult and child members of the public, respectively.

Table 6-27 presents the cumulative risk to members of the public from the subset of proposed chemicals that are more likely than the others to be used in a given year. In this case, the cumulative hazard indices are 0.000543 and 0.000889 for adult and child members of the public, respectively; and there are no cancer risks for members of the public.

For workers (see Table 6-28), the highest cumulative exposure could occur if one employee was involved in all pesticide applications. In this case, the cumulative hazard index for workers is

4,230, and the cumulative cancer risk is 2.55 in 100 thousand. It is important to note that this scenario includes the unlikely case in which all pesticides that target every pest problem are called for during the season. The highest contributor to the cumulative hazard index is dimethoate (4,220) for an individual applying the chemical by a backpack sprayer and conducting irrigation system maintenance activities. The estimated cumulative cancer risk to workers is 2.55×10^{-5}. The main contributor to this risk is propargite, which is associated with a 2.54×10^{-5} cancer risk for an individual conducting backpack application and irrigation system maintenance activities.

Table 6-29 presents the cumulative risk to workers from the subset of proposed chemicals that are more likely than the others to be used in a given year. In this case, the cumulative hazard index is 0.138, and the cumulative cancer risk is zero.

6.5 Dermal and Eye Effects

If a pesticide comes into contact with unprotected skin or eyes, the effects may range from no effect or mild irritation to severe tissue damage. Table 6-30 summarizes the skin and eye irritation conclusions for each chemical that are presented throughout Section 4; detailed information can be found in the chemical-specific discussions in that section.

6.6 Uncertainties in the Human Health Risk Assessment

The risks summarized in this assessment are not probabilistic estimates of risk, but are conditional estimates. That is, these risks are likely only if all exposure scenario assumptions that were described are met. In addition, the methodology applied to estimating risks is not definitive, since uncertainty in the final risk estimates is introduced in almost every step of the assessment. Some of the primary areas of uncertainty are as follows:

- The accuracy of the RfDs in approximating doses to humans that pose negligible risk of health effects, without either under- or overestimating these doses: The RfDs are derived from tests in laboratory animals. Extrapolating the results of animal tests to human health hazards has an inherent level of uncertainty associated with it. Further discussion of this issue can be found in references such as Roloff et al. (1987) and Clewell and Anderson (1987).

- The use of the conservative approach, recommended by EPA, that chronic toxic data be used in estimating risks from occasional (or, at most, subchronic) exposures to the chemicals proposed for use at the seed orchard.

- The cancer slope factors, in providing a good approximation of the chemical's carcinogenic potency in humans: Updated methods for estimating cancer risks are in progress that may provide a different approach to estimating cancer risks for some of the chemicals evaluated in this report. Reassessment of the carcinogenic mechanism and application of an appropriate strategy for cancer risk assessment for any one chemical may be years away. This analysis uses the cancer risk approach currently used by the U.S. EPA for estimating the cancer potency of each chemical.

- The equations and studies on which the dose estimations are based: Many monitoring studies have been conducted since the 1970s that measure exposures to pesticides in a range of situations. This risk assessment relies on those that (1) are most relevant to the types of applications at the seed orchard, (2) incorporated sound methodology to provide a degree of confidence in the reported results, and (3) monitored, correlated, and reported a sufficient number of parameters to allow extrapolation to other situations.

All together, it is likely that the uncertainty in the risk estimates predicted in this assessment spans at least an order of magnitude. For example, for a hazard index estimated to be 0.0035, the true value is likely to be within the range of 0.035 to 0.00035, as a result of the uncertainties described here.

Table 6-1. Ingestion of Groundwater

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Diazinon	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Diazinon	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dimethoate	HHW	-0-	5.46E-007	-0-	-0-	8.51E-007	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	5.46E-007	-0-	-0-	8.51E-007	-0-
Dimethoate	Backpack	-0-	5.46E-007	-0-	-0-	8.51E-007	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	5.46E-007	-0-	-0-	8.51E-007	-0-
Esfenvalerate	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Horticultural Oil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Propargite	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Propargite	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Propargite	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Chlorothalonil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Chlorothalonil	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	2.48E-009	-0-	-0-	3.86E-009	-0-
Hexazinone	Backpack	-0-	2.48E-009	-0-	-0-	3.86E-009	-0-
Picloram	HHW	-0-	2.00E-009	-0-	-0-	3.11E-009	-0-
Hexachlorobenzene		-0-	-0-	6.16E-015	-0-	-0-	9.61E-015
Picloram	Backpack	-0-	2.00E-009	-0-	-0-	3.11E-009	-0-
Hexachlorobenzene		-0-	-0-	6.16E-015	-0-	-0-	9.61E-015
Triclopyr butoxyethyl ester	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Fertilizer (N as nitrate)	Spreader	-0-	-0-	-0-	-0-	-0-	-0-

*HI = Hazard Index

Table 6-2. Ingestion of Surface Water from Williams Creek

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	4.34E-007	-0-	-0-	6.77E-007	-0-
Chlorpyrifos	HHW	-0-	4.34E-007	-0-	-0-	6.77E-007	-0-
Diazinon	HPHS	-0-	3.41E-007	-0-	-0-	5.32E-007	-0-
Diazinon	HHW	-0-	3.41E-007	-0-	-0-	5.32E-007	-0-
Dimethoate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	1.68E-010	-0-	-0-	2.62E-010	-0-
Additive Risk		-0-	1.68E-010	-0-	-0-	2.62E-010	-0-
Dimethoate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	1.68E-010	-0-	-0-	2.62E-010	-0-
Additive Risk		-0-	1.68E-010	-0-	-0-	2.62E-010	-0-
Esfenvalerate	HPHS	-0-	8.33E-010	-0-	-0-	1.30E-009	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	8.33E-010	-0-	-0-	1.30E-009	-0-
Esfenvalerate	HHW	-0-	1.05E-009	-0-	-0-	1.64E-009	-0-
Ethylbenzene		-0-	1.13E-011	-0-	-0-	1.76E-011	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.06E-009	-0-	-0-	1.66E-009	-0-
Esfenvalerate	Backpack	-0-	1.05E-009	-0-	-0-	1.64E-009	-0-
Ethylbenzene		-0-	1.13E-011	-0-	-0-	1.76E-011	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.06E-009	-0-	-0-	1.66E-009	-0-
Horticultural Oil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HPHS	-0-	2.81E-010	7.39E-018	-0-	4.38E-010	1.15E-017
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	1.75E-009	-0-	-0-	2.73E-009	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	2.03E-009	7.39E-018	-0-	3.17E-009	1.15E-017
Permethrin	HHW	-0-	7.37E-012	1.94E-019	-0-	1.15E-011	3.02E-019
Ethylbenzene		-0-	6.74E-012	-0-	-0-	1.05E-011	-0-
Light aromatic solvent naphtha		-0-	4.18E-010	-0-	-0-	6.51E-010	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.32E-010	1.94E-019	-0-	6.73E-010	3.02E-019
Permethrin	Backpack	-0-	7.37E-012	1.94E-019	-0-	1.15E-011	3.02E-019
Ethylbenzene		-0-	6.74E-012	-0-	-0-	1.05E-011	-0-
Light aromatic solvent naphtha		-0-	4.18E-010	-0-	-0-	6.51E-010	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.32E-010	1.94E-019	-0-	6.73E-010	3.02E-019
Propargite	HPHS	-0-	2.34E-009	3.25E-016	-0-	3.65E-009	5.07E-016
Propargite	HHW	-0-	2.34E-009	3.25E-016	-0-	3.65E-009	5.07E-016
Propargite	Backpack	-0-	2.34E-009	3.25E-016	-0-	3.65E-009	5.07E-016
Chlorothalonil	HPHS	-0-	9.20E-009	3.56E-017	-0-	1.43E-008	5.55E-017
Chlorothalonil	HHW	-0-	9.20E-009	3.56E-017	-0-	1.43E-008	5.55E-017
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	6.63E-012	-0-	-0-	1.03E-011	-0-
Glyphosate	Backpack	-0-	6.63E-012	-0-	-0-	1.03E-011	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	6.64E-012	-0-	-0-	1.04E-011	-0-
Hexazinone	Backpack	-0-	6.64E-012	-0-	-0-	1.04E-011	-0-
Picloram	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Picloram	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW	-0-	1.08E-011	-0-	-0-	1.69E-011	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	1.08E-011	-0-	-0-	1.69E-011	-0-
Fertilizer (N as nitrate)	Spreader	-0-	1.34E-005	-0-	-0-	2.08E-005	-0-

*HI = Hazard Index

Table 6-3. Ingestion of Surface Water from Applegate River

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	1.69E-007	-0-	-0-	2.63E-007	-0-
Chlorpyrifos	HHW	-0-	1.69E-007	-0-	-0-	2.63E-007	-0-
Diazinon	HPHS	-0-	2.22E-008	-0-	-0-	3.46E-008	-0-
Diazinon	HHW	-0-	2.22E-008	-0-	-0-	3.46E-008	-0-
Dimethoate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	6.54E-011	-0-	-0-	1.02E-010	-0-
Additive Risk		-0-	6.54E-011	-0-	-0-	1.02E-010	-0-
Dimethoate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	6.54E-011	-0-	-0-	1.02E-010	-0-
Additive Risk		-0-	6.54E-011	-0-	-0-	1.02E-010	-0-
Esfenvalerate	HPHS	-0-	1.28E-009	-0-	-0-	1.99E-009	-0-
Ethylbenzene		-0-	1.83E-013	-0-	-0-	2.85E-013	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.28E-009	-0-	-0-	1.99E-009	-0-
Esfenvalerate	HHW	-0-	4.10E-010	-0-	-0-	6.39E-010	-0-
Ethylbenzene		-0-	7.34E-013	-0-	-0-	1.14E-012	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.10E-010	-0-	-0-	6.40E-010	-0-
Esfenvalerate	Backpack	-0-	4.10E-010	-0-	-0-	6.39E-010	-0-
Ethylbenzene		-0-	7.34E-013	-0-	-0-	1.14E-012	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.10E-010	-0-	-0-	6.40E-010	-0-
Horticultural Oil	HPHS	-0-	1.19E-011	-0-	-0-	1.86E-011	-0-
Permethrin	HPHS	-0-	4.88E-010	1.28E-017	-0-	7.60E-010	2.00E-017
Ethylbenzene		-0-	8.13E-013	-0-	-0-	1.27E-012	-0-
Light aromatic solvent naphtha		-0-	3.19E-009	-0-	-0-	4.97E-009	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	3.68E-009	1.28E-017	-0-	5.74E-009	2.00E-017
Permethrin	HHW	-0-	2.87E-012	7.54E-020	-0-	4.47E-012	1.18E-019
Ethylbenzene		-0-	4.39E-013	-0-	-0-	6.84E-013	-0-
Light aromatic solvent naphtha		-0-	1.63E-010	-0-	-0-	2.53E-010	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.66E-010	7.54E-020	-0-	2.59E-010	1.18E-019
Permethrin	Backpack	-0-	2.87E-012	7.54E-020	-0-	4.47E-012	1.18E-019
Ethylbenzene		-0-	4.39E-013	-0-	-0-	6.84E-013	-0-
Light aromatic solvent naphtha		-0-	1.63E-010	-0-	-0-	2.53E-010	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.66E-010	7.54E-020	-0-	2.59E-010	1.18E-019
Propargite	HPHS	-0-	3.91E-009	5.43E-016	-0-	6.10E-009	8.46E-016
Propargite	HHW	-0-	3.91E-009	5.43E-016	-0-	6.10E-009	8.46E-016
Propargite	Backpack	-0-	3.91E-009	5.43E-016	-0-	6.10E-009	8.46E-016
Chlorothalonil	HPHS	-0-	4.82E-009	1.87E-017	-0-	7.52E-009	2.91E-017
Chlorothalonil	HHW	-0-	4.82E-009	1.87E-017	-0-	7.52E-009	2.91E-017
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	2.58E-012	-0-	-0-	4.02E-012	-0-
Glyphosate	Backpack	-0-	2.58E-012	-0-	-0-	4.02E-012	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	4.32E-013	-0-	-0-	6.74E-013	-0-
Hexazinone	Backpack	-0-	4.32E-013	-0-	-0-	6.74E-013	-0-
Picloram	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Picloram	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW	-0-	4.21E-012	-0-	-0-	6.56E-012	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	4.21E-012	-0-	-0-	6.56E-012	-0-
Fertilizer (N as nitrate)	Spreader	-0-	6.31E-006	-0-	-0-	9.84E-006	-0-

*HI = Hazard Index

Table 6-4. Ingestion of Fish from Williams Creek

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	1.01E-004	-0-	-0-	3.22E-004	-0-
Chlorpyrifos	HHW	-0-	1.01E-004	-0-	-0-	3.22E-004	-0-
Diazinon	HPHS	-0-	1.58E-005	-0-	-0-	5.03E-005	-0-
Diazinon	HHW	-0-	1.58E-005	-0-	-0-	5.03E-005	-0-
Dimethoate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	6.61E-010	-0-	-0-	2.10E-009	-0-
Additive Risk		-0-	6.61E-010	-0-	-0-	2.10E-009	-0-
Dimethoate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	6.61E-010	-0-	-0-	2.10E-009	-0-
Additive Risk		-0-	6.61E-010	-0-	-0-	2.10E-009	-0-
Esfenvalerate	HPHS	-0-	9.98E-008	-0-	-0-	3.17E-007	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	9.98E-008	-0-	-0-	3.17E-007	-0-
Esfenvalerate	HHW	-0-	1.26E-007	-0-	-0-	4.01E-007	-0-
Ethylbenzene		-0-	1.45E-011	-0-	-0-	4.60E-011	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.26E-007	-0-	-0-	4.01E-007	-0-
Esfenvalerate	Backpack	-0-	1.26E-007	-0-	-0-	4.01E-007	-0-
Ethylbenzene		-0-	1.45E-011	-0-	-0-	4.60E-011	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.26E-007	-0-	-0-	4.01E-007	-0-
Horticultural Oil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HPHS	-0-	1.15E-008	3.04E-016	-0-	3.67E-008	9.64E-016
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	1.50E-007	-0-	-0-	4.76E-007	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.61E-007	3.04E-016	-0-	5.12E-007	9.64E-016
Permethrin	HHW	-0-	3.03E-010	7.96E-018	-0-	9.61E-010	2.53E-017
Ethylbenzene		-0-	8.65E-012	-0-	-0-	2.75E-011	-0-
Light aromatic solvent naphtha		-0-	3.57E-008	-0-	-0-	1.14E-007	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	3.60E-008	7.96E-018	-0-	1.15E-007	2.53E-017
Permethrin	Backpack	-0-	3.03E-010	7.96E-018	-0-	9.61E-010	2.53E-017
Ethylbenzene		-0-	8.65E-012	-0-	-0-	2.75E-011	-0-
Light aromatic solvent naphtha		-0-	3.57E-008	-0-	-0-	1.14E-007	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	3.60E-008	7.96E-018	-0-	1.15E-007	2.53E-017
Propargite	HPHS	-0-	1.55E-007	2.16E-014	-0-	4.94E-007	6.85E-014
Propargite	HHW	-0-	1.55E-007	2.16E-014	-0-	4.94E-007	6.85E-014
Propargite	Backpack	-0-	1.55E-007	2.16E-014	-0-	4.94E-007	6.85E-014
Chlorothalonil	HPHS	-0-	2.08E-007	8.04E-016	-0-	6.60E-007	2.55E-015
Chlorothalonil	HHW	-0-	2.08E-007	8.04E-016	-0-	6.60E-007	2.55E-015
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	2.95E-013	-0-	-0-	9.37E-013	-0-
Glyphosate	Backpack	-0-	2.95E-013	-0-	-0-	9.37E-013	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	1.14E-012	-0-	-0-	3.61E-012	-0-
Hexazinone	Backpack	-0-	1.14E-012	-0-	-0-	3.61E-012	-0-
Picloram	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Picloram	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW	-0-	9.99E-013	-0-	-0-	3.17E-012	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	9.99E-013	-0-	-0-	3.17E-012	-0-

*HI = Hazard Index

Table 6-5. Ingestion of Fish from Applegate River

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	3.94E-005	-0-	-0-	1.25E-004	-0-
Chlorpyrifos	HHW	-0-	3.94E-005	-0-	-0-	1.25E-004	-0-
Diazinon	HPHS	-0-	1.03E-006	-0-	-0-	3.27E-006	-0-
Diazinon	HHW	-0-	1.03E-006	-0-	-0-	3.27E-006	-0-
Dimethoate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	2.57E-010	-0-	-0-	8.18E-010	-0-
Additive Risk		-0-	2.57E-010	-0-	-0-	8.18E-010	-0-
Dimethoate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	2.57E-010	-0-	-0-	8.18E-010	-0-
Additive Risk		-0-	2.57E-010	-0-	-0-	8.18E-010	-0-
Esfenvalerate	HPHS	-0-	1.53E-007	-0-	-0-	4.86E-007	-0-
Ethylbenzene		-0-	2.35E-013	-0-	-0-	7.46E-013	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.53E-007	-0-	-0-	4.86E-007	-0-
Esfenvalerate	HHW	-0-	4.91E-008	-0-	-0-	1.56E-007	-0-
Ethylbenzene		-0-	9.41E-013	-0-	-0-	2.99E-012	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.91E-008	-0-	-0-	1.56E-007	-0-
Esfenvalerate	Backpack	-0-	4.91E-008	-0-	-0-	1.56E-007	-0-
Ethylbenzene		-0-	9.41E-013	-0-	-0-	2.99E-012	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	4.91E-008	-0-	-0-	1.56E-007	-0-
Horticultural Oil	HPHS	-0-	4.69E-011	-0-	-0-	1.49E-010	-0-
Permethrin	HPHS	-0-	2.00E-008	5.27E-016	-0-	6.36E-008	1.67E-015
Ethylbenzene		-0-	1.04E-012	-0-	-0-	3.32E-012	-0-
Light aromatic solvent naphtha		-0-	2.73E-007	-0-	-0-	8.67E-007	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	2.93E-007	5.27E-016	-0-	9.31E-007	1.67E-015
Permethrin	HHW	-0-	1.18E-010	3.10E-018	-0-	3.74E-010	9.84E-018
Ethylbenzene		-0-	5.63E-013	-0-	-0-	1.79E-012	-0-
Light aromatic solvent naphtha		-0-	1.39E-008	-0-	-0-	4.42E-008	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.40E-008	3.10E-018	-0-	4.45E-008	9.84E-018
Permethrin	Backpack	-0-	1.18E-010	3.10E-018	-0-	3.74E-010	9.84E-018
Ethylbenzene		-0-	5.63E-013	-0-	-0-	1.79E-012	-0-
Light aromatic solvent naphtha		-0-	1.39E-008	-0-	-0-	4.42E-008	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	1.40E-008	3.10E-018	-0-	4.45E-008	9.84E-018
Propargite	HPHS	-0-	2.59E-007	3.60E-014	-0-	8.24E-007	1.14E-013
Propargite	HHW	-0-	2.59E-007	3.60E-014	-0-	8.24E-007	1.14E-013
Propargite	Backpack	-0-	2.59E-007	3.60E-014	-0-	8.24E-007	1.14E-013
Chlorothalonil	HPHS	-0-	1.09E-007	4.22E-016	-0-	3.46E-007	1.34E-015
Chlorothalonil	HHW	-0-	1.09E-007	4.22E-016	-0-	3.46E-007	1.34E-015
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	1.15E-013	-0-	-0-	3.65E-013	-0-
Glyphosate	Backpack	-0-	1.15E-013	-0-	-0-	3.65E-013	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	7.40E-014	-0-	-0-	2.35E-013	-0-
Hexazinone	Backpack	-0-	7.40E-014	-0-	-0-	2.35E-013	-0-
Picloram	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Picloram	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW	-0-	3.89E-013	-0-	-0-	1.23E-012	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	3.89E-013	-0-	-0-	1.23E-012	-0-

*HI = Hazard Index

Table 6-6. Ingestion of Fish from Onsite Pond

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	-0-	7.96E-005	-0-	-0-	2.53E-004	-0-
Chlorpyrifos	HHW	-0-	7.96E-005	-0-	-0-	2.53E-004	-0-
Diazinon	HPHS	-0-	1.42E-006	-0-	-0-	4.52E-006	-0-
Diazinon	HHW	-0-	1.42E-006	-0-	-0-	4.52E-006	-0-
Dimethoate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Dimethoate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Cyclohexanone		-0-	-0-	-0-	-0-	-0-	-0-
Petroleum distillate		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Esfenvalerate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Horticultural Oil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Permethrin	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Ethylbenzene		-0-	-0-	-0-	-0-	-0-	-0-
Light aromatic solvent naphtha		-0-	-0-	-0-	-0-	-0-	-0-
Xylene		-0-	-0-	-0-	-0-	-0-	-0-
Additive Risk		-0-	-0-	-0-	-0-	-0-	-0-
Propargite	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Propargite	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Propargite	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Chlorothalonil	HPHS	-0-	-0-	-0-	-0-	-0-	-0-
Chlorothalonil	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Dicamba	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Boom	-0-	-0-	-0-	-0-	-0-	-0-
Glyphosate	Wick	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexazinone	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Picloram	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Picloram	Backpack	-0-	-0-	-0-	-0-	-0-	-0-
Hexachlorobenzene		-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	HHW	-0-	-0-	-0-	-0-	-0-	-0-
Triclopyr butoxyethyl ester	Backpack	-0-	-0-	-0-	-0-	-0-	-0-

*HI = Hazard Index

Table 6-7. Ingestion of Venison

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	2.24E-004	1.32E-003	-0-	3.58E-004	2.10E-003	-0-
Chlorpyrifos	HHW	2.24E-004	1.32E-003	-0-	3.58E-004	2.10E-003	-0-
Diazinon	HPHS	2.69E-006	1.23E-004	-0-	4.29E-006	1.96E-004	-0-
Diazinon	HHW	2.69E-006	1.23E-004	-0-	4.29E-006	1.96E-004	-0-
Dimethoate	HHW	3.78E-006	1.86E-004	-0-	6.03E-006	2.96E-004	-0-
Cyclohexanone		3.75E-012	1.69E-010	-0-	5.98E-012	2.69E-010	-0-
Petroleum distillate		2.48E-004	1.44E-003	-0-	3.96E-004	2.30E-003	-0-
Additive Risk		2.52E-004	1.63E-003	-0-	4.02E-004	2.59E-003	-0-
Dimethoate	Backpack	3.78E-006	1.86E-004	-0-	6.03E-006	2.96E-004	-0-
Cyclohexanone		3.75E-012	1.69E-010	-0-	5.98E-012	2.69E-010	-0-
Petroleum distillate		2.48E-004	1.44E-003	-0-	3.96E-004	2.30E-003	-0-
Additive Risk		2.52E-004	1.63E-003	-0-	4.02E-004	2.59E-003	-0-
Esfenvalerate	HPHS	1.22E-007	6.71E-007	-0-	1.95E-007	1.07E-006	-0-
Ethylbenzene		1.37E-010	9.88E-010	-0-	2.18E-010	1.58E-009	-0-
Xylene		8.46E-010	4.97E-009	-0-	1.35E-009	7.92E-009	-0-
Additive Risk		1.23E-007	6.77E-007	-0-	1.97E-007	1.08E-006	-0-
Esfenvalerate	HHW	1.22E-007	6.71E-007	-0-	1.95E-007	1.07E-006	-0-
Ethylbenzene		1.37E-010	9.88E-010	-0-	2.18E-010	1.58E-009	-0-
Xylene		8.46E-010	4.97E-009	-0-	1.35E-009	7.92E-009	-0-
Additive Risk		1.23E-007	6.77E-007	-0-	1.97E-007	1.08E-006	-0-
Esfenvalerate	Backpack	1.22E-007	6.71E-007	-0-	1.95E-007	1.07E-006	-0-
Ethylbenzene		1.37E-010	9.88E-010	-0-	2.18E-010	1.58E-009	-0-
Xylene		8.46E-010	4.97E-009	-0-	1.35E-009	7.92E-009	-0-
Additive Risk		1.23E-007	6.77E-007	-0-	1.97E-007	1.08E-006	-0-
Horticultural Oil	HPHS	1.71E-005	1.17E-004	-0-	2.73E-005	1.86E-004	-0-
Permethrin	HPHS	6.02E-008	3.32E-007	3.88E-014	9.60E-008	5.29E-007	6.19E-014
Ethylbenzene		6.16E-010	4.33E-009	-0-	9.82E-010	6.90E-009	-0-
Light aromatic solvent naphtha		4.69E-009	1.32E-006	-0-	7.47E-009	2.10E-006	-0-
Xylene		1.49E-011	1.66E-009	-0-	2.37E-011	2.64E-009	-0-
Additive Risk		6.56E-008	1.66E-006	3.88E-014	1.05E-007	2.64E-006	6.19E-014
Permethrin	HHW	1.76E-009	2.43E-008	1.52E-015	2.81E-009	3.88E-008	2.42E-015
Ethylbenzene		1.80E-011	3.89E-010	-0-	2.87E-011	6.21E-010	-0-
Light aromatic solvent naphtha		1.37E-010	1.97E-007	-0-	2.18E-010	3.14E-007	-0-
Xylene		4.34E-013	2.45E-010	-0-	6.92E-013	3.90E-010	-0-
Additive Risk		1.92E-009	2.22E-007	1.52E-015	3.06E-009	3.54E-007	2.42E-015
Permethrin	Backpack	1.76E-009	2.43E-008	1.52E-015	2.81E-009	3.88E-008	2.42E-015
Ethylbenzene		1.80E-011	3.89E-010	-0-	2.87E-011	6.21E-010	-0-
Light aromatic solvent naphtha		1.37E-010	1.97E-007	-0-	2.18E-010	3.14E-007	-0-
Xylene		4.34E-013	2.45E-010	-0-	6.92E-013	3.90E-010	-0-
Additive Risk		1.92E-009	2.22E-007	1.52E-015	3.06E-009	3.54E-007	2.42E-015
Propargite	HPHS	1.12E-006	3.44E-005	7.72E-012	1.78E-006	5.48E-005	1.23E-011
Propargite	HHW	1.12E-006	3.44E-005	7.72E-012	1.78E-006	5.48E-005	1.23E-011
Propargite	Backpack	1.12E-006	3.44E-005	7.72E-012	1.78E-006	5.48E-005	1.23E-011
Chlorothalonil	HPHS	3.58E-007	3.47E-006	3.98E-014	5.71E-007	5.52E-006	6.34E-014
Chlorothalonil	HHW	3.58E-007	3.47E-006	3.98E-014	5.71E-007	5.52E-006	6.34E-014
Dicamba	HHW	3.74E-009	2.87E-007	-0-	5.96E-009	4.57E-007	-0-
Dicamba	Backpack	3.74E-009	2.87E-007	-0-	5.96E-009	4.57E-007	-0-
Dicamba	Boom	3.74E-009	2.87E-007	-0-	5.96E-009	4.57E-007	-0-
Dicamba	Wick	2.49E-009	2.87E-007	-0-	3.97E-009	4.57E-007	-0-
Glyphosate	HHW	3.37E-016	3.11E-015	-0-	5.37E-016	4.96E-015	-0-
Glyphosate	Backpack	3.37E-016	1.75E-015	-0-	5.37E-016	2.79E-015	-0-
Glyphosate	Boom	8.42E-017	1.75E-015	-0-	1.34E-016	2.79E-015	-0-
Glyphosate	Wick	3.76E-017	1.40E-015	-0-	5.99E-017	2.23E-015	-0-
Hexazinone	HHW	2.42E-016	2.55E-016	-0-	3.86E-016	4.07E-016	-0-
Hexazinone	Backpack	2.42E-016	6.62E-015	-0-	3.86E-016	1.06E-014	-0-
Picloram	HHW	5.30E-011	1.04E-008	-0-	8.45E-011	1.66E-008	-0-
Hexachlorobenzene		-0-	-0-	5.33E-015	-0-	-0-	8.49E-015
Picloram	Backpack	5.30E-011	1.47E-008	-0-	8.45E-011	2.34E-008	-0-
Hexachlorobenzene		-0-	-0-	7.30E-015	-0-	-0-	1.16E-014
Triclopyr butoxyethyl ester	HHW	6.24E-009	2.07E-007	-0-	9.95E-009	3.30E-007	-0-
Triclopyr butoxyethyl ester	Backpack	6.24E-009	2.07E-007	-0-	9.95E-009	3.30E-007	-0-

*HI = Hazard Index

Table 6-8. Ingestion of Quail

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	1.43E-011	8.20E-021	-0-	2.40E-011	1.56E-019
Chlorpyrifos	HPHS	2.36E-003	9.43E-003	-0-	3.95E-003	1.58E-002	-0-
Chlorpyrifos	HHW	2.36E-003	9.43E-003	-0-	3.95E-003	1.58E-002	-0-
Diazinon	HPHS	2.83E-005	8.49E-004	-0-	4.74E-005	1.42E-003	-0-
Diazinon	HHW	2.83E-005	8.49E-004	-0-	4.74E-005	1.42E-003	-0-
Dimethoate	HHW	8.93E-005	4.68E-004	-0-	1.50E-004	7.83E-004	-0-
Cyclohexanone		8.85E-011	4.63E-010	-0-	1.48E-010	7.76E-010	-0-
Petroleum distillate		6.70E-005	3.51E-004	-0-	1.12E-004	5.87E-004	-0-
Additive Risk		1.56E-004	8.18E-004	-0-	2.62E-004	1.37E-003	-0-
Dimethoate	Backpack	8.93E-005	4.68E-004	-0-	1.50E-004	7.83E-004	-0-
Cyclohexanone		8.85E-011	4.63E-010	-0-	1.48E-010	7.76E-010	-0-
Petroleum distillate		6.70E-005	3.51E-004	-0-	1.12E-004	5.87E-004	-0-
Additive Risk		1.56E-004	8.18E-004	-0-	2.62E-004	1.37E-003	-0-
Esfenvalerate	HPHS	1.09E-006	4.48E-006	-0-	1.82E-006	7.50E-006	-0-
Ethylbenzene		1.22E-009	5.01E-009	-0-	2.04E-009	8.39E-009	-0-
Xylene		2.95E-010	1.21E-009	-0-	4.93E-010	2.03E-009	-0-
Additive Risk		1.09E-006	4.49E-006	-0-	1.83E-006	7.51E-006	-0-
Esfenvalerate	HHW	1.09E-006	4.48E-006	-0-	1.82E-006	7.50E-006	-0-
Ethylbenzene		1.22E-009	5.01E-009	-0-	2.04E-009	8.39E-009	-0-
Xylene		2.95E-010	1.21E-009	-0-	4.93E-010	2.03E-009	-0-
Additive Risk		1.09E-006	4.49E-006	-0-	1.83E-006	7.51E-006	-0-
Esfenvalerate	Backpack	1.09E-006	4.48E-006	-0-	1.82E-006	7.50E-006	-0-
Ethylbenzene		1.22E-009	5.01E-009	-0-	2.04E-009	8.39E-009	-0-
Xylene		2.95E-010	1.21E-009	-0-	4.93E-010	2.03E-009	-0-
Additive Risk		1.09E-006	4.49E-006	-0-	1.83E-006	7.51E-006	-0-
Horticultural Oil	HPHS	4.04E-004	1.35E-003	-0-	6.77E-004	2.25E-003	-0-
Permethrin	HPHS	5.36E-007	2.15E-006	3.24E-013	8.98E-007	3.59E-006	1.10E-012
Ethylbenzene		5.48E-009	2.19E-008	-0-	9.18E-009	3.67E-008	-0-
Light aromatic solvent naphtha		7.12E-007	2.85E-006	-0-	1.19E-006	4.77E-006	-0-
Xylene		2.26E-009	9.03E-009	-0-	3.78E-009	1.51E-008	-0-
Additive Risk		1.26E-006	5.02E-006	3.24E-013	2.10E-006	8.41E-006	1.10E-012
Permethrin	HHW	4.16E-008	2.51E-007	2.74E-014	6.96E-008	4.20E-007	1.27E-013
Ethylbenzene		4.25E-010	2.56E-009	-0-	7.12E-010	4.29E-009	-0-
Light aromatic solvent naphtha		5.52E-008	3.33E-007	-0-	9.24E-008	5.57E-007	-0-
Xylene		1.75E-010	1.05E-009	-0-	2.93E-010	1.77E-009	-0-
Additive Risk		9.74E-008	5.87E-007	2.74E-014	1.63E-007	9.83E-007	1.27E-013
Permethrin	Backpack	4.16E-008	2.51E-007	2.74E-014	6.96E-008	4.20E-007	1.27E-013
Ethylbenzene		4.25E-010	2.56E-009	-0-	7.12E-010	4.29E-009	-0-
Light aromatic solvent naphtha		5.52E-008	3.33E-007	-0-	9.24E-008	5.57E-007	-0-
Xylene		1.75E-010	1.05E-009	-0-	2.93E-010	1.77E-009	-0-
Additive Risk		9.74E-008	5.87E-007	2.74E-014	1.63E-007	9.83E-007	1.27E-013
Propargite	HPHS	2.64E-005	1.41E-004	8.93E-011	4.42E-005	2.37E-004	3.79E-010
Propargite	HHW	2.64E-005	1.41E-004	8.93E-011	4.42E-005	2.37E-004	3.79E-010
Propargite	Backpack	2.64E-005	1.41E-004	8.93E-011	4.42E-005	2.37E-004	3.79E-010
Chlorothalonil	HPHS	8.45E-006	5.28E-005	8.26E-013	1.42E-005	8.84E-005	3.94E-012
Chlorothalonil	HHW	8.45E-006	5.28E-005	8.26E-013	1.42E-005	8.84E-005	3.94E-012
Dicamba	HHW	8.83E-008	5.89E-007	-0-	1.48E-007	9.86E-007	-0-
Dicamba	Backpack	8.83E-008	5.89E-007	-0-	1.48E-007	9.86E-007	-0-
Dicamba	Boom	8.83E-008	5.89E-007	-0-	1.48E-007	9.86E-007	-0-
Dicamba	Wick	5.89E-008	1.77E-007	-0-	9.86E-008	2.96E-007	-0-
Glyphosate	HHW	7.95E-015	3.31E-014	-0-	1.33E-014	5.54E-014	-0-
Glyphosate	Backpack	7.95E-015	3.31E-014	-0-	1.33E-014	5.54E-014	-0-
Glyphosate	Boom	1.99E-015	2.65E-014	-0-	3.33E-015	4.44E-014	-0-
Glyphosate	Wick	8.87E-016	2.66E-015	-0-	1.49E-015	4.46E-015	-0-
Hexazinone	HHW	5.72E-015	7.63E-014	-0-	9.58E-015	1.28E-013	-0-
Hexazinone	Backpack	5.72E-015	7.63E-014	-0-	9.58E-015	1.28E-013	-0-
Picloram	HHW	1.25E-009	1.67E-008	-0-	2.10E-009	2.79E-008	-0-
Hexachlorobenzene		-0-	-0-	1.88E-014	-0-	-0-	1.49E-013
Picloram	Backpack	1.25E-009	1.67E-008	-0-	2.10E-009	2.79E-008	-0-
Hexachlorobenzene		-0-	-0-	1.88E-014	-0-	-0-	1.49E-013
Triclopyr butoxyethyl ester	HHW	1.47E-007	2.62E-006	-0-	2.47E-007	4.39E-006	-0-
Triclopyr butoxyethyl ester	Backpack	1.47E-007	2.62E-006	-0-	2.47E-007	4.39E-006	-0-

*HI = Hazard Index

Table 6-9. Ingestion of Goose

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	4.50E-009	2.57E-018	-0-	7.53E-009	4.31E-018
Chlorpyrifos	HPHS	4.73E-005	2.78E-004	-0-	7.93E-005	4.65E-004	-0-
Chlorpyrifos	HHW	4.73E-005	2.78E-004	-0-	7.93E-005	4.65E-004	-0-
Diazinon	HPHS	5.68E-007	2.60E-005	-0-	9.51E-007	4.35E-005	-0-
Diazinon	HHW	5.68E-007	2.60E-005	-0-	9.51E-007	4.35E-005	-0-
Dimethoate	HHW	7.98E-007	3.91E-005	-0-	1.34E-006	6.56E-005	-0-
Cyclohexanone		7.91E-013	3.56E-011	-0-	1.32E-012	5.97E-011	-0-
Petroleum distillate		5.23E-005	3.04E-004	-0-	8.77E-005	5.09E-004	-0-
Additive Risk		5.31E-005	3.43E-004	-0-	8.90E-005	5.75E-004	-0-
Dimethoate	Backpack	7.98E-007	3.91E-005	-0-	1.34E-006	6.56E-005	-0-
Cyclohexanone		7.91E-013	3.56E-011	-0-	1.32E-012	5.97E-011	-0-
Petroleum distillate		5.23E-005	3.04E-004	-0-	8.77E-005	5.09E-004	-0-
Additive Risk		5.31E-005	3.43E-004	-0-	8.90E-005	5.75E-004	-0-
Esfenvalerate	HPHS	2.58E-008	1.42E-007	-0-	4.32E-008	2.37E-007	-0-
Ethylbenzene		2.88E-011	2.08E-010	-0-	4.83E-011	3.49E-010	-0-
Xylene		1.78E-010	1.05E-009	-0-	2.99E-010	1.75E-009	-0-
Additive Risk		2.60E-008	1.43E-007	-0-	4.36E-008	2.39E-007	-0-
Esfenvalerate	HHW	2.58E-008	1.42E-007	-0-	4.32E-008	2.37E-007	-0-
Ethylbenzene		2.88E-011	2.08E-010	-0-	4.83E-011	3.49E-010	-0-
Xylene		1.78E-010	1.05E-009	-0-	2.99E-010	1.75E-009	-0-
Additive Risk		2.60E-008	1.43E-007	-0-	4.36E-008	2.39E-007	-0-
Esfenvalerate	Backpack	2.58E-008	1.42E-007	-0-	4.32E-008	2.37E-007	-0-
Ethylbenzene		2.88E-011	2.08E-010	-0-	4.83E-011	3.49E-010	-0-
Xylene		1.78E-010	1.05E-009	-0-	2.99E-010	1.75E-009	-0-
Additive Risk		2.60E-008	1.43E-007	-0-	4.36E-008	2.39E-007	-0-
Horticultural Oil	HPHS	3.61E-006	2.47E-005	-0-	6.05E-006	4.13E-005	-0-
Permethrin	HPHS	1.27E-008	7.01E-008	8.19E-015	2.13E-008	1.17E-007	1.37E-014
Ethylbenzene		1.30E-010	9.13E-010	-0-	2.18E-010	1.53E-009	-0-
Light aromatic solvent naphtha		9.88E-010	2.78E-007	-0-	1.66E-009	4.66E-007	-0-
Xylene		3.13E-012	3.49E-010	-0-	5.25E-012	5.85E-010	-0-
Additive Risk		1.38E-008	3.49E-007	8.19E-015	2.32E-008	5.85E-007	1.37E-014
Permethrin	HHW	3.72E-010	5.14E-009	3.21E-016	6.22E-010	8.60E-009	5.37E-016
Ethylbenzene		3.80E-012	8.21E-011	-0-	6.36E-012	1.38E-010	-0-
Light aromatic solvent naphtha		2.89E-011	4.16E-008	-0-	4.84E-011	6.96E-008	-0-
Xylene		9.16E-014	5.16E-011	-0-	1.53E-013	8.65E-011	-0-
Additive Risk		4.04E-010	4.69E-008	3.21E-016	6.77E-010	7.85E-008	5.37E-016
Permethrin	Backpack	3.72E-010	5.14E-009	3.21E-016	6.22E-010	8.60E-009	5.37E-016
Ethylbenzene		3.80E-012	8.21E-011	-0-	6.36E-012	1.38E-010	-0-
Light aromatic solvent naphtha		2.89E-011	4.16E-008	-0-	4.84E-011	6.96E-008	-0-
Xylene		9.16E-014	5.16E-011	-0-	1.53E-013	8.65E-011	-0-
Additive Risk		4.04E-010	4.69E-008	3.21E-016	6.77E-010	7.85E-008	5.37E-016
Propargite	HPHS	2.36E-007	7.25E-006	1.63E-012	3.95E-007	1.21E-005	2.73E-012
Propargite	HHW	2.36E-007	7.25E-006	1.63E-012	3.95E-007	1.21E-005	2.73E-012
Propargite	Backpack	2.36E-007	7.25E-006	1.63E-012	3.95E-007	1.21E-005	2.73E-012
Chlorothalonil	HPHS	7.56E-008	7.31E-007	8.39E-015	1.27E-007	1.22E-006	1.40E-014
Chlorothalonil	HHW	7.56E-008	7.31E-007	8.39E-015	1.27E-007	1.22E-006	1.40E-014
Dicamba	HHW	7.89E-010	6.05E-008	-0-	1.32E-009	1.01E-007	-0-
Dicamba	Backpack	7.89E-010	6.05E-008	-0-	1.32E-009	1.01E-007	-0-
Dicamba	Boom	7.89E-010	6.05E-008	-0-	1.32E-009	1.01E-007	-0-
Dicamba	Wick	5.26E-010	2.92E-008	-0-	8.81E-010	4.89E-008	-0-
Glyphosate	HHW	7.10E-017	3.69E-016	-0-	1.19E-016	6.19E-016	-0-
Glyphosate	Backpack	7.10E-017	3.69E-016	-0-	1.19E-016	6.19E-016	-0-
Glyphosate	Boom	1.78E-017	2.96E-016	-0-	2.97E-017	4.95E-016	-0-
Glyphosate	Wick	7.93E-018	3.36E-017	-0-	1.33E-017	5.63E-017	-0-
Hexazinone	HHW	5.11E-017	1.40E-015	-0-	8.56E-017	2.34E-015	-0-
Hexazinone	Backpack	5.11E-017	1.40E-015	-0-	8.56E-017	2.34E-015	-0-
Picloram	HHW	1.12E-011	2.20E-013	-0-	1.87E-011	3.69E-013	-0-
Hexachlorobenzene		-0-	-0-	1.44E-011	-0-	-0-	2.41E-011
Picloram	Backpack	1.12E-011	3.09E-013	-0-	1.87E-011	5.18E-013	-0-
Hexachlorobenzene		-0-	-0-	1.44E-011	-0-	-0-	2.41E-011
Triclopyr butoxyethyl ester	HHW	1.32E-009	4.37E-008	-0-	2.20E-009	7.31E-008	-0-
Triclopyr butoxyethyl ester	Backpack	1.32E-009	4.37E-008	-0-	2.20E-009	7.31E-008	-0-

*HI = Hazard Index

Table 6-10. Ingestion of Blackberries

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	6.78E-003	1.36E-003	-0-	3.95E-003	7.91E-003	-0-
Chlorpyrifos	HHW	4.44E-006	8.88E-007	-0-	2.59E-006	5.18E-006	-0-
Diazinon	HPHS	7.62E-003	1.14E-002	-0-	4.45E-003	6.67E-002	-0-
Diazinon	HHW	1.66E-006	2.50E-006	-0-	9.71E-007	1.46E-005	-0-
Dimethoate	HHW	8.35E-005	2.18E-005	-0-	4.87E-005	1.27E-004	-0-
Cyclohexanone		6.72E-009	1.76E-009	-0-	3.92E-009	1.03E-008	-0-
Petroleum distillate		8.14E-009	2.13E-009	-0-	4.75E-009	1.24E-008	-0-
Additive Risk		8.35E-005	2.18E-005	-0-	4.87E-005	1.27E-004	-0-
Dimethoate	Backpack	8.35E-006	2.18E-006	-0-	4.87E-006	1.27E-005	-0-
Cyclohexanone		6.72E-010	1.76E-010	-0-	3.92E-010	1.03E-009	-0-
Petroleum distillate		8.14E-010	2.13E-010	-0-	4.75E-010	1.24E-009	-0-
Additive Risk		8.35E-006	2.18E-006	-0-	4.87E-006	1.27E-005	-0-
Esfenvalerate	HPHS	5.08E-006	1.05E-006	-0-	2.97E-006	6.10E-006	-0-
Ethylbenzene		1.21E-007	2.49E-008	-0-	7.06E-008	1.45E-007	-0-
Xylene		1.81E-008	3.73E-009	-0-	1.06E-008	2.18E-008	-0-
Additive Risk		5.22E-006	1.07E-006	-0-	3.05E-006	6.27E-006	-0-
Esfenvalerate	HHW	1.55E-008	3.20E-009	-0-	9.07E-009	1.86E-008	-0-
Ethylbenzene		3.70E-010	7.61E-011	-0-	2.16E-010	4.44E-010	-0-
Xylene		5.55E-011	1.14E-011	-0-	3.24E-011	6.66E-011	-0-
Additive Risk		1.60E-008	3.28E-009	-0-	9.31E-009	1.92E-008	-0-
Esfenvalerate	Backpack	1.55E-009	3.20E-010	-0-	9.07E-010	1.86E-009	-0-
Ethylbenzene		3.70E-011	7.61E-012	-0-	2.16E-011	4.44E-011	-0-
Xylene		5.55E-012	1.14E-012	-0-	3.24E-012	6.66E-012	-0-
Additive Risk		1.60E-009	3.28E-010	-0-	9.31E-010	1.92E-009	-0-
Horticultural Oil	HPHS	2.22E-005	3.69E-006	-0-	1.29E-005	2.15E-005	-0-
Permethrin	HPHS	2.03E-005	4.07E-006	1.03E-011	1.19E-005	2.37E-005	2.34E-012
Ethylbenzene		5.30E-007	1.06E-007	-0-	3.09E-007	6.18E-007	-0-
Light aromatic solvent naphtha		4.26E-005	8.53E-006	-0-	2.49E-005	4.98E-005	-0-
Xylene		1.35E-007	2.70E-008	-0-	7.89E-008	1.58E-007	-0-
Additive Risk		6.36E-005	1.27E-005	1.03E-011	3.71E-005	7.43E-005	2.34E-012
Permethrin	HHW	6.39E-009	1.93E-009	3.25E-015	3.73E-009	1.12E-008	1.06E-015
Ethylbenzene		1.67E-010	5.02E-011	-0-	9.72E-011	2.93E-010	-0-
Light aromatic solvent naphtha		1.34E-008	4.04E-009	-0-	7.82E-009	2.36E-008	-0-
Xylene		4.25E-011	1.28E-011	-0-	2.48E-011	7.47E-011	-0-
Additive Risk		2.00E-008	6.03E-009	3.25E-015	1.17E-008	3.52E-008	1.06E-015
Permethrin	Backpack	6.39E-010	1.93E-010	3.25E-016	3.73E-010	1.12E-009	1.06E-016
Ethylbenzene		1.67E-011	5.02E-012	-0-	9.72E-012	2.93E-011	-0-
Light aromatic solvent naphtha		1.34E-009	4.04E-010	-0-	7.82E-010	2.36E-009	-0-
Xylene		4.25E-012	1.28E-012	-0-	2.48E-012	7.47E-012	-0-
Additive Risk		2.00E-009	6.03E-010	3.25E-016	1.17E-009	3.52E-009	1.06E-016
Propargite	HPHS	8.08E-005	1.39E-005	2.15E-010	4.72E-005	8.08E-005	4.31E-011
Propargite	HHW	1.55E-007	2.66E-008	4.13E-013	9.07E-008	1.55E-007	8.28E-014
Propargite	Backpack	1.55E-008	2.66E-009	4.13E-014	9.07E-009	1.55E-008	8.28E-015
Chlorothalonil	HPHS	3.23E-004	6.47E-005	2.40E-011	1.89E-004	3.77E-004	5.49E-012
Chlorothalonil	HHW	6.22E-007	1.24E-007	4.62E-014	3.63E-007	7.25E-007	1.05E-014
Dicamba	HHW	9.87E-008	1.97E-003	-0-	5.76E-008	1.15E-003	-0-
Dicamba	Backpack	9.87E-009	1.97E-003	-0-	5.76E-009	1.15E-003	-0-
Dicamba	Boom	3.28E-005	1.97E-003	-0-	1.91E-005	1.15E-003	-0-
Dicamba	Wick	-0-	9.87E-004	-0-	-0-	5.76E-004	-0-
Glyphosate	HHW	8.88E-009	1.11E-004	-0-	5.18E-009	6.48E-005	-0-
Glyphosate	Backpack	8.88E-010	1.11E-004	-0-	5.18E-010	6.48E-005	-0-
Glyphosate	Boom	7.37E-007	8.88E-005	-0-	4.30E-007	5.18E-005	-0-
Glyphosate	Wick	-0-	1.49E-005	-0-	-0-	8.68E-006	-0-
Hexazinone	HHW	1.60E-007	6.39E-003	-0-	9.32E-008	3.73E-003	-0-
Hexazinone	Backpack	1.60E-008	6.39E-003	-0-	9.32E-009	3.73E-003	-0-
Picloram	HHW	5.55E-009	2.22E-004	-0-	3.24E-009	1.30E-004	-0-
Hexachlorobenzene		-0-	-0-	1.30E-013	-0-	-0-	2.48E-012
Picloram	Backpack	5.55E-010	2.22E-004	-0-	3.24E-010	1.30E-004	-0-
Hexachlorobenzene		-0-	-0-	1.30E-013	-0-	-0-	2.48E-012
Triclopyr butoxyethyl ester	HHW	1.33E-008	7.10E-004	-0-	7.77E-009	4.14E-004	-0-
Triclopyr butoxyethyl ester	Backpack	1.33E-009	7.10E-004	-0-	7.77E-010	4.14E-004	-0-

*HI = Hazard Index

Table 6-11. Recreational Hiking

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	4.59E-003	1.84E-002	-0-	5.94E-003	2.37E-002	-0-
Chlorpyrifos	HHW	3.01E-006	1.20E-005	-0-	3.89E-006	1.56E-005	-0-
Diazinon	HPHS	5.80E-003	1.74E-001	-0-	7.50E-003	2.25E-001	-0-
Diazinon	HHW	1.27E-006	3.80E-005	-0-	1.64E-006	4.92E-005	-0-
Dimethoate	HHW	3.49E-004	1.83E-003	-0-	4.52E-004	2.36E-003	-0-
Cyclohexanone		2.56E-008	1.34E-007	-0-	3.31E-008	1.73E-007	-0-
Petroleum distillate		3.10E-009	1.62E-008	-0-	4.00E-009	2.10E-008	-0-
Additive Risk		3.49E-004	1.83E-003	-0-	4.52E-004	2.36E-003	-0-
Dimethoate	Backpack	3.49E-005	1.83E-004	-0-	4.52E-005	2.36E-004	-0-
Cyclohexanone		2.56E-009	1.34E-008	-0-	3.31E-009	1.73E-008	-0-
Petroleum distillate		3.10E-010	1.62E-009	-0-	4.00E-010	2.10E-009	-0-
Additive Risk		3.49E-005	1.83E-004	-0-	4.52E-005	2.36E-004	-0-
Esfenvalerate	HPHS	2.90E-006	1.19E-005	-0-	3.75E-006	1.54E-005	-0-
Ethylbenzene		1.56E-007	6.44E-007	-0-	2.02E-007	8.33E-007	-0-
Xylene		2.69E-008	1.11E-007	-0-	3.48E-008	1.43E-007	-0-
Additive Risk		3.08E-006	1.27E-005	-0-	3.99E-006	1.64E-005	-0-
Esfenvalerate	HHW	8.87E-009	3.65E-008	-0-	1.15E-008	4.72E-008	-0-
Ethylbenzene		4.78E-010	1.97E-009	-0-	6.19E-010	2.55E-009	-0-
Xylene		8.23E-011	3.39E-010	-0-	1.06E-010	4.38E-010	-0-
Additive Risk		9.43E-009	3.88E-008	-0-	1.22E-008	5.02E-008	-0-
Esfenvalerate	Backpack	8.87E-010	3.65E-009	-0-	1.15E-009	4.72E-009	-0-
Ethylbenzene		4.78E-011	1.97E-010	-0-	6.19E-011	2.55E-010	-0-
Xylene		8.23E-012	3.39E-011	-0-	1.06E-011	4.38E-011	-0-
Additive Risk		9.43E-010	3.88E-009	-0-	1.22E-009	5.02E-009	-0-
Horticultural Oil	HPHS	8.43E-006	2.81E-005	-0-	1.09E-005	3.63E-005	-0-
Permethrin	HPHS	1.31E-005	5.26E-005	7.95E-012	1.70E-005	6.80E-005	1.03E-011
Ethylbenzene		6.85E-007	2.74E-006	-0-	8.86E-007	3.54E-006	-0-
Light aromatic solvent naphtha		1.62E-004	6.49E-004	-0-	2.10E-004	8.40E-004	-0-
Xylene		2.01E-007	8.02E-007	-0-	2.60E-007	1.04E-006	-0-
Additive Risk		1.76E-004	7.05E-004	7.95E-012	2.28E-004	9.12E-004	1.03E-011
Permethrin	HHW	4.13E-009	2.49E-008	2.72E-015	5.35E-009	3.22E-008	3.52E-015
Ethylbenzene		2.15E-010	1.30E-009	-0-	2.79E-010	1.68E-009	-0-
Light aromatic solvent naphtha		5.10E-008	3.07E-007	-0-	6.60E-008	3.98E-007	-0-
Xylene		6.31E-011	3.80E-010	-0-	8.16E-011	4.92E-010	-0-
Additive Risk		5.54E-008	3.34E-007	2.72E-015	7.17E-008	4.32E-007	3.52E-015
Permethrin	Backpack	4.13E-010	2.49E-009	2.72E-016	5.35E-010	3.22E-009	3.52E-016
Ethylbenzene		2.15E-011	1.30E-010	-0-	2.79E-011	1.68E-010	-0-
Light aromatic solvent naphtha		5.10E-009	3.07E-008	-0-	6.60E-009	3.98E-008	-0-
Xylene		6.31E-012	3.80E-011	-0-	8.16E-012	4.92E-011	-0-
Additive Risk		5.54E-009	3.34E-008	2.72E-016	7.17E-009	4.32E-008	3.52E-016
Propargite	HPHS	2.23E-004	7.64E-004	6.94E-010	2.88E-004	9.89E-004	8.98E-010
Propargite	HHW	4.29E-007	1.47E-006	1.33E-012	5.54E-007	1.90E-006	1.73E-012
Propargite	Backpack	4.29E-008	1.47E-007	1.33E-013	5.54E-008	1.90E-007	1.73E-013
Chlorothalonil	HPHS	1.84E-005	7.38E-005	1.64E-012	2.39E-005	9.55E-005	2.13E-012
Chlorothalonil	HHW	3.55E-008	1.42E-007	3.16E-015	4.59E-008	1.84E-007	4.09E-015
Dicamba	HHW	3.75E-003	1.50E-002	-0-	4.85E-003	1.94E-002	-0-
Dicamba	Backpack	3.75E-003	1.50E-002	-0-	4.85E-003	1.94E-002	-0-
Dicamba	Boom	3.75E-003	1.50E-002	-0-	4.85E-003	1.94E-002	-0-
Dicamba	Wick	3.75E-003	7.51E-003	-0-	4.85E-003	9.71E-003	-0-
Glyphosate	HHW	7.99E-006	2.00E-005	-0-	1.03E-005	2.59E-005	-0-
Glyphosate	Backpack	7.99E-006	2.00E-005	-0-	1.03E-005	2.59E-005	-0-
Glyphosate	Boom	2.00E-006	1.60E-005	-0-	2.59E-006	2.07E-005	-0-
Glyphosate	Wick	1.34E-006	2.68E-006	-0-	1.73E-006	3.46E-006	-0-
Hexazinone	HHW	6.08E-004	4.86E-003	-0-	7.87E-004	6.29E-003	-0-
Hexazinone	Backpack	6.08E-004	4.86E-003	-0-	7.87E-004	6.29E-003	-0-
Picloram	HHW	4.22E-006	3.38E-005	-0-	5.46E-006	4.37E-005	-0-
Hexachlorobenzene		-0-	-0-	7.69E-012	-0-	-0-	9.95E-012
Picloram	Backpack	4.22E-006	3.38E-005	-0-	5.46E-006	4.37E-005	-0-
Hexachlorobenzene		-0-	-0-	7.69E-012	-0-	-0-	9.95E-012
Triclopyr butoxyethyl ester	HHW	4.18E-005	4.46E-004	-0-	5.41E-005	5.77E-004	-0-
Triclopyr butoxyethyl ester	Backpack	4.18E-005	4.46E-004	-0-	5.41E-005	5.77E-004	-0-

*HI = Hazard Index

Table 6-12. Petting Dog with Residues

		Adult			Child		
Chemical	**Application Method**	**Typ HI***	**Max HI**	**Cancer Risk**	**Typ HI**	**Max HI**	**Cancer Risk**
Acephate	implant	-0-	-0-	-0-	-0-	-0-	-0-
Chlorpyrifos	HPHS	1.70E-003	5.11E-003	-0-	5.41E-003	1.62E-002	-0-
Chlorpyrifos	HHW	1.12E-006	3.35E-006	-0-	3.54E-006	1.06E-005	-0-
Diazinon	HPHS	2.15E-003	4.84E-002	-0-	6.84E-003	1.54E-001	-0-
Diazinon	HHW	4.70E-007	1.06E-005	-0-	1.49E-006	3.36E-005	-0-
Dimethoate	HHW	1.30E-004	5.09E-004	-0-	4.12E-004	1.62E-003	-0-
Cyclohexanone		9.49E-009	3.72E-008	-0-	3.01E-008	1.18E-007	-0-
Petroleum distillate		1.15E-009	4.51E-009	-0-	3.65E-009	1.43E-008	-0-
Additive Risk		1.30E-004	5.09E-004	-0-	4.12E-004	1.62E-003	-0-
Dimethoate	Backpack	1.30E-005	5.09E-005	-0-	4.12E-005	1.62E-004	-0-
Cyclohexanone		9.49E-010	3.72E-009	-0-	3.01E-009	1.18E-008	-0-
Petroleum distillate		1.15E-010	4.51E-010	-0-	3.65E-010	1.43E-009	-0-
Additive Risk		1.30E-005	5.09E-005	-0-	4.12E-005	1.62E-004	-0-
Esfenvalerate	HPHS	1.08E-006	3.32E-006	-0-	3.42E-006	1.06E-005	-0-
Ethylbenzene		5.81E-008	1.79E-007	-0-	1.84E-007	5.69E-007	-0-
Xylene		9.99E-009	3.08E-008	-0-	3.17E-008	9.79E-008	-0-
Additive Risk		1.14E-006	3.53E-006	-0-	3.64E-006	1.12E-005	-0-
Esfenvalerate	HHW	3.29E-009	1.02E-008	-0-	1.05E-008	3.23E-008	-0-
Ethylbenzene		1.78E-010	5.48E-010	-0-	5.64E-010	1.74E-009	-0-
Xylene		3.05E-011	9.42E-011	-0-	9.70E-011	2.99E-010	-0-
Additive Risk		3.50E-009	1.08E-008	-0-	1.11E-008	3.43E-008	-0-
Esfenvalerate	Backpack	3.29E-010	1.02E-009	-0-	1.05E-009	3.23E-009	-0-
Ethylbenzene		1.78E-011	5.48E-011	-0-	5.64E-011	1.74E-010	-0-
Xylene		3.05E-012	9.42E-012	-0-	9.70E-012	2.99E-011	-0-
Additive Risk		3.50E-010	1.08E-009	-0-	1.11E-009	3.43E-009	-0-
Horticultural Oil	HPHS	3.13E-006	7.81E-006	-0-	9.94E-006	2.48E-005	-0-
Permethrin	HPHS	4.88E-006	1.46E-005	2.82E-012	1.55E-005	4.65E-005	8.97E-012
Ethylbenzene		2.54E-007	7.63E-007	-0-	8.08E-007	2.42E-006	-0-
Light aromatic solvent naphtha		6.02E-005	1.81E-004	-0-	1.91E-004	5.74E-004	-0-
Xylene		7.44E-008	2.23E-007	-0-	2.37E-007	7.10E-007	-0-
Additive Risk		6.54E-005	1.96E-004	2.82E-012	2.08E-004	6.23E-004	8.97E-012
Permethrin	HHW	1.53E-009	6.94E-009	9.49E-016	4.87E-009	2.20E-008	3.02E-015
Ethylbenzene		7.99E-011	3.61E-010	-0-	2.54E-010	1.15E-009	-0-
Light aromatic solvent naphtha		1.89E-008	8.56E-008	-0-	6.01E-008	2.72E-007	-0-
Xylene		2.34E-011	1.06E-010	-0-	7.44E-011	3.36E-010	-0-
Additive Risk		2.06E-008	9.30E-008	9.49E-016	6.53E-008	2.95E-007	3.02E-015
Permethrin	Backpack	1.53E-010	6.94E-010	9.49E-017	4.87E-010	2.20E-009	3.02E-016
Ethylbenzene		7.99E-012	3.61E-011	-0-	2.54E-011	1.15E-010	-0-
Light aromatic solvent naphtha		1.89E-009	8.56E-009	-0-	6.01E-009	2.72E-008	-0-
Xylene		2.34E-012	1.06E-011	-0-	7.44E-012	3.36E-011	-0-
Additive Risk		2.06E-009	9.30E-009	9.49E-017	6.53E-009	2.95E-008	3.02E-016
Propargite	HPHS	8.27E-005	2.13E-004	2.48E-010	2.63E-004	6.76E-004	7.87E-010
Propargite	HHW	1.59E-007	4.09E-007	4.76E-013	5.05E-007	1.30E-006	1.51E-012
Propargite	Backpack	1.59E-008	4.09E-008	4.76E-014	5.05E-008	1.30E-007	1.51E-013
Chlorothalonil	HPHS	6.85E-006	2.05E-005	5.83E-013	2.17E-005	6.52E-005	1.85E-012
Chlorothalonil	HHW	1.32E-008	3.95E-008	1.12E-015	4.18E-008	1.25E-007	3.56E-015
Dicamba	HHW	1.39E-003	4.18E-003	-0-	4.42E-003	1.33E-002	-0-
Dicamba	Backpack	1.39E-003	4.18E-003	-0-	4.42E-003	1.33E-002	-0-
Dicamba	Boom	1.39E-003	4.18E-003	-0-	4.42E-003	1.33E-002	-0-
Dicamba	Wick	1.39E-003	2.09E-003	-0-	4.42E-003	6.64E-003	-0-
Glyphosate	HHW	2.97E-006	5.56E-006	-0-	9.42E-006	1.77E-005	-0-
Glyphosate	Backpack	2.97E-006	5.56E-006	-0-	9.42E-006	1.77E-005	-0-
Glyphosate	Boom	7.42E-007	4.45E-006	-0-	2.36E-006	1.41E-005	-0-
Glyphosate	Wick	4.97E-007	7.45E-007	-0-	1.58E-006	2.37E-006	-0-
Hexazinone	HHW	2.26E-004	1.35E-003	-0-	7.17E-004	4.30E-003	-0-
Hexazinone	Backpack	2.26E-004	1.35E-003	-0-	7.17E-004	4.30E-003	-0-
Picloram	HHW	1.57E-006	9.40E-006	-0-	4.98E-006	2.99E-005	-0-
Hexachlorobenzene		-0-	-0-	2.64E-012	-0-	-0-	8.40E-012
Picloram	Backpack	1.57E-006	9.40E-006	-0-	4.98E-006	2.99E-005	-0-
Hexachlorobenzene		-0-	-0-	2.64E-012	-0-	-0-	8.40E-012
Triclopyr butoxyethyl ester	HHW	1.55E-005	1.24E-004	-0-	4.93E-005	3.94E-004	-0-
Triclopyr butoxyethyl ester	Backpack	1.55E-005	1.24E-004	-0-	4.93E-005	3.94E-004	-0-

*HI = Hazard Index

Table 6-13. High-Pressure Hydraulic Sprayer Mixer/Loader/Applicator

Chemical	Typ HI*	Max HI	Cancer Risk
Chlorpyrifos	2.31E-001	7.40E-001	-0-
Diazinon	4.87E-001	**3.89E+000**	-0-
Esfenvalerate	2.43E-004	7.79E-004	-0-
Ethylbenzene	1.31E-005	4.20E-005	-0-
Xylene	2.26E-006	7.23E-006	-0-
Additive Risk	2.59E-004	8.28E-004	-0-
Horticultural Oil	7.07E-004	1.88E-003	-0-
Permethrin	1.10E-003	3.53E-003	8.52E-009
Ethylbenzene	5.75E-005	1.84E-004	-0-
Light aromatic solvent naphtha	1.36E-002	4.36E-002	-0-
Xylene	1.68E-005	5.39E-005	-0-
Additive Risk	1.48E-002	4.73E-002	8.52E-009
Propargite	1.12E-002	3.23E-002	7.39E-008
Chlorothalonil	1.55E-003	5.19E-003	2.83E-010

*HI = Hazard Index

Table 6-14. Hydraulic Sprayer with Hand-Held Wand Mixer/Loader/Applicator

Chemical	Typ HI*	Max HI	Cancer Risk
Chlorpyrifos	9.26E-003	1.85E-002	-0-
Diazinon	1.61E-001	**1.61E+000**	-0-
Dimethoate	**6.13E+000**	**1.61E+001**	-0-
Cyclohexanone	4.49E-004	1.17E-003	-0-
Petroleum distillate	5.44E-005	1.42E-004	-0-
Additive Risk	**6.13E+000**	**1.61E+001**	-0-
Esfenvalerate	1.57E-004	3.22E-004	-0-
Ethylbenzene	8.45E-006	1.74E-005	-0-
Xylene	1.41E-005	2.89E-005	-0-
Additive Risk	1.79E-004	3.69E-004	-0-
Permethrin	1.45E-004	4.38E-004	8.17E-011
Ethylbenzene	7.57E-006	2.28E-005	-0-
Light aromatic solvent naphtha	1.79E-003	5.40E-003	-0-
Xylene	2.22E-006	6.68E-006	-0-
Additive Risk	1.95E-003	5.87E-003	8.17E-011
Propargite	4.51E-004	8.02E-004	1.41E-009
Chlorothalonil	1.03E-003	2.13E-003	8.88E-011
Dicamba	2.39E-003	4.78E-003	-0-
Glyphosate	5.09E-006	6.36E-006	-0-
Hexazinone	3.87E-004	1.55E-003	-0-
Picloram	2.69E-006	1.07E-005	-0-
Hexachlorobenzene	-0-	-0-	1.03E-011
Triclopyr butoxyethyl ester	2.66E-005	1.42E-004	-0-

*HI = Hazard Index

Table 6-15. Tractor-Pulled Boom Mixer/Loader/Applicator

Chemical	Typ HI*	Max HI	Cancer Risk
Dicamba	1.37E-002	2.28E-002	-0-
Glyphosate	7.29E-006	4.86E-005	-0-

*HI = Hazard Index

Table 6-16. Backpack Sprayer

Chemical	Typ HI*	Max HI	Cancer Risk
Dimethoate	**4.22E+003**	**1.10E+004**	-0-
Cyclohexanone	3.09E-001	8.08E-001	-0-
Petroleum distillate	3.74E-002	9.79E-002	-0-
Additive Risk	**4.22E+003**	**1.10E+004**	-0-
Esfenvalerate	1.08E-001	2.22E-001	-0-
Ethylbenzene	5.81E-003	1.20E-002	-0-
Xylene	1.00E-003	2.06E-003	-0-
Additive Risk	1.15E-001	2.36E-001	-0-
Permethrin	9.99E-002	3.01E-001	5.62E-008
Ethylbenzene	5.21E-003	1.57E-002	-0-
Light aromatic solvent nap	**1.23E+000**	**3.72E+000**	-0-
Xylene	1.52E-003	4.59E-003	-0-
Additive Risk	**1.34E+000**	**4.04E+000**	5.62E-008
Propargite	**8.56E+000**	**1.52E+001**	**2.54E-005**
Dicamba	**1.64E+000**	**3.29E+000**	-0-
Glyphosate	3.50E-003	4.37E-003	-0-
Hexazinone	2.66E-001	**1.06E+000**	-0-
Picloram	1.85E-003	7.39E-003	-0-
Hexachlorobenzene	-0-	-0-	6.92E-009
Triclopyr butoxyethyl ester	1.83E-002	9.76E-002	-0-

*HI = Hazard Index

Table 6-17. Hand-Held Wick Mixer/Loader/Applicator

Chemical	Typ HI*	Max HI	Cancer Risk
Dicamba	7.36E-001	7.36E-001	-0-
Glyphosate	2.62E-004	2.62E-004	-0-

*HI = Hazard Index

Table 6-18. Irrigation System Maintenance Worker

Chemical	Application Method	Typ HI*	Max HI	Cancer Risk
Chlorpyrifos	HPHS	3.84E-001	**2.92E+000**	-0-
Chlorpyrifos	HHW	1.50E-004	1.14E-003	-0-
Diazinon	HPHS	3.88E-001	**2.73E+001**	-0-
Diazinon	HHW	5.07E-005	3.56E-003	-0-
Dimethoate	HHW	9.08E-003	1.24E-001	-0-
Cyclohexanone		3.67E-007	7.68E-006	-0-
Petroleum distillate		2.74E-008	8.10E-007	-0-
Additive Risk		9.08E-003	1.24E-001	-0-
Dimethoate	Backpack	7.26E-004	9.95E-003	-0-
Cyclohexanone		2.94E-008	6.15E-007	-0-
Petroleum distillate		2.19E-009	6.48E-008	-0-
Additive Risk		7.26E-004	9.95E-003	-0-
Esfenvalerate	HPHS	3.43E-004	1.95E-003	-0-
Ethylbenzene		2.28E-005	1.07E-004	-0-
Xylene		3.52E-008	1.31E-005	-0-
Additive Risk		3.66E-004	2.07E-003	-0-
Esfenvalerate	HHW	6.27E-007	3.56E-006	-0-
Ethylbenzene		4.16E-008	1.95E-007	-0-
Xylene		6.44E-011	2.39E-008	-0-
Additive Risk		6.69E-007	3.78E-006	-0-
Esfenvalerate	Backpack	5.02E-008	2.85E-007	-0-
Ethylbenzene		3.33E-009	1.56E-008	-0-
Xylene		5.15E-012	1.92E-009	-0-
Additive Risk		5.35E-008	3.02E-007	-0-
Horticultural Oil	HPHS	1.25E-004	3.95E-003	-0-
Permethrin	HPHS	1.35E-003	8.50E-003	7.00E-010
Ethylbenzene		9.97E-005	4.54E-004	-0-
Light aromatic solvent naphtha		2.22E-002	1.07E-001	-0-
Xylene		2.62E-007	9.49E-005	-0-
Additive Risk		2.36E-002	1.16E-001	7.00E-010
Permethrin	HHW	2.55E-007	2.41E-006	1.48E-013
Ethylbenzene		1.88E-008	1.29E-007	-0-
Light aromatic solvent naphtha		4.17E-006	3.03E-005	-0-
Xylene		4.93E-011	2.69E-008	-0-
Additive Risk		4.44E-006	3.29E-005	1.48E-013
Permethrin	Backpack	2.04E-008	1.93E-007	1.19E-014
Ethylbenzene		1.50E-009	1.03E-008	-0-
Light aromatic solvent naphtha		3.33E-007	2.42E-006	-0-
Xylene		3.95E-012	2.15E-009	-0-
Additive Risk		3.55E-007	2.63E-006	1.19E-014
Propargite	HPHS	2.57E-002	8.80E-002	5.92E-008
Propargite	HHW	2.95E-005	1.01E-004	6.80E-011
Propargite	Backpack	2.36E-006	8.09E-006	5.44E-012
Chlorothalonil	HPHS	2.46E-003	1.16E-002	1.71E-010
Chlorothalonil	HHW	2.82E-006	1.33E-005	1.97E-013
Dicamba	HHW	2.19E-003	2.78E-002	-0-
Dicamba	Backpack	2.19E-003	2.78E-002	-0-
Glyphosate	HHW	4.36E-006	2.39E-005	-0-
Glyphosate	Backpack	4.36E-006	2.39E-005	-0-
Hexazinone	HHW	5.17E-004	1.90E-002	-0-
Hexazinone	Backpack	5.17E-004	1.90E-002	-0-
Picloram	HHW	2.30E-006	1.29E-004	-0-
Hexachlorobenzene		-0-	-0-	1.07E-011
Picloram	Backpack	2.30E-006	1.29E-004	-0-
Hexachlorobenzene		-0-	-0-	1.07E-011
Triclopyr butoxyethyl ester	HHW	3.02E-005	2.32E-003	-0-
Triclopyr butoxyethyl ester	Backpack	3.02E-005	2.32E-003	-0-

*HI = Hazard Index

Table 6-19. Groundwater Ingestion after Spill of Concentrate at Mixing Area

	Adult		Child	
Chemical	**HI***	**Cancer Risk**	**HI**	**Cancer Risk**
Acephate-Acecap	4.05E-006	5.15E-015	6.31E-006	8.02E-015
Chlorpyrifos	3.26E-004	-0-	5.08E-004	-0-
Diazinon	2.80E-003	-0-	4.35E-003	-0-
Dimethoate	4.46E-003	-0-	6.94E-003	-0-
Cyclohexanone	3.60E-007	-0-	5.61E-007	-0-
Petroleum distillate	4.35E-007	-0-	6.78E-007	-0-
Additive Risk	4.46E-003	-0-	6.94E-003	-0-
Esfenvalerate	3.69E-006	-0-	5.75E-006	-0-
Ethylbenzene	8.77E-008	-0-	1.37E-007	-0-
Xylene	1.31E-008	-0-	2.05E-008	-0-
Additive Risk	3.79E-006	-0-	5.90E-006	-0-
Horticultural Oil	4.06E-006	-0-	6.32E-006	-0-
Permethrin	7.15E-006	2.09E-013	1.11E-005	3.25E-013
Ethylbenzene	1.86E-007	-0-	2.90E-007	-0-
Light aromatic solvent naphtha	1.50E-005	-0-	2.34E-005	-0-
Xylene	4.75E-008	-0-	7.40E-008	-0-
Additive Risk	2.24E-005	2.09E-013	3.49E-005	3.25E-013
Propargite	2.68E-006	7.88E-013	4.17E-006	1.23E-012
Chlorothalonil	1.56E-004	6.53E-013	2.42E-004	1.02E-012
Dicamba	9.95E-006	-0-	1.55E-005	-0-
Glyphosate	1.11E-006	-0-	1.74E-006	-0-
Hexazinone	2.01E-005	-0-	3.13E-005	-0-
Picloram	1.11E-006	-0-	1.74E-006	-0-
Hexachlorobenzene	-0-	1.38E-015	-0-	2.16E-015
Triclopyr butoxyethyl ester	4.48E-006	-0-	6.97E-006	-0-
Fertilizer (N as nitrate)	4.04E-005	-0-	6.30E-005	-0-

*HI = Hazard Index

Table 6-20. Fish and Surface Water Ingestion after Spill of Concentrate at Mixing Area

	Adult		Child	
Chemical	**HI***	**Cancer Risk**	**HI**	**Cancer Risk**
Acephate-Acecap	9.71E-005	1.23E-013	1.55E-004	1.97E-013
Chlorpyrifos	**1.81E+000**	-0-	**5.72E+000**	-0-
Diazinon	**3.13E+000**	-0-	**9.85E+000**	-0-
Dimethoate	1.26E-001	-0-	2.29E-001	-0-
Cyclohexanone	1.10E-005	-0-	2.13E-005	-0-
Petroleum distillate	5.08E-005	-0-	1.45E-004	-0-
Additive Risk	1.26E-001	-0-	2.30E-001	-0-
Esfenvalerate	1.05E-002	-0-	3.33E-002	-0-
Ethylbenzene	4.70E-006	-0-	1.16E-005	-0-
Xylene	7.08E-007	-0-	1.75E-006	-0-
Additive Risk	1.05E-002	-0-	3.33E-002	-0-
Horticultural Oil	4.67E-004	-0-	1.33E-003	-0-
Permethrin	7.07E-003	2.07E-010	2.22E-002	6.48E-010
Ethylbenzene	9.98E-006	-0-	2.46E-005	-0-
Light aromatic solvent naphtha	3.09E-002	-0-	9.76E-002	-0-
Xylene	2.57E-006	-0-	6.33E-006	-0-
Additive Risk	3.80E-002	2.07E-010	1.20E-001	6.48E-010
Propargite	4.24E-003	1.25E-009	1.34E-002	3.93E-009
Chlorothalonil	8.59E-002	3.60E-010	2.67E-001	1.12E-009
Dicamba	7.93E-004	-0-	2.14E-003	-0-
Glyphosate	2.75E-005	-0-	4.46E-005	-0-
Hexazinone	5.42E-004	-0-	9.72E-004	-0-
Picloram	2.75E-005	-0-	4.48E-005	-0-
Hexachlorobenzene	-0-	2.17E-011	-0-	6.88E-011
Triclopyr butoxyethyl ester	1.16E-004	-0-	1.96E-004	-0-
Fertilizer (N as nitrate)	9.58E-004	-0-	1.49E-003	-0-

*HI = Hazard Index

Table 6-21. Fish and Water Ingestion after Spill of Tank Mix into Bridge Point Ditch

		Adult		Child	
Chemical	**Application Method**	**HI***	**Cancer Risk**	**HI**	**Cancer Risk**
Chlorpyrifos	HPHS	**2.46E+002**	-0-	**7.80E+002**	-0-
Chlorpyrifos	HHW	**1.97E+001**	-0-	**6.24E+001**	-0-
Diazinon	HPHS	**6.96E+001**	-0-	**2.19E+002**	-0-
Diazinon	HHW	**5.57E+000**	-0-	**1.75E+001**	-0-
Dimethoate	HHW	3.12E-001	-0-	5.69E-001	-0-
Cyclohexanone		2.75E-005	-0-	5.33E-005	-0-
Petroleum distillate		1.14E-004	-0-	3.25E-004	-0-
Additive Risk		3.12E-001	-0-	5.69E-001	-0-
Dimethoate	Backpack	7.80E-002	-0-	1.42E-001	-0-
Cyclohexanone		6.87E-006	-0-	1.33E-005	-0-
Petroleum distillate		2.85E-005	-0-	8.12E-005	-0-
Additive Risk		7.80E-002	-0-	1.42E-001	-0-
Esfenvalerate	HPHS	5.83E-002	-0-	1.85E-001	-0-
Ethylbenzene		2.59E-005	-0-	6.39E-005	-0-
Xylene		3.84E-006	-0-	9.47E-006	-0-
Additive Risk		5.84E-002	-0-	1.85E-001	-0-
Esfenvalerate	HHW	4.67E-003	-0-	1.48E-002	-0-
Ethylbenzene		2.07E-006	-0-	5.11E-006	-0-
Xylene		3.07E-007	-0-	7.58E-007	-0-
Additive Risk		4.67E-003	-0-	1.48E-002	-0-
Esfenvalerate	Backpack	1.17E-003	-0-	3.69E-003	-0-
Ethylbenzene		5.18E-007	-0-	1.28E-006	-0-
Xylene		7.68E-008	-0-	1.89E-007	-0-
Additive Risk		1.17E-003	-0-	3.69E-003	-0-
Horticultural Oil	HPHS	8.92E-004	-0-	2.54E-003	-0-
Permethrin	HPHS	3.18E-002	9.29E-010	9.98E-002	2.92E-009
Ethylbenzene		4.54E-005	-0-	1.12E-004	-0-
Light aromatic solvent naphtha		1.40E-001	-0-	4.42E-001	-0-
Xylene		1.14E-005	-0-	2.82E-005	-0-
Additive Risk		1.72E-001	9.29E-010	5.42E-001	2.92E-009
Permethrin	HHW	2.54E-003	7.44E-011	7.98E-003	2.33E-010
Ethylbenzene		3.63E-006	-0-	8.95E-006	-0-
Light aromatic solvent naphtha		1.12E-002	-0-	3.54E-002	-0-
Xylene		9.15E-007	-0-	2.26E-006	-0-
Additive Risk		1.37E-002	7.44E-011	4.34E-002	2.33E-010
Permethrin	Backpack	6.36E-004	1.86E-011	2.00E-003	5.83E-011
Ethylbenzene		9.07E-007	-0-	2.24E-006	-0-
Light aromatic solvent naphtha		2.80E-003	-0-	8.84E-003	-0-
Xylene		2.29E-007	-0-	5.64E-007	-0-
Additive Risk		3.44E-003	1.86E-011	1.08E-002	5.83E-011
Propargite	HPHS	7.77E-001	2.28E-007	**2.45E+000**	7.20E-007
Propargite	HHW	6.22E-002	1.83E-008	1.96E-001	5.76E-008
Propargite	Backpack	1.55E-002	4.57E-009	4.90E-002	1.44E-008
Chlorothalonil	HPHS	**1.29E+000**	5.41E-009	**4.00E+000**	1.68E-008
Chlorothalonil	HHW	1.03E-001	4.32E-010	3.20E-001	1.34E-009
Dicamba	HHW	2.38E-002	-0-	6.42E-002	-0-
Dicamba	Backpack	5.94E-003	-0-	1.60E-002	-0-
Dicamba	Boom	1.19E-001	-0-	3.21E-001	-0-
Dicamba	Wick	5.94E-003	-0-	1.60E-002	-0-
Glyphosate	HHW	2.97E-004	-0-	4.82E-004	-0-
Glyphosate	Backpack	7.41E-005	-0-	1.21E-004	-0-
Glyphosate	Boom	1.19E-003	-0-	1.93E-003	-0-
Glyphosate	Wick	1.98E-005	-0-	3.21E-005	-0-
Hexazinone	HHW	1.03E-002	-0-	1.85E-002	-0-
Hexazinone	Backpack	2.59E-003	-0-	4.64E-003	-0-
Picloram	HHW	8.03E-004	-0-	1.31E-003	-0-
Hexachlorobenzene		-0-	3.73E-011	-0-	1.18E-010
Picloram	Backpack	2.01E-004	-0-	3.27E-004	-0-
Hexachlorobenzene		-0-	9.32E-012	-0-	2.96E-011
Triclopyr butoxyethyl ester	HHW	2.68E-003	-0-	4.53E-003	-0-
Triclopyr butoxyethyl ester	Backpack	6.69E-004	-0-	1.13E-003	-0-

*HI = Hazard Index

Table 6-22. Fish and Water Ingestion after Spill of Tank Mix into Williams Creek

		Adult		Child	
Chemical	**Application Method**	**HI***	**Cancer Risk**	**HI**	**Cancer Risk**
Chlorpyrifos	HPHS	**2.63E+002**	-0-	**8.33E+002**	-0-
Chlorpyrifos	HHW	**2.10E+001**	-0-	**6.66E+001**	-0-
Diazinon	HPHS	**6.96E+001**	-0-	**2.19E+002**	-0-
Diazinon	HHW	**5.57E+000**	-0-	**1.75E+001**	-0-
Dimethoate	HHW	3.17E-001	-0-	5.78E-001	-0-
Cyclohexanone		2.80E-005	-0-	5.44E-005	-0-
Petroleum distillate		1.24E-004	-0-	3.54E-004	-0-
Additive Risk		3.17E-001	-0-	5.78E-001	-0-
Dimethoate	Backpack	7.93E-002	-0-	1.44E-001	-0-
Cyclohexanone		7.01E-006	-0-	1.36E-005	-0-
Petroleum distillate		3.11E-005	-0-	8.86E-005	-0-
Additive Risk		7.93E-002	-0-	1.45E-001	-0-
Esfenvalerate	HPHS	5.83E-002	-0-	1.85E-001	-0-
Ethylbenzene		2.64E-005	-0-	6.51E-005	-0-
Xylene		3.84E-006	-0-	9.47E-006	-0-
Additive Risk		5.84E-002	-0-	1.85E-001	-0-
Esfenvalerate	HHW	4.67E-003	-0-	1.48E-002	-0-
Ethylbenzene		2.11E-006	-0-	5.21E-006	-0-
Xylene		3.07E-007	-0-	7.58E-007	-0-
Additive Risk		4.67E-003	-0-	1.48E-002	-0-
Esfenvalerate	Backpack	1.17E-003	-0-	3.69E-003	-0-
Ethylbenzene		5.28E-007	-0-	1.30E-006	-0-
Xylene		7.68E-008	-0-	1.89E-007	-0-
Additive Risk		1.17E-003	-0-	3.69E-003	-0-
Horticultural Oil	HPHS	9.12E-004	-0-	2.60E-003	-0-
Permethrin	HPHS	3.18E-002	9.29E-010	9.98E-002	2.92E-009
Ethylbenzene		4.62E-005	-0-	1.14E-004	-0-
Light aromatic solvent naphtha		1.45E-001	-0-	4.59E-001	-0-
Xylene		1.14E-005	-0-	2.82E-005	-0-
Additive Risk		1.77E-001	9.29E-010	5.59E-001	2.92E-009
Permethrin	HHW	2.54E-003	7.44E-011	7.98E-003	2.33E-010
Ethylbenzene		3.70E-006	-0-	9.12E-006	-0-
Light aromatic solvent naphtha		1.16E-002	-0-	3.67E-002	-0-
Xylene		9.15E-007	-0-	2.26E-006	-0-
Additive Risk		1.42E-002	7.44E-011	4.47E-002	2.33E-010
Permethrin	Backpack	6.36E-004	1.86E-011	2.00E-003	5.83E-011
Ethylbenzene		9.24E-007	-0-	2.28E-006	-0-
Light aromatic solvent naphtha		2.91E-003	-0-	9.19E-003	-0-
Xylene		2.29E-007	-0-	5.64E-007	-0-
Additive Risk		3.55E-003	1.86E-011	1.12E-002	5.83E-011
Propargite	HPHS	7.77E-001	2.28E-007	**2.45E+000**	7.20E-007
Propargite	HHW	6.22E-002	1.83E-008	1.96E-001	5.76E-008
Propargite	Backpack	1.55E-002	4.57E-009	4.90E-002	1.44E-008
Chlorothalonil	HPHS	**1.29E+000**	5.41E-009	**4.00E+000**	1.68E-008
Chlorothalonil	HHW	1.03E-001	4.32E-010	3.20E-001	1.34E-009
Dicamba	HHW	2.38E-002	-0-	6.42E-002	-0-
Dicamba	Backpack	5.94E-003	-0-	1.60E-002	-0-
Dicamba	Boom	1.19E-001	-0-	3.21E-001	-0-
Dicamba	Wick	5.94E-003	-0-	1.60E-002	-0-
Glyphosate	HHW	3.18E-004	-0-	5.18E-004	-0-
Glyphosate	Backpack	7.96E-005	-0-	1.29E-004	-0-
Glyphosate	Boom	1.27E-003	-0-	2.07E-003	-0-
Glyphosate	Wick	2.12E-005	-0-	3.45E-005	-0-
Hexazinone	HHW	1.03E-002	-0-	1.85E-002	-0-
Hexazinone	Backpack	2.59E-003	-0-	4.64E-003	-0-
Picloram	HHW	8.14E-004	-0-	1.33E-003	-0-
Hexachlorobenzene		-0-	6.24E-011	-0-	1.98E-010
Picloram	Backpack	2.04E-004	-0-	3.31E-004	-0-
Hexachlorobenzene		-0-	1.56E-011	-0-	4.96E-011
Triclopyr-4	HHW	2.74E-003	-0-	4.65E-003	-0-
Triclopyr-4	Backpack	6.86E-004	-0-	1.16E-003	-0-

*HI = Hazard Index

Table 6-23. Spill of Concentrate onto Worker

Chemical	HI*	Cancer Risk
Acephate	**4.39E+001**	1.95E-008
Dimethoate	**1.01E+004**	-0-
Cyclohexanone	7.39E-001	-0-
Petroleum distillate	8.95E-002	-0-
Additive Risk	**1.01E+004**	-0-
Esfenvalerate	**5.68E+000**	-0-
Ethylbenzene	3.06E-001	-0-
Xylene	5.27E-002	-0-
Additive Risk	**6.04E+000**	-0-
Horticultural Oil	8.33E-001	-0-
Permethrin	**1.25E+001**	3.65E-007
Ethylbenzene	6.50E-001	-0-
Light aromatic solvent naphtha	**1.54E+002**	-0-
Xylene	1.90E-001	-0-
Additive Risk	**1.67E+002**	3.65E-007
Chlorothalonil	**4.78E+000**	2.01E-008
Dicamba	**1.02E+002**	-0-
Glyphosate	5.43E-002	-0-
Picloram	2.29E-001	-0-
Hexachlorobenzene	-0-	3.28E-008
Triclopyr butoxyethyl ester	7.57E-001	-0-

*HI = Hazard Index

Table 6-24. Spill of Tank Mix onto Worker

Chemical	Application Method	HI*	Cancer Risk
Chlorpyrifos	HPHS	**1.36E+001**	-0-
Chlorpyrifos	HHW	**1.36E+001**	-0-
Diazinon	HPHS	**1.72E+001**	-0-
Diazinon	HHW	**1.72E+001**	-0-
Dimethoate	HHW	**2.15E+002**	-0-
Cyclohexanone		1.57E-002	-0-
Petroleum distillate		1.90E-003	-0-
Additive Risk		**2.15E+002**	-0-
Dimethoate	Backpack	**2.15E+002**	-0-
Cyclohexanone		1.57E-002	-0-
Petroleum distillate		1.90E-003	-0-
Additive Risk		**2.15E+002**	-0-
Esfenvalerate	HPHS	4.30E-003	-0-
Ethylbenzene		2.32E-004	-0-
Xylene		3.99E-005	-0-
Additive Risk		4.57E-003	-0-
Esfenvalerate	HHW	4.30E-003	-0-
Ethylbenzene		2.32E-004	-0-
Xylene		3.99E-005	-0-
Additive Risk		4.57E-003	-0-
Esfenvalerate	Backpack	4.30E-003	-0-
Ethylbenzene		2.32E-004	-0-
Xylene		3.99E-005	-0-
Additive Risk		4.57E-003	-0-
Horticultural Oil	HPHS	1.15E-003	-0-
Permethrin	HPHS	7.80E-003	2.28E-010
Ethylbenzene		4.06E-004	-0-
Light aromatic solvent naphtha		9.63E-002	-0-
Xylene		1.19E-004	-0-
Additive Risk		1.05E-001	2.28E-010
Permethrin	HHW	7.80E-003	2.28E-010
Ethylbenzene		4.06E-004	-0-
Light aromatic solvent naphtha		9.63E-002	-0-
Xylene		1.19E-004	-0-
Additive Risk		1.05E-001	2.28E-010
Permethrin	Backpack	7.80E-003	2.28E-010
Ethylbenzene		4.06E-004	-0-
Light aromatic solvent naphtha		9.63E-002	-0-
Xylene		1.19E-004	-0-
Additive Risk		1.05E-001	2.28E-010
Propargite	HPHS	4.99E-001	1.47E-007
Propargite	HHW	4.99E-001	1.47E-007
Propargite	Backpack	4.99E-001	1.47E-007
Chlorothalonil	HPHS	4.82E-002	2.02E-010
Chlorothalonil	HHW	4.82E-002	2.02E-010
Dicamba	HHW	**5.10E+000**	-0-
Dicamba	Backpack	**5.10E+000**	-0-
Dicamba	Boom	**5.10E+000**	-0-
Dicamba	Wick	**5.10E+001**	-0-
Glyphosate	HHW	6.79E-003	-0-
Glyphosate	Backpack	6.79E-003	-0-
Glyphosate	Boom	1.09E-003	-0-
Glyphosate	Wick	1.81E-002	-0-
Hexazinone	HHW	6.61E-001	-0-
Hexazinone	Backpack	6.61E-001	-0-
Picloram	HHW	1.15E-002	-0-
Hexachlorobenzene		-0-	1.64E-009
Picloram	Backpack	1.15E-002	-0-
Hexachlorobenzene		-0-	1.64E-009
Triclopyr butoxyethyl ester	HHW	1.51E-001	-0-
Triclopyr butoxyethyl ester	Backpack	1.51E-001	-0-

*HI = Hazard Index

Table 6-25. Spray of Worker with Tank Mix

Chemical	Application Method	HI*	Cancer Risk
Chlorpyrifos	HPHS	6.74E-001	-0-
Chlorpyrifos	HHW	6.74E-001	-0-
Diazinon	HPHS	2.84E-001	-0-
Diazinon	HHW	2.84E-001	-0-
Dimethoate	HHW	**7.83E+001**	-0-
Cyclohexanone		5.73E-003	-0-
Petroleum distillate		6.94E-004	-0-
Additive Risk		**7.83E+001**	-0-
Dimethoate	Backpack	**7.83E+001**	-0-
Cyclohexanone		5.73E-003	-0-
Petroleum distillate		6.94E-004	-0-
Additive Risk		**7.83E+001**	-0-
Esfenvalerate	HPHS	1.99E-003	-0-
Ethylbenzene		1.07E-004	-0-
Xylene		1.84E-005	-0-
Additive Risk		2.11E-003	-0-
Esfenvalerate	HHW	1.99E-003	-0-
Ethylbenzene		1.07E-004	-0-
Xylene		1.84E-005	-0-
Additive Risk		2.11E-003	-0-
Esfenvalerate	Backpack	1.99E-003	-0-
Ethylbenzene		1.07E-004	-0-
Xylene		1.84E-005	-0-
Additive Risk		2.11E-003	-0-
Horticultural Oil	HPHS	5.78E-003	-0-
Permethrin	HPHS	9.27E-003	2.71E-010
Ethylbenzene		4.83E-004	-0-
Light aromatic solvent naphtha		1.14E-001	-0-
Xylene		1.41E-004	-0-
Additive Risk		1.24E-001	2.71E-010
Permethrin	HHW	9.27E-004	2.71E-011
Ethylbenzene		4.83E-005	-0-
Light aromatic solvent naphtha		1.14E-002	-0-
Xylene		1.41E-005	-0-
Additive Risk		1.24E-002	2.71E-011
Permethrin	Backpack	9.27E-004	2.71E-011
Ethylbenzene		4.83E-005	-0-
Light aromatic solvent naphtha		1.14E-002	-0-
Xylene		1.41E-005	-0-
Additive Risk		1.24E-002	2.71E-011
Propargite	HPHS	9.61E-002	2.82E-008
Propargite	HHW	9.61E-002	2.82E-008
Propargite	Backpack	9.61E-002	2.82E-008
Chlorothalonil	HPHS	7.95E-003	3.34E-011
Chlorothalonil	HHW	7.95E-003	3.34E-011
Dicamba	HHW	8.41E-002	-0-
Dicamba	Backpack	8.41E-002	-0-
Dicamba	Boom	8.41E-002	-0-
Glyphosate	HHW	1.79E-004	-0-
Glyphosate	Backpack	1.79E-004	-0-
Glyphosate	Boom	4.48E-005	-0-
Hexazinone	HHW	1.36E-002	-0-
Hexazinone	Backpack	1.36E-002	-0-
Picloram	HHW	9.46E-005	-0-
Hexachlorobenzene		-0-	1.35E-011
Picloram	Backpack	9.46E-005	-0-
Hexachlorobenzene		-0-	1.35E-011
Triclopyr butoxyethyl ester	HHW	9.37E-004	-0-
Triclopyr butoxyethyl ester	Backpack	9.37E-004	-0-

*HI = Hazard Index

Table 6-26 Cumulative Risks to Members of the Public

	Aggregated Risk from All Routes of Exposure			
	Adult		Child	
Chemical	**HI***	**Cancer Risk**	**HI***	**Cancer Risk**
Acephate	-0-	2.58E-018	-0-	4.47E-018
Chlorpyrifos	1.57E-002	-0-	1.97E-002	-0-
Diazinon	1.56E-002	-0-	1.88E-002	-0-
Dimethoate	1.02E-003	-0-	1.66E-003	-0-
Esfenvalerate	1.07E-005	-0-	1.27E-005	-0-
Horticultural Oil	4.59E-004	-0-	7.44E-004	-0-
Permethrin	3.07E-004	2.15E-011	4.76E-004	2.29E-011
Propargite	4.14E-004	1.26E-009	6.45E-004	2.12E-009
Chlorothalonil	3.57E-004	2.71E-011	2.49E-004	1.35E-011
Dicamba	1.55E-002	-0-	2.79E-002	-0-
Glyphosate	1.63E-005	-0-	2.85E-005	-0-
Hexazinone	8.34E-004	-0-	1.50E-003	-0-
Picloram	5.80E-006	-0-	1.04E-005	-0-
Hexachlorobenzene	-0-	2.49E-011	-0-	4.51E-011
Triclopyr butoxyethyl ester	5.75E-005	-0-	1.04E-004	-0-
Fertilizers	-0-	-0-	-0-	-0-
Cumulative Risk	5.03E-002	1.33E-009	7.18E-002	2.20E-009

*HI = Hazard Index

Table 6-27 Cumulative Risks to Members of the Public from Chemicals More Likely to be Used

	Aggregated Risk from All Routes of Exposure			
	Adult		Child	
Chemical	**HI***	**Cancer Risk**	**HI***	**Cancer Risk**
Esfenvalerate	1.07E-005	-0-	1.27E-005	-0-
Horticultural Oil	4.59E-004	-0-	7.44E-004	-0-
Glyphosate	1.63E-005	-0-	2.85E-005	-0-
Triclopyr butoxyethyl ester	5.75E-005	-0-	1.04E-004	-0-
Fertilizers	-0-	-0-	-0-	-0-
Cumulative Risk	5.43E-004	-0-	8.89E-004	-0-

*HI = Hazard Index

Table 6-28. Cumulative Risk to Workers

Chemical	Application Method	HI*	Cancer Risk
Acephate	implant	-0-	1.95E-008
Chlorpyrifos	HPHS	6.15E-001	-0-
Diazinon	HPHS	8.75E-001	-0-
Dimethoate	Backpack	**4.22E+003**	-0-
Cyclohexanone		3.09E-001	-0-
Petroleum distillate		3.74E-002	-0-
Esfenvalerate	Backpack	1.08E-001	-0-
Ethylbenzene		5.81E-003	-0-
Xylene		1.00E-003	-0-
Horticultural Oil	HPHS	8.32E-004	-0-
Permethrin	HPHS	2.46E-003	9.22E-009
Ethylbenzene		1.57E-004	-0-
Light aromatic solvent naphtha		3.58E-002	-0-
Xylene		1.71E-005	-0-
Permethrin	Backpack	9.99E-002	5.62E-008
Ethylbenzene		5.21E-003	-0-
Light aromatic solvent naphtha		**1.23E+000**	-0-
Xylene		1.52E-003	-0-
Propargite	Backpack	**8.56E+000**	**2.54E-005**
Chlorothalonil	HHW	1.03E-003	8.90E-011
Dicamba	Backpack	**1.65E+000**	-0-
Dicamba	Boom	1.37E-002	-0-
Dicamba	Wick	7.36E-001	-0-
Glyphosate	Backpack	3.50E-003	-0-
Glyphosate	Boom	7.29E-006	-0-
Glyphosate	Wick	2.62E-004	-0-
Hexazinone	Backpack	2.67E-001	-0-
Picloram	Backpack	1.85E-003	-0-
Hexachlorobenzene			
Triclopyr butoxyethyl ester	Backpack	1.83E-002	-0-
Cumulative Risk		**4.23E+003**	**2.55E-005**

*HI = Hazard Index

Table 6-29. Cumulative Risk to Workers from Chemicals More Likely to be Used

Chemical	Application Method	HI*	Cancer Risk
Esfenvalerate	Backpack	1.08E-001	-0-
Ethylbenzene		5.81E-003	-0-
Xylene		1.00E-003	-0-
Horticultural Oil	HPHS	8.32E-004	-0-
Glyphosate	Backpack	3.50E-003	-0-
Glyphosate	Boom	7.29E-006	-0-
Glyphosate	Wick	2.62E-004	-0-
Triclopyr butoxyethyl ester	Backpack	1.83E-002	-0-
Cumulative Risk		1.38E-001	-0-

*HI = Hazard Index

Table 6-30. Dermal and Eye Effects

Chemical	Dermal Effect	Eye Effect
Acephate	Non-irritating, non-sensitizing	Minimal irritant
Chlorothalonil	Irritant, potential sensitizer	Severe irritant
Chlorpyrifos	Mild irritant, non-sensitizing	Slight irritant
Diazinon	Slightly irritating, potential sensitizer	Slightly irritating
Dicamba	Mild to moderate irritant, non-sensitizing	Mild to moderate irritant; Banvel® formulation may be corrosive
Dimethoate	Dermatitis following high occupational exposures	Severe irritant in manufacturing workers
Esfenvalerate	Can cause paraesthesia (an abnormal sensation such as burning or prickling); not a sensitizer	Slightly irritating
Glyphosate	Non-irritating, non-sensitizing	Mild irritant
Hexazinone	Mild irritant, non-sensitizing	Severe irritant
Horticultural oil	Slight irritant, possible sensitizer	Mild irritant
Permethrin	Irritating, can cause numbness, burning, and tingling	Irritating
Picloram	Non-irritating; non-sensitizing, but Tordon® 22K formulation may be a sensitizer	Moderate irritant; Tordon® 22K formulation is a severe irritant
Propargite	Corrosive, causes dermal sensitization	Corrosive
Triclopyr butoxyethyl ester	Irritating, dermal sensitizer	Minimal irritant
Cyclohexanone	Irritating, potential sensitizer	Irritating
Ethylbenzene	Irritating	Slight irritant
Light aromatic solvent naphtha	Irritating, dermal sensitizer	Irritating, linked to cataracts
Xylene	Irritating	Irritating
Fertilizer salts	Irritating	Irritating

6.6 References

Clewell, H.J., III, and M.E. Anderson. 1987. Dose, species, route extrapolation using physiologically based pharmacokinetic models. In *Drinking Water and Health*, Vol. 8. National Academy Press. Washington, DC.

EPA. See U.S. Environmental Protection Agency.

HSDB. See Hazardous Substances Databank.

Hazardous Substances Databank. 2000. On-line database. National Library of Medicine. Bethesda, MD.

Roloff, M.V., A.G.E. Wilson, W.E. Ribelin, W.P. Ridley, and F.A. Ruecker, eds. 1987. *Human Risk Assessment—The Role of Animal Selection and Extrapolation.* Taylor & Francis. New York.

U.S. Environmental Protection Agency. 1986a. Guidelines for carcinogen risk assessment. EPA 630/R-00/002. Risk Assessment Forum. Washington, DC.

U.S. Environmental Protection Agency . 1986b. Guidelines for the health risk assessment of chemical mixtures. EPA 630/R-98/002. Risk Assessment Forum. Washington, DC.

7.0 NON-TARGET SPECIES PROBLEM FORMULATION

This section presents the results of the non-target species problem formulation, in which the purpose of the non-target species risk assessment is provided, the problem is defined, and a plan for analyzing and characterizing risk is determined. Section 7.1, integrating available information, identifies and characterizes the stressors, the ecological effects expected or observed, the receptors, and ecosystem potentially affected. Section 7.2 describes the assessment endpoints for the non-target species risk assessment. Section 7.3 presents the conceptual model describing key relationships between the stressors and assessment endpoints. Section 7.4 summarizes the analysis plan that includes the design of the assessment, data needs, measures that will be used to evaluate risk hypotheses, and methods for conducting the analysis phase of the assessment.

7.1 Integration of Available Information

In this non-target species risk assessment, the potential stressors are the pesticides or fertilizers that may be used at Provolt. Detailed information was developed on the exact formulations of the pesticide chemicals, chemical nature of the fertilizer compounds, potential application methods, application rates, timing and frequency of application, and sites that could be candidates for treatment, and is provided in Section 2.0 of this risk assessment. These data provide a thorough description of the potential sources of pesticide or fertilizer release to the environment at the seed orchard.

The ecological effects that may be associated with the chemical pesticides and fertilizers are those associated with direct toxicity to non-target species that encounter the chemical. Permanent or persistent exposures through environmental pathways are not expected, since the half-lives of these chemicals are on the order of one month or less. Control of certain pests and vegetation in and of itself is not expected to affect the area's wildlife, since the seed orchard is a managed area, and has been managed for tree species preservation and seed production for 20 years.

The receptors in this non-target species risk assessment were selected to represent the range of species present at or near Provolt, along with specific evaluation of endangered, threatened, or sensitive species that may inhabit or visit the site. These receptors include mammals, birds, reptiles, amphibians, fish, and aquatic vertebrates for which quantitative risk estimates can be made, based on the program description data in Section 2.0 and the environmental fate and transport predictions described in Section 3.0. In addition, endangered, threatened, and sensitive species were also identified and evaluated for potential risks.

Provolt Seed Orchard comprises 294 acres. An eight-foot-high woven wire big game exclosure fence surrounds the orchard units and serves as a partial barrier for many wildlife species. The seed orchards, fields, and roadsides consist mostly of introduced grasses and weedy species. These areas serve as hiding and nesting cover for birds and mammals. In addition, the orchard units provide good to excellent hunting and foraging areas, which could attract bald eagles or northern spotted owls (both Federally listed threatened species). Perennial pasture grasses provide a thick cover. There are scattered native trees, including Ponderosa pine, incense cedar, Oregon white oak, and Douglas-fir. Blackberry thickets are prominent on the roadsides and along fencelines. Irrigation ditches provide riparian habitat. Gravel bars and terraces along the rivers support weedy species.

One special status plant species is known to occur at Provolt, the California smilax (*Smilax californica*), a Bureau watch species. Scattered individual plants have been identified along Williams Creek and the Applegate River, north of Highway 238.

The Applegate River, a Class I stream, flows along the northern boundary of the seed orchard property. Williams Creek, also a Class I stream, flows near the western side of the property and crosses through the property near the northern boundary, where it flows into the Applegate River. There are three irrigation ditches on the seed orchard property, two of which (Bridge Point and Laurel Hill Ditches) are diverted from the Applegate River; the third ditch (Spencer Ditch) is fed by a small reservoir in the south central part of the orchard. There is also a pond near the orchard office with a control gate to manage outflows as needed. The onsite irrigation ditches may contain aquatic invertebrates and aquatic stages of amphibians, and the ponds may contain warmwater fish such as bluegill and perch. Several special status and threatened aquatic species are known to be present in the Applegate River, and possibly in Williams Creek: coho salmon, a Federally listed threatened species; steelhead, chinook salmon, and cutthroat trout, all Federal candidate species; and Pacific lamprey, a Federal species of concern.

7.2 Assessment Endpoints

Assessment endpoints are selected based on three criteria: ecological relevance, susceptibility to stressors, and relevance to management goals (EPA 1998). For species that are endangered, threatened, or sensitive, the assessment endpoint selected is individual survival, growth, and reproduction. For non-sensitive species present at the seed orchard, the assessment endpoint selected is the survival of populations.

Scenarios describing the potential impacts of pesticide and fertilizer use at the seed orchard on the assessment endpoints are developed in the conceptual model described in the next section.

7.3 Conceptual Model

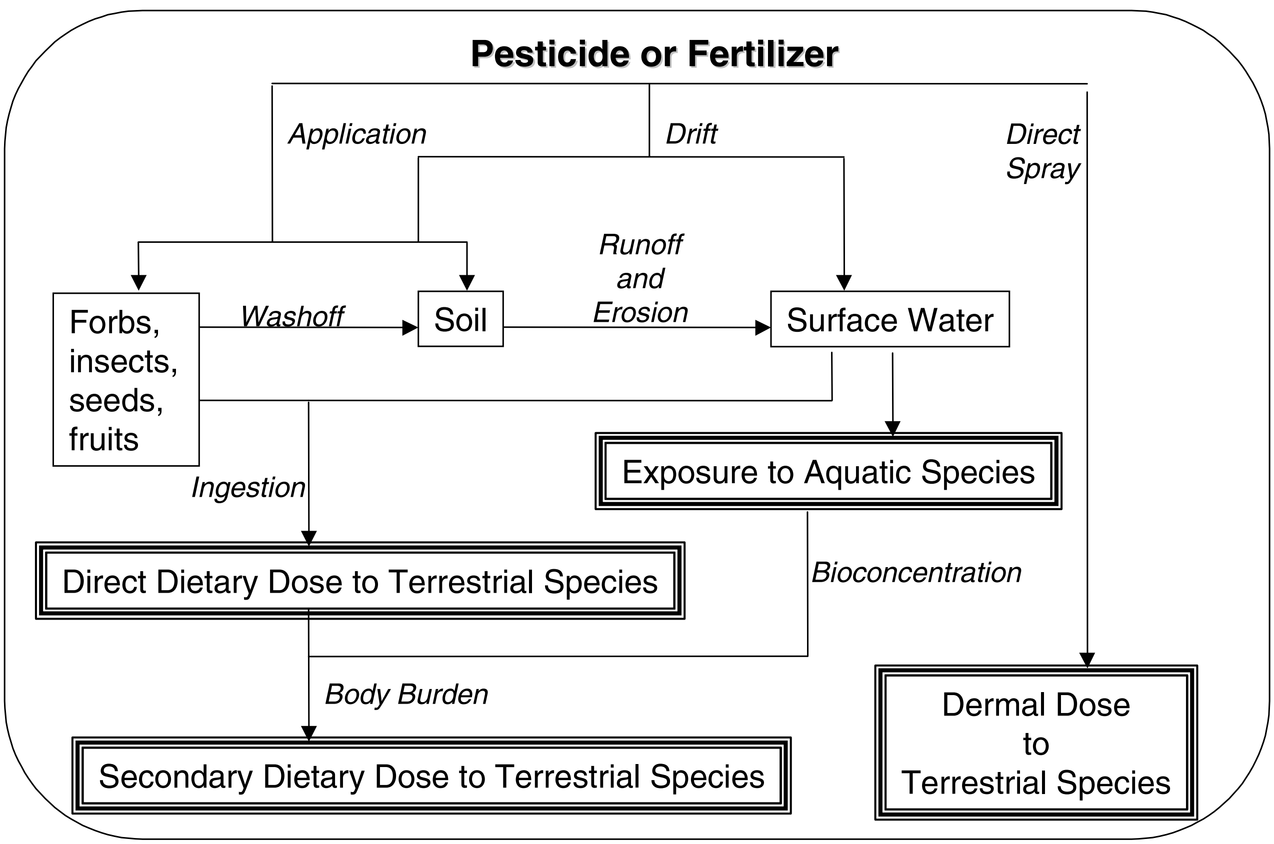
A conceptual model consists of a risk hypothesis that describe relationships between the stressor, exposure, and assessment endpoint response; and a diagram illustrating these relationships. For the proposed chemical use at Provolt, the risk hypothesis is as follows.

Risk Hypothesis

Pesticide chemicals have demonstrated toxicity to wildlife species, based on laboratory and field tests that have characterized exposure-response relationships. Similarly, fertilizers have shown the potential for wildlife toxicity in some situations. The associated hypothesis is that use of pesticides and fertilizers as proposed in the Program Description for Provolt Seed Orchard will cause chemical toxicity resulting in adverse effects to the individual's survival, growth, and reproduction for sensitive species, or to the survival of populations of non-sensitive species.

To test this hypothesis, a conceptual model was developed to illustrate the relationships between stressors, exposure routes, and receptors. The conceptual model is presented in Figure 7-1.

Figure 7-1. Conceptual Model



7.4 Analysis Plan

Based on the conceptual model, typical and maximum exposure scenarios were selected to evaluate risks to terrestrial and aquatic wildlife species. Representative terrestrial and aquatic species and their characteristics were identified, illustrating the various types of exposure that wildlife species may have to chemicals used at the seed orchard. Using the results of the environmental fate assessment described in Section 3.0, environmental exposures were estimated, in terms of dose (mg/kg) for terrestrial species or concentration (mg/L) for aquatic species.

The toxic properties of each pesticide, other ingredient, and fertilizer to wildlife species were researched and summarized, using data available in scientific journals, reference sources, and studies submitted to EPA for registration of the pesticides under FIFRA. Endpoints were identified, including median lethal doses (LD_{50}s), median lethal concentrations (LC_{50}s), and maximum acceptable toxicant concentrations (MATCs).

The doses and concentrations identified in the exposure characterization were compared to the toxic properties identified in the effects characterization, using the guidelines specified by EPA's Office of Pesticide Programs for interpreting risk estimates to general wildlife and to endangered, threatened, or sensitive species.

7.5 References

EPA. See U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency. 1998. Guidelines for ecological risk assessment. Risk Assessment Forum. Washington, DC.

8.0 NON-TARGET SPECIES ANALYSIS

8.1 Data and Models for Analysis

A combination of laboratory study data, field study data, and modeling outputs were used in the non-target species risk assessment.

A large body of quantitative dose-response information for a range of wildlife species has been generated for pesticide chemicals in laboratory studies, in response to the regulatory requirements of FIFRA and environmental concerns about possible hazards posed by pesticides. These data have generally been peer-reviewed by EPA or are published in scientific journals with peer-review protocols. These studies were selected to generate the LD_{50}s (median lethal doses) and LC_{50}s (median lethal concentrations) that are used in the non-target species risk assessment, along with many of the maximum acceptable toxicant concentrations (MATCs).

For some chemicals, sublethal or longer-term effects on aquatic species have been studied in laboratory and field trials, generating no-observed-effect concentrations (NOECs) and lowest-observed-effect concentrations (LOECs). The geometric mean of these two values is the MATC, and is particularly useful if sensitive species may be present.

The GLEAMS model, described in detail in Section 3.2, was used to estimate runoff of pesticides and fertilizers from treated areas into streams, possibly exposing aquatic species as well as terrestrial species (through drinking water). Residue levels on foliage and other wildlife diet items were estimated using the results of field studies.

8.2 Characterization of Exposure

8.2.1 Terrestrial Species

The terrestrial species exposure scenarios postulate that a variety of terrestrial wildlife species use Provolt at various times. The scenarios further postulate that these terrestrial species may be exposed to any applied pesticides or fertilizers through ingestion of contaminated food and water and, in the maximum scenario, direct dermal spray as a result of being in an area as a treatment is occurring.

The list of representative species is as follows:

Mammals
- Deer (large herbivore)
- Coyote (carnivore)
- Long-tailed vole (small herbivore)
- Pocket gopher (subterranean herbivore)
- Raccoon (omnivore)
- Long-eared myotis (insectivore)
- Dog (domestic)

Birds
- Black-capped chickadee (conifer seed-eater)
- Western bluebird (insectivore)
- Tree swallow (insect- and fruit-eater)
- Canada goose (herbivore)
- Mallard duck (water fowl)
- Great blue heron
- Common barn owl (raptor)
- Osprey (piscivore)
- Song sparrow (seed-eater)

Reptiles/Amphibians
- Pacific chorus frog
- Western pond turtle
- Gopher snake
- Western fence lizard

These particular wildlife species were selected because they represent the majority of the species present, or the seed orchard has suitable habitat and is within their range (e.g., selection of black-capped chickadee as conifer seed-eater), and because they represent several types of coverage: a range of phylogenetic classes, body sizes, foraging habitat, and diets for which parameters are generally available. Other common species include the western gray squirrel, turkey, pheasant, quail, and grouse. In addition, several sensitive terrestrial species were evaluated for potential risk:

- Bald eagles, a Federally listed threatened species, may hunt at Provolt.

- Vagrant northern spotted owls may also occasionally use the site for roosting during dispersal.

- The common kingsnake and western pond turtle are state-listed species known to occur at the site.

For each species, characteristics were identified that were used in estimating doses of pesticides, other ingredients, and fertilizers. These characteristics include body weight, surface area, water intake, dietary intake, composition of diet, and home range/foraging area.

For terrestrial wildlife, exposures were assumed to occur through one or more of the following routes for each species/application type, as appropriate:

- Ingestion of sprayed forbs, berries, insects, seeds in treated area
- Ingestion of food with residues or body burden
- Ingestion of water from onsite irrigation ditches
- Direct dermal spray (maximum scenario only)

Spray or drift residues on food items were estimated using the results of field studies by Hoerger and Kenaga (1972), as updated by Fletcher et al. (1994, as cited in Pfleeger et al. 1996). Table 8-1 lists the residue levels predicted.

Table 8-1. Residue Levels

Item	Residue (ppm per lb/acre)
Grass	175[a]
Leaves	135
Forage	135
Small insects	135[b]
Fruits	15
Pod containing seeds	12
Large insects	12[b]

[a]Mean of short range grass and long grass.
[b]EPA's Office of Pesticide Programs groups small insects with broadleaf/forage plants and large insects with fruits, pods, and seeds (EPA 1999).

Predators that feed on other animals were assumed to receive the total body burden that each of the prey species received. Wildlife that feed on aquatic species were assumed to receive residue levels based on the pesticide concentrations in water and pesticide-specific bioconcentration factors, summarized in the environmental fate profiles in Section 3.1.

Chemical concentrations in drinking water sources for wildlife were assumed to be those predicted for the onsite irrigation ditches, presented in Section 3.0.

To calculate typical scenario doses for terrestrial wildlife, the doses from the exposure routes described in the preceding paragraphs were summed, as follows:

$$\mathrm{DOSE} = \mathrm{AREA} \times \mathrm{FRAC} \times \mathrm{DIET} \times \sum_{i=1}^{n} \mathrm{RES}_i \times \mathrm{INT}_i + (\mathrm{AREA} \times \mathrm{H2O} \times \mathrm{CONC}) \div \mathrm{BW}$$

where:

DOSE = dose to wildlife species (mg/kg)
AREA = species' foraging area as a fraction of treatment area (maximum value = 1)
FRAC = fraction of diet assumed to be contaminated (0.5 in typical scenario, 1 in maximum scenario)
DIET = mass of total daily dietary intake (kg)
RES_i = chemical residues on food item *i* (mg residues per kg food item)
INT_i = fraction of daily diet consisting of food item *i*
H2O = daily water intake (L)
CONC = concentration of chemical in drinking water (mg/L)
BW = body weight (kg)

In addition to the dietary doses described above for the typical scenario, the maximum scenario includes direct spray of one-half of the animal's surface area during application. This is only assumed for the maximum scenario, since animals are likely to leave the spray area during any type of disturbance, and the applicators would avoid spraying any animals that are present. Dermal penetration rates were assumed to be 1, 1.5, 3, and 0.5 times the human dermal penetration rates identified in the human health hazard assessment of each chemical (Section 4.4) for mammals, birds, amphibians, and reptiles, respectively. Most dermal penetration tests identified in the human health hazard assessment were conducted in laboratory mammals, so the same rate would be appropriate. Birds may have a slightly higher penetration rate, as feathers serve as excellent conduits to the skin and some birds have a featherless brood patch during incubation (Hope 1995). Amphibians are likely to have significantly increased uptake through their moist, respiring dermis. Reptiles, on the other hand, generally have a drier exterior that would decrease the relative dermal uptake.

Typical and maximum scenarios incorporate different inputs for percent of diet contaminated (half in the typical scenario and all in the maximum scenario), as well as chemical application rate, area treated, and frequency of treatment (see Section 2.2 for details). The scenarios were chosen to be representative of the various combinations of pesticides/fertilizers and application methods that may be used, and to provide an average to conservative picture of the potential range of exposures.

8.2.2 Aquatic Species

The aquatic species exposure scenarios postulate that tadpoles, and aquatic invertebrates in the onsite irrigation ditches; and fish, tadpoles, and aquatic invertebrates in Williams Creek and the Applegate River may be exposed to pesticides or fertilizers through either contaminated runoff coming directly off the fields or drift from pesticide applications.

For each chemical, risks were estimated for aquatic species for which ecotoxicity data are available: rainbow trout as a representative coldwater fish species, the water flea *Daphnia magna* as a representative aquatic invertebrate, and tadpoles of the Pacific chorus frog as a representative amphibian aquatic stage. In addition, five sensitive species known to be present in the Williams Creek watershed and Applegate River sub-basin were evaluated:

- Coho salmon is a Federally listed threatened and state-listed critical species.

- Steelhead and cutthroat trout are state-listed vulnerable species. Chinook salmon is a state-listed critical species. Steelhead trout and chinook salmon were Federal candidate species found to not warrant listing.

- Pacific lamprey is a Federal species of concern.

Also, the spill scenarios described in Section 3.2.5 were evaluated for the risk that would be posed to aquatic species in the case these accidents were to occur.

The concentrations of the proposed chemicals in streams were estimated using the environmental fate and transport modeling methodologies described in Section 3.0. For *Daphnia* and Pacific chorus frog tadpoles, stream concentrations were estimated in the onsite irrigation ditches. For rainbow trout, tadpoles, aquatic invertebrates, and the threatened and sensitive species known to be present in the watershed, concentrations were estimated in both Williams Creek and the Applegate River.

8.3 Characterization of Ecological Effects: Ecological Response Analysis and Stressor-Response Profiles

The most commonly used measurement of terrestrial species toxicity in ecological risk assessments is the acute toxicity test. Acute toxicity studies are used primarily to determine the toxicity reference level known as the median lethal dose (LD_{50}), which is the dose that kills 50 percent of the test animals within 14 days of administering a substance. The lower the LD_{50}, the greater the toxicity of the chemical. Toxic symptoms displayed by the animals are recorded throughout the study, and tissues and organs may be examined for abnormalities at the end of the test. In many cases, toxicity studies with laboratory animals such as rats and mice have been used because of the lack of specific wildlife studies. The results of laboratory animal studies are considered to be representative of the effects that would occur in similar species in the wild. Acute toxicity studies are also conducted on common avian species, such as mallard ducks and bobwhite quail. The toxicity values reported in the following section include oral LD_{50}s for lethal effects, and no-observed-effect levels (NOELs) and lowest-observed-effect levels (LOELs) for studies of sublethal effects. For feeding studies, LD_{50}s, NOELs, and LOELs may be expressed in terms of parts per million (ppm), representing milligrams of chemical per kilogram of food consumed by the animals during the study.

For aquatic species, the LC_{50} is the water concentration that is lethal to half the test population, and is presented in terms of milligrams per liter (mg/L). Another common endpoint is the median effective concentration (EC_{50}), which is the concentration of a toxicant that produces a specific effect on 50 percent of the test organisms; it is often used with animals for which determining mortality is difficult, such as daphnid species. The EC_{50} is also expressed in units of mg/L. In some cases, no-observed-effect concentrations (NOECs) and lowest-observed-effect concentrations (LOECs) are identified for studies in which non-lethal observations of aquatic toxicity were recorded.

If no information on a group of animals is included in a stressor-response profile for a particular chemical, it is because no data are available in the literature.

8.3.1 Acephate

Toxicity to Terrestrial Species

In general, acephate is moderately toxic to mammals and birds. A summary of acephate's toxicity to terrestrial species is found in Table 8-2.

Table 8-2. Toxicity of Acephate to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Mouse	361	EPA 1984a
Rat	605	Lambert 1983
White mice*	720	Clark and Rattner 1987
Dog	>681	Lambert 1983
Rabbit*	700	Lambert 1983
Little brown bats*	>1,500	Clark and Rattner 1987
Mallard duck	350	EPA 1984a
Dark-eyed junco*	106	Zinkle et al. 1981
Chicken	568	Lambert 1983
Ring-necked pheasant	140	EPA 1984b

*Substance tested was a 75% formulation.

In an acute study in little brown bats, 24 hours after the study, nine of the 30 bats tested could not right themselves after being placed on their backs. The investigators calculated an ED_{50} (median effective dose) of 687 mg/kg for this observation. The investigators believed that this was a useful measurement, since bats unable to right themselves would be helpless and subject to predation (Clark and Rattner 1987).

When single doses of acephate were administered to buffalo calves, 100% mortality was observed at a dose of 800 mg/kg. No mortality, but significant cholinesterase inhibition, occurred at a dose of 400 mg/kg (Singh and Sandhu 1999).

Effects to songbirds from forestry applications of acephate have been reported in several studies. In eastern Canada, acephate was applied to control spruce budworm at two sites at rates of 0.5 and 1.0 lb/acre. Actual measured deposition ranged from 0.09 to 6.5 lb/acre. Daily surveys detected no adverse effects to songbirds from these applications (Lambert 1983). In another study in Maine, acephate was applied at a rate of 0.5 lb/acre to a 60,000-acre block of forest. No affected birds were identified during surveys taken up to 35 days post-spraying. Significantly inhibited brain cholinesterase levels were found in some bird species (evening grosbeaks and magnolia warblers); however, there was also a great deal of variability in brain cholinesterase among individuals and between species (Lambert 1983). In reviewing these and other studies, Lambert (1983) concluded that cholinesterase inhibition greater than 50 percent may be lethal, the effects from summer applications tend to last longer than those from fall applications, and birds preferring open spaces or crown foliage for their foraging are likely to receive higher doses.

When adult white-throated sparrows were exposed to 256 ppm dietary acephate for 14 days, their ability to establish a preferred migratory orientation was impaired (Vyas et al. 1995). Because this effect was not observed in juvenile sparrows, the authors suggested that it may have been due to an effect on memory, as opposed to motor system effects.

Toxicity to Aquatic Species

Acephate is slightly toxic to freshwater fish. A summary of toxicity values published in the literature is found in Table 8-3.

Table 8-3. Toxicity of Acephate to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	895-1,050 (24-hr)	Lambert 1983
Rainbow trout	1,100 (96-hr)	EPA 1984a
Rainbow trout*	730 (96-hr)	EPA 1984a
Rainbow trout*	>4.7 (20-day)	Davies et al. 1994
Cutthroat trout	>100 (96-hr)	Lambert 1983, EPA 1984a
Brook trout	>100 (96-hr)	EPA 1984a
Fathead minnow	>,1000 (96-hr)	EPA 1984a
Bluegill sunfish*	2,050	Valent 2000
Channel catfish	>1,000 (96-hr)	EPA 1984a
Green frog tadpole	6,433 (24-hr)	Lambert 1983
Green frog tadpole	>5,000 (24-hr)	EPA 1984a
Northwestern salamander, larvae	8,816 (96-hr)	Pauli et al. 2000
Stonefly naiad	9.5 (96-hr)	USDA 1989
Scud	>50 (96-hr)	USDA 1989
*Daphnia magna**	1.3 (48-hr)	EPA 1984a
Midge	>1,000 (96-hr)	USDA 1989

*Value reported is for 75% formulation of acephate.

In a study in a small stream in British Columbia, the toxicity of low doses of acephate was studied in caged rainbow trout, insect nymphs, and benthic insects. Four study sites were selected: one site upstream (the control site), and three sites downstream (at 150, 300, and 2,000 m) from the site of the acephate introduction. Acephate was applied to the creek for five hours at a concentration of 1.000 mg/L. Maximum concentrations in the stream were 1.199 mg/L at the first site at three hours, 0.987 mg/L at the second site at five hours, and 0.169 mg/L at the third site at eight hours. No mortality was noted in the caged fish or insect nymphs during the 96-hour exposure (Geen et al. 1981).

Studies in freshwater fish and crustaceans in Australia found a consistent NOEC for cholinesterase inhibition and elevated blood glucose of 1.3 mg/L for 10 days exposure across all species tested, with a LOEC of 4.4 mg/L. However, a LOEC of 0.19 mg/L was observed for specific endpoints in some individual species (Davies et al. 1994).

Laboratory tests have demonstrated that acephate is of low toxicity to aquatic invertebrates. EPA (1984b) reported that a 21-day exposure of daphnia to a 75-percent formulation of acephate was not toxic to adult organisms (test levels ranged from 0.019 to 1.50 mg/L), although the number of offspring produced per female was reduced at test concentrations of 0.375 mg/L and higher.

Egg hatch in the northwestern salamander was not affected by acephate concentrations up to 798 mg/L; however, decreased growth and increased mortality of larvae were observed at a concentration of 382 mg/L (Pauli et al. 2000). In another study, all tadpoles of the bullfrog *Rana catesbeiana* survived after exposure to 5 mg/L (Pauli et al. 2000). The NOEC and LOEC for decreased activity in green frog larvae exposed to acephate were 500 and 1,000 mg/L, respectively (Pauli et al. 2000).

8.3.2 Chlorothalonil

Toxicity to Terrestrial Species

Zeneca (1998) reported an oral LD_{50} in rats of 4,200 mg/kg. Subchronic oral studies in mice, rats, and beagle dogs resulted in NOELs of 2.1, 3.0, and 15 mg/kg/day, respectively (EPA 1999). Terrestrial acute toxicity data are summarized in Table 8-4.

Table 8-4. Toxicity of Chlorothalonil to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	4,200	Zeneca 1998
Japanese quail	>2,000	EPA 1999
Mallard	>4,640	EPA 1999
Mallard	5,000	Extoxnet 2000

No adverse effects were observed in a 4-day feeding study in which Holstein cows were fed 0.144 mg/kg/day (Caux et al. 1996).

In subacute feeding studies, dietary LC_{50}s were reported as >10,000 ppm in northern bobwhite, and >21,500 and >10,000 ppm in two studies in mallards (EPA 1999). A reproductive NOEL of 1,000 ppm was determined in a feeding study in bobwhite, where overt toxicity and reduced reproduction were observed at the LOEL of 5,000 ppm (EPA 1999).

Toxicity to Aquatic Species

Acute aquatic toxicity data for chlorothalonil are summarized in Table 8-5.

A full life-cycle aquatic toxicity test with chlorothalonil resulted in a NOEC of 0.003 mg/L in fathead minnows, with hatching success and survivability affected at the LOEC of 0.0065 mg/L (EPA 1999). Caux et al. (1996) reported a 21-day NOEC and LOEC of 0.0023 and 0.0049 mg/L, respectively, for rainbow trout mortality and behavioral effects.

Table 8-5. Toxicity of Chlorothalonil to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	0.0423 (96-hr)	EPA 1999
Rainbow trout	0.25	Extoxnet 2000
Rainbow trout	>0.0082 (10-day)	Davies et al. 1994
Bluegill	0.051 (96-hr)	EPA 1999
Bluegill	0.3	Extoxnet 2000
Channel catfish	0.048 (96-hr)	EPA 1999
Channel catfish	0.43	Extoxnet 2000
Channel catfish	0.052 (96-hr)	Gallagher et al. 1992
Fathead minnow	0.023 (96-hr)	EPA 1999
Daphnia magna	0.068	EPA 1999
Frog	0.16 (48-hr)	Caux et al. 1996
Clawed toad, embryo	0.09 (96-hr)	Pauli et al. 2000
Indian rice frog, adult	0.25 (48-hr)	Pauli et al. 2000

Acute toxicity to caged aquatic species was examined in a field study summarized by Caux et al. (1996). Cumulative water concentrations ranging from 0.171 to 0.883 mg/L were produced by three aerial applications of Bravo® 500 (40.4% a.i.) to a 0.2-hectare pond that was 0.5 meters deep. Mortality to the aquatic insect water boatman and the fish species threespine stickleback was observed.

Studies in freshwater fish and crustaceans in Australia found a consistent NOEC for elevated liver enzymes of 0.0008 mg/L for 10 days exposure across all species tested, with a LOEC of 0.0014 mg/L. However, a LOEC of 0.0003 mg/L was observed for specific non-lethal toxicity endpoints in some individual species (Davies et al. 1994).

A 96-hour EC_{50} for malformations in embryos of the clawed toad was measured as 0.02 mg/L (Pauli et al. 2000).

8.3.3 Chlorpyrifos

Toxicity to Terrestrial Species

The acute toxicity of chlorpyrifos to mammals and bird species is summarized in Table 8-6.

Table 8-6. Toxicity of Chlorpyrifos to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	223	EPA 2000a
Rat	97	EPA 2000b
Mouse	62.5	EPA 2000a
Rabbit	1,000 to 2,000	EPA 2000a
Guinea pig	504	EPA 2000a
Domestic goat	500 to 1,000	HSDB 2001
Chickens	32	Extoxnet 2000
House sparrow	10	EPA 2000b
Ring-necked pheasant	8.41	EPA 2000b
Northern bobwhite	32	EPA 2000b
Mallard duck	75.6	EPA 2000b
Red-winged blackbird	31.1	EPA 2000b
Coturnix quail	13.3	EPA 2000b
California quail	68.3	EPA 2000b
Sandhill crane	25 to50	EPA 2000b
Rock dove	26.9	EPA 2000b
White leghorn cockerel	34.8	EPA 2000b
Canada goose	40 to 80	EPA 2000b
Common grackle	5.62	EPA 2000b
Common pigeon	10.0	EPA 2000b
Chukar	61	EPA 2000b
Starling	75	EPA 2000b
Bull frog	>400	EPA 2000b

Subacute feeding studies in birds resulted in dietary LC_{50}s of 492, 387, and 803 ppm for coturnix quail, northern bobwhite, and mallard ducks, respectively (EPA 2000b). Chronic feeding studies in birds have also been conducted for chlorpyrifos (EPA 2000b). Among three studies in mallard ducks, the lowest LOEL was 60 ppm in the diet, producing reduced body weight and reduced number of eggs. The highest NOEL in these three studies was 46 ppm. Reduced eggs were also observed in northern bobwhite at a dietary level of 130 ppm, with a NOEL of 40 ppm.

Toxicity to Aquatic Species

Chlorpyrifos is highly to very highly toxic to fish and aquatic invertebrates. LC_{50}s for aquatic species are summarized in Table 8-7.

Table 8-7. Toxicity of Chlorpyrifos to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Bluegill sunfish	0.0018 (96-hr)	EPA 2000b
Rainbow trout	0.003 (96-hr)	EPA 2000b
Cutthroat trout	0.0134 (96-hr)	EPA 2000b
Channel catfish	0.0134 (96-hr)	EPA 2000b
Lake trout	0.098 (96-hr)	EPA 2000b
Fathead minnow	0.203 (96-hr)	EPA 2000b
Green sunfish	0.0225 (36-hr)	EPA 2000b
Golden shiner	0.035 (36-hr)	EPA 2000b
Mosquito fish	0.215 (36-hr)	EPA 2000b
Daphnia magna	0.00010 (48-hr)	EPA 2000b
Daphnia magna	0.0006 (48-hr)	Moore et al. 1998
Scud	0.00011 (96-hr)	EPA 2000b
Stonefly	0.0082 (96-hr)	EPA 2000b
Toad, tadpole	0.001 (24-hr)	EPA 2000b
Leopard frog, tadpole	3 (24-hr)	EPA 2000b
Leopard frog, adult	30 (24-hr)	EPA 2000b
Indian bullfrog, tadpole	0.177 (24-hour)	Barron and Woodburn 1995
Indian bullfrog, tadpole	0.010 (6-day)	Barron and Woodburn 1995

The 96-hour EC_{10} for locomotion and behavior was determined to be greater than 0.096 mg/L (highest concentration tested) in the newt *Triturus vulgaris* (van Wijngaarden et al. 1993).

A 30-day early life stage toxicity test in fathead minnows resulted in a NOEC of 0.00129 mg/L, with spinal deformities observed at the LOEC of 0.0021 mg/L (EPA 2000b). A full life-cycle test in fathead minnows resulted in a NOEC of 0.00057 mg/L, with decreased survival at the LOEC of 0.00109 mg/L (EPA 2000b).

8.3.4 Diazinon

Toxicity to Terrestrial Species

The acute toxicity of diazinon is summarized in Table 8-8.

Birds seem to be more sensitive than mammals to diazinon poisoning (Stone and Gradoni 1985). Technical diazinon is characterized as very highly toxic to waterfowl on an acute oral basis, with an LD_{50} of 3.5 mg/kg for mallard ducks (Hudson et al. 1984). Diazinon has a potential for causing acute avian poisoning episodes (Schafer et al. 1983). Kills of Canada geese, brant, mallard, American black duck, other species of waterfowl, and songbirds have all been associated with consumption of grass or grain shortly after diazinon application (Schobert 1974, Zinkle et al. 1978, Stone 1980, and Stone and Knoch 1982). Fatal diazinon poisonings have also been recorded in domestic ducklings and goslings (Egyed et al. 1974, as cited in Eisler 1986; Egyed et al. 1976).

Table 8-8. Toxicity of Diazinon to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	505	EPA 2001
Rat	1,960	Platte Chemical Co. 1994
Pig	300	Machin et al. 1975
Guinea pig	300	Machin et al. 1975
Dog (beagle)	>300	Earl et al. 1971
Sheep	>1,000	Machin et al. 1975
Mallard	3.5	Hudson et al. 1984
Mallard	1.44	EPA 2001
Northern bobwhite	5.2	EPA 2001
Ring-necked pheasant	4.3	Hudson et al. 1984
European quail	4.2	Schafer et al. 1983
House sparrow	7.5	Schafer et al. 1983
Red-winged blackbird	3.2	EPA 2001
Brown-headed cowbird	69.0	EPA 2001
Canada goose	6.16	EPA 2001
Bullfrog	>2,000	Hudson et al. 1984

Avian dietary studies characterized diazinon as highly toxic for upland game birds with a dietary LC_{50} of 245 ppm for bobwhite quail. Diazinon was very highly toxic to waterfowl with a dietary LC_{50} less of 32 ppm for mallard ducks; the NOEL was 16 ppm in this study (EPA 2001). Results of 5-day feeding trials with 2-week old Japanese quail followed by 3 days on untreated feed showed a dietary LC_{50} of 167 ppm. No deaths were observed at a dietary level of 85 ppm, but 53 percent died at 170 ppm and 87 percent died at 240 ppm (Hill and Camardese 1986). The 8-day dietary LC_{50} for the brown-headed cowbird was determined to be 38 ppm in food, with a NOEL of 8 ppm (EPA 2001). In Canada goose and ring-necked pheasant, 8-day assays yielded LC_{50}s of 3,912 and 244 ppm, respectively (EPA 2001). Reduced egg production, decreased food consumption, and loss in body weight have been observed in ring-necked pheasants at daily diazinon intakes greater than 1.05 mg/bird (Stromborg 1977). In dietary studies, oral doses above 50 mg/kg were associated with reduced food consumption, weight loss, and reduced egg production in northern bobwhites (Eisler 1986).

Two avian reproduction studies were reported by EPA (2001). A significant reduction in the number of 14-day hatchling survivors was observed at the LOEL of 16.33 ppm diazinon fed to mallard ducks, with a NOEL of 8.3 ppm. No adverse effects were observed at the highest dietary level tested in northern bobwhite quail of 32 ppm.

Diazinon adversely affects survival of developing mallard embryos when the eggshell surface is subjected for 30 seconds to concentrations 25 to 34 times higher than recommended field application rates. These findings suggest that eggs of mallards, and probably other birds, may remain unaffected when diazinon is applied according to label directions (Hoffman and Eastin 1981, Eisler 1986).

Toxicity to Aquatic Species

Technical diazinon and its end-use formulations are highly to very highly toxic to aquatic organisms. The acute toxicity of diazinon has been determined in many species of freshwater fish, and is summarized in Table 8-9.

Chronic testing in fish did not determine a NOEC, as adverse effects were observed at the lowest concentrations tested of 0.00055 mg/L in brook trout for 8 months, and 0.0032 mg/L in fathead minnows for 25 days (EPA 2001). An acute NOEC of 0.00056 mg/L was determined for daphnids (EPA 2001). In a 21-day study in *Daphnia*, a subchronic NOEC of 0.00017 was identified (EPA 2001).

Table 8-9. Toxicity of Diazinon to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	0.090 (96-hr)	Johnson and Finley 1980
Cutthroat trout	1.7 (96-hr)	Johnson and Finley 1980
Brook trout	0.770 (96-hr)	Allison and Hermanutz 1977
Lake trout	0.602 (96-hr)	Johnson and Finley 1980
Bluegill sunfish	0.022 (96-hr)	Cope 1965
Bluegill sunfish	0.168 (96-hr)	Johnson and Finley 1980
Bluegill sunfish	0.136	EPA 2001
Bluegill sunfish	0.460	EPA 2001
Fathead minnow	10.3 (96-hr)	Meier et al. 1979
Fathead minnow	7.8	EPA 2001
Daphnia sp.	0.0008 (48-hr)	Johnson and Finley 1980
Daphnia sp.	0.00083	EPA 2001
Daphnia sp.	0.0014 (48-hr)	Johnson and Finley 1980
Scud	0.0002 (96-hr)	Johnson and Finley 1980
Scud	0.80 (24-hr) 0.50 (48-hr) 0.20 (96-hr)	Sanders 1969 Sanders 1969 Sanders 1969
Stonefly	0.025 (96-hr)	Johnson and Finley 1980
Green frog	<0.05 (96-hr) 0.005 (16-day)	Harris et al. 1998

8.3.5 Dicamba

Toxicity to Terrestrial Species

Dicamba is slightly toxic to mammalian and bird species. The results of acute studies are summarized in Table 8-10.

In a 15-week feeding study in rats, the NOEL was 316 ppm (19 to 43 mg/kg/day), based on increased relative liver weights at a dietary level of 1,000 ppm (Edson and Sanderson 1965). The authors also reported the results of other studies in which 20,000 ppm in the diet of a heifer caused no adverse effects, and a study in which sheep were unaffected by doses of 250 mg/kg for 10 days, but were killed by doses of 500 mg/kg after 2 days.

Table 8-10. Toxicity of Dicamba to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	2,740	EPA 1999
Rat	2,629	Micro Flo 1999
Rat	757 (male) 1,414 (female)	Edson and Sanderson 1965
Mouse	1,189	Edson and Sanderson 1965
Guinea pig	566	Edson and Sanderson 1965
Rabbit	566	Edson and Sanderson 1965
Hen	673	Edson and Sanderson 1965
Pheasant	800 (male) 673 (female)	Caux et al. 1993
Mallard ducks	2,009	Extoxnet 2000

Dietary studies in birds resulted in a 5-day LC_{50} >5,000 ppm in Japanese quail (Hill and Camardese 1986), and 8-day dietary LC_{50}s >10,000 ppm in bobwhite quail and mallard ducks (Extoxnet 2000).

Toxicity to Aquatic Species

Dicamba exhibits slight to moderate toxicity to aquatic species. Rainbow trout are the most sensitive fish. Acute study data are presented in Table 8-11.

Table 8-11. Toxicity of Dicamba to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Trout	>1,000 (96-hr)	Micro Flo 1999
Rainbow trout	35 (24-hr) 28 (96-hr)	Mayer and Ellersieck 1986
Cutthroat trout	>50 (96-hr)	Caux et al. 1993
Coho salmon	>109 (6-day)	Caux et al. 1993
Bluegill	>1,000 (96-hr)	Micro Flo 1999
Bluegill	20 mg/L (48-hr)	Verschueren 1983
Bluegill	>50 (96-hr)	Mayer and Ellersieck 1986
Mosquitofish	516 (24-hr) 510 (48-hr) 465 (96-hr)	Caux et al. 1993
Carp	465 (48-hr)	Extoxnet 2000
Daphnia sp.	1,600 (48-hr)	Micro Flo 1999
Daphnia sp.	>100 (48-hr)	Mayer and Ellersieck 1986
Scud	3.9 (96-hr)	Verschueren 1983
Scud	>100 (96-hr)	Mayer and Ellersieck 1986
Scud	>100 (96-hr)	Mayer and Ellersieck 1986
Aquatic sow bug	>100 (96-hr)	Mayer and Ellersieck 1986
Tusked frog *Adelotus brevis*	220 (24-hr) 202 (48-hr) 185 (96-hr)	Caux et al. 1993
Striped marsh frog *Limnodynastes peroni*	205 (24-hr) 166 (48-hr) 106 (96-hr)	Caux et al. 1993

8.3.6 Dimethoate

Toxicity to Terrestrial Species

Dimethoate is moderately toxic to mammals and highly toxic to avian species. Acute data are summarized in Table 8-12.

Oral doses of dimethoate at 0.2 mg/kg were given to ewes 3 times per week for 36 days. Serum insulin concentrations were increased at the conclusion of the study, although no overt signs of toxicity were observed (Rawlings et al. 1998).

Table 8-12. Toxicity of Dimethoate to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	387	EPA 1999a
Mouse	120	EPA 1999b
Rabbit	400 to500	Extoxnet 2000
Guinea pig	350 to 400	Extoxnet 2000
Mule deer	>200	EPA 1999b
Dog	400	Khan and Dev 1982
Red-winged blackbird	5.4	EPA 1999b
Ring-necked pheasant	20	EPA 1999b
European starling	32	EPA 1999b
Mallard	41.7	EPA 1999b
Domestic chicken	50	EPA 1999b

In 5-day feeding studies, dimethoate produced dietary LC_{50}s of 332, 346, and 1,011 ppm in ring-necked pheasant, Japanese quail, and mallard ducks, respectively (EPA 1999b). Longer-term feedings studies have also been conducted. A NOEL of 4 ppm was found for northern bobwhite in a 147-day study, in which reduced weights were observed in surviving offspring at a dietary level of 10.1 ppm. In northern bobwhite, reduced hatchlings and increased cracked eggs were found at a dietary level of 30 ppm for 196 days. Effects on egg production and survivability of offspring were found at a level of 152 ppm in a 154-day feeding study in mallards, with a NOEL of 35.4 ppm (EPA 1999b).

Pradhan and Dasgupta (1993) gave single oral doses of dimethoate at 450 mg/kg to male toads *Bufo melanostictus*, and evaluated ascorbic acid concentrations in the kidney, liver, and testes for 21 days. The authors stated that normal levels in the liver and kidney had not recovered by the conclusion of the test.

Toxicity to Aquatic Species

Acute studies of dimethoate's effects on aquatic species are summarized in Table 8-13.

In a chronic study with rainbow trout, growth was affected at a concentration of 0.84 mg/L; no adverse effects were observed at a level of 0.43 mg/L (EPA 1999b).

In a 21-day study in *Daphnia magna*, reproduction, growth, and survival were affected at a concentration of 0.1 mg/L, with no effects at 0.04 mg/L. In a 28-day study, *Daphnia* exhibited decreased reproduction and survival at 0.45 mg/L, and were unaffected at 0.23 mg/L (EPA 1999b).

Table 8-13. Toxicity of Dimethoate to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	6.2 (96-hr)	EPA 1999b
Bluegill sunfish	6.0 (96-hr)	EPA 1999b
Daphnia	3.32 (48-hr)	EPA 1999b
Stonefly	0.043 (96-hr)	EPA 1999b
Scud	0.2 (96-hr)	EPA 1999b
Indian bullfrog, egg	8	Khan and Dev 1982
Indian bullfrog, larvae	5	Khan and Dev 1982
Skipper frog *Rana cyanophlyctis*, adult	39 (male) 36 (female)	Mudgall and Patil 1987

In tests in the clawed toad, NOECs for dimethoate's lethality, developmental effects, and effects on growth after 100 days of exposure were 1, 32, and 32 mg/L, respectively (Devillers and Exbrayat 1992).

8.3.7 Esfenvalerate

Toxicity to Terrestrial Species

Esfenvalerate is slightly to moderately toxic to mammals and birds. Toxicity data for terrestrial species are summarized in Table 8-14.

Table 8-14. Toxicity of Esfenvalerate to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	87.2	EPA 1997
Rat*	458	Du Pont 1999
Mouse	100 to 300	Cabral and Galendo 1990
Mallard duck	>2,250	Extoxnet 2000
Bobwhite quail	1,312	Extoxnet 2000
Bobwhite quail	381	Du Pont 1999

*Value is for Asana® XL formulation.

For avian species, dietary LC_{50} values of >5,620 ppm in bobwhite quail and 5,247 ppm in mallard ducks were reported (Du Pont 1999). Single oral doses up to 4,000 mg/kg were not lethal to the American kestrel, although signs of mild intoxication were observed (Eisler 1992).

Toxicity to Aquatic Species

Esfenvalerate is very highly toxic to aquatic species. Acute aquatic toxicity data for esfenvalerate and fenvalerate are summarized in Table 8-15.

Table 8-15. Toxicity of Esfenvalerate and Fenvalerate to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	0.00026 (96-hr)	Du Pont 1999
Rainbow trout	0.076 (24-hr)	Coats and O'Donnell-Jeffery 1979
Steelhead trout, juvenile	0.000088 (96-hr intermittent, mean) 0.000172 (96-hr continuous)	Curtis et al. 1985
Atlantic salmon	0.0000012 (96-hr)	McLeese et al. 1980
Bluegill sunfish	0.00026 (96-hr)	Du Pont 1999
Bluegill sunfish	0.00031 (96-hr)	Fairchild et al. 1982
Common carp	0.0001 (96-hr)	Extoxnet 2000
Killifish	0.0002 (96-hr)	Extoxnet 2000
Fathead minnow	0.00113 (48-hr)	Bradbury et al. 1987
Fathead minnow	0.00018 (96-hr)	Du Pont 1999
Daphnia sp.	0.00027 (48-hr)	Fairchild et al. 1992
Amphipod *Gammarus*	0.00003 (96-hr)	Anderson 1982
Mayflies	0.093 (96-hr)	Anderson 1982
Rhagonid fly	0.00032 (96-hr)	Anderson 1982
Leopard frog, tadpole	0.00729 (96-hr)	Materna et al. 1995

Holcombe et al. (1982) observed sublethal effects in acute toxicity studies of fenvalerate to rainbow trout and fathead minnows. The 48-hour NOEC for rainbow trout was 0.0007 mg/L, with rapid gill movements and a pattern of swimming at the water surface observed at the LOEC of 0.0013 mg/L. The 48-hour NOEC in fathead minnows was 0.0023 mg/L, with absence of schooling behavior at the LOEC of 0.0025 mg/L.

Barry et al. (1995) reported a study of esfenvalerate exposure to the Australian crimson-spotted rainbowfish *Melanotaenia fluviatilis*. Adult fish were pulse-exposed to initial concentrations of 0.001, 0.0032, 0.010, 0.032, and 0.100 mg/L esfenvalerate, and the concentrations declined to less than one percent of initial levels by 24 hours, through constant fresh water flow into the aquaria. At a concentration of 0.100 mg/L, 100% of males and 70% of females died within the first 24 hours. At the lowest concentration tested of 0.001 mg/L, there was a significant decrease in the number of larvae hatching per spawning day, compared to controls. Hatchability of eggs, increased incidence of abnormalities in larvae, and liver weights in females were significantly affected at a concentration of 0.032 mg/L. Larval size was not significantly affected at concentrations up to 0.032 mg/L (the effective highest concentration tested). The only concentration tested (0.032 mg/L) in two additional assays affected hepatic cytochrome P450 activity in males, and the mitogenic activity of kidney lymphocytes in females. Overall, this study indicates that fish reproduction can be affected by initial pulse-exposure concentrations of esfenvalerate as low as 0.001 mg/L.

In an aquatic mesocosm study on esfenvalerate, measurements of bluegill sunfish survival, biomass, sex ratios, and reproductive success decreased with increasing concentrations of esfenvalerate (Fairchild et al. 1992). Reproductive success in bluegills was significantly lower in mesocosms with concentrations of 0.00067 and 0.00171 mg/L of esfenvalerate. Macroinvertebrate populations were significantly reduced at all concentrations. In a reproductive study in bluegill sunfish in littoral enclosures conducted by Tanner and Knuth (1996), applied concentrations of esfenvalerate one month apart as low as 0.00008 mg/L affected growth of young. At 0.001 mg/L, spawning was delayed for 15 days and most larvae died.

Woin (1998) found structural changes in the macroinvertebrate community over a period of more than two years following a single addition of fenvalerate to mesocosms. Concentrations in the different mesocosms one week after application were 0.00002, 0.0002, 0.002, and 0.02 mg/L. No effects were seen at the two lowest concentrations.

Berrill et al. (1993) evaluated the effect of fenvalerate on three amphibian species at two water temperatures. Mortality did not occur with 22-hour exposures of 0.01 and 0.1 mg/L in embryos and newly hatched tadpoles of the green frog *Rana clamitans*, the leopard frog *Rana pipiens*, and the spotted salamander *Ambystoma maculatum*. However, sublethal effects were indicated by abnormal behaviors in the frogs, with recovery occurring within three to nine days, depending on temperature, after exposure to 0.01 mg/L. Complete recovery was not accomplished after exposure to 0.1 mg/L by the end of the 11-day observation period in frog tadpoles held at 15 °C, while those at 20 °C recovered by day 6. Salamander larvae were more sensitive, with little recovery evident by day 11 after exposure to 0.01 mg/L at 15 °C, although most had recovered by that time in the 20 °C test group.

Devillers and Exbrayat (1992) reported an EC_{50} of 0.00485 mg/L for leopard frog tadpoles. The observed effect was twitching and twisting while staying on the bottom of the test chamber.

8.3.8 Glyphosate

Toxicity to Terrestrial Species

Glyphosate is slightly toxic to mammals. Toxicity data for terrestrial species are summarized in Table 8-16.

Table 8-16. Toxicity of Glyphosate to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	>4,320	EPA 1993
Rat	2,047	Giesy et al. 2000
Rabbit	3,800	Smith et al. 1992
Goat	3,500	Giesy et al. 2000
Bobwhite quail	>2,000	EPA 1993
Bobwhite quail	>3,851	Giesy et al. 2000

Glyphosate is slightly toxic to avian species. An eight-day dietary LC_{50} greater than 4,640 ppm was reported for mallards and bobwhites (EPA 1993), and a dietary LC_{50} value was greater than 5,000 ppm in Japanese quail (Hill and Camardese 1986). Three avian reproduction studies reviewed by EPA (1993) indicated that glyphosate was not expected to cause any reproductive impairment in birds; the highest dietary levels tested were 1,000 ppm in a study in bobwhite quail, and 30 and 1,000 ppm in two studies in mallard ducks.

Black-tailed deer in pens showed no gross adverse health effects after exposure to glyphosate applied at rates used for vegetation management (2.2 kg/ha). Browse treated with glyphosate was readily eaten by the deer, and was actually preferred as forage in two trials (Sullivan and Sullivan 1979, as cited in Sullivan 1985).

Studies evaluating the effects of 2.2-kg/ha (2-lb/acre) glyphosate treatments on small wild mammals (deer mice, voles, chipmunks, and shrews) in coniferous forests found little or no adverse effect on reproduction, growth, or survival in populations during the year following field treatments (Sullivan and Sullivan 1982, as cited in Sullivan 1985). However, a slight decrease in habitat population would be expected after glyphosate application due to vegetative succession and interactions among the various communities (Smith et al. 1992).

Toxicity to Aquatic Species

The active ingredient glyphosate is moderately toxic to aquatic species. The Roundup® formulation of glyphosate is more toxic to aquatic organisms than technical glyphosate due to the surfactant in the formulation. The Rodeo® formulation, which is the only glyphosate formulation used at Provolt, does not contain this ingredient. The toxicity of glyphosate and its formulations to aquatic species is summarized in Table 8-17.

In 96-hour studies in fathead minnows and plains minnows using the Rodeo® formulation, no effects on survival were observed at a concentration of 1,000 mg Rodeo®/L (Beyers 1995).

Table 8-17. Toxicity of Glyphosate to Aquatic Species

Species	Material	LC_{50} (mg/L)	Reference
Rainbow trout	glyphosate Roundup	86 (96-hr) 8.2 (96-hr)	EPA 1993
Rainbow trout	Rodeo	>1,000 (96-hr)	Monsanto 2000
Chinook salmon	Roundup	20 (96-hr)	Giesy et al. 2000
Chinook salmon	glyphosate	30 to 211 (96-hr)	Giesy et al. 2000
Coho salmon	Roundup	22 (96-hr)	Giesy et al. 2000
Coho salmon	glyphosate	36 to 174 (96-hr)	Giesy et al. 2000
Fathead minnow	glyphosate Roundup	97 (96-hr) 2.3 (96-hr)	Folmar et al. 1979
Fathead minnow	glyphosate	84.9 (96-hr)	EPA 1993
Channel catfish	glyphosate Roundup	130 (96-hr) 3.3 (96-hr)	Folmar et al. 1979
Bluegill sunfish	glyphosate Roundup	140 (96-hr) 5.0 (96-hr)	Folmar et al. 1979
Bluegill sunfish	glyphosate	120 (96-hr)	EPA 1993
Bluegill sunfish	Rodeo	>1,000 (96-hr)	Monsanto 2000
Daphnia sp.	glyphosate Roundup	780 (48-hr) 3.0 (48-hr)	EPA 1993 Folmar et al. 1979
Daphnia sp.	Rodeo	930 (48-hr)	Monsanto 2000
Scud	glyphosate	43 (96-hr)	Folmar et al. 1979
Midge *Chironomus plumosus*	glyphosate	55 (48-hr)	EPA 1993
Frog (*Crinia insignifera)*, newly emerged	glyphosate Roundup	83.6 (48-hr) 144 (48-hr)	Mann and Bidwell 1999, as cited in Geisy et al. 2000
Frog (*Crinia insignifera)*, adult	glyphosate Roundup	78 (96-hr) 96.8 (96-hr)	Bidwell and Gorrie 1995, as cited in Geisy et al. 2000
Frog (*Heleioporus eyrei*), tadpole	glyphosate Roundup	>373 (48-hr) 17.5 (48-hr)	Mann and Bidwell 1999, as cited in Geisy et al. 2000
Frog (*Limnodynastes dorsalis*), tadpole	glyphosate Roundup	>400 (48-hr) 8.3 (48-hr)	Mann and Bidwell 1999, as cited in Geisy et al. 2000
Frog (*Litoria moorei*), tadpole	glyphosate Roundup	>400 (48-hr) 8.1 (48-hr)	Mann and Bidwell 1999, as cited in Geisy et al. 2000

Roundup® produced no effects on fecundity or maturation in rainbow trout exposed to 0.02, 0.2, and 2.0 mg/L for 12 hours. Also, no effects were observed on fecundity or maturation of gonads in test fish after being held in freshwater for 30 days (Folmar et al. 1979). In 21-day tests in rainbow trout, NOECs of 52 and 2.4 mg/L were reported for glyphosate and Roundup® exposure, respectively

(Giesy et al. 2000). In a full life cycle study of glyphosate in fathead minnows, no effects were observed at the concentration tested of 25.7 mg/L (EPA 1993).

Midge larvae were exposed to 0.02, 0.2, and 2.0 mg/L of Roundup®. Significant increases in stream drift (dead or dying individuals floating on surface) of the larvae were observed at the highest concentration (Folmar et al. 1979). In a chronic test of glyphosate in *Daphnia*, reduced reproductive success was observed at a concentration of 96 mg/L, with no effects at 50 mg/L (EPA 1993).

Perkins et al. (2000) tested the Roundup® and Rodeo® formulations of glyphosate in a frog (*Xenopus laevis*) embryo teratogenesis assay. Rodeo® produced an LC_5 (concentration lethal to 5% of the test species) of 3,779 mg glyphosate acid equivalent (a.e.)/L, and an LC_{50} of 5,407 mg a.e./L. Roundup® resulted in an LC_5 of 6.4 mg a.e./L and an LC_{50} of 9.4 mg a.e./L.

8.3.9 Hexazinone

Toxicity to Terrestrial Species

Hexazinone has low acute toxicity to the terrestrial species tested. Data are presented in Table 8-18.

Subacute 5-day feeding studies in bobwhite quail and mallard ducks produced dietary LC_{50}s >10,000 ppm for both species (USDA 1984). In a reproduction study in bobwhite quail, effects on body weight were observed at the lowest dietary level tested of 100 ppm (EPA 1994). No adverse effects were observed at the highest dietary level tested of 1,000 ppm in a reproduction study in mallard ducks (EPA 1994).

Table 8-18. Toxicity of Hexazinone to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	1,200	EPA 1994
Rat	1,100	Du Pont 1998
Rat	1,690	Kennedy 1984
Guinea pig	860	Kennedy 1984
Beagle dog	>3,400	Kennedy 1984
Bobwhite quail	2,258	Kennedy 1984

Toxicity to Aquatic Species

Hexazinone is moderately toxic to aquatic species on an acute basis. Data are summarized in Table 8-19.

Table 8-19. Toxicity of Hexazinone to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	>320 (96-hr)	EPA 1994
Rainbow trout	401 (24-hr) 388 (48-hr) 320 to 420 (96-hr)	Kennedy 1984
Rainbow trout, juvenile	320 (24-hr) 286 (48-hr) 271 (72-hr) 257 (96-hr)	Wan et al. 1988
Coho salmon, juvenile	290 (24-hr) 282 (48-hr) 265 (72-hr) 246 (96-hr)	Wan et al. 1988
Chum salmon, juvenile	321 (24-hr) 288 (48-hr) 288 (72-hr) 285 (96-hr)	Wan et al. 1988
Chinook salmon, juvenile	394 (24-hr) 323 (48-hr) 318 (72-hr) 317 (96-hr)	Wan et al. 1988
Pink salmon, juvenile	309 (24-hr) 280 (48-hr) 280 (72-hr) 236 (96-hr)	Wan et al. 1988
Sockeye salmon, juvenile	363 (24-hr) 332 (48-hr) 318 (72-hr) 317 (96-hr)	Wan et al. 1988
Bluegill sunfish	505 (96-hr)	EPA 1994
Bluegill sunfish	425 (24-hr) 370 to 420 (48-hr) 370 to 420 (96-hr)	Kennedy 1984
Fathead minnow	453 (24-hr) 370 to 490 (48-hr) 274 (96-hr)	Kennedy 1984
Daphnia magna	152 (48-hr)	Kennedy 1984

In an early lifestage test using the fathead minnow, a NOEC of 17 mg/L was determined, with fish length affected at the LOEC of 35.5 mg/L (EPA 1994). No effects were found in bluegill sunfish exposed to 0.01 or 1 mg/L hexazinone for 4 weeks (Rhodes 1980).

In two life-cycle studies using *Daphnia magna*, MATCs of 48.5 mg/L and 20 to 50 mg/L were calculated, based on survival and reproduction endpoints, respectively (EPA 1994).

A 12-hour exposure to 2.7 mg/L hexazinone in stream channels resulted in no difference in invertebrate drift compared to controls, and no differences in macroinvertebrate community structure 14 days after exposure (Kreutzweiser et al. 1995). In an earlier test, an avoidance response by the mayfly *Isonychia* sp. was observed after a one-hour exposure to 80 mg/L, but all of the displaced organisms survived (Kreutzweiser et al. 1992). Drift of 12 other aquatic insect species and survival of all 13 tested aquatic insect species was unaffected.

A nine-day exposure to a hexazinone concentration of 100 mg/L had no effect on the hatching success of embryos, or on the mortality, ability to swim away when prodded, or total body length of tadpoles of leopard frogs and green frogs. Bullfrog tadpoles exhibited occasional decreased response to prodding, but recovered over the exposure duration (Berrill et al. 1994).

8.3.10 Horticultural Oil

Toxicity to Terrestrial Species

The oral LD_{50} in rats is >5,050 mg/kg (Riverside/Terra 1995).

An oral LD_{50} >2,250 mg/kg was determined for northern bobwhite (Wildlife International 1990). Christens et al. (1995) documented that spraying Canada goose eggs with white mineral oil as a proposed bird population control measure caused failure to hatch in all cases. The oil blocks the pores in the eggshell, asphyxiating the developing embryo.

Toxicity to Aquatic Species

No mortality or indications of toxicity were observed in 96-hour studies in which rainbow trout and bluegill sunfish, and juvenile rainbow trout, were exposed to horticultural oil at a concentration of 100 mg/L (Valent USA 1983a, Wildlife International 1991). Although no LC_{50}s were determined, the value of 100 mg/L is used as the toxicity data point for fish species in the risk assessment of horticultural oil, due to the lack of additional exposure-response information.

A 48-hour LC_{50} of 2.2 mg/L was determined for the aquatic invertebrate *Daphnia magna* (Valent USA 1983b).

8.3.11 Permethrin

Toxicity to Terrestrial Species

Permethrin is only slightly toxic to mammals and birds. Toxicity data for terrestrial species are summarized in Table 8-20.

Table 8-20. Toxicity of Permethrin to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	430	WHO 1990
Rat	1,030*	FMC 1995
Mouse	540	WHO 1990
Rabbit	>4,000	WHO 1990
Guinea pig	>4,000	WHO 1990
Chicken	>3,000	WHO 1990
Ring-necked pheasant	>13,500	WHO 1990
Japanese quail	>13,500	WHO 1990
Mallard duck	9,900	Braithwaite 1984

*Pounce 3.2EC formulation

A field study of the effects of two 17.5-g/ha (0.016 lb/acre) applications of permethrin, six days apart, to a forest stream ecosystem was conducted in Ontario (Kingsbury and McLeod 1979). No effects were observed on breeding songbirds and small mammals. However, there was a large knockdown of both target and non-target insects.

The five-day dietary LC_{50} for permethrin is >5,000 ppm in Japanese quail (Hill and Camardese 1986), >27,500 ppm in mallard ducks and ring-necked pheasants, and >38,000 ppm in starlings (WHO 1990).

Toxicity to Aquatic Species

Permethrin is highly to very highly toxic to aquatic species. Toxicity data are summarized in Table 8-21.

Anderson (1982) studied the effects of permethrin on aquatic invertebrates. The investigator noticed behavioral changes in caddisflies after exposures as short as 48 hours to 0.000064 mg/L.

A 21-day LC_{50} of 0.00017 mg/L was calculated for caddisflies. In a test using stoneflies, the insects were immobilized within five hours of exposure to 0.00021 mg/L. At the lowest concentration tested of 0.000029 mg/L, no effects were observed in stoneflies after 21 days; the LOEC was 0.000042 mg/L. Ibrahim et al. (1998) found a small but statistically significant decrease in acetylcholinesterase activity in chironomids exposed to 0.032 mg/L permethrin for 24 hours.

In a study of the effects of permethrin on zooplankton, a series of enclosures (limnocorrals) were placed in a lake in southern Ontario. Permethrin was applied to achieve concentrations of 0.0005, 0.005, and 0.050 mg/L. Results indicated that macrozooplankton (cladocerans and copepods) were more susceptible to permethrin than microzooplankton (rotifers), showing the

Table 8-21. Toxicity of Permethrin to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	0.135 (24-hr)	Coates & O'Donnell-Jeffrey 1979
Rainbow trout	0.008 (24-hr) 0.006 (48-hr)	Mulla et al. 1978
Rainbow trout	0.0070 (96-hr)	Holcombe et al. 1982
Rainbow trout	0.0043 to 0.0092 (24-hr) 0.0029 to 0.0082 (96-hr)	Mayer and Ellersieck 1986
Brook trout	0.00032 (96-hr)	Mayer and Ellersieck 1986
Largemouth bass	0.0085 (96-hr)	Jolly et al. 1978
Bluegill sunfish	0.0076 to 0.014 (24-hr) 0.0045 to 0.008 (96-hr)	Mayer and Ellersieck 1986
Fathead minnow	0.0156 (96-hr)	Holcombe et al. 1982
Fathead minnow	0.0057 (96-hr)	Mayer and Ellersieck 1986
Channel catfish	0.00110 (96-hr)	Jolly et al. 1978
Channel catfish	0.0072 (24-hr) 0.0072 (96-hr)	Mayer and Ellersieck 1986
Common carp	0.132 (48-hr)	Reddy et al. 1995
Mosquitofish	0.015 (96-hr)	Jolly et al. 1978
Mosquitofish	0.100 (24-hr) 0.097 (48-hr)	Mulla et al. 1978
Desert pupfish	0.007 (24-hr) 0.005 (48-hr)	Mulla et al. 1978
Tilapia	0.050 (24-hr) 0.044 (48-hr)	Mulla et al. 1978
Daphnia sp.	0.00126 (48-hr)	Mayer and Ellersieck 1986
Midge *Chironomus riparius*	0.0166 (24-hr)	Ibrahim et al. 1998
Crayfish, newly hatched	0.00039 (96-hr)	Jolly et al. 1978
Crayfish, juvenile	0.00062 (96-hr)	Jolly et al. 1978
Bullfrog tadpole	0.115 (96-hr)	Devillers and Exbrayat 1992
Bullfrog tadpole	7.033 (96-hr)	Jolly et al. 1978

effects of acute toxicity at all concentrations, while microzooplankton showed acute toxicity only at the high concentration. Initial direct toxicity, followed by the indirect effects of release from predator and competitive interactions, led to changes in relative abundance of the various species over time. In general, the investigators found that application of permethrin reduced the overall zooplankton diversity in the enclosures (Kaushik et al. 1985).

In a field study in Ontario, permethrin was applied aerially at a nominal rate of 70 g/ha to a small stream where caged native fish were placed (Kingsbury 1976). The actual deposition rate measured was 13.4 g/ha (0.012 lb/acre). This study reported little impact to caged or other native fish. Significant disturbance to aquatic insects was indicated by the high number of insects drifting downstream for more than a day after the application. No changes in populations of benthic fauna were found in samples taken. Significant numbers of terrestrial insects were knocked down by the permethrin treatment, fell into the stream, and were eaten by the fish. In the second part of this same study (Kingsbury and Kreutzweiser 1979), two applications of 17.5 g/ha (0.016 lb/acre) were made six days apart. The authors noted a significant reduction in aquatic insect populations, and longer recovery time after the second application. No fish mortality was observed, but the diet of the slimy sculpin shifted from various aquatic insects to midge larvae almost exclusively. The overall findings of these and other studies in the series (Kingsbury and Kreutzweiser 1987) indicated that, at application rates up to 70 g/ha (0.062 lb/acre), no lethality to fish was observed. However, trout and salmon diets were significantly changed due to effects on aquatic insects. These changes lasted from several months to over a year, depending upon the application rate. This effect is believed to have caused migration away from the treated areas in salmon nursery streams.

Berrill et al. (1993) evaluated the effect of permethrin on three amphibian species at two water temperatures. Exposure of embryos of the green frog *Rana clamitans* for 96 hours to concentrations up to 2 mg/L permethrin did not result in any concentration-related mortality; however, a concentration of 0.1 mg/L resulted in abnormal behavior and slowed growth for two to three weeks following exposure, and 1 mg/L was associated with a deformed tail. Newly hatched tadpoles exposed to the same concentrations for 96 hours showed no mortality, but again showed abnormal behavior and decreased growth at three weeks to the lowest concentration tested of 0.1 mg/L. No effects on hatching success or abnormalities were found when embryos of green frogs, wood frogs (*R. silvatica*), leopard frogs (*R. pipiens*), or American toads (*Bufo americanus*) were exposed to 0.01 or 0.1 mg/L for 22 hours. Mortality at metamorphosis occurred in green frog and toad tadpoles exposed to 0.05 mg/L for 22 hours. Sublethal effects were indicated by abnormal behaviors in green frog and leopard frog tadpoles, with significant recovery occurring by the end of the 11-day study period after exposure to 0.01 mg/L. Complete recovery was not accomplished after exposure to 0.1 mg/L by the end of the observation period in frog tadpoles held at 15 °C, while those at 20 °C recovered by day 8. Larvae of the spotted salamander *Ambystoma maculatum* were more sensitive, with little recovery evident by day 11 after exposure to 0.01 mg/L at 15 °C, although most had recovered by that time in the 20 °C test group.

8.3.12 Picloram

Toxicity to Terrestrial Species

Picloram is only slightly toxic to terrestrial species on an acute basis, as illustrated by the studies summarized in Table 8-22.

Table 8-22. Toxicity of Picloram to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	>5,000 (males) 4,012 (females)	EPA 1995
Mouse	2,000 to 4,000	Lynn 1965
Rabbit	2,000	Lynn 1965
Guinea pig	3,000	Lynn 1965
Sheep	>720	Jackson 1966
Cattle	>540	Jackson 1966
Cattle	>750	HSDB 2001
Mallard duck	>2,150	EPA 1995
Mallard duck	>1,935	EPA 1995
Mallard duck	>1,720*	Hudson et al. 1984
Bobwhite quail	>1,935	EPA 1995
Pheasant	>2,000	Hudson et al. 1984
Chicken	6,000	Lynn 1965

*Study conducted with potassium salt of picloram; converted to acid equivalent for comparability to other values by applying factor of 0.86 (EPA 1995).

No signs of toxicity were observed in a 33-day feeding study in sheep at a picloram dose of 18 mg/kg/day (Jackson 1966). In the same study, no adverse effects resulted from 30-day dosing with 72 mg/kg/day of Tordon® 22K (equivalent to 15 mg/kg/day picloram acid).

In bobwhite quail, Japanese quail, mallard ducks, and ring-necked pheasant, five-day feeding studies resulted in dietary LC_{50}s >5,000 ppm in all cases (HSDB 2001). In a two-week feeding study with Japanese quail, no effects were noted at the highest dietary concentration of 1,000 ppm (Lynn 1965).

Toxicity to Aquatic Species

Picloram is moderately toxic to fish and slightly toxic to aquatic invertebrates. Acute studies are summarized in Table 8-23.

In a 60-day early lifestage test using picloram, the NOEC was 0.55 mg/L and the LOEC was 0.88 mg/L, based on decreased weight and length in rainbow trout (Mayes et al. 1987). In a chronic exposure study, the lowest concentration tested of 0.035 mg/L was associated with decreased survival and growth of lake trout fry when exposed from 10 days before hatching to 60 days after hatching (Woodward 1976).

Table 8-23. Toxicity of Picloram to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	5.50	EPA 1995
Rainbow trout	11*	EPA 1995
Rainbow trout	3.1 to 17.0 (24-hr) 3.1 to 14 (96-hr)	Mayer and Ellersieck 1986
Rainbow trout	18 (96-hr)	Mayes and Dill 1984
Rainbow trout	15.6 (96-hr) 14.0 (8-day)	Mayes et al. 1987
Cutthroat trout	3.4 to 12.5 (24-hr) 1.5 to 8.6 (96-hr)	Mayer and Ellersieck 1986
Cutthroat trout	3.45 to 8.60 (96-hr) 1.475 (8-day)	Woodward 1976
Lake trout	1.55 to 4.95 (96-hr) 1.3 (12-day)	Woodward 1976
Lake trout	1.8 to 16.8 (24-hr) 1.6 to 16.8 (96-hr)	Mayer and Ellersieck 1986
Bluegill sunfish	14.5 to 19.4	EPA 1995
Bluegill sunfish	21*	EPA 1995
Bluegill sunfish	30 to >100 (24-hr) 13.5 to 33 (96-hr)	Mayer and Ellersieck 1986
Bluegill sunfish	51 (96-hr)	Mayes and Dill 1984
Fathead minnow	55.3 (96-hr) 75 (96-hr)*	Mayes and Dill 1984
Channel catfish	3.2 to 44 (24-hr) 1.4 to 22 (96-hr)	Mayer and Ellersieck 1986
Daphnia sp.	34.4	EPA 1995
Daphnia magna	76 (48-hr)	Mayer and Ellersieck 1986
Daphnia sp.	58.7*	EPA 1995
Daphnia magna	50.7 (48-hr) 79 (48-hr)*	Mayes and Dill 1984
Amphipod *Gammarus fasciatus*	50 (24-hr) 27 (96-hr)	Mayer and Ellersieck 1986
Amphipod *Gammarus pseudolimnaeus*	20 (24-hr) 16.5 (96-hr)	Mayer and Ellersieck 1986
Stonefly *Pteronarcys californica*	140 (24-hr) 48 (96-hr)	Mayer and Ellersieck 1986

Table 8-23. Toxicity of Picloram to Aquatic Species (continued)

Species	LC_{50} (mg/L)	Reference
Tusked frog *Adelotus brevis*	143 to 210 (24-hr) 123 to 182 (48-hr) 95 to 154 (96-hr)	Pauli et al. 2000
Brown-striped frog *Limnodynastes peronii*	120 (24-hr) 116 (48-hr) 105 (96-hr)	Pauli et al. 2000

*Study conducted with potassium salt of picloram; converted to acid equivalent for comparability to other values by applying factor of 0.86 (EPA 1995) if not converted in study report.

Simulated field runoff studies were conducted, with four 48-hour metered picloram applications to test aquaria containing cutthroat trout fry made over 25 days. The results demonstrate that concentrations ranging up to 0.290 mg/L over the test period did not affect growth and survival of cutthroat trout. Concentrations ranging up to 0.790 mg/L over the test period led to statistically significant decreases in fry weight and length. Concentrations ranging up to 1.6 mg/L decreased survival and growth (Woodward 1979).

A 21-day life-cycle test using picloram in *Daphnia magna* resulted in a NOEC of 11.8 mg/L and a LOEC of 18.1 mg/L, based on decreased reproduction endpoints (Gersich et al. 1985). The associated MATC is 14.6 mg/L.

8.3.13 Propargite

Toxicity to Terrestrial Species

Propargite is only slightly toxic to mammalian and avian species on an acute basis, as demonstrated by the results of the studies summarized in Table 8-24.

Avian feeding studies resulted in dietary LC_{50}s of 3,401 ppm for the northern bobwhite quail, and >4,640 for mallard ducks (EPA 2000). In reproduction studies, the dietary NOELs were 84.7 and 43.2 ppm for bobwhite and mallards, respectively. Both species exhibited reduction in eggs laid and reduced hatchling survival and weights at a dietary level of 288 ppm. In addition, mallard females showed reduced body weight at a level of 84.7 ppm (EPA 2000, EPA undated).

Table 8-24. Toxicity of Propargite to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	2,639	EPA 2000
Rat	1,480	Hayes and Laws 1991
Mallard duck	>4,640	EPA 2000

Toxicity to Aquatic Species

Propargite is highly toxic to aquatic species. Acute study data are summarized in Table 8-25.

Table 8-25. Toxicity of Propargite to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	0.118 to 0.143 (96-hr)	EPA 2000
Bluegill sunfish	0.168 (96-hr)	Uniroyal 1998
Catfish	0.04	Uniroyal 1998
Minnow	0.06	Uniroyal 1998
Daphnia magna	0.074 to 0.091 (48-hr)	EPA 2000

A chronic test in fathead minnows showed that propargite affected growth, survival, and reproduction parameters at a concentration 0.028 mg/L; the NOEC was 0.016 mg/L (EPA 2000).

In a life-cycle test in *Daphnia magna*, reproduction was affected at 0.014 mg/L, with a NOEC of 0.009 mg/L (EPA 2000).

EPA (2000) stated, "Based on the high toxicity of propargite to fish, propargite is also expected to demonstrate high toxicity to amphibians, particular to early life stages that are primarily aquatic and where respiration is dependent on gills (such as tadpoles) or where later adult stages retain external gill structures (primitive salamanders). Amphibians often inhabit shallow littoral areas where incoming runoff concentrations may be the highest."

8.3.14 Triclopyr

Toxicity to Terrestrial Species

Triclopyr is slightly to moderately toxic to mammals and avian species. Toxicity values for triclopyr to terrestrial species are given in Table 8-26.

Ponies exposed to four daily doses of 60 mg/kg of triclopyr acid exhibited no adverse effects; however, exposure to four daily doses of 300 mg/kg caused depression, recumbency (lying down), decreased gastrointestinal activity, and respiratory and muscular distress (Osweiler 1983).

Triclopyr acid feeding studies in bird species resulted in dietary LC_{50} values of 2,934 ppm in bobwhite quail, 3,272 ppm in Japanese quail, and 5,620 ppm in mallard ducks (EPA 1998). A 64.7% formulation of triclopyr triethylamine salt produced dietary LC_{50}s of 11,622 and >10,000 ppm in bobwhite quail and mallard ducks, respectively (EPA 1998). Studies using the butoxyethyl ester of triclopyr resulted in dietary LC_{50}s of 5,401 ppm in bobwhite quail and >10,000 ppm in mallard ducks (EPA 1998).

Table 8-26. Toxicity of Triclopyr to Terrestrial Species

Species	Material*	LD_{50} (mg/kg)	Reference
Rat	acid	729 (males) 630 (females)	EPA 1998
Rat	BEE	803	EPA 1998
Mouse	acid	471	EPA 1989
Rabbit	acid	550	WSSA 1989
Guinea pig	acid	310	WSSA 1989
Northern bobwhite	BEE	735 to 849	EPA 1998
Mallard duck	acid	1,698	Kenaga 1979
Mallard duck	TEA	2,055	EPA 1998

*acid = triclopyr acid; TEA = triclopyr triethylamine salt; BEE = triclopyr butoxyethyl ester

The 8-day dietary LC_{50} for the butoxyethyl ester was determined to be 1,923 ppm in zebra finches (Holmes et al. 1994). Exposure of zebra finches to the ester in the diet for 29 days had no significant effect at a concentration of 150 ppm, but caused decreased body weight and food consumption at a level of 500 ppm.

In avian reproduction studies using triclopyr acid, no effects were seen at the highest dietary concentration of 500 ppm for bobwhite quail, but there was a decrease in the number of 14-day survivors in mallard ducks at a dietary level of 200 ppm; the NOEL was 100 ppm (EPA 1998).

Toxicity to Aquatic Species

The toxicity of triclopyr to aquatic species is summarized in Table 8-27. The acid and triethylamine salt exhibit moderate to low toxicity to aquatic species. However, the butoxyethyl ester, which is proposed for use at Provolt, is moderately to highly toxic.

An assay using early life-stages of fathead minnows exposed to the triethylamine salt of triclopyr produced a NOEC of 72.7 mg/L and a LOEC of 114 mg/L, based on decreased survival (Mayes et al. 1984). The corresponding MATC is 91 mg/L.

The MATC for daphnia exposed to the triethylamine salt of triclopyr was reported as 110 mg/L using a reproductive endpoint, with a NOEC and LOEC of 80.7 and 149 mg/L, respectively, for effects on total young and mean brood size (Gersich et al. 1984).

In 8-day studies, Berrill et al. (1994) reported that triclopyr ester exposure of 4.8 mg/L did not affect the hatching success of embryos of green frogs, leopard frogs, or bullfrogs. Newly hatched tadpoles of these species exhibited behavioral effects at a concentration of 1.2 mg/L. At a concentration of 2.4 mg/L, all green frog and bullfrog tadpoles died. Pauli et al. (2000) reported the results of a study in which the Garlon® 3A (triethylamine salt) formulation had no significant effects at a concentration of 100 mg/L in a test using embryos of the clawed toad.

Table 8-27. Toxicity of Triclopyr to Aquatic Species

Species	Material	LC_{50} (mg/L)	Reference
Rainbow trout	acid	117 (96-hr)	EPA 1998
Rainbow trout	64.7% TEA	613	EPA 1998
Rainbow trout	BEE	0.65	EPA 1998
Rainbow trout	BEE**	22.5 (1-hr) 1.95 (6-hr) 0.79 (24-hr)	Kreutzweiser et al. 1994
Chinook salmon	BEE**	34.6 (1-hr) 4.7 (6-hr) 1.76 (24-hr)	Kreutzweiser et al. 1994
Coho salmon	BEE	0.26 (96-hr) (alevin) 1.3 (96-hr) (juvenile)	Mayes et al. 1986
Bluegill sunfish	acid	148 (96-hr)	EPA 1998
Bluegill sunfish	64.7% TEA	893	EPA 1998
Bluegill sunfish	BEE	0.36	EPA 1998
Fathead minnow	64.7% TEA	947	EPA 1998
Fathead minnow	TEA	120 (96-hr) 101 (8-day)	Mayes et al. 1984
Fathead minnow	BEE	2.4 (24-hr)	EPA 1998
Daphnia magna	acid	133 (48-hr)	Kenaga 1979
Daphnia magna	TEA	1,170 (48-hr) 1,140 (21-day)	Gersich et al. 1984
Daphnia magna	BEE	1.7 to 12	EPA 1998
Mayfly *Isonychia*	BEE**	37.0 (9-hr) 8.8 (24-hr)	Kreutzweiser et al. 1994
Caddisfly *Hydropsyche*	BEE**	14.9 (9-hr) 4.0 (24-hr)	Kreutzweiser et al. 1994
Clawed toad *Xenopus laevis*, embryo	TEA**	162.5	Perkins et al. 2000
Clawed toad *Xenopus laevis*, embryo	BEE**	9.3	Perkins et al. 2000

*acid = triclopyr acid; TEA = triclopyr triethylamine salt; BEE= triclopyr butoxyethyl ester
**expressed as equivalent acid concentration

8.3.15 Other Ingredients

The following paragraphs present the ecotoxicity hazard analysis for the List 2 other ("inert") ingredients in the seed orchard pesticide formulations.

Cyclohexanone

Cyclohexanone is only slightly toxic to mammals, with acute oral LD_{50}s of 1,180 mg/kg in rats, and 2,070 (male) and 2,110 (female) mg/kg in mice (Gupta et al. 1979).

Table 8-28 lists acute LC_{50}s for aquatic species.

Table 8-28. Toxicity of Cyclohexanone to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Fathead minnow	527 (96-hr)	HSDB 2001
Fathead minnow	481 to 770 (96-hr)	EPA 2001
Daphnia magna	820 (24-hr)	EPA 2001
Daphnia magna	800 (24-hr)	EPA 2001

A 48-hour exposure to a cyclohexanone concentration of 757 mg/L was lethal to all rainbow trout tested, whereas no mortality was observed at a concentration of 30.3 mg/L (EPA 2001). Behavioral effects were noted in rainbow trout and bluegill sunfish at a concentration of 5 mg/L for 24 hours (EPA 2001).

No mortality was observed in *Daphnia magna* after a 24-hour exposure to 526 mg/L. Exposure for the same duration to 1,240 mg/L caused complete lethality (EPA 2001).

Ethylbenzene

Ethylbenzene is slightly toxic to mammals, with reported oral LD_{50}s in rats of 5,460 mg/kg (HSDB 2001) and 3,500 mg/kg (Von Burg 1992).

Ethylbenzene is moderately toxic to aquatic species. Aquatic species toxicity values are summarized in Table 8-29.

Table 8-29. Toxicity of Ethylbenzene to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	14 (24-hr)	Mayer and Ellersieck 1986
Rainbow trout	4.2 (96-hr)	WHO 1996
Bluegill sunfish	32 (96-hr)	Von Burg 1992
Bluegill sunfish	100 (24-hr) 84 (96-hr)	Mayer and Ellersieck 1986
Goldfish	94.44 (96-hr)	Von Burg 1992
Channel catfish	210 (24-hr)	Mayer and Ellersieck 1986
Fathead minnow	12.1 (96-hr)	HSDB 2001
Fathead minnow	42.3 to 48.5 (96-hr)	HSDB 2001
Guppy	97.1 (96-hr)	Von Burg 1992
Daphnia sp.	1.8 (48-hr)	WHO 1996

Light Aromatic Solvent Naphtha

Light aromatic solvent naphtha is slightly toxic to terrestrial species, as illustrated by the data summarized in Table 8-30.

Table 8-30. Toxicity of Light Aromatic Solvent Naphtha (as Naphthalene) to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	2,200 to 2,600	HSDB 2001
Mouse	533 (male) 710 (female)	HSDB 2001
Bobwhite quail	2,690	EPA 2001

The 8-day dietary LC_{50} for naphthalene in bobwhite quail was >5,620 ppm (EPA 2001).

Table 8-31 summarizes the aquatic species toxicity values identified for naphthalene.

EPA (2001) reported the NOEC and LOEC for mortality of naphthalene to coho salmon as 1.8 and 3.2 mg/L, respectively.

In clawed toads, six hours of exposure to naphthalene resulted in a behavioral EC_{50} of 1.7 mg/L (EPA 2001).

Table 8-31. Toxicity of Light Aromatic Solvent Naphtha (as Naphthalene) to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	1.6 to 5.5 (96-hr) 0.12 (23-day) 0.110 (27-day)	EPA 2001
Coho salmon, fry	3.2 (96-hr)	Eisler 1987
Mosquitofish	150 (96-hr)	Eisler 1987
Fathead minnow	7.76 (24-hr) 6.35 (48-hr) 6.08 (72-hr) 1.99 to 7.90 (96-hr)	EPA 2001
Daphnia magna	6.6 to 17.0 (24-hr) 2.16 to 22.6 (48-hr)	EPA 2001
Midge *Chironomus attenuatus*	13.0 to 13.9 (24-hr) 2.81 (48-hr)	EPA 2001
Clawed toad	2.1 (96-hr)	EPA 2001

Xylene

Xylene is slightly toxic to mammals, as summarized in Table 8-32.

Table 8-32. Toxicity of Xylene to Terrestrial Species

Species	LD_{50} (mg/kg)	Reference
Rat	4,300	HSDB 2001
Rat	3,523 to 8,600	HSDB 2001
Mouse	1,590	HSDB 2001
Mouse	5,251 (female) 5,627 (male)	HSDB 2001

Xylene's 5-day dietary LC_{50} for Japanese quail was >20,000 ppm (Hill and Camardese 1986).

Xylene is slightly to moderately toxic to aquatic species. The toxicity values reported for aquatic species are listed in Table 8-33.

Table 8-33. Toxicity of Xylene to Aquatic Species

Species	LC_{50} (mg/L)	Reference
Rainbow trout	8.3 to 13.5 (24-hr) 8.2 (96-hr)	Mayer and Ellersieck 1986
Bluegill sunfish	14 (24-hr) 13.5 (96-hr)	Mayer and Ellersieck 1986
Striped bass	2.0 to 11 (96-hr)	Verschueren 1983
Goldfish	13 to 18 (96-hr)	Verschueren 1983
Fathead minnow	26.7 to 42 (24- to 96-hr)	Verschueren 1983
Daphnia magna	75.5 (24-hr)	Calleja et al. 1994
Daphnia magna	100 to 1,000 (24-hr)	Verschueren 1983
Rotifer *Brachionus calyciflorus*	253 (24-hr)	Ferrando and Andreu-Moliner 1992
Clawed toad	73 (48-hr)	Devillers and Exbrayat 1992

8.3.16 Fertilizers

Fertilizers proposed for use at Provolt are ammonium nitrate, ammonium phosphate, ammonium sulfate, and potassium nitrate. The following paragraphs provide information on the ecotoxicity of these fertilizers.

Ammonium Nitrate

In water, ammonium nitrate degrades to form ammonium and nitrate ions. In addition, ammonia is oxidized to nitrate by algae and bacteria. In water, the ammonium ion can exist in its ionized form (NH_4^+), and in its un-ionized form as ammonia (NH_3). The equilibrium between these two forms is largely dependent on pH and temperature. Ammonia demonstrates greater toxicity to aquatic species than does the ammonium ion, and this toxicity increases with decreases in pH and temperature.

Acute toxicity values for ammonium nitrate, ammonia, and nitrate are summarized in Table 8-34.

Ruminant animals such as cows have been affected, and sometimes killed, by over-exposure to ammonium nitrate fertilizer, with several incidents reported in the open literature (Horner 1982, Jones 1982, Bruning-Fann and Kaneene 1993).

Westin (1974, as cited in Norris et al. 1991) reported a median tolerance limit for nitrate of 5,800 mg/L for chinook salmon fingerlings, and 6,000 mg/L for rainbow trout fingerlings.

Table 8-34. Acute Toxicity of Ammonium Nitrate, Ammonia, and Nitrate

Species	Endpoint	Result	Reference
Ammonium nitrate			
Rat	oral LD_{50}	4,500 mg/kg	HSDB 2001
Mouse	oral LD_{50}	2,085 mg/kg	Nechkina 1992
Pacific tree frog, tadpole	96-hr LC_{50} 10-day LC_{50}	774 mg/L 315 mg/L	Schuytema and Nebeker 1999a
Pacific tree frog, embryo	96-hr LC_{50} 10-day LC_{50}	23.4 mg/L 14.3 mg/L	Schuytema and Nebeker 1999b
Clawed toad, tadpole	96-hr LC_{50} 10-day LC_{50}	575 mg/L 302 mg/L	Schuytema and Nebeker 1999a
Clawed toad, embryo	5-day LC_{50}	250 mg/L	Schuytema and Nebeker 1999b
Common toad, tadpole	96-hour LC_{50} 7-day LC_{50}	2,199 mg/L 2,112 mg/L	Xu and Oldham 1997
Red-legged frog, embryo	16-day LC_{50}	411 mg/L	Schuytema and Nebeker 1999c
Western chorus frog, tadpole	96-hr LC_{50}	17 mg/L	Pauli et al. 2000
Green frog, tadpole	96-hr LC_{50}	32.4 mg/L	Pauli et al. 2000
Northern leopard frog, tadpole	96-hr LC_{50}	22.6 mg/L	Pauli et al. 2000
American toad, tadpole	96-hr LC_{50}	39.3 mg/L	Pauli et al. 2000
Ammonia (as NH_3)			
Rainbow trout	96-hr LC_{50}	0.53 mg/L	Arthur et al. 1987
Atlantic salmon, parr	96-hr LC_{50}	0.037 to 0.178 mg/L	Knoph 1992
Channel catfish	96-hr LC_{50}	0.86 mg/L	Arthur et al. 1987
Fathead minnow	96-hr LC_{50}	2.17 mg/L	Arthur et al. 1987
Daphnia magna	48-hr LC_{50}	3.57 mg/L	Gersich and Hopkins 1986
Fingernail clam	96-hr LC_{50}	1.10 mg/L	Arthur et al. 1987
Caddisfly	96-hr LC_{50}	10.1 mg/L	Arthur et al. 1987
Crayfish	96-hr LC_{50}	18.3	Arthur et al. 1987
Nitrate (as NO_3)			
Cattle	LD_{50}	1,468 mg/kg	Bruning-Fann and Kaneene 1993
Rainbow trout, egg and fry	LC_{46}	10.2 mg/L	Rouse et al. 1999
Cutthroat trout, egg and fry	LC_{41}	19.9 mg/L	Rouse et al. 1999
Caddisfly, larvae	96-hr LC_{50}	431 to 502 mg/L	Rouse et al. 1999

Schuytema and Nebeker (1999a) identified a 10-day NOEC and LOEC for ammonium nitrate in Pacific treefrog tadpoles of 141 and 280 mg/L, respectively, based on decreased length and weight; corresponding values for the clawed toad were 280 and 569 mg/L. In a follow-on study of toxicity to the embryos of the same species, Schuytema and Nebeker (1999b) identified a 10-day NOEC and LOEC in Pacific treefrog embryos of 19 and 39 mg/L, and a 5-day NOEC and LOEC in clawed toad embryos of 19 and 39 mg/L. In an additional study, Schuytema and Nebeker (1999c) identified a 16-day ammonium nitrate NOEC and LOEC for embryos of the red-legged frog as 36.6 and 75.4 mg/L, respectively.

Xu and Oldham (1997) studied ammonium nitrate exposures to tadpoles of the common toad *Bufo bufo*. A concentration of 100 mg/L for up to 72 hours decreased the activity of the tadpoles, but did not affect food consumption or delay development. Exposure to 50 mg/L for 15 days resulted in significantly *increased* length at metamorphosis compared to controls; this was not interpreted as an adverse effect. Exposure to 100 mg/L for 30 days resulted in 21% mortality and 17% failure to resorb tails at metamorphosis. No adverse effects were reported for exposure of smooth newt (*Triturus vulgaris*) larvae to ammonium nitrate at a concentration of 50 mg/L for up to 72 hours (Watt and Oldham 1995). Feeding rate increased at 100 mg/L, and exposure to 200 mg/L for 24 hours led to decreased size at metamorphosis.

In an experiment designed to replicate field conditions, application of ammonium nitrate to damp soil at a rate of 6.2 g/m^2 (55.3 lb/acre) led to signs of toxicity in exposed adult common frogs (*Rana temporaria*) in as little as 60 minutes (Oldham et al. 1997). In associated field studies, the investigators found that application at a rate of 10.8 g/m^2 (96.4 lb/acre) to a wheat field and 19.9 g/m^2 (178 lb/acre) to grass caused acute toxicity symptoms within 5 and 24 minutes, respectively. The authors also noted that there was no evidence of a toxic effect once the fertilizer granules had dissolved, in one to two hours after application.

Ammonia. EPA (1999) recommended ambient water quality criteria for ammonia for protection of freshwater aquatic life, both for the presence and absence of salmonids (1-hour average) and early life stages of fish (30-day average). These criteria are dependent on site-specific pH, as follows:

- One-hour average (mg N/L), salmonids present: $\frac{0.275}{1+10^{7.204-pH}}+\frac{39.0}{1+10^{pH-7.204}}$
- One-hour average (mg N/L), salmonids absent: $\frac{0.411}{1+10^{7.204-pH}}+\frac{58.4}{1+10^{pH-7.204}}$
- 30-day average (mg N/L), early life stages present, where T = temperature (°C):

$$\frac{0.0557}{1+10^{7.688-pH}}+\frac{2.487}{1+10^{pH-7.688}} \times MIN\left(2.85, 1.45\times 10^{0.028\times(25-T)}\right)$$

- 30-day average (mg N/L), early life stages absent, where T = temperature (°C):

$$\left(\frac{0.0557}{1+10^{7.688-pH}}+\frac{2.487}{1+10^{pH-7.688}}\right)\times 1.45\times 10^{0.028\times(25-MAX(T,7))}$$

Ammonium Sulfate

Acute toxicity data for ammonium sulfate are summarized in Table 8-35.

Table 8-35. Acute Toxicity of Ammonium Sulfate

Species	Endpoint	Result	Reference
Rat	oral LD_{50}	3,000 mg/kg	NIOSH 1987
Catfish	24-hr LC_{50} 48-hr LC_{50} 72-hr LC_{50} 96-hr LC_{50}	9,414 mg/L 4,781 mg/L 4,469 mg/L 3,748 mg/L	Banerjee 1993
Daphnia magna	24-hr LC_{50}	423 mg/L	HSDB 2001
Freshwater snail *Helisoma trivolvis*	24-hr LC_{50}	558 mg/L (eggs) 393 mg/L (juveniles) 701 mg/L (adults)	HSDB 2001
Freshwater snail *Biomphalaria havanensis*	24-hr LC_{50}	669 mg/L (eggs) 526 mg/L (juveniles) 657 mg/L (adults)	HSDB 2001
Pacific treefrog, tadpoles	96-hr LC_{50} 10-day LC_{50}	1,088 mg/L 846 mg/L	Schuytema and Nebeker 1999a
Pacific treefrog, embryos	96-hr LC_{50} 10-day LC_{50}	>971 mg/L 306 mg/L	Schuytema and Nebeker 1999b
Clawed toad, tadpoles	96-hr LC_{50} 10-day LC_{50}	575 mg/L 302 mg/L	Schuytema and Nebeker 1999a
Clawed toad, embryos	5-day LC_{50}	259 mg/L	Schuytema and Nebeker 1999b

Oral ammonium sulfate doses of 40,000 and 150,000 mg were lethal to a heifer and a cow, respectively (HSDB 2001). An incident was reported in which accidental ammonium sulfate ingestion was fatal to dairy cows (HSDB 2001).

Schuytema and Nebeker (1999a) identified a 10-day NOEC and LOEC for ammonium sulfate in Pacific treefrog tadpoles of 116 and 232 mg/L, respectively, based on decreased length; no adverse effects on length or weight were observed in the clawed toad at the highest concentration tested of 939 mg/L. In a follow-up study, the same investigators (Schuytema and Nebeker 1999b) identified a 10-day NOEC and LOEC in Pacific treefrog embryos of 58 and 110 mg/L, and a 5-day NOEC and LOEC in clawed toad embryos of 24 and 58 mg/L.

A sulfate concentration of 200 mg/L was reported to be lethal to bluegill exposed for 180 days (EPA 2001). The ammonia data reported under "Ammonium Nitrate" are also relevant to the ecotoxicity of ammonium sulfate.

Ammonium Phosphate

Phosphate in aquatic systems can contribute to eutrophication if phosphorus is a limiting nutrient in the system. The effects of the ammonium component of mono- and diammonium phosphate are addressed under "Ammonium Nitrate." Acute toxicity data reported for mono- and diammonium phosphate are presented in Table 8-36.

Table 8-36. Acute Toxicity of Monoammonium Phosphate and Diammonium Phosphate

Species	Endpoint	Result	Reference
Monoammonium Phosphate			
Rat	oral LD_{50}	5,750 mg/kg	Monsanto 1991a
Diammonium Phosphate			
Rat	oral LD_{50}	6,500 mg/kg	Monsanto 1991b
Rainbow trout, juvenile	24- to 96-hr LC_{50}s	93 to 283 mg/L*	Blahm and Snyder 1973
Rainbow trout	96-hr LC_{50}	160 to 230 mg/L	HSDB 2001
Coho salmon	96-hr LC_{50}	245 to 320 mg/L	HSDB 2001
Fathead minnow	24-hr LC_{50} 48-hr LC_{50} 72-hr LC_{50} 96-hr LC_{50}	225 mg/L 169 mg/L 155 mg/L 155 mg/L	Inman 1974

*89% fire retardant formulation of diammonium phosphate

Potassium Nitrate

Potassium nitrate quickly breaks down to potassium and nitrate in the environment. In its intact form, a dose of 1,000 mg/kg is toxic to horses and lethal to sheep (HSDB 2001). Aquatic species LC_{50}s for potassium nitrate are 760 to 2,100 mg/L (24-hr) and 420 to 1,200 mg/L (96-hr) in bluegill sunfish; 58.5 to 162 mg/L (24-hr) and 22.5 to 62 mg/L (96-hr) in western mosquitofish; and 68 to 190 mg/L (48-hr) in *Daphnia magna* (EPA 2001).

In tests summarized by EPA (2001), potassium produced LC_{50}s in the long fingernail clam *Musculium transversum* of 518 to 2,700 mg/L for 48 hours and 185 to 530 mg/L for 96 hours. In the scud *Gammarus lacustris*, the 96-hour LC_{50} was 53.2 mg/L (EPA 2001). Toxicity associated with the nitrate component is described under "Ammonium Nitrate," above.

8.3.17 Aquatic Species LC_{50}s Used in Risk Assessment

The stream concentrations estimated by the fate and transport modeling represent 24-hour average concentrations. In cases in which the most species-appropriate LC_{50} resulted from a study that was other than 24 hours in duration, the reported LC_{50} was adjusted in a linear fashion, as described in Suter (1993), to provide better comparability between the toxicity and exposure data. Table 8-39 summarizes the LC_{50}s used.

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Acephate	Rainbow trout	Rainbow trout	895	24	895
	Daphnia magna	Daphnia magna	1.3/0.75=1.73	48	3.47
	Pacific chorus frog tadpole	Green frog tadpole	6433	24	6433
	Coho salmon	Rainbow trout	895	24	895
	Chinook salmon	Rainbow trout	895	24	895
	Cutthroat trout	Rainbow trout	895	24	895
	Steelhead	Rainbow trout	895	24	895
	Pacific lamprey	Rainbow trout	895	24	895
Chlorpyrifos	Rainbow trout	Rainbow trout	0.003	96	0.012
	Daphnia magna	Daphnia magna	0.0001	48	0.0002
	Pacific chorus frog tadpole	Leopard frog tadpole	3	24	3
	Coho salmon	Rainbow trout	0.003	96	0.012
	Chinook salmon	Rainbow trout	0.003	96	0.012
	Cutthroat trout	Cutthroat trout	0.0134	96	0.0536
	Steelhead	Rainbow trout	0.003	96	0.012
	Pacific lamprey	Rainbow trout	0.003	96	0.012
Diazinon	Rainbow trout	Rainbow trout	0.09	96	0.36
	Daphnia magna	Daphnia	0.0008	48	0.0016
	Pacific chorus frog tadpole	Green frog	<0.05	96	0.2
	Coho salmon	Rainbow trout	0.09	96	0.36
	Chinook salmon	Rainbow trout	0.09	96	0.36
	Cutthroat trout	Cutthroat trout	1.7	96	6.8
	Steelhead	Rainbow trout	0.09	96	0.36
	Pacific lamprey	Rainbow trout	0.09	96	0.36
Dimethoate	Rainbow trout	Rainbow trout	6.2	96	24.8
	Daphnia magna	Daphnia	3.32	48	6.64

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Dimethoate	Pacific chorus frog tadpole	Indian bullfrog larvae	5	96	20
	Coho salmon	Rainbow trout	6.2	96	24.8
	Chinook salmon	Rainbow trout	6.2	96	24.8
	Cutthroat trout	Rainbow trout	6.2	96	24.8
	Steelhead	Rainbow trout	6.2	96	24.8
	Pacific lamprey	Rainbow trout	6.2	96	24.8
Esfenvalerate	Rainbow trout	Rainbow trout	0.00026	96	0.00104
	Daphnia magna	Daphnia	0.00027	48	0.00054
	Pacific chorus frog tadpole	Leopard frog tadpole	0.00729	96	0.02916
	Coho salmon	Steelhead trout, juvenile	0.000088	96	0.000352
	Chinook salmon	Steelhead trout, juvenile	0.000088	96	0.000352
	Cutthroat trout	Steelhead trout, juvenile	0.000088	96	0.000352
	Steelhead	Steelhead trout, juvenile	0.000088	96	0.000352
	Pacific lamprey	Steelhead trout, juvenile	0.000088	96	0.000352
Horticultural oil and petroleum distillates	Rainbow trout	Rainbow trout, juvenile	100	96	400
	Daphnia magna	Daphnia magna	2.2	48	4.4
	Pacific chorus frog tadpole	Daphnia magna	2.2	48	4.4
	Coho salmon	Rainbow trout, juvenile	100	96	400
	Chinook salmon	Rainbow trout, juvenile	100	96	400
	Cutthroat trout	Rainbow trout, juvenile	100	96	400

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Horticultural oil and petroleum distillates	Steelhead	Rainbow trout, juvenile	100	96	400
	Pacific lamprey	Rainbow trout, juvenile	100	96	400
Permethrin	Rainbow trout	Rainbow trout	0.0043	24	0.0043
	Daphnia magna	Daphnia	0.00126	48	0.00252
	Pacific chorus frog tadpole	Bullfrog tadpole	0.115	96	0.46
	Coho salmon	Rainbow trout	0.0043	24	0.0043
	Chinook salmon	Rainbow trout	0.0043	24	0.0043
	Cutthroat trout	Rainbow trout	0.0043	24	0.0043
	Steelhead	Rainbow trout	0.0043	24	0.0043
	Pacific lamprey	Rainbow trout	0.0043	24	0.0043
Propargite	Rainbow trout	Rainbow trout	0.118	96	0.472
	Daphnia magna	Daphnia magna	0.074	48	0.148
	Pacific chorus frog tadpole	Daphnia magna	0.074	48	0.148
	Coho salmon	Rainbow trout	0.118	96	0.472
	Chinook salmon	Rainbow trout	0.118	96	0.472
	Cutthroat trout	Rainbow trout	0.118	96	0.472
	Steelhead	Rainbow trout	0.118	96	0.472
	Pacific lamprey	Rainbow trout	0.118	96	0.472
Chlorothalonil	Rainbow trout	Rainbow trout	0.0423	96	0.169
	Daphnia magna	Daphnia magna	0.068	48	0.136
	Pacific chorus frog tadpole	Frog	0.16	48	0.32
	Coho salmon	Rainbow trout	0.0423	96	0.169
	Chinook salmon	Rainbow trout	0.0423	96	0.169
	Cutthroat trout	Rainbow trout	0.0423	96	0.169
	Steelhead	Rainbow trout	0.0423	96	0.169

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Chlorothalonil	Pacific lamprey	Rainbow trout	0.0423	96	0.169
Dicamba	Rainbow trout	Rainbow trout	35	24	35
	Daphnia magna	Daphnia	1600	48	3200
	Pacific chorus frog tadpole	Striped marsh frog	205	24	205
	Coho salmon	Rainbow trout	35	24	35
	Chinook salmon	Rainbow trout	35	24	35
	Cutthroat trout	Cutthroat trout	50	96	200
	Steelhead	Rainbow trout	35	24	35
	Pacific lamprey	Rainbow trout	35	24	35
Glyphosate (Rodeo)	Rainbow trout	Rainbow trout	1000	96	4000
	Daphnia magna	Daphnia sp.	930	48	1860
	Pacific chorus frog tadpole	Banjo frog	400/0.538 = 743	48	1487
	Coho salmon	Coho salmon	36/0.538 = 66.9	96	268
	Chinook salmon	Chinook salmon	30/0.538 = 55.8	96	223
	Cutthroat trout	Chinook salmon	30/0.538 = 55.8	96	223
	Steelhead	Chinook salmon	30/0.538 = 55.8	96	223
	Pacific lamprey	Chinook salmon	30/0.538 = 55.8	96	223
Hexazinone	Rainbow trout	Rainbow trout	320	24	320
	Daphnia magna	Daphnia magna	152	48	304
	Pacific chorus frog tadpole	Daphnia magna	152	48	304
	Coho salmon	Coho salmon, juvenile	290	24	290

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Hexazinone	Chinook salmon	Coho salmon, juvenile	290	24	290
	Cutthroat trout	Coho salmon, juvenile	290	24	290
	Steelhead	Coho salmon, juvenile	290	24	290
	Pacific lamprey	Coho salmon, juvenile	290	24	290
Picloram	Rainbow trout	Rainbow trout	3.1	24	3.1
	Daphnia magna	Daphnia	34.4	48	68.8
	Pacific chorus frog tadpole	Brown striped frog	120	24	120
	Coho salmon	Rainbow trout	3.1	24	3.1
	Chinook salmon	Rainbow trout	3.1	24	3.1
	Cutthroat trout	Rainbow trout	3.1	24	3.1
	Steelhead	Rainbow trout	3.1	24	3.1
	Pacific lamprey	Rainbow trout	3.1	24	3.1
Triclopyr butoxyethyl ester	Rainbow trout	Rainbow trout	0.65	24	0.65
	Daphnia magna	Daphnia magna	1.7	48	3.4
	Pacific chorus frog tadpole	Clawed toad embryo	6.69	24	6.69
	Coho salmon	Coho salmon	0.26	96	1.04
	Chinook salmon	Chinook salmon	1.3	96	5.2
	Cutthroat trout	Rainbow trout	0.65	24	0.65
	Steelhead	Rainbow trout	0.65	24	0.65
	Pacific lamprey	Rainbow trout	0.65	24	0.65
Cyclohexanone	Rainbow trout	Fathead minnow	481	96	1924
	Daphnia magna	Daphnia magna	800	24	800
	Pacific chorus frog tadpole	Fathead minnow	481	96	1924

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Cyclohexanone	Coho salmon	Fathead minnow	481	96	1924
	Chinook salmon	Fathead minnow	481	96	1924
	Cutthroat trout	Fathead minnow	481	96	1924
	Steelhead	Fathead minnow	481	96	1924
	Pacific lamprey	Fathead minnow	481	96	1924
Ethylbenzene	Rainbow trout	Rainbow trout	14	24	14
	Daphnia magna	Daphnia	1.8	48	3.6
	Pacific chorus frog tadpole	Daphnia	1.8	48	3.6
	Coho salmon	Rainbow trout	14	24	14
	Chinook salmon	Rainbow trout	14	24	14
	Cutthroat trout	Rainbow trout	14	24	14
	Steelhead	Rainbow trout	14	24	14
	Pacific lamprey	Rainbow trout	14	24	14
Light aromatic solvent naphtha	Rainbow trout	Rainbow trout	1.6	96	6.4
	Daphnia magna	Daphnia magna	6.6	24	6.6
	Pacific chorus frog tadpole	Clawed toad	2.1	96	8.4
	Coho salmon	Coho salmon fry	3.2	96	12.8
	Chinook salmon	Rainbow trout	1.6	96	6.4
	Cutthroat trout	Rainbow trout	1.6	96	6.4
	Steelhead	Rainbow trout	1.6	96	6.4
	Pacific lamprey	Rainbow trout	1.6	96	6.4
Xylene	Rainbow trout	Rainbow trout	8.3	24	8.3
	Daphnia magna	Daphnia magna	75.5	24	75.5
	Pacific chorus frog tadpole	Clawed toad	73	48	146
	Coho salmon	Rainbow trout	8.3	24	8.3

Table 8-37. Aquatic Species LC_{50}s Used in Risk Assessment (continued)

Chemical	Representative Species	Study Species	Study LC_{50} (mg/L)	Duration (hours)	Adjusted LC_{50} (mg/L)
Xylene	Chinook salmon	Rainbow trout	8.3	24	8.3
	Cutthroat trout	Rainbow trout	8.3	24	8.3
	Steelhead	Rainbow trout	8.3	24	8.3
	Pacific lamprey	Rainbow trout	8.3	24	8.3
Ammonia	Rainbow trout	Rainbow trout	0.53	96	2.12
	Daphnia magna	Daphnia magna	3.57	48	7.14
	Pacific chorus frog tadpole	Daphnia magna	3.57	48	7.14
	Coho salmon	Rainbow trout	0.53	96	2.12
	Chinook salmon	Rainbow trout	0.53	96	2.12
	Cutthroat trout	Rainbow trout	0.53	96	2.12
	Steelhead	Rainbow trout	0.53	96	2.12
	Pacific lamprey	Rainbow trout	0.53	96	2.12
Nitrate	Rainbow trout	Rainbow trout egg and fry	10.2	96	40.8
	Daphnia magna	Caddisfly larvae	431	96	1724
	Pacific chorus frog tadpole	Caddisfly larvae	431	96	1724
	Coho salmon	Rainbow trout egg and fry	10.2	96	40.8
	Chinook salmon	Rainbow trout egg and fry	10.2	96	40.8
	Cutthroat trout	Cutthroat trout	19.9	96	79.6
	Steelhead	Rainbow trout egg and fry	10.2	96	40.8
	Pacific lamprey	Rainbow trout egg and fry	10.2	96	40.8

8.4 References

EPA. See U. S. Environmental Protection Agency.

HSDB. See Hazardous Substances Databank.

NIOSH. See National Institute for Occupational Safety and Health.

USDA. See U.S. Department of Agriculture.

WHO. See World Health Organization.

WSSA. See Weed Science Society of America.

Section 8.2.1

Hoerger, F., and E.E. Kenaga. 1972. Pesticide residues on plants: Correlation of representative data as a basis for estimation of their magnitude in the environment. In *Environmental Quality and Safety: V1–Global Aspects of Toxicology and Technology as Applied to the Environment.* F. Coulston, ed. Academica Press. New York, NY.

Hope, B.K. 1995. A review of models for estimating terrestrial ecological receptor exposure to chemical contaminants. Chemosphere 30(12):2267-2287.

Pfleeger, T.G., A. Fong, R. Hayes, H. Ratsch, and C. Wickliff. 1996. Field evaluation of the EPA (Kenaga) nomogram, a method for estimating wildlife exposure to pesticide residues on plants. Environmental Toxicology and Chemistry 15(4):535-543.

U.S. Environmental Protection Agency. 1999. Reregistration eligibility decision (RED): Chlorothalonil. EPA 738-R-99-004. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Section 8.3

<u>Acephate (8.3.1)</u>

Clark, D.R., and B.A. Rattner. 1987. Orthene® toxicity to little brown bats (*Myotis lucifugus*): acetylcholinesterase inhibition, coordination loss and mortality. Environmental Toxicology and Chemistry 6:705-708.

Davies, P.E., L.S.J. Cook, and D. Goenarso. 1994. Sublethal responses to pesticides of several species of Australian freshwater fish and crustaceans and rainbow trout. Environmental Toxicology and Chemistry 13(8):1341-1354.

Geen, G.H., M.A. Hussain, P.C. Oloffs, and B.A. McKeown. 1981. Fate and toxicity of acephate (Orthene®) added to a coastal B.C. stream. Journal of Environmental Science and Health B16(3):253-271.

Lambert, W.P. 1983. Orthene®: A profile of its behavior in the environment. U.S. Department of Agriculture, Animal and Plant Health Inspection Service. Contract #53-6395-1-151.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters. Hull, Québec, Canada.

Singh, R., and H.S. Sandhu. 1999. Acute toxicity of acephate and its effects on biochemical parameters in buffalo calves. Abstract: Indian Journal of Animal Sciences 69(11):935-937.

U.S. Department of Agriculture. 1989. Pesticide background statements: Volume IV—Insecticides. Agriculture Handbook No. 685. U.S. Forest Service. Washington, DC.

U.S. Environmental Protection Agency. 1984a. Health and environmental effects profile for acephate. Office of Research and Development. Cincinnati, OH.

U.S. Environmental Protection Agency. 1984b. Supplement to acephate registration standard. Office of Pesticides and Toxic Substances. Washington, DC.

Valent USA Corporation. 2000. Material safety data sheet: Orthene® Turf, Tree & Ornamental Spray (insecticide). Walnut Creek, CA.

Vyas, N.B., W.J. Kuenzel, E.F. Hill, and J.R. Sauer. 1995. Acephate affects migratory orientation of the white-throated sparrow (*Zonotrichia albicollis*). Environmental Toxicology and Chemistry 14(11):1961-1965.

Zinkle, J.G., R.B. Roberts, P.J. Shea, and J. Lasmanis. 1981. Toxicity of acephate and methamidophos to dark-eyed juncos. Archives of Environmental Contamination and Toxicology 10:185-192.

Chlorothalonil (8.3.2)

Caux, P.Y., R.A. Kent, G.T. Fan, and G.L. Stephenson. 1996. Environmental fate and effects of chlorothalonil: A Canadian perspective. Critical Reviews in Environmental Science and Technology 26(1):45-93.

Davies, P.E., L.S.J. Cook, and D. Goenarso. 1994. Sublethal responses to pesticides of several species of Australian freshwater fish and crustaceans and rainbow trout. Environmental Toxicology and Chemistry 13(8):1341-1354.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of

Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Gallagher, E.P., R.C. Catley, and R.T. DiGiulio. 1992. The acute toxicity and sublethal effects of chlorothalonil in channel catfish (Ictalurus punctatus). Chemosphere 24(1):3-10.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters. Hull, Québec, Canada.

U.S. Environmental Protection Agency. 1999. Reregistration eligibility decision (RED): Chlorothalonil. EPA 738-R-99-004. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Zeneca Ag Products. 1998. Material safety data sheet: Bravo 500. Wilmington, DE.

Chlorpyrifos (8.3.3)

Barron, M.G., and K.B. Woodburn. 1995. Ecotoxicology of chlorpyrifos. Reviews of Environmental Contamination and Toxicology 144:1-93.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

Moore, M.T., D.B. Huggett, W.B. Gillespie, Jr., J.H. Rodgers, Jr., and C.M. Cooper. 1998. Comparative toxicity of chlordane, chlorpyrifos, and aldicarb to four aquatic testing organisms. Archives of Environmental Contamination and Toxicology 34:152-157.

U.S. Environmental Protection Agency. 2000a. Toxicology chapter for chlorpyrifos. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000b. Reregistration eligibility science chapter for chlorpyrifos: Fate and environmental risk assessment chapter. Office of Pesticide Programs. Washington, DC.

van Wijngaarden, R., P. Leeuwangh, W.G.H. Lucassen, K. Romijn, R. Ronday, R. van der Velde, and W. Willigenburg. 1993. Acute toxicity of chlorpyrifos to fish, a newt, and aquatic invertebrates. Bulletin of Environmental Contamination and Toxicology 51:716-723.

Diazinon (8.3.4)

Allison, D.T., and R.O. Hermanutz. 1977. Toxicity of diazinon to brook trout and fathead minnows. EPA 600/3-77-060. National Environmental Research Center, Ecological Research Services, U.S. Environmental Protection Agency. Washington, DC.

Cope, O.B. 1965. Effects of pesticides on fish and wildlife. 1964 Research Findings of the Fish and Wildlife Service. Circular 226. U.S. Department of the Interior Fish and Wildlife Service. Washington, DC.

Earl, F.L., B.E. Melveger, J.E. Reinwall, G.W. Bierbower, and J.M. Curtis. 1971. Diazinon toxicity—comparative studies in dogs and miniature swine. Toxicology and Applied Pharmacology 18:285-295.

Egyed, M.N., A. Shlosberg, M. Malkinson, and A. Eilat. 1976. Some considerations in the treatment of diazinon poisoned goslings. Clinical Toxicology 9:245-249.

Eisler, R. 1986. Diazinon hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.9). Patuxent Wildlife Research Center. U.S. Fish and Wildlife Service. Laurel, MD.

Harris, M.L., C.A. Bishop, J. Struger, B. Ripley, and J.P. Bogart. 1998. The functional integrity of northern leopard frog (*Rana pipiens*) and green frog (*Rana clamitans*) populations in orchard wetlands. II: Effects of pesticides and eutrophic conditions on early life stage development. Environmental Toxicology and Chemistry 17(7):1351-1363.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to coturnix. U.S. Fish and Wildlife Service Tech. Rep. 2. Washington, DC.

Hoffman, D.J., and W.C. Eastin, Jr. 1981. Effects of malathion, diazinon, and parathion on mallard embryo development and cholinesterase activity. Environmental Research 26:472-485.

Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. U.S. Fish and Wildlife Service Resource Publication 153. Washington, DC.

Johnson, W.W., and M.T. Finley. 1980. Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. Summaries of toxicity tests conducted at Columbia National Fisheries Research Laboratory, 1965-1978. Resource Publication 137. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Machin, A.F., H. Rogers, A.J. Cross, M.P. Quick, L.C. Howells, and N.F. Janes. 1975. Metabolic aspects of the toxicology of diazinon—I: Hepatic metabolism in the sheep, cow, pig, guinea-pig, rat, turkey, chicken and duck. Pesticide Science 6:461-473.

Meier, E.P., W.H. Dennis, A.B. Rosencrance, W.F. Randall, W.J. Cooper, and M.C. Warner. 1979. Sulfotepp, a toxic impurity in formulations of diazinon. Bulletin of Environmental Contamination and Toxicology 23(1-2):158-164.

Platte Chemical Co. 1994. Material safety data sheet: Clean Crop Diazinon 50W Insecticide. Fremont, NE.

Sanders, H.O. 1969. Toxicity of pesticides to the crustacea, Gammarus lacustris. Technical Paper No. 25, Bureau of Sports Fisheries and Wildlife.

Schafer, E.W., Jr., W.A. Bowles, Jr., and J. Hurlbert. 1983. The acute oral toxicity, repellence, and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Archives of Environmental Contamination and Toxicology 12:355-382.

Schobert, E.E. 1974. Ingested insecticide poisoning in waterfowl. Journal of Zoo and Animal Medicine 5:20.

Stone, W.B. 1980. Bird deaths caused by pesticides used on turfgrass. N.Y. State Turfgrass Conference Proceedings 4:58-62.

Stone, W.B., and P.B. Gradoni. 1985. Wildlife mortality related to use of the pesticide diazinon. Northeastern Environmental Science 4(1):30-38.

Stone, W.B., and H. Knoch. 1982. American brant killed on golf courses by diazinon. New York Fish and Game Journal 29:95-96.

Stromborg, K.L. 1977. Seed treatment pesticide effects on pheasant reproduction at sublethal doses. Journal of Wildlife Management 41:632-642.

U.S. Environmental Protection Agency. 2001. Environmental risk assessment for diazinon. Office of Pesticide Programs. Washington, DC.

Zinkle, J.G., J. Rathert, and R.R. Hudson. 1978. Diazinon poisoning in wild Canada geese. Journal of Wildlife Management 42:406-408.

<u>Dicamba (8.3.5)</u>

Caux, P.-Y., R.A. Kent, M. Taché, C. Grande, G.T. Fan, and D.D. MacDonald. 1993. Environmental fate and effects of dicamba: A Canadian perspective. Reviews of Environmental Contamination and Toxicology 133:1-59.

Edson, E.F., and D.M. Sanderson. 1965. Toxicity of the herbicides, 2-methoxy-3,6-dichlorobenzoic acid (dicamba) and 2-methoxy-3,5,6-trichlorobenzoic acid (tricamba). Food and Cosmetic Toxicology 3:299-304.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to *Coturnix*. Fish and Wildlife Technical Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Mayer, F.L., Jr., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Micro Flo Company. 1999. Material safety data sheet: Banvel®. Memphis, TN.

U.S. Environmental Protection Agency. 1999. Dicamba (3,6-dichloro-o-anisic acid): Pesticide tolerance. Federal Register 64(3):759-769. Office of Pesticide Programs. Washington, DC.

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals.* 2nd ed. Van Nostrand Reinhold. New York.

Dimethoate (8.3.6)

Devillers, J., and J.M. Exbrayat. 1992. *Ecotoxicity of Chemicals to Amphibians.* Gordon and Breach Science Publishers. Philadelphia, PA.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Khan, R.R., and B. Dev. 1982. Toxicology data sheets on chemicals: Dimethoate. Industrial Toxicology Research Centre. Lucknow, India.

Mudgall, C.F., and H.S. Patil. 1987. Toxic effects of dimethoate and methyl parathion on glycogen reserves of male and female *Rana cyanophlyctis*. Journal of Environmental Biology 8(3):237-244.

Pradhan, P.K., and S. Dasgupta. 1993. Effects of dimethoate on ascorbic acid in some metabolically active tissues of male toad, Bufo melanostictus. Abstract: Geobis (Jodhpur):20(2):57-60.

Rawlings, N.C., S.J. Cook, and D. Waldbillig. 1998. Effects of the pesticides carbofuran, chlorpyrifos, dimethoate, lindane, triallate, trifluralin, 2,4-D, and pentachlorophenol on the metabolic endocrine and reproductive endocrine system in ewes. Abstract: Journal of Toxicology and Environmental Health 54(1):21-36.

U.S. Environmental Protection Agency. 1999a. Dimethoate: The updated, revised HED chapter of the Reregistration Eligibility Decision Document (RED). Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1999b. Environmental Fate and Effects Division's revised chapter for the dimethoate RED. Office of Pesticide Programs. Washington, DC.

Esfenvalerate (8.3.7)

Anderson, R.L. 1982. Toxicity of fenvalerate and permethrin to several nontarget aquatic invertebrates. Environmental Entomology 11:1251-1257.

Barry, M.J., K. O'Halloran, D.C. Logan, J.T. Ahokas, and D.A. Holdway. 1995. Sublethal effects of esfenvalerate pulse-exposure on spawning and non-spawning Australian crimson-spotted rainbowfish (*Melanotaenia fluviatilis*). Archives of Environmental Contamination and Toxicology 28:459-463.

Berrill, M., S. Bertram, A. Wilson, S. Louis, D. Brigham, and C. Stromberg. 1993. Lethal and sublethal impacts of pyrethroid insecticides on amphibian embryos and tadpoles. Environmental Toxicology and Chemistry 12:525-539.

Bradbury, S.P., D.M. Symonik, J.R. Coats, and G.J. Atchison. 1987. Toxicity of fenvalerate and its constituent isomers to the fathead minnow, Pimephales promelas, and bluegill, Lepomis macrochirus. Bulletin of Environmental Contamination and Toxicology 38:727-735.

Cabral, J.R.P., and D. Galendo. 1990. Carcinogenicity study of the pesticide fenvalerate in mice. Cancer Letters 49:13-18.

Coats, J.R., and N.L. O'Donnell-Jeffery. 1979. Toxicity of four synthetic pyrethroid insecticides to rainbow trout. Bulletin of Environmental Contamination and Toxicology 23:250-255.

Curtis, L.R., W.K. Seim, and G.A. Chapman. 1985. Toxicity of fenvalerate to developing steelhead trout following continuous or intermittent exposure. Journal of Toxicology and Environmental Health 15:445-457.

Devillers, J., and J.M. Exbrayat. 1992. *Ecotoxicity of Chemicals to Amphibians.* Gordon and Breach Science Publishers. Philadelphia, PA.

Du Pont. 1999. Material safety data sheet: "Asana" XL insecticide. Wilmington, DE.

Eisler, R. 1992. Fenvalerate hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Extoxnet. 2000. Extension Toxicology Network (database of pesticide information profiles). Cooperative Extension Offices of Cornell University, Oregon State University, University of Idaho, and University of California at Davis; and Institute for Environmental Toxicology, Michigan State University. Major funding by USDA Extension Service. http://ace.orst.edu/info/extoxnet.

Fairchild, J.F., T.W. La Point, J.L. Zajicek, M.K. Nelson, F.J. Dwyer, and P.A. Lovely. 1992. Population-, community- and ecosystem-level responses of aquatic mesocosms to pulsed doses of a pyrethroid insecticide. Environmental Toxicology and Chemistry 11(1):115-129.

Holcombe, G.W., G.L. Phipps, and D.K. Tanner. 1982. The acute toxicity of Kelthane, Dursban, disulfoton, Pydrin, and permethrin to fathead minnows *Pimephales promelas* and rainbow trout *Salmo gairdneri*. Environmental Pollution (Series A) 29:167-178.

Materna, E.J., C.F. Rabeni, and T.W. LaPoint. 1995. Effects of the synthetic pyrethroid insecticide, esfenvalerate, on larval leopard frogs (Rana spp.). Abstract: Environmental Toxicology and Chemistry 14 (4):613-622.

McLeese, D.W., C.D. Metcalfe, and V. Zitko. 1980. Lethality of permethrin, cypermethrin and fenvalerate to salmon, lobster and shrimp. Bulletin of Environmental Contamination and Toxicology 25:950-955.

Tanner, D.K., and M.L. Knuth. 1996. Effects of esfenvalerate on the reproductive success of the bluegill sunfish, *Lepomis macrochirus* in littoral enclosures. Archives of Environmental Contamination and Toxicology 31:244-251.

U.S. Environmental Protection Agency. 1997. Fenvalerate; Pesticide tolerances. Federal Register 62(228):63019-63027. Office of Pesticide Programs. Washington, DC.

Woin, P. 1998. Short- and long-term effects of the pyrethroid insecticide fenvalerate on an invertebrate pond community. Ecotoxicology and Environmental Safety 41:137-156.

<u>Glyphosate (8.3.8)</u>

Beyers, D.W. 1995. Acute toxicity of Rodeo herbicide to Rio Grande silvery minnow as estimated by surrogate species: Plains minnow and fathead minnow. Archives of Environmental Contamination & Toxicology 29(1):24-26.

Folmar, L.C., H.O. Sanders, and A.M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. Archives of Environmental Contamination and Toxicology 8:269-278.

Giesy, J.P., S. Dobson, and K.R. Solomon. 2000. Ecotoxicological risk assessment for Roundup® herbicide. Reviews of Environmental Contamination and Toxicology 167:35-120.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to coturnix. Fish and Wildlife Technical Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Monsanto Company. 2000. Material safety data sheet: Rodeo® emerged aquatic weed and brush herbicide. St. Louis, MO.

Perkins, P.J., H.J. Boermans, and G.R. Stephenson. 2000. Toxicity of glyphosate and triclopyr using the Frog Embryo Teratogenesis Assay--Xenopus. Abstract: Environmental Toxicology and Chemistry 9(4):940-945.

Smith, E.A., and F.W. Oehme. 1992. The biological activity of glyphosate to plants and animals: A literature review. Veterinary and Human Toxicology 34 (6) 531-543.

Sullivan, T.P. 1985. Effects of glyphosate on selected species of wildlife. In *The Herbicide Glyphosate*. E. Grossbard and D. Atkinson (eds.). Butterworths, Boston, MA.

U.S. Environmental Protection Agency. 1993. Registration eligibility decision: Glyphosate. EPA 738-R-93-014. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

Hexazinone (8.3.9)

Berrill, M., S. Bertram, L. McGillivray, M. Kolohon, and B. Pauli. 1994. Effects of low concentrations of forest-use pesticides on frog embryos and tadpoles. Environmental Toxicology and Chemistry 13(4):657-664.

Du Pont. 1998. Material safety data sheet: "Velpar" herbicide. Wilmington, DE.

Kennedy, G.L. 1984. Acute and environmental toxicity studies with hexazinone. Fundamental and Applied Toxicology 4:603-611.

Kreutzweiser, D.P., S.B. Holmes, and D.J. Behmer. 1992. Effects of the herbicides hexazinone and triclopyr ester on aquatic insects. Ecotoxicology and Environmental Safety 23:364-374.

Kreutzweiser, D.P., S.S. Capell, and B.C. Sousa. 1995. Hexazinone effects on stream periphyton and invertebrate communities. Environmental Toxicology and Chemistry 14(9):1521-1527.

Rhodes, R.C. 1980. Studies with ^{14}C-labeled hexazinone in water and bluegill sunfish. Journal of Agricultural and Food Chemistry 28:306-310.

U.S. Department of Agriculture. 1984. Pesticide background statements, Volume I: Herbicides. Agriculture Handbook Number 633. U.S. Forest Service. Washington, DC.

U.S. Environmental Protection Agency. 1994. Reregistration eligibility decision (RED): Hexazinone. EPA 738-R-94-022. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Wan, M.T., R.G. Watts, and D.J. Moul. 1988. Evaluation of the acute toxicity to juvenile Pacific salmonids of hexazinone and its formulated products: Pronone 10G, Velpar® L, and their carriers. Bulletin of Environmental Contamination and Toxicology 41:609-616.

Horticultural Oil (8.3.10)

Christens, E., H. Blokpoel, G. Rason, and S.W.D. Jarvie. 1995. Spraying white mineral oil on Canada goose eggs to prevent hatching. Wildlife Society Bulletin 23(2):228-230.

Riverside/Terra Corp. 1995. Material safety data sheet: Dormant Oil 435. Sioux City, IA.

Valent USA Corporation. 1983a. 96-hour aquatic toxicity study in rainbow trout and bluegill sunfish with 70 Orchard Spray. Walnut Creek, CA. Performed by Gulf Life Sciences Center. Pittsburg, PA.

Valent USA Corporation. 1983b. 48-hour aquatic toxicity study in Daphnia with 70 Orchard Spray. Walnut Creek, CA. Performed by Gulf Life Sciences Center. Pittsburg, PA.

Wildlife International Ltd. 1990. 90 Neutral Oil: An acute oral toxicity study with the northern bobwhite. Easton, MD. Performed on behalf of PureGro Company (Unocal Corporation), West Sacramento, CA.

Wildlife International Ltd. 1991. 90 Neutral Oil: A 96-hour static acute toxicity test with the rainbow trout (Oncohrynchus mykiss). Easton, MD. Submitted to Unocal Corporation, Los Angeles, CA.

Permethrin (8.3.11)

Anderson, R.L. 1982. Toxicity of fenvalerate and permethrin to several nontarget aquatic invertebrates. Environmental Entomology 11:1251-1257.

Berrill, M., S. Bertram, A. Wilson, S. Louis, D. Brigham, and C. Stromberg. 1993. Lethal and sublethal impacts of pyrethroid insecticides on amphibian embryos and tadpoles. Environmental Toxicology and Chemistry 12:525-539.

Braithwaite, G.B. 1984. Development and registration of permethrin insecticide. Agri-Practice 5(4):16-22.

Coates, J.R., and N.L. O'Donnell-Jeffrey. 1979. Toxicity of four synthetic pyrethroid insecticides to rainbow trout. Bulletin of Environmental Contamination and Toxicology 23:250-255.

Devillers, J., and J.M. Exbrayat. 1992. *Ecotoxicity of Chemicals to Amphibians.* Gordon and Breach Science Publishers. Philadelphia, PA.

FMC Corporation. 1995. Material safety data sheet: Pounce® 3.2 EC Insecticide. Philadelphia, PA.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to coturnix. Fish and Wildlife Technical Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Holcombe, G.W., G.L. Phipps, and D.K. Tanner. 1982. The acute toxicity of kelthane, dursban, disulfoton, pydrin, and permethrin to fathead minnows *Pimephales promelas* and rainbow trout *Salmo gairdneri*. Environmental Pollution (Series A) 29:167-187.

Ibrahim, H., R. Kheir, S. Helmi, J. Lewis, and M. Crane. 1998. Effects of organophosphorus, carbamate, pyrethroid and organochlorine pesticides, and a heavy metal on survival and cholinesterase activity of *Chironomus riparius* Meigen. Bulletin of Environmental Contamination and Toxicology 60:448-455.

Jolly, A.L., J.W. Avault, Jr., K.L. Koonce, and J.B. Graves. 1978. Acute toxicity of permethrin to several aquatic animals. Transactions of the American Fisheries Society 107(6):825-827.

Kaushik, N.K., G.L. Stephenson, K.R. Solomon, and K.E. Day. 1985. Impact of permethrin on zooplankton communities in limnocorrals. Canadian Journal of Fisheries and Aquatic Sciences 42(1):77-85.

Kingsbury, P.D. 1976. Effects of an aerial application of the synthetic pyrethroid permethrin on a forest stream. The Manitoba Entomologist 10:9-17.

Kingsbury, P.D., and D.P. Kreutzweiser. 1979. Impacts of double applications of permethrin on forest stream and ponds. Report FPM-X-27. Forest Pest Management Institute. Canadian Forestry Service, Sault Ste. Marie, Ontario.

Kingsbury, P.D., and D.P. Kreutzweiser. 1987. Permethrin treatments in Canadian forests. Part I: Impacts on stream fish. Pesticide Science 19:35-48.

Kingsbury, P.D., and B.B. McLeod. 1979. Terrestrial impact studies in forest ecosystems treated with double applications of permethrin. Report FPM-X-28. Forest Pest Management Institute. Canadian Forestry Service, Sault Ste. Marie, Ontario.

Mayer, F.L., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Fish and Wildlife Service. Washington, DC.

Mulla, M.S., H.A. Navvab-Gojrati, and H.A. Darwazeh. 1978. Toxicity of mosquito larvicidal pyrethroids to four species of freshwater fish. Entomology 7(3):428-430.

Reddy, P.M., S.S. Naik, and M.D. Bashamohideen. 1995. Toxicity of cypermethrin and permethrin to fish *Cyprinus carpio*. Environment and Ecology 13(1):30-33.

World Health Organization. 1990. Environmental health criteria 94: Permethrin. Geneva, Switzerland.

Picloram (8.3.12)

Gersich, F.M., D.L. Hopkins, and D.P. Milazzo. 1985. Acute and chronic toxicity of technical picloram (4-amino-3,5,6-trichloropicolinic acid) to *Daphnia magna* Straus. Bulletin of Environmental Contamination and Toxicology 35:121-126.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

Hudson, R.H., R.K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. Resource Publication 153. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Jackson, J.B. 1966. Toxicologic studies on a new herbicide in sheep and cattle. American Journal of Veterinary Research 27(113):821-824.

Lynn, G.E. 1965. A review of toxicological information on TORDON herbicides. Down to Earth, Spring 1965:6-8 (publication of Dow Chemical Company).

Mayer, F.L., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Fish and Wildlife Service. Washington, DC.

Mayes, M.A., and D.C. Dill. 1984. The acute toxicity of picloram, picloram potassium salt, and picloram triisopropanolamine salt to aquatic organisms. Environmental Toxicology and Chemistry 3:263-269.

Mayes, M.A., D.L. Hopkins, and D.C. Dill. 1987. Toxicity of picloram (4-amino-3,5,6-trichloropicolinic acid) to life stages of the rainbow trout. Bulletin of Environmental Contamination and Toxicology 38:653-660.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters. Hull, Québec, Canada.

U.S. Environmental Protection Agency. 1995. Reregistration eligibility decision (RED): Picloram. EPA 738-R95-019. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

Woodward, D.F. 1976. Toxicity of the herbicides dinoseb and picloram to cutthroat (*Salmo clarki*) and lake trout (*Salvelinus namaycush*). Journal of the Fisheries Research Board of Canada 33(8):1671-1676.

Woodward, D.F. 1979. Assessing the hazard of picloram to cutthroat trout. Journal of Range Management 32(3):230-232.

Propargite (8.3.13)

Hayes, W.H., Jr., and E.R. Laws, Jr., eds. 1991. *Handbook of Pesticide Toxicology.* Academic Press, Inc. San Diego, CA.

Uniroyal Chemical Company. 1998. Material safety data sheet: Omite® CR. Middlebury, CT.

U.S. Environmental Protection Agency. Undated. Memorandum: Response to comments from Uniroyal Chemical Company on the EFED chapter of the draft reregistration eligibility document for propargite. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000. Environmental Fate and Effects Division science chapter for Reregistration Eligibility Document for propargite. Office of Pesticide Programs. Washington, DC.

Triclopyr (8.3.14)

Berrill, M., S. Bertram, L. McGillivray, M. Kolohon, and B. Pauli. 1994. Effects of low concentrations of forest-use pesticides on frog embryos and tadpoles. Environmental Toxicology and Chemistry 13(4):657-664.

Gersich, F.M., C.G. Mendoza, D.L. Hopkins, and K.M. Bodner. 1984. Acute and chronic toxicity of triclopyr triethylamine salt to *Daphnia magna* Straus. Bulletin of Environmental Contamination and Toxicology 32:497-502.

Holmes, S.B., D.G. Thompson, K.L. Wainio-Keizer, S.S. Capell, and B. Staznik. 1994. Effects of lethal and sublethal concentrations of the herbicide, triclopyr butoxyethyl ester, in the diet of zebra finches. Journal of Wildlife Diseases 30(3):319-327.

Kenaga, E.E. 1979. Acute and chronic toxicity of 75 pesticides to various animal species. Down to Earth 35(2):25–31.

Kreutzweiser, D.P., S.B. Holmes, and D.C. Eichenberg. 1994. Influence of exposure duration on the toxicity of triclopyr ester to fish and aquatic insects. Archives of Environmental Contamination and Toxicology 26:124-129.

Mayes, M.A., D.C. Dill, K.M. Bodner, and C.G. Mendoza. 1984. Triclopyr triethylamine salt toxicity to life stages of the fathead minnow (*Pimephales promelas* Rafinesque). Bulletin of Environmental Contamination and Toxicology 33:339–347.

Mayes, M.A., P.G. Murphy, D.L. Hopkins, F.M. Gersich, and F.A. Blanchard. 1986. The toxicity and metabolism of triclopyr butoxyethyl ester: Coho salmon. The Toxicologist 6(1):102.

Osweiler, G.D. 1983. Toxicology of triclopyr herbicide to equine. 20th Annual Proceedings, American Association of Veterinary Laboratory Diagnosticians, pp. 193-201.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters. Hull, Québec, Canada.

Perkins, P.J., H.J. Boermans, and G.R. Stephenson. 2000. Toxicity of glyphosate and triclopyr using the Frog Embryo Teratogenesis Assay--Xenopus. Abstract: Environmental Toxicology and Chemistry 19(4):940-945.

U.S. Environmental Protection Agency. 1989. Tox one-liner: Summary of results of studies submitted in support of the registration of triclopyr. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1998. Reregistration eligibility decision (RED): Triclopyr. EPA 738-R-98-011. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

Weed Science Society of America. 1989. *Herbicide Handbook of the Weed Science Society of America.* 6th ed. Champaign, IL.

Other Ingredients (8.3.15)

Cyclohexanone

Gupta, P.K., W.H. Lawrence, J.E. Turner, and J. Autian. 1979. Toxicological aspects of cyclohexanone. Toxicology and Applied Pharmacology 49:525-533.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

U.S. Environmental Protection Agency. 2001. Ecotox: Ecotoxicology database. Office of Research and Development, National Health and Environmental Effects Research Laboratory, Mid-Continent Ecology Division. Duluth, MN.

Ethylbenzene

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

Mayer, F.L., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Fish and Wildlife Service. Washington, DC.

Von Burg, R. 1992. Toxicology update: Ethylbenzene. Journal of Applied Toxicology 12(1):69-71.

World Health Organization. 1996. Ethylbenzene. Abstract: Environmental Health Criteria 186. Geneva, Switzerland.

Light Aromatic Solvent Naphtha

Eisler, R. 1987. Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: A synoptic review. U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center. Laurel, MD.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

U.S. Environmental Protection Agency. 2001. Ecotox: Ecotoxicology database. Office of Research and Development, National Health and Environmental Effects Research Laboratory, Mid-Continent Ecology Division. Duluth, MN.

Xylene

Calleja, M.C., G. Persone, and P. Geladi. 1994. Comparative acute toxicity of the first 50 multicentre evaluation of *in vitro* cytotoxicity chemicals to aquatic non-vertebrates. Environmental Contamination and Toxicology 26:69-78.

Devillers, J., and J.M. Exbrayat. 1992. *Ecotoxicity of Chemicals to Amphibians.* Gordon and Breach Science Publishers. Philadelphia, PA.

Ferrando, M.D., and E. Andreu-Moliner. 1992. Acute toxicity of toluene, hexane, xylene, and benzene to the rotifers *Brachionus calyciflorus* and *Brachionus pilcatilis*. Bulletin of Environmental Contamination and Toxicology 49:266-271.

Hazardous Substances Databank. 2001. On-line database. National Library of Medicine. Bethesda, MD.

Hill, E.F., and M.B. Camardese. 1986. Lethal dietary toxicities of environmental contaminants and pesticides to coturnix. Fish and Wildlife Technical Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

Mayer, F.L., and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Fish and Wildlife Service. Washington, DC.

Verschueren, K. 1983. *Handbook of Environmental Data on Organic Chemicals.* 2nd ed. Van Nostrand Reinhold. New York.

Fertilizers (8.3.16)

Arthur, J.W., C.W. West, K.N. Allen, and S.F. Hedtke. 1987. Seasonal toxicity of ammonia to five fish and nine invertebrate species. Bulletin of Environmental Contamination and Toxicology 38:324-331.

Banerjee, T.K. 1993. Estimation of acute toxicity of ammonium sulphate to the fresh water catfish, *Heteropneustes fossilis* I. Analysis of LC_{50} values determined by various methods. Biomedical and Environmental Sciences 6:31-36.

Blahm, T.H., and G.R. Snyder. 1973. Effect of chemical fire retardants on the survival of juvenile salmonids. National Marine Fisheries Service, Environmental Facility. Prescott, OR.

Bruning-Fann, C.S., and J.B. Kaneene. 1993. The effects of nitrate, nitrite, and *N*-nitroso compounds on animal health. Veterinary and Human Toxicology 35(3):237-253.

Gersich, F.M., and D.L. Hopkins. 1986. Site-specific acute and chronic toxicity of ammonia to *Daphnia magna* Straus. Environmental Toxicology and Chemistry 5:443-447.

Hazardous Substances Databank (HSDB). 2001. On-line database. National Library of Medicine. Bethesda, MD.

Horner, R.F. 1982. Suspected ammonium nitrate fertiliser poisoning in cattle. The Veterinary Record 110:472-474.

Inman, R.C. 1974. Acute toxicity of Phos-Check® 202 and diammonium phosphate to fathead minnows. USAF Environmental Health Laboratory. Kelly AFB, TX.

Jones, T.O. 1982. Letter: Suspected ammonium nitrate fertiliser poisoning in cattle. The Veterinary Record 111:211-212.

Knoph, M.B. 1992. Acute toxicity of ammonia to Atlantic salmon (*Salmo salar*) parr. Comparative Biochemistry and Physiology 101C(2):275-282.

Monsanto Company. 1991a. Material safety data sheet: Monoammonium phosphate anhydrous. St. Louis, MO.

Monsanto Company. 1991b. Material safety data sheet: Diammonium phosphate anhydrous. St. Louis, MO.

National Institute of Occupational Safety and Health (NIOSH). 1987. *Registry of Toxic Effects of Chemical Substances.* U.S. Department of Health and Human Services. U.S. Government Printing Office. Washington, DC.

Nechkina, M.A. 1992. Assessment of mutagenic effect of ammonium nitrate and 2,4-D acid. Gig Sanit 0(2):66-67.

Norris, L.A., H.W. Lorz, and S.V. Gregory. 1991. Forest chemicals. pp. 207-296 in *Influences of Forest and Rangeland Management on Salmonid Fishes and Their Habitats*. American Fisheries Society Special Publication 19.

Oldham, R.S., D.M. Latham, D. Hilton-Brown, M. Towns, A.S. Cooke, and A. Burn. 1997. The effect of ammonium nitrate fertiliser on frog (*Rana temporaria*) survival. Agriculture, Ecosystems & Environment 61:69-74.

Pauli, B.D., J.A. Perrault, and S.L. Money. 2000. RATL: A database of reptile and amphibian toxicology literature. Technical Report Series No. 357. Canadian Wildlife Service, Headquarters. Hull, Québec, Canada.

Rouse, J.D., C.A. Bishop, and J. Struger. 1999. Nitrogen pollution: An assessment of its threat to amphibian survival. Environmental Health Perspectives 107(10):799-803.

Schuytema, G.S., and A.V. Nebeker. 1999a. Comparative toxicity of ammonium and nitrate compounds to Pacific treefrog and African clawed frog tadpoles. Environmental Toxicology and Chemistry 18(10):2251-2257.

Schuytema, G.S., and A.V. Nebeker. 1999b. Comparative effects of ammonium and nitrate compounds on Pacific treefrog and African clawed frog embryos. Archives of Environmental Contamination and Toxicology 36:200-206.

Schuytema, G.S., and A.V. Nebeker. 1999c. Effects of ammonium and nitrate, sodium nitrate, and urea on red-legged frogs, Pacific treefrog, and African clawed frogs. Bulletin of Environmental Contamination and Toxicology 63:357-364.

U.S. Environmental Protection Agency (EPA). 1999. 1999 update of ambient water quality criteria for ammonia. EPA 822-R-99-014. Office of Water. Washington, DC.

U.S. Environmental Protection Agency (EPA). 2001. Ecotox: Ecotoxicology database. Office of Research and Development, National Health and Environmental Effects Research Laboratory, Mid-Continent Ecology Division. Duluth, MN.

Watt, P.J., and R.S. Oldham. 1995. The effect of ammonium nitrate on the feeding and development of larvae of the smooth newt, Triturus vulgaris (L.), and on the behaviour of its food source, Daphnia. Abstract: Freshwater Biology 33(2):319-324.

Xu, Q., and R.S. Oldham. 1997. Lethal and sublethal effects of nitrogen fertilizer ammonium nitrate on common toad (*Bufo bufo*) tadpoles. Archives of Environmental Contamination and Toxicology 32:298-303.

Aquatic Species LC_{50}s Used in the Risk Assessment (8.3.17)

Suter, G.W. II. 1993. Organism-level effects. In *Ecological Risk Assessment*, G.W. Suter II, ed. Lewis Publishers. Chelsea, MI.

9.0 NON-TARGET SPECIES RISK CHARACTERIZATION

Risk characterization is the last step in the ecological risk assessment process. The exposure profile is compared to the stressor-response profile, to estimate the likelihood of adverse effects.

9.1 Risk Estimation

By comparing the exposure profile data (estimated dose or water concentration) to the stressor-response profile data (LD_{50}s, LC_{50}s, MATCs), an estimate of the possibility of adverse effects can be made. The levels of concern are determined following the quotient methodology used by EPA's Office of Pesticide Programs. The quotient is the ratio of the exposure level to the hazard level. For acute exposures, the levels of concern at which a quotient is concluded to reflect risk to non-target species are as follows:

- Terrestrial species (general): 0.5, where dose equals one-half the LD_{50}.
- Terrestrial species (endangered, threatened, sensitive): 0.1, where dose equals one-tenth the LD_{50}.
- Aquatic species (general): 0.5, where water concentration equals one-half the LC_{50}.
- Aquatic species (endangered, threatened, sensitive): 0.05, where water concentration equals one-twentieth the LC_{50}.

Due to the high level of concern for protecting threatened salmonids in the watershed, the predicted water concentrations are also compared to the MATC for a chemical, if available.

Tables 9-1 to 9-42, at the end of this chapter, summarize the estimated risks to non-target species from each type of proposed pesticide or fertilizer application at Provolt. Tables 9-43 to 9-46 present the estimated risks to wildlife species from accidents.

The risk tables in this section use scientific notation, since many of the values are very small. For example, the notation 3.63E-001 represents 3.63×10^{-1}, or 0.363. Similarly, 4.65E-009 represents 4.65×10^{-9}, or 0.00000000465.

9.2 Risk Discussion

9.2.1 Estimated Risks to Terrestrial Wildlife

Risks to General Species

Risks are predicted from chlorpyrifos for the black-capped chickadee in the typical and maximum scenarios.

Risks are predicted from diazinon for the black-capped chickadee, western bluebird, and song sparrow in the maximum scenario.

Dimethoate was estimated to present risks to the pocket gopher, black-capped chickadee, western bluebird, song sparrow, and Pacific chorus frog in the typical scenario, and to these same species plus the long-tailed vole, long-eared myotis, mallard duck, great blue heron, tree swallow, Canada goose, gopher snake, and western fence lizard in the maximum scenario.

In most cases, little or no adverse impact to terrestrial wildlife populations is expected from the pesticides and fertilizers proposed for use at Provolt under typical conditions of use, with the possible exception of impacts to subterranean mammal, bird, reptile, and amphibian species from applications of two of the insecticides (chlorpyrifos and dimethoate). Most of the estimated doses are extremely low, with risk quotients several orders of magnitude below the levels of concern. A margin for error is provided by the methodology applied, which uses reasonable assumptions that tend toward overstating potential exposures to wildlife, in the absence of site-specific data on potential exposure patterns. In addition, all of the chemicals have relatively short half-lives (see Section 3.0) and are not expected to remain in the environment for significant periods of time.

Although some terrestrial insects onsite may be affected by the insecticide applications, and may constitute a portion of the dose to insectivorous species, populations of beneficial insects as a whole are not expected to suffer adverse impacts because the proposed seed orchard applications are localized.

Roberts and Dorough (1985) summarized data on the risk posed by agricultural pesticides to terrestrial invertebrates, primarily the earthworm. Earthworms come into contact with chemicals in the terrestrial environment by direct exposure as they move through the soil or when feeding on the surface. The ingestion of contaminated leaf litter and organic debris is another route of exposure. The authors stated that more studies are needed, and that the assessment of the comparative toxicities of chemicals to earthworms under field conditions poses a challenging research problem, because the toxicity of chemicals varies with the type of soil, method of application, and prevailing environmental conditions. It is also difficult to determine the adverse effects of chemicals on natural earthworm populations because the populations fluctuate throughout the year and because difficulties are encountered in obtaining reliable samples. In addition, there is little consistency among protocols for both field and laboratory studies, limiting the validity of comparisons of the relative toxicity of chemicals to earthworms. Of the proposed seed orchard insecticides named in the review, fenvalerate was very toxic, acephate was moderately toxic, and permethrin was relatively nontoxic, based on the results of the studies reported. Therefore, it appears that insecticide applications may have adverse impacts on local earthworm populations. Any possible impacts are expected to be reversible, given that these chemicals are not persistent in the soil and that limited areas would be treated only on an as-needed basis in any growing season, allowing for re-population from adjacent untreated areas.

Risks to Endangered, Threatened, and Sensitive Species

Risks are predicted from chlorpyrifos for the western pond turtle in the typical and maximum scenarios, and for the common kingsnake in the maximum scenario.

Risks are predicted from diazinon for the western pond turtle and common kingsnake in the maximum scenario.

Dimethoate was estimated to present risks to the western pond turtle and common kingsnake in the typical scenario, and to these same species plus the spotted owl and bald eagle in the maximum scenario.

9.2.2 Estimated Risks to Aquatic Wildlife

Stream concentrations, summarized in Table 3-4, are compared to the LC_{50}s presented in Table 8-37, to calculate the risk quotients for aquatic species.

Risks to General Species

No risks were predicted for any aquatic invertebrates or tadpoles in the onsite irrigation ditches; nor for any coldwater fish species (represented by rainbow trout) in Williams Creek; nor for any coldwater fish, aquatic invertebrates, or tadpoles in the Applegate River from any pesticides or fertilizers proposed for use at Provolt.

Risks to Endangered, Threatened, and Sensitive Species

No risks to sensitive species in Williams Creek or the Applegate River were predicted from any pesticides or fertilizers proposed for use at Provolt.

9.2.3 Risks from Accidents

Risks are predicted for all terrestrial species except the deer, coyote, raccoon, and dog in the accident scenario in which an animal ingests an acephate implant capsule.

Aquatic invertebrates are at risk from a spill of chlorpyrifos concentrate at the mixing area, and sensitive aquatic species are at risk from a spill of esfenvalerate or permethrin concentrate. Spills of tank mix directly into streams were predicted to pose risks to coldwater fish (represented by rainbow trout) from chlorpyrifos; to aquatic invertebrates from chlorpyrifos, diazinon, esfenvalerate, and permethrin; and to sensitive species from chlorpyrifos, esfenvalerate, permethrin, chlorothalonil, and triclopyr.

9.2.4 Risks to Plants

Terrestrial Plants

The proposed herbicides will be variously toxic to any plants with which they come into contact. One sensitive plant species has been identified on site at the seed orchard. Herbicide-free buffer zones will be implemented for the protection of this species. Mechanical control of nearby weeds could be accomplished through mowing. Broadcast applications of herbicides are only proposed for intensively managed or disturbed areas such as along roads and fences, within orchard units, or around facilities, while spot applications will be used to control weed species in less disturbed areas. Only spot hand applications would be conducted within the buffer zones. Insecticides, fungicides, and fertilizers are only proposed for use in cultivated areas (seed orchard blocks), so no direct contact with plant species in other areas is expected.

Aquatic Plants

Aquatic plants may be present in streams and ponds that receive runoff from treated areas. A literature review was conducted to identify the levels at which any of the proposed chemicals may pose a hazard to aquatic plants. For many chemicals, tests in algae were the only available data, and are expected to provide a sensitive endpoint for hazards to aquatic plants. For each chemical, the estimated water concentrations were compared to the levels of concern. This analysis is summarized in the following paragraphs.

The EC_{50} for acephate to the saltwater diatom *Skeletonema costatum* is >50 mg/L, which led EPA (undated) to conclude that no further testing of impacts to aquatic plants was warranted.

Algae EC_{50}s for chlorpyrifos ranged from 0.14 to 0.3 mg/L (EPA 2000a).

The EC_{50} for diazinon in algae was 3.7 mg/L (EPA 2001).

The 96-hour EC_{50}s for dimethoate in algae species ranged from 9.5 to 12.5 mg/L (EPA 1984).

The 96-hour EC_{50} for growth inhibition in four species of marine algae was >1 mg/L for fenvalerate (Eisler 1992).

No data were available on the toxicity of horticultural oil to aquatic plant species.

An EC_{50} of 1.6 to 5.0 mg/L was found for permethrin's effects in cyanobacteria (blue-green algae). In green algae, no effects on growth were observed at 10 mg/L, and no effects on photosynthesis or acetylene reduction were observed at 100 mg/L, the highest concentration tested in each case (Stratton and Corke 1982).

Propargite was tested in several aquatic plants. For duckweed, the EC_{50} was 75 mg/L. In nonvascular plants (algae and diatoms), EC_{50}s ranged from 0.0194 to 105.5 mg/L (EPA 2000b).

EPA (1999) reported an EC_{50}, LOEC, and NOEC of 0.19, 0.1, and 0.05 mg/L, respectively, for chlorothalonil's effects on freshwater algae.

Freshwater green algae exhibited chronic EC_{50}s of 0.1 to 10 mg/L when exposed to dicamba (Caux et al. 1993).

EPA (1993) reported 96-hour EC_{50}s of 0.85 to 39.9 mg/L for glyphosate's effects on four aquatic plant species. These results led EPA to conclude that glyphosate may have adverse effects on aquatic plants under some conditions.

Five studies of hexazinone's effects on aquatic plants were reported by EPA (1994), with EC_{50}s ranging from 0.007 to 0.12 mg/L. These results indicate there may be effects on aquatic plants, particularly in ponds or lakes, if runoff or drift occurs.

Test results of picloram potassium salt's toxicity to aquatic plant species were reported in EPA (1995) as an EC_{50} of 52.6 mg/L and a NOEC of 13.1 mg/L.

EPA (1998) concluded that concentrations of triclopyr triethylamine salt greater than 8.8 mg/L may cause detrimental effects to vascular aquatic plants, and concentrations greater than 5.9 mg/L may affect algae. For the butoxyethyl ester of triclopyr, corresponding levels of concern are 0.88 mg/L for vascular aquatic plants, and 0.10 mg/L for effects on algae.

None of the predicted concentrations in onsite ditches, Williams Creek, or the Applegate River exceed the effects criteria equivalent to 50% of the values reported in the literature summarized in the preceding paragraphs. Therefore, no adverse effects to aquatic plants are expected under typical or maximum conditions of pesticide or fertilizer application at Provolt.

Table 9-1. Terrestrial Risks from Chlorpyrifos

	Risk Quotient			
	HPHS*		HHW**	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	1.71E-005	1.00E-004	1.71E-005	1.00E-004
Coyote	8.02E-006	4.92E-004	8.02E-006	4.92E-004
Long-tailed vole	4.13E-003	1.97E-002	4.13E-003	1.97E-002
Long-eared myotis	4.21E-005	5.99E-003	4.21E-005	5.99E-003
Pocket gopher	4.06E-002	1.65E-001	4.06E-002	1.65E-001
Raccoon	2.15E-004	1.41E-003	2.15E-004	1.41E-003
Dog	-0-	3.80E-004	-0-	3.80E-004
Black-capped chickadee	**5.51E-001**	**2.28E+000**	**5.51E-001**	**2.28E+000**
Western bluebird	6.70E-002	3.20E-001	6.70E-002	3.20E-001
Mallard duck	1.46E-004	1.72E-003	1.46E-004	1.72E-003
Great blue heron	2.36E-004	1.27E-002	2.36E-004	1.27E-002
Song sparrow	4.10E-002	1.97E-001	4.10E-002	1.97E-001
Tree swallow	4.70E-006	5.89E-002	4.70E-006	5.89E-002
Canada goose	1.38E-004	1.76E-003	1.38E-004	1.76E-003
Common barn owl	4.65E-004	9.30E-004	4.65E-004	9.30E-004
Osprey	-0-	1.27E-006	-0-	1.27E-006
Pacific chorus frog	6.69E-003	2.71E-002	6.69E-003	2.71E-002
Gopher snake	9.37E-004	4.75E-002	9.37E-004	4.75E-002
Western fence lizard	8.68E-003	3.49E-002	8.68E-003	3.49E-002
E&T and Sensitive Terrestrial Species				
Spotted owl	2.27E-004	1.93E-002	2.27E-004	1.93E-002
Bald eagle	3.55E-005	1.02E-002	3.55E-005	1.02E-002
Western pond turtle	**1.25E-001**	**5.02E-001**	**1.25E-001**	**5.02E-001**
Common kingsnake	2.49E-002	**4.90E-001**	2.49E-002	**4.90E-001**

*HPHS = High-Pressure Hydraulic Sprayer
**HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-2. Terrestrial Risks from Diazinon

	Risk Quotient			
	HPHS*		HHW**	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	2.14E-006	9.78E-005	2.14E-006	9.78E-005
Coyote	4.18E-007	2.12E-004	4.18E-007	2.12E-004
Long-tailed vole	2.15E-004	7.87E-003	2.15E-004	7.87E-003
Long-eared myotis	2.19E-006	2.62E-003	2.19E-006	2.62E-003
Pocket gopher	2.12E-003	6.46E-002	2.12E-003	6.46E-002
Raccoon	1.12E-005	5.77E-004	1.12E-005	5.77E-004
Dog	-0-	1.67E-004	-0-	1.67E-004
Black-capped chickadee	2.42E-001	**7.52E+000**	2.42E-001	**7.52E+000**
Western bluebird	2.94E-002	**1.08E+000**	2.94E-002	**1.08E+000**
Mallard duck	1.91E-003	1.84E-001	1.91E-003	1.84E-001
Great blue heron	1.04E-004	4.56E-002	1.04E-004	4.56E-002
Song sparrow	1.37E-002	**5.03E-001**	1.37E-002	**5.03E-001**
Tree swallow	2.07E-006	2.18E-001	2.07E-006	2.18E-001
Canada goose	2.24E-004	2.33E-002	2.24E-004	2.33E-002
Common barn owl	2.04E-004	4.08E-004	2.04E-004	4.08E-004
Osprey	-0-	2.65E-008	-0-	2.65E-008
Pacific chorus frog	3.34E-004	1.02E-002	3.34E-004	1.02E-002
Gopher snake	4.12E-004	1.70E-001	4.12E-004	1.70E-001
Western fence lizard	3.81E-003	1.15E-001	3.81E-003	1.15E-001
E&T and Sensitive Terrestrial Species				
Spotted owl	9.98E-005	7.01E-002	9.98E-005	7.01E-002
Bald eagle	1.56E-005	3.76E-002	1.56E-005	3.76E-002
Western pond turtle	5.49E-002	**1.65E+000**	5.49E-002	**1.65E+000**
Common kingsnake	1.09E-002	**1.65E+000**	1.09E-002	**1.65E+000**

*HPHS = High-Pressure Hydraulic Sprayer
**HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-3. Terrestrial Risks from Dimethoate*

	Risk Quotient			
	HHW**		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	5.29E-004	2.55E-002	5.29E-004	2.55E-002
Coyote	1.75E-005	2.33E-002	1.75E-005	2.33E-002
Long-tailed vole	7.32E-002	**8.40E-001**	7.32E-002	**8.40E-001**
Long-eared myotis	2.57E-004	**8.27E-001**	2.57E-004	**8.27E-001**
Pocket gopher	**7.20E-001**	**4.14E+000**	**7.20E-001**	**4.14E+000**
Raccoon	1.31E-003	8.52E-002	1.31E-003	8.52E-002
Dog	-0-	1.88E-002	-0-	1.88E-002
Black-capped chickadee	**1.04E+001**	**7.33E+001**	**1.04E+001**	**7.33E+001**
Western bluebird	**2.20E+000**	**2.53E+001**	**2.20E+000**	**2.53E+001**
Mallard duck	2.95E-003	**5.53E-001**	2.95E-003	**5.53E-001**
Great blue heron	7.73E-003	**3.26E+000**	7.73E-003	**3.26E+000**
Song sparrow	**2.39E+000**	**2.81E+001**	**2.39E+000**	**2.81E+001**
Tree swallow	5.31E-005	**1.56E+001**	5.31E-005	**1.56E+001**
Canada goose	1.11E-002	**2.34E+000**	1.11E-002	**2.34E+000**
Common barn owl	5.25E-003	1.05E-002	5.25E-003	1.05E-002
Osprey	-0-	2.95E-012	-0-	2.95E-012
Pacific chorus frog	**1.56E+001**	**8.81E+001**	**1.56E+001**	**8.81E+001**
Gopher snake	1.39E-002	**1.21E+001**	1.39E-002	**1.21E+001**
Western fence lizard	2.84E-001	**1.53E+000**	2.84E-001	**1.53E+000**
E&T and Sensitive Terrestrial Species				
Spotted owl	2.57E-003	**5.00E+000**	2.57E-003	**5.00E+000**
Bald eagle	4.01E-004	**2.69E+000**	4.01E-004	**2.69E+000**
Western pond turtle	**4.09E+000**	**2.18E+001**	**4.09E+000**	**2.18E+001**
Common kingsnake	**8.14E-001**	**1.18E+002**	**8.14E-001**	**1.18E+002**

*Includes additive risks from other ingredients in formulation.

**HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-4. Terrestrial Risks from Esfenvalerate*

	Risk Quotient					
	HPHS**		HHW***		Backpack	
Animal	Typ	Max	Typ	Max	Typ	Max
General Terrestrial Species						
Deer	2.76E-005	1.53E-004	2.76E-005	1.53E-004	2.76E-005	1.53E-004
Coyote	1.62E-006	7.67E-005	1.62E-006	7.67E-005	1.62E-006	7.67E-005
Long-tailed vole	7.06E-004	3.40E-003	7.06E-004	3.40E-003	7.06E-004	3.40E-003
Long-eared myotis	8.49E-006	9.27E-004	8.49E-006	9.27E-004	8.49E-006	9.27E-004
Pocket gopher	6.95E-003	2.90E-002	6.95E-003	2.90E-002	6.95E-003	2.90E-002
Raccoon	4.33E-005	2.63E-004	4.33E-005	2.63E-004	4.33E-005	2.63E-004
Dog	-0-	5.83E-005	-0-	5.83E-005	-0-	5.83E-005
Black-capped chickadee	2.08E-003	8.84E-003	2.08E-003	8.84E-003	2.08E-003	8.84E-003
Western bluebird	2.53E-004	1.24E-003	2.53E-004	1.24E-003	2.53E-004	1.24E-003
Mallard duck	2.13E-006	2.80E-005	2.13E-006	2.80E-005	2.13E-006	2.80E-005
Great blue heron	8.93E-007	4.76E-005	8.93E-007	4.76E-005	8.93E-007	4.76E-005
Song sparrow	2.76E-004	1.36E-003	2.76E-004	1.36E-003	2.76E-004	1.36E-003
Tree swallow	2.10E-008	2.20E-004	2.10E-008	2.20E-004	2.10E-008	2.20E-004
Canada goose	4.38E-006	5.02E-005	4.38E-006	5.02E-005	4.38E-006	5.02E-005
Common barn owl	2.07E-006	4.15E-006	2.07E-006	4.15E-006	2.07E-006	4.15E-006
Osprey	-0-	5.76E-010	-0-	2.15E-010	-0-	2.15E-010
Pacific chorus frog	1.80E-003	7.50E-003	1.80E-003	7.50E-003	1.80E-003	7.50E-003
Gopher snake	3.54E-006	1.78E-004	3.54E-006	1.78E-004	3.54E-006	1.78E-004
Western fence lizard	3.28E-005	1.36E-004	3.28E-005	1.36E-004	3.28E-005	1.36E-004
E&T and Sensitive Terrestrial Species						
Spotted owl	1.01E-006	7.26E-005	1.01E-006	7.26E-005	1.01E-006	7.26E-005
Bald eagle	1.58E-007	3.82E-005	1.58E-007	3.82E-005	1.58E-007	3.82E-005
Western pond turtle	4.73E-004	1.95E-003	4.73E-004	1.95E-003	4.73E-004	1.95E-003
Common kingsnake	9.40E-005	1.83E-003	9.40E-005	1.83E-003	9.40E-005	1.83E-003

*Includes additive risks from other ingredients in formulation.
**HPHS = High-Pressure Hydraulic Sprayer
***HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-5. Terrestrial Risks from Horticultural Oil--HPHS*

	Risk Quotient	
Animal	**Typ**	**Max**
General Terrestrial Species		
Deer	2.15E-005	1.47E-004
Coyote	1.26E-006	1.41E-004
Long-tailed vole	2.60E-003	9.61E-003
Long-eared myotis	6.62E-006	1.74E-003
Pocket gopher	2.56E-002	8.59E-002
Raccoon	3.38E-005	2.75E-004
Dog	-0-	1.12E-004
Black-capped chickadee	2.92E-002	1.01E-001
Western bluebird	8.52E-003	3.15E-002
Mallard duck	6.23E-005	1.12E-003
Great blue heron	3.00E-005	7.89E-004
Song sparrow	9.28E-003	3.44E-002
Tree swallow	1.49E-007	3.50E-003
Canada goose	3.12E-005	6.16E-004
Common barn owl	1.48E-005	2.95E-005
Osprey	-0-	-0-
Pacific chorus frog	6.05E-002	2.03E-001
Gopher snake	3.90E-005	2.79E-003
Western fence lizard	1.10E-003	3.68E-003
E&T and Sensitive Terrestrial Species		
Spotted owl	7.22E-006	1.14E-003
Bald eagle	1.13E-006	6.06E-004
Western pond turtle	1.59E-002	5.30E-002
Common kingsnake	3.16E-003	3.25E-002

*HPHS = High-Pressure Hydraulic Sprayer

Table 9-6. Terrestrial Risks from Permethrin*

	Risk Quotient					
	HPHS**		HHW***		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species						
Deer	9.89E-005	8.35E-004	2.89E-006	8.37E-005	2.89E-006	8.37E-005
Coyote	5.78E-006	8.23E-004	1.71E-007	1.22E-004	1.69E-007	1.22E-004
Long-tailed vole	2.53E-003	1.57E-002	2.53E-004	2.36E-003	2.53E-004	2.36E-003
Long-eared myotis	3.04E-005	1.01E-002	8.89E-007	1.51E-003	8.89E-007	1.51E-003
Pocket gopher	2.49E-002	1.04E-001	2.49E-003	1.57E-002	2.49E-003	1.57E-002
Raccoon	1.55E-004	1.57E-003	4.54E-006	1.70E-004	4.53E-006	1.70E-004
Dog	-0-	6.53E-004	-0-	9.85E-005	-0-	9.85E-005
Black-capped chickadee	4.22E-003	1.90E-002	2.43E-004	1.79E-003	2.43E-004	1.79E-003
Western bluebird	5.14E-004	3.61E-003	5.14E-005	5.44E-004	5.14E-005	5.44E-004
Mallard duck	1.86E-005	5.39E-004	5.43E-007	7.32E-005	5.43E-007	7.32E-005
Great blue heron	1.76E-006	3.70E-004	3.86E-007	5.61E-005	1.81E-007	5.56E-005
Song sparrow	5.60E-004	4.00E-003	5.60E-005	6.03E-004	5.60E-005	6.03E-004
Tree swallow	4.25E-008	1.76E-003	1.24E-009	2.66E-004	1.24E-009	2.66E-004
Canada goose	8.88E-006	2.93E-004	2.60E-007	4.04E-005	2.60E-007	4.04E-005
Common barn owl	4.10E-006	8.19E-006	1.34E-007	2.69E-007	1.23E-007	2.46E-007
Osprey	-0-	1.27E-010	-0-	2.54E-012	-0-	2.54E-012
Pacific chorus frog	3.65E-003	1.53E-002	3.65E-004	2.31E-003	3.65E-004	2.31E-003
Gopher snake	6.99E-006	1.38E-003	4.16E-007	2.07E-004	3.25E-007	2.06E-004
Western fence lizard	6.65E-005	2.71E-004	6.65E-006	4.09E-005	6.65E-006	4.09E-005
E&T and Sensitive Terrestrial Species						
Spotted owl	2.00E-006	5.69E-004	6.86E-008	8.52E-005	6.01E-008	8.52E-005
Bald eagle	3.12E-007	3.04E-004	1.56E-008	4.58E-005	9.37E-009	4.58E-005
Western pond turtle	9.58E-004	3.88E-003	9.58E-005	5.84E-004	9.58E-005	5.84E-004
Common kingsnake	1.85E-004	1.35E-002	2.08E-005	2.03E-003	1.90E-005	2.02E-003

*Includes additive risks from other ingredients in formulation.
**HPHS = High-Pressure Hydraulic Sprayer
***HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-7. Terrestrial Risks from Propargite

	Risk Quotient					
	HPHS*		HHW**		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species						
Deer	7.69E-006	2.36E-004	7.69E-006	2.36E-004	7.69E-006	2.36E-004
Coyote	4.50E-007	3.25E-004	4.50E-007	3.25E-004	4.50E-007	3.25E-004
Long-tailed vole	8.14E-004	5.01E-003	8.14E-004	5.01E-003	8.14E-004	5.01E-003
Long-eared myotis	2.36E-006	4.02E-003	2.36E-006	4.02E-003	2.36E-006	4.02E-003
Pocket gopher	8.01E-003	2.92E-002	8.01E-003	2.92E-002	8.01E-003	2.92E-002
Raccoon	1.21E-005	4.74E-004	1.21E-005	4.74E-004	1.21E-005	4.74E-004
Dog	-0-	2.62E-004	-0-	2.62E-004	-0-	2.62E-004
Black-capped chickadee	1.48E-003	1.21E-002	1.48E-003	1.21E-002	1.48E-003	1.21E-002
Western bluebird	3.79E-004	2.33E-003	3.79E-004	2.33E-003	3.79E-004	2.33E-003
Mallard duck	3.16E-006	3.27E-004	3.16E-006	3.27E-004	3.16E-006	3.27E-004
Great blue heron	1.33E-006	2.45E-004	1.33E-006	2.45E-004	1.33E-006	2.45E-004
Song sparrow	4.13E-004	2.58E-003	4.13E-004	2.58E-003	4.13E-004	2.58E-003
Tree swallow	7.58E-009	1.16E-003	7.58E-009	1.16E-003	7.58E-009	1.16E-003
Canada goose	1.58E-006	1.82E-004	1.58E-006	1.82E-004	1.58E-006	1.82E-004
Common barn owl	7.49E-007	3.42E-006	7.49E-007	3.42E-006	7.49E-007	3.42E-006
Osprey	-0-	1.03E-010	-0-	1.03E-010	-0-	1.03E-010
Pacific chorus frog	2.69E-003	9.71E-003	2.69E-003	9.71E-003	2.69E-003	9.71E-003
Gopher snake	1.98E-006	9.10E-004	1.98E-006	9.10E-004	1.98E-006	9.10E-004
Western fence lizard	4.90E-005	1.72E-004	4.90E-005	1.72E-004	4.90E-005	1.72E-004
E&T and Sensitive Terrestrial Species						
Spotted owl	3.66E-007	3.74E-004	3.66E-007	3.74E-004	3.66E-007	3.74E-004
Bald eagle	5.71E-008	2.01E-004	5.71E-008	2.01E-004	5.71E-008	2.01E-004
Western pond turtle	7.07E-004	2.45E-003	7.07E-004	2.45E-003	7.07E-004	2.45E-003
Common kingsnake	1.40E-004	8.97E-003	1.40E-004	8.97E-003	1.40E-004	8.97E-003

*HPHS = High-Pressure Hydraulic Sprayer

**HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-8. Terrestrial Risks from Chlorothalonil

	Risk Quotient			
	HPHS*		HHW**	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	4.06E-006	3.93E-005	4.06E-006	3.93E-005
Coyote	2.38E-007	5.71E-006	2.38E-007	5.71E-006
Long-tailed vole	4.30E-004	1.75E-003	4.30E-004	1.75E-003
Long-eared myotis	1.25E-006	6.25E-005	1.25E-006	6.25E-005
Pocket gopher	4.23E-003	1.69E-002	4.23E-003	1.69E-002
Raccoon	6.37E-006	6.29E-005	6.37E-006	6.29E-005
Dog	-0-	3.34E-006	-0-	3.34E-006
Black-capped chickadee	5.15E-003	4.35E-002	5.15E-003	4.35E-002
Western bluebird	1.32E-003	5.36E-003	1.32E-003	5.36E-003
Mallard duck	4.40E-006	5.03E-005	4.40E-006	5.03E-005
Great blue heron	4.65E-006	2.96E-005	4.65E-006	2.96E-005
Song sparrow	1.44E-003	5.84E-003	1.44E-003	5.84E-003
Tree swallow	2.64E-008	9.79E-005	2.64E-008	9.79E-005
Canada goose	5.50E-006	6.45E-005	5.50E-006	6.45E-005
Common barn owl	2.61E-006	1.19E-005	2.61E-006	1.19E-005
Osprey	-0-	3.77E-011	-0-	-0-
Pacific chorus frog	9.36E-003	3.75E-002	9.36E-003	3.75E-002
Gopher snake	6.88E-006	1.07E-004	6.88E-006	1.07E-004
Western fence lizard	1.71E-004	6.83E-004	1.71E-004	6.83E-004
E&T and Sensitive Terrestrial Species				
Spotted owl	1.27E-006	3.73E-005	1.27E-006	3.73E-005
Bald eagle	1.99E-007	1.77E-005	1.99E-007	1.77E-005
Western pond turtle	2.46E-003	9.84E-003	2.46E-003	9.84E-003
Common kingsnake	4.89E-004	1.71E-003	4.89E-004	1.71E-003

*HPHS = High-Pressure Hydraulic Sprayer

**HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-9. Terrestrial Risks from Dicamba

	Risk Quotient							
	HHW*		Backpack		Boom		Wick	
Animal	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species								
Deer	7.56E-006	5.79E-004	7.56E-006	5.79E-004	7.56E-006	5.79E-004	5.04E-006	2.80E-004
Coyote	4.43E-007	9.72E-004	4.43E-007	9.72E-004	4.43E-007	9.72E-004	2.95E-007	4.86E-004
Long-tailed vole	1.52E-003	1.27E-002	1.52E-003	1.27E-002	1.52E-003	1.27E-002	1.52E-003	6.37E-003
Long-eared myotis	2.32E-006	1.20E-002	2.32E-006	1.20E-002	2.32E-006	1.20E-002	1.55E-006	6.02E-003
Pocket gopher	1.50E-002	6.52E-002	1.50E-002	6.52E-002	1.50E-002	6.52E-002	1.50E-002	3.26E-002
Raccoon	1.18E-005	1.22E-003	1.18E-005	1.22E-003	1.18E-005	1.22E-003	7.90E-006	5.95E-004
Dog	-0-	7.86E-004	-0-	7.86E-004	-0-	7.86E-004	-0-	3.93E-004
Black-capped chickadee	3.83E-003	3.68E-002	3.83E-003	3.68E-002	3.83E-003	3.68E-002	2.56E-003	1.33E-002
Western bluebird	1.87E-003	1.56E-002	1.87E-003	1.56E-002	1.87E-003	1.56E-002	1.87E-003	7.80E-003
Mallard duck	2.74E-006	8.22E-004	2.74E-006	8.22E-004	2.74E-006	8.22E-004	1.83E-006	4.07E-004
Great blue heron	6.57E-006	1.93E-003	6.57E-006	1.93E-003	6.58E-006	1.93E-003	6.57E-006	9.72E-004
Song sparrow	2.03E-003	1.73E-002	2.03E-003	1.73E-002	2.03E-003	1.73E-002	2.03E-003	8.67E-003
Tree swallow	1.96E-008	9.21E-003	1.96E-008	9.21E-003	1.98E-008	9.21E-003	1.31E-008	4.61E-003
Canada goose	4.10E-006	1.37E-003	4.10E-006	1.37E-003	4.10E-006	1.37E-003	2.73E-006	6.81E-004
Common barn owl	1.94E-006	6.47E-006	1.94E-006	6.47E-006	1.94E-006	6.47E-006	1.29E-006	3.88E-006
Osprey	-0-	-0-	-0-	-0-	1.26E-010	7.03E-010	-0-	-0-
Pacific chorus frog	1.33E-002	5.68E-002	1.33E-002	5.68E-002	1.33E-002	5.68E-002	1.33E-002	2.84E-002
Gopher snake	5.13E-006	7.15E-003	5.13E-006	7.15E-003	5.13E-006	7.15E-003	3.42E-006	3.58E-003
Western fence lizard	2.42E-004	9.94E-004	2.42E-004	9.94E-004	2.42E-004	9.94E-004	2.42E-004	4.97E-004
E&T and Sensitive Terrestrial Species								
Spotted owl	9.49E-007	2.96E-003	9.49E-007	2.96E-003	9.49E-007	2.96E-003	6.33E-007	1.48E-003
Bald eagle	1.48E-007	1.59E-003	1.48E-007	1.59E-003	1.48E-007	1.59E-003	9.87E-008	7.95E-004
Western pond turtle	3.48E-003	1.41E-002	3.48E-003	1.41E-002	3.48E-003	1.41E-002	2.71E-003	7.07E-003
Common kingsnake	5.01E-004	7.02E-002	5.01E-004	7.02E-002	5.01E-004	7.02E-002	3.34E-004	3.54E-002

*HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-10. Terrestrial Risks from Glyphosate

	Risk Quotient							
	HHW*		Backpack		Boom		Wick	
Animal	**Typ**	**Max**			**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species								
Deer	4.89E-006	2.54E-005	4.89E-006	2.54E-005	1.22E-006	2.03E-005	5.46E-007	2.32E-006
Coyote	4.90E-007	1.77E-005	4.90E-007	1.77E-005	1.22E-007	1.37E-005	5.47E-008	2.29E-006
Long-tailed vole	1.68E-003	4.31E-003	1.68E-003	4.31E-003	4.20E-004	3.45E-003	2.82E-004	5.78E-004
Long-eared myotis	2.57E-006	2.08E-004	2.57E-006	2.08E-004	6.42E-007	1.66E-004	2.87E-007	2.72E-005
Pocket gopher	1.65E-002	4.14E-002	1.65E-002	4.14E-002	4.13E-003	3.31E-002	2.77E-003	5.55E-003
Raccoon	1.31E-005	7.32E-005	1.31E-005	7.32E-005	3.28E-006	5.85E-005	1.46E-006	6.89E-006
Dog	-0-	1.29E-005	-0-	1.29E-005	-0-	1.03E-005	-0-	1.72E-006
Black-capped chickadee	2.68E-003	1.13E-002	2.68E-003	1.13E-002	6.70E-004	9.03E-003	2.99E-004	9.13E-004
Western bluebird	1.30E-003	3.35E-003	1.30E-003	3.35E-003	3.26E-004	2.68E-003	2.18E-004	4.48E-004
Mallard duck	5.72E-006	4.86E-005	5.72E-006	4.86E-005	1.43E-006	3.89E-005	6.39E-007	5.24E-006
Great blue heron	4.60E-006	2.90E-005	4.60E-006	2.90E-005	1.15E-006	1.82E-005	7.70E-007	4.20E-006
Song sparrow	1.42E-003	3.65E-003	1.42E-003	3.65E-003	3.55E-004	2.92E-003	2.38E-004	4.89E-004
Tree swallow	1.37E-008	9.53E-005	1.37E-008	9.53E-005	3.46E-009	7.63E-005	1.53E-009	1.28E-005
Canada goose	2.87E-006	2.59E-005	2.87E-006	2.59E-005	7.16E-007	2.07E-005	3.20E-007	2.83E-006
Common barn owl	1.36E-006	4.52E-006	1.36E-006	4.52E-006	3.39E-007	1.13E-006	1.52E-007	4.55E-007
Osprey	-0-	7.86E-015	-0-	8.91E-015	6.98E-012	3.00E-011	-0-	-0-
Pacific chorus frog	9.26E-003	2.32E-002	9.26E-003	2.32E-002	2.32E-003	1.86E-002	1.55E-003	3.11E-003
Gopher snake	3.58E-006	8.57E-005	3.58E-006	8.57E-005	8.96E-007	6.20E-005	4.00E-007	1.11E-005
Western fence lizard	1.69E-004	4.22E-004	1.69E-004	4.22E-004	4.22E-005	3.38E-004	2.83E-005	5.66E-005
E&T and Sensitive Terrestrial Species								
Spotted owl	6.63E-007	3.28E-005	6.63E-007	3.28E-005	1.66E-007	2.50E-005	7.41E-008	4.31E-006
Bald eagle	1.03E-007	1.68E-005	1.03E-007	1.68E-005	2.59E-008	1.32E-005	1.16E-008	2.24E-006
Western pond turtle	2.43E-003	6.09E-003	2.43E-003	6.09E-003	6.08E-004	4.87E-003	3.17E-004	8.15E-004
Common kingsnake	3.50E-004	1.68E-003	3.50E-004	1.68E-003	8.76E-005	8.11E-004	3.91E-005	2.13E-004

*HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-11. Terrestrial Risks from Hexazinone

	Risk Quotient			
	HHW*		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	8.95E-006	2.45E-004	8.95E-006	2.45E-004
Coyote	1.33E-007	5.92E-005	1.33E-007	5.92E-005
Long-tailed vole	1.80E-003	1.60E-002	1.80E-003	1.60E-002
Long-eared myotis	2.75E-006	2.89E-003	2.75E-006	2.89E-003
Pocket gopher	1.77E-002	1.43E-001	1.77E-002	1.43E-001
Raccoon	1.40E-005	4.57E-004	1.40E-005	4.57E-004
Dog	-0-	4.71E-005	-0-	4.71E-005
Black-capped chickadee	2.06E-003	2.86E-002	2.06E-003	2.86E-002
Western bluebird	1.00E-003	8.88E-003	1.00E-003	8.88E-003
Mallard duck	4.39E-006	3.16E-004	4.39E-006	3.16E-004
Great blue heron	3.53E-006	2.13E-004	3.53E-006	2.13E-004
Song sparrow	1.09E-003	9.71E-003	1.09E-003	9.71E-003
Tree swallow	1.05E-008	9.89E-004	1.05E-008	9.89E-004
Canada goose	2.20E-006	1.74E-004	2.20E-006	1.74E-004
Common barn owl	1.04E-006	3.47E-006	1.04E-006	3.47E-006
Osprey	-0-	3.12E-016	-0-	3.12E-016
Pacific chorus frog	7.11E-003	5.73E-002	7.11E-003	5.73E-002
Gopher snake	2.75E-006	7.75E-004	2.75E-006	7.75E-004
Western fence lizard	1.30E-004	1.04E-003	1.30E-004	1.04E-003
E&T and Sensitive Terrestrial Species				
Spotted owl	5.09E-007	3.19E-004	5.09E-007	3.19E-004
Bald eagle	7.94E-008	1.71E-004	7.94E-008	1.71E-004
Western pond turtle	1.87E-003	1.50E-002	1.87E-003	1.50E-002
Common kingsnake	2.69E-004	8.13E-003	2.69E-004	8.13E-003

*HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-12. Terrestrial Risks from Picloram

	Risk Quotient			
	HHW*		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	1.43E-006	2.30E-005	1.43E-006	2.30E-005
Coyote	3.13E-008	3.00E-006	3.13E-008	3.00E-006
Long-tailed vole	1.08E-004	8.79E-004	1.08E-004	8.79E-004
Long-eared myotis	1.64E-007	3.62E-005	1.64E-007	3.62E-005
Pocket gopher	1.06E-003	8.48E-003	1.06E-003	8.48E-003
Raccoon	8.38E-007	1.44E-005	8.38E-007	1.44E-005
Dog	-0-	2.22E-006	-0-	2.22E-006
Black-capped chickadee	1.08E-004	1.45E-003	1.08E-004	1.45E-003
Western bluebird	5.23E-005	4.28E-004	5.23E-005	4.28E-004
Mallard duck	6.41E-007	1.61E-005	6.41E-007	1.61E-005
Great blue heron	1.84E-007	2.52E-006	1.84E-007	2.52E-006
Song sparrow	5.70E-005	4.66E-004	5.70E-005	4.66E-004
Tree swallow	5.51E-010	1.03E-005	5.51E-010	1.03E-005
Canada goose	1.15E-007	3.04E-006	1.15E-007	3.04E-006
Common barn owl	5.45E-008	1.82E-007	5.45E-008	1.82E-007
Osprey	-0-	-0-	-0-	-0-
Pacific chorus frog	3.72E-004	2.98E-003	3.72E-004	2.98E-003
Gopher snake	1.44E-007	8.48E-006	1.44E-007	8.48E-006
Western fence lizard	6.77E-006	5.42E-005	6.77E-006	5.42E-005
E&T and Sensitive Terrestrial Species				
Spotted owl	2.66E-008	3.41E-006	2.66E-008	3.41E-006
Bald eagle	4.15E-009	1.80E-006	4.15E-009	1.80E-006
Western pond turtle	9.76E-005	7.81E-004	9.76E-005	7.81E-004
Common kingsnake	1.41E-005	1.16E-004	1.41E-005	1.16E-004

*HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-13. Terrestrial Risks from Triclopyr

	Risk Quotient			
	HHW*		Backpack	
Animal	**Typ**	**Max**	**Typ**	**Max**
General Terrestrial Species				
Deer	1.11E-005	3.69E-004	1.11E-005	3.69E-004
Coyote	6.51E-007	3.20E-004	6.51E-007	3.20E-004
Long-tailed vole	2.23E-003	2.60E-002	2.23E-003	2.60E-002
Long-eared myotis	3.42E-006	3.95E-003	3.42E-006	3.95E-003
Pocket gopher	2.20E-002	2.36E-001	2.20E-002	2.36E-001
Raccoon	1.74E-005	6.78E-004	1.74E-005	6.78E-004
Dog	-0-	2.54E-004	-0-	2.54E-004
Black-capped chickadee	7.32E-003	1.35E-001	7.32E-003	1.35E-001
Western bluebird	3.56E-003	4.14E-002	3.56E-003	4.14E-002
Mallard duck	4.87E-006	4.00E-004	4.87E-006	4.00E-004
Great blue heron	1.26E-005	8.31E-004	1.26E-005	8.31E-004
Song sparrow	3.88E-003	4.53E-002	3.88E-003	4.53E-002
Tree swallow	3.75E-008	3.87E-003	3.75E-008	3.87E-003
Canada goose	7.83E-006	7.05E-004	7.83E-006	7.05E-004
Common barn owl	3.71E-006	1.24E-005	3.71E-006	1.24E-005
Osprey	-0-	2.98E-014	-0-	2.98E-014
Pacific chorus frog	2.53E-002	2.72E-001	2.53E-002	2.72E-001
Gopher snake	9.79E-006	3.03E-003	9.79E-006	3.03E-003
Western fence lizard	4.61E-004	4.93E-003	4.61E-004	4.93E-003
E&T and Sensitive Terrestrial Species				
Spotted owl	1.81E-006	1.25E-003	1.81E-006	1.25E-003
Bald eagle	2.83E-007	6.68E-004	2.83E-007	6.68E-004
Western pond turtle	6.65E-003	7.10E-002	6.65E-003	7.10E-002
Common kingsnake	9.57E-004	3.16E-002	9.57E-004	3.16E-002

*HHW = Hydraulic Sprayer with Hand-Held Wand

Table 9-14. Terrestrial Risks from Fertilizers*

Animal	Risk Quotient Typ	Max
General Terrestrial Species		
Deer	8.41E-004	1.53E-003
Coyote	3.17E-004	4.59E-004
Long-tailed vole	6.16E-006	7.70E-003
Long-eared myotis	-0-	2.15E-003
Pocket gopher	4.16E-002	7.57E-002
Raccoon	1.50E-002	2.16E-002
Dog	8.10E-005	1.16E-004
Black-capped chickadee	4.14E-003	7.54E-003
Western bluebird	2.81E-003	4.54E-003
Mallard duck	1.56E-003	2.65E-003
Great blue heron	3.86E-004	5.51E-004
Song sparrow	-0-	2.92E-012
Tree swallow	2.60E-007	4.73E-007
Canada goose	5.43E-004	9.87E-004
Common barn owl	1.93E-004	2.76E-004
Osprey	-0-	7.07E-013
Pacific chorus frog	-0-	5.78E-012
Gopher snake	-0-	1.06E-012
Western fence lizard	-0-	3.51E-012
E&T and Sensitive Terrestrial Species		
Spotted owl	-0-	9.44E-013
Bald eagle	-0-	4.89E-013
Western pond turtle	1.15E-002	2.10E-002
Common kingsnake	4.10E-003	5.86E-003

*Additive risk from all fertilizer chemicals used.

Table 9-15. Aquatic Risks from Acephate--Implant Capsules

			Exceeds MATC?*	
Species	**Typ**	**Max**	**Typ**	**Max**
Irrigation Ditches				
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	no	no
Williams Creek				
Rainbow trout	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND
Applegate River				
Rainbow trout	-0-	-0-	ND	ND
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	no	no
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND

*ND= No data

Table 9-16. Aquatic Risks from Chlorpyrifos-High-Pressure Hydraulic Sprayer

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	2.44E-004	ND	ND
Pacific chorus frog tadpole	-0-	1.62E-008	no	no
Williams Creek				
Rainbow trout	-0-	5.17E-007	no	no
Coho salmon	-0-	5.17E-007	no	no
Chinook salmon	-0-	5.17E-007	no	no
Cutthroat trout	-0-	1.16E-007	no	no
Steelhead	-0-	5.17E-007	no	no
Pacific lamprey	-0-	5.17E-007	no	no
Applegate River				
Rainbow trout	-0-	2.01E-007	no	no
Daphnia magna	-0-	1.21E-005	ND	ND
Pacific chorus frog tadpole	-0-	8.05E-010	no	no
Coho salmon	-0-	2.01E-007	no	no
Chinook salmon	-0-	2.01E-007	no	no
Cutthroat trout	-0-	4.50E-008	no	no
Steelhead	-0-	2.01E-007	no	no
Pacific lamprey	-0-	2.01E-007	no	no

*ND= No data

Table 9-17. Aquatic Risks from Chlorpyrifos-Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	2.44E-004	ND	ND
Pacific chorus frog tadpole	-0-	1.62E-008	no	no
Williams Creek				
Rainbow trout	-0-	5.17E-007	no	no
Coho salmon	-0-	5.17E-007	no	no
Chinook salmon	-0-	5.17E-007	no	no
Cutthroat trout	-0-	1.16E-007	no	no
Steelhead	-0-	5.17E-007	no	no
Pacific lamprey	-0-	5.17E-007	no	no
Applegate River				
Rainbow trout	-0-	2.01E-007	no	no
Daphnia magna	-0-	1.21E-005	ND	ND
Pacific chorus frog tadpole	-0-	8.05E-010	no	no
Coho salmon	-0-	2.01E-007	no	no
Chinook salmon	-0-	2.01E-007	no	no
Cutthroat trout	-0-	4.50E-008	no	no
Steelhead	-0-	2.01E-007	no	no
Pacific lamprey	-0-	2.01E-007	no	no

*ND= No data

Table 9-18. Aquatic Risks from Diazinon-High-Pressure Hydraulic Sprayer

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.83E-006	no	no
Pacific chorus frog tadpole	-0-	1.46E-008	ND	ND
Williams Creek				
Rainbow trout	-0-	9.03E-009	no	no
Coho salmon	-0-	9.03E-009	no	no
Chinook salmon	-0-	9.03E-009	no	no
Cutthroat trout	-0-	4.78E-010	no	no
Steelhead	-0-	9.03E-009	no	no
Pacific lamprey	-0-	9.03E-009	ND	ND
Applegate River				
Rainbow trout	-0-	5.88E-010	no	no
Daphnia magna	-0-	1.32E-007	no	no
Pacific chorus frog tadpole	-0-	1.06E-009	ND	ND
Coho salmon	-0-	5.88E-010	no	no
Chinook salmon	-0-	5.88E-010	no	no
Cutthroat trout	-0-	3.11E-011	no	no
Steelhead	-0-	5.88E-010	no	no
Pacific lamprey	-0-	5.88E-010	ND	ND

*ND= No data

Table 9-19. Aquatic Risks from Diazinon-Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.83E-006	no	no
Pacific chorus frog tadpole	-0-	1.46E-008	ND	ND
Williams Creek				
Rainbow trout	-0-	9.03E-009	no	no
Coho salmon	-0-	9.03E-009	no	no
Chinook salmon	-0-	9.03E-009	no	no
Cutthroat trout	-0-	4.78E-010	no	no
Steelhead	-0-	9.03E-009	no	no
Pacific lamprey	-0-	9.03E-009	ND	ND
Applegate River				
Rainbow trout	-0-	5.88E-010	no	no
Daphnia magna	-0-	1.32E-007	no	no
Pacific chorus frog tadpole	-0-	1.06E-009	ND	ND
Coho salmon	-0-	5.88E-010	no	no
Chinook salmon	-0-	5.88E-010	no	no
Cutthroat trout	-0-	3.11E-011	no	no
Steelhead	-0-	5.88E-010	no	no
Pacific lamprey	-0-	5.88E-010	ND	ND

*ND= No data

Table 9-20. Aquatic Risks from Dimethoate*--Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.68E-009	ID	ID
Pacific chorus frog tadpole	-0-	8.39E-010	ID	ID
Williams Creek				
Rainbow trout	-0-	2.00E-011	ID	ID
Coho salmon	-0-	2.00E-011	ID	ID
Chinook salmon	-0-	2.00E-011	ID	ID
Cutthroat trout	-0-	2.00E-011	ID	ID
Steelhead	-0-	2.00E-011	ID	ID
Pacific lamprey	-0-	2.00E-011	ID	ID
Applegate River				
Rainbow trout	-0-	7.78E-012	ID	ID
Daphnia magna	-0-	7.08E-010	ID	ID
Pacific chorus frog tadpole	-0-	3.54E-010	ID	ID
Coho salmon	-0-	7.78E-012	ID	ID
Chinook salmon	-0-	7.78E-012	ID	ID
Cutthroat trout	-0-	7.78E-012	ID	ID
Steelhead	-0-	7.78E-012	ID	ID
Pacific lamprey	-0-	7.78E-012	ID	ID

*Includes additive risks from other ingredients in formulation.
**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-21. Aquatic Risks from Dimethoate*--Backpack

Species	Typical	Maximum	Exceeds MATC?** Typical	Exceeds MATC?** Maximum
Irrigation Ditches				
Daphnia magna	-0-	1.68E-009	ID	ID
Pacific chorus frog tadpole	-0-	8.39E-010	ID	ID
Williams Creek				
Rainbow trout	-0-	2.00E-011	ID	ID
Coho salmon	-0-	2.00E-011	ID	ID
Chinook salmon	-0-	2.00E-011	ID	ID
Cutthroat trout	-0-	2.00E-011	ID	ID
Steelhead	-0-	2.00E-011	ID	ID
Pacific lamprey	-0-	2.00E-011	ID	ID
Applegate River				
Rainbow trout	-0-	7.78E-012	ID	ID
Daphnia magna	-0-	7.08E-010	ID	ID
Pacific chorus frog tadpole	-0-	3.54E-010	ID	ID
Coho salmon	-0-	7.78E-012	ID	ID
Chinook salmon	-0-	7.78E-012	ID	ID
Cutthroat trout	-0-	7.78E-012	ID	ID
Steelhead	-0-	7.78E-012	ID	ID
Pacific lamprey	-0-	7.78E-012	ID	ID

*Includes additive risks from other ingredients in formulation.

**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-22. Aquatic Risks from Esfenvalerate*--High-Pressure Hydraulic Sprayer

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	4.60E-006	ID	ID
Pacific chorus frog tadpole	-0-	8.52E-008	ID	ID
Williams Creek				
Rainbow trout	-0-	7.63E-007	ID	ID
Coho salmon	-0-	2.25E-006	ID	ID
Chinook salmon	-0-	2.25E-006	ID	ID
Cutthroat trout	-0-	2.25E-006	ID	ID
Steelhead	-0-	2.25E-006	ID	ID
Pacific lamprey	-0-	2.25E-006	ID	ID
Applegate River				
Rainbow trout	-0-	6.23E-014	ID	ID
Daphnia magna	-0-	2.42E-013	ID	ID
Pacific chorus frog tadpole	-0-	2.42E-013	ID	ID
Coho salmon	-0-	6.23E-014	ID	ID
Chinook salmon	-0-	6.23E-014	ID	ID
Cutthroat trout	-0-	6.23E-014	ID	ID
Steelhead	-0-	6.23E-014	ID	ID
Pacific lamprey	-0-	6.23E-014	ID	ID

*Includes additive risks from other ingredients in formulation.

**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-23. Aquatic Risks from Esfenvalerate*--Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.71E-006	ID	ID
Pacific chorus frog tadpole	-0-	3.20E-008	ID	ID
Williams Creek				
Rainbow trout	-0-	9.64E-007	ID	ID
Coho salmon	-0-	2.85E-006	ID	ID
Chinook salmon	-0-	2.85E-006	ID	ID
Cutthroat trout	-0-	2.85E-006	ID	ID
Steelhead	-0-	2.85E-006	ID	ID
Pacific lamprey	-0-	2.85E-006	ID	ID
Applegate River				
Rainbow trout	-0-	3.75E-007	ID	ID
Daphnia magna	-0-	7.23E-007	ID	ID
Pacific chorus frog tadpole	-0-	1.34E-008	ID	ID
Coho salmon	-0-	1.11E-006	ID	ID
Chinook salmon	-0-	1.11E-006	ID	ID
Cutthroat trout	-0-	1.11E-006	ID	ID
Steelhead	-0-	1.11E-006	ID	ID
Pacific lamprey	-0-	1.11E-006	ID	ID

*Includes additive risks from other ingredients in formulation.
**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-24. Aquatic Risks from Esfenvalerate*--Backpack

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.71E-006	ID	ID
Pacific chorus frog tadpol	-0-	3.17E-008	ID	ID
Williams Creek				
Rainbow trout	-0-	9.64E-007	ID	ID
Coho salmon	-0-	2.85E-006	ID	ID
Chinook salmon	-0-	2.85E-006	ID	ID
Cutthroat trout	-0-	2.85E-006	ID	ID
Steelhead	-0-	2.85E-006	ID	ID
Pacific lamprey	-0-	2.85E-006	ID	ID
Applegate River				
Rainbow trout	-0-	3.75E-007	ID	ID
Daphnia magna	-0-	7.23E-007	ID	ID
Pacific chorus frog tadpol	-0-	1.34E-008	ID	ID
Coho salmon	-0-	1.11E-006	ID	ID
Chinook salmon	-0-	1.11E-006	ID	ID
Cutthroat trout	-0-	1.11E-006	ID	ID
Steelhead	-0-	1.11E-006	ID	ID
Pacific lamprey	-0-	1.11E-006	ID	ID

*Includes additive risks from other ingredients in formulation.
**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-25. Aquatic Risks from Horticultural Oil--High-Pressure Hydraulic Sprayer

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND
Applegate River				
Rainbow trout	-0-	1.42E-012	ND	ND
Daphnia magna	-0-	1.29E-010	ND	ND
Pacific chorus frog tadpole	-0-	6.46E-011	ND	ND
Coho salmon	-0-	1.42E-012	ND	ND
Chinook salmon	-0-	1.42E-012	ND	ND
Cutthroat trout	-0-	1.42E-012	ND	ND
Steelhead	-0-	1.42E-012	ND	ND
Pacific lamprey	-0-	1.42E-012	ND	ND

*ND= No data

Table 9-26. Aquatic Risks from Permethrin*--High-Pressure Hydraulic Sprayer

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	8.32E-007	ID	ID
Pacific chorus frog tadpole	-0-	5.18E-009	ID	ID
Williams Creek				
Rainbow trout	-0-	1.56E-007	ID	ID
Coho salmon	-0-	1.56E-007	ID	ID
Chinook salmon	-0-	1.56E-007	ID	ID
Cutthroat trout	-0-	1.56E-007	ID	ID
Steelhead	-0-	1.56E-007	ID	ID
Pacific lamprey	-0-	1.56E-007	ID	ID
Applegate River				
Rainbow trout	-0-	2.70E-007	ID	ID
Daphnia magna	-0-	4.61E-007	ID	ID
Pacific chorus frog tadpole	-0-	2.89E-009	ID	ID
Coho salmon	-0-	2.70E-007	ID	ID
Chinook salmon	-0-	2.70E-007	ID	ID
Cutthroat trout	-0-	2.70E-007	ID	ID
Steelhead	-0-	2.70E-007	ID	ID
Pacific lamprey	-0-	2.70E-007	ID	ID

*Includes additive risks from other ingredients in formulation.

**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-27. Aquatic Risks from Permethrin*--Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	6.48E-009	ID	ID
Pacific chorus frog tadpole	-0-	8.39E-011	ID	ID
Williams Creek				
Rainbow trout	-0-	4.14E-009	ID	ID
Coho salmon	-0-	4.11E-009	ID	ID
Chinook salmon	-0-	4.14E-009	ID	ID
Cutthroat trout	-0-	4.14E-009	ID	ID
Steelhead	-0-	4.14E-009	ID	ID
Pacific lamprey	-0-	4.14E-009	ID	ID
Applegate River				
Rainbow trout	-0-	1.61E-009	ID	ID
Daphnia magna	-0-	2.73E-009	ID	ID
Pacific chorus frog tadpole	-0-	3.38E-011	ID	ID
Coho salmon	-0-	1.60E-009	ID	ID
Chinook salmon	-0-	1.61E-009	ID	ID
Cutthroat trout	-0-	1.61E-009	ID	ID
Steelhead	-0-	1.61E-009	ID	ID
Pacific lamprey	-0-	1.61E-009	ID	ID

*Includes additive risks from other ingredients in formulation.

**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-28. Aquatic Risks from Permethrin*--Backpack

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	6.48E-009	ID	ID
Pacific chorus frog tadpole	-0-	8.39E-011	ID	ID
Williams Creek				
Rainbow trout	-0-	4.14E-009	ID	ID
Coho salmon	-0-	4.11E-009	ID	ID
Chinook salmon	-0-	4.14E-009	ID	ID
Cutthroat trout	-0-	4.14E-009	ID	ID
Steelhead	-0-	4.14E-009	ID	ID
Pacific lamprey	-0-	4.14E-009	ID	ID
Applegate River				
Rainbow trout	-0-	1.61E-009	ID	ID
Daphnia magna	-0-	2.73E-009	ID	ID
Pacific chorus frog tadpole	-0-	3.38E-011	ID	ID
Coho salmon	-0-	1.60E-009	ID	ID
Chinook salmon	-0-	1.61E-009	ID	ID
Cutthroat trout	-0-	1.61E-009	ID	ID
Steelhead	-0-	1.61E-009	ID	ID
Pacific lamprey	-0-	1.61E-009	ID	ID

*Includes additive risks from other ingredients in formulation.

**ND= No data; ID = Incomplete data: known MATCs not exceeded, but MATCs not available for all ingredients assessed

Table 9-29. Aquatic Risks from Propargite--High-Pressure Hydraulic Sprayer

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.19E-007	no	no
Pacific chorus frog tadpole	-0-	1.19E-007	ND	ND
Williams Creek				
Rainbow trout	-0-	9.46E-009	no	no
Coho salmon	-0-	9.46E-009	no	no
Chinook salmon	-0-	9.46E-009	no	no
Cutthroat trout	-0-	9.46E-009	no	no
Steelhead	-0-	9.46E-009	no	no
Pacific lamprey	-0-	9.46E-009	no	no
Applegate River				
Rainbow trout	-0-	1.58E-008	no	no
Daphnia magna	-0-	5.03E-008	no	no
Pacific chorus frog tadpole	-0-	5.03E-008	ND	ND
Coho salmon	-0-	1.58E-008	no	no
Chinook salmon	-0-	1.58E-008	no	no
Cutthroat trout	-0-	1.58E-008	no	no
Steelhead	-0-	1.58E-008	no	no
Pacific lamprey	-0-	1.58E-008	no	no

*ND= No data

Table 9-30. Aquatic Risks from Propargite--Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	1.19E-007	no	no
Pacific chorus frog tadpole	-0-	1.19E-007	ND	ND
Williams Creek				
Rainbow trout	-0-	9.46E-009	no	no
Coho salmon	-0-	9.46E-009	no	no
Chinook salmon	-0-	9.46E-009	no	no
Cutthroat trout	-0-	9.46E-009	no	no
Steelhead	-0-	9.46E-009	no	no
Pacific lamprey	-0-	9.46E-009	no	no
Applegate River				
Rainbow trout	-0-	1.58E-008	no	no
Daphnia magna	-0-	5.03E-008	no	no
Pacific chorus frog tadpole	-0-	5.03E-008	ND	ND
Coho salmon	-0-	1.58E-008	no	no
Chinook salmon	-0-	1.58E-008	no	no
Cutthroat trout	-0-	1.58E-008	no	no
Steelhead	-0-	1.58E-008	no	no
Pacific lamprey	-0-	1.58E-008	no	no

*ND= No data

Table 9-31. Aquatic Risks from Propargite--Backpack

Species	Typical	Maximum	Exceeds MATC?* Typical	Exceeds MATC?* Maximum
Irrigation Ditches				
Daphnia magna	-0-	1.19E-007	no	no
Pacific chorus frog tadpole	-0-	1.19E-007	ND	ND
Williams Creek				
Rainbow trout	-0-	9.46E-009	no	no
Coho salmon	-0-	9.46E-009	no	no
Chinook salmon	-0-	9.46E-009	no	no
Cutthroat trout	-0-	9.46E-009	no	no
Steelhead	-0-	9.46E-009	no	no
Pacific lamprey	-0-	9.46E-009	no	no
Applegate River				
Rainbow trout	-0-	1.58E-008	no	no
Daphnia magna	-0-	5.03E-008	no	no
Pacific chorus frog tadpole	-0-	5.03E-008	ND	ND
Coho salmon	-0-	1.58E-008	no	no
Chinook salmon	-0-	1.58E-008	no	no
Cutthroat trout	-0-	1.58E-008	no	no
Steelhead	-0-	1.58E-008	no	no
Pacific lamprey	-0-	1.58E-008	no	no

*ND= No data

Table 9-32. Aquatic Risks from Chlorothalonil--High-Pressure Hydraulic Sprayer

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	6.01E-008	ND	ND
Pacific chorus frog tadpole	-0-	2.55E-008	ND	ND
Williams Creek				
Rainbow trout	-0-	3.88E-008	no	no
Coho salmon	-0-	3.88E-008	no	no
Chinook salmon	-0-	3.88E-008	no	no
Cutthroat trout	-0-	3.88E-008	no	no
Steelhead	-0-	3.88E-008	no	no
Pacific lamprey	-0-	3.88E-008	no	no
Applegate River				
Rainbow trout	-0-	2.04E-008	no	no
Daphnia magna	-0-	2.53E-008	ND	ND
Pacific chorus frog tadpole	-0-	1.08E-008	ND	ND
Coho salmon	-0-	2.04E-008	no	no
Chinook salmon	-0-	2.04E-008	no	no
Cutthroat trout	-0-	2.04E-008	no	no
Steelhead	-0-	2.04E-008	no	no
Pacific lamprey	-0-	2.04E-008	no	no

*ND= No data

Table 9-33. Aquatic Risks from Chlorothalonil--Hydraulic Sprayer with Hand-Held Wand

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Williams Creek				
Rainbow trout	-0-	3.88E-008	no	no
Coho salmon	-0-	3.88E-008	no	no
Chinook salmon	-0-	3.88E-008	no	no
Cutthroat trout	-0-	3.88E-008	no	no
Steelhead	-0-	3.88E-008	no	no
Pacific lamprey	-0-	3.88E-008	no	no
Applegate River				
Rainbow trout	-0-	2.04E-008	no	no
Daphnia magna	-0-	2.53E-008	ND	ND
Pacific chorus frog tadpole	-0-	1.08E-008	ND	ND
Coho salmon	-0-	2.04E-008	no	no
Chinook salmon	-0-	2.04E-008	no	no
Cutthroat trout	-0-	2.04E-008	no	no
Steelhead	-0-	2.04E-008	no	no
Pacific lamprey	-0-	2.04E-008	no	no

*ND= No data

Table 9-34. Aquatic Risks from Dicamba--HHW*, Backpack, or Wick

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND
Applegate River				
Rainbow trout	-0-	-0-	ND	ND
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND

*HHW = Hydraulic Sprayer with Hand-Held Wand
**ND= No data

Table 9-35. Aquatic Risks from Dicamba--Tractor-Pulled Spray Boom

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	1.61E-010	3.47E-010	ND	ND
Pacific chorus frog tadpole	2.52E-009	5.42E-009	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND
Applegate River				
Rainbow trout	-0-	-0-	ND	ND
Daphnia magna	-0-	-0-	ND	ND
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	ND	ND
Chinook salmon	-0-	-0-	ND	ND
Cutthroat trout	-0-	-0-	ND	ND
Steelhead	-0-	-0-	ND	ND
Pacific lamprey	-0-	-0-	ND	ND

*ND= No data

Table 9-36. Aquatic Risks from Glyphosate--HHW* or Backpack

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	3.13E-013	no	no
Pacific chorus frog tadpole	-0-	3.92E-013	ND	ND
Williams Creek				
Rainbow trout	-0-	1.58E-013	no	no
Coho salmon	-0-	2.36E-012	no	no
Chinook salmon	-0-	2.83E-012	no	no
Cutthroat trout	-0-	2.83E-012	no	no
Steelhead	-0-	2.83E-012	no	no
Pacific lamprey	-0-	2.83E-012	no	no
Applegate River				
Rainbow trout	-0-	6.14E-014	no	no
Daphnia magna	-0-	1.32E-013	no	no
Pacific chorus frog tadpole	-0-	1.65E-013	ND	ND
Coho salmon	-0-	9.18E-013	no	no
Chinook salmon	-0-	1.10E-012	no	no
Cutthroat trout	-0-	1.10E-012	no	no
Steelhead	-0-	1.10E-012	no	no
Pacific lamprey	-0-	1.10E-012	no	no

*HHW = Hydraulic Sprayer with Hand-Held Wand
**ND= No data

Table 9-37. Aquatic Risks from Glyphosate--Tractor-Pulled Spray Boom

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	2.78E-010	1.19E-009	no	no
Pacific chorus frog tadpole	3.47E-010	1.49E-009	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	no	no
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no
Applegate River				
Rainbow trout	-0-	-0-	no	no
Daphnia magna	-0-	-0-	no	no
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no

*ND= No data

Table 9-38. Aquatic Risks from Glyphosate--Wick

			Exceeds MATC?*	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	-0-	no	no
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	no	no
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no
Applegate River				
Rainbow trout	-0-	-0-	no	no
Daphnia magna	-0-	-0-	no	no
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no

*ND= No data

Table 9-39. Aquatic Risks from Hexazinone--HHW* or Backpack

Species	Typical	Maximum	Exceeds MATC?** Typical	Exceeds MATC?** Maximum
Irrigation Ditches				
Daphnia magna	-0-	2.94E-014	no	no
Pacific chorus frog tadpole	-0-	2.94E-014	no	no
Williams Creek				
Rainbow trout	-0-	4.94E-014	no	no
Coho salmon	-0-	5.45E-014	no	no
Chinook salmon	-0-	5.45E-014	no	no
Cutthroat trout	-0-	5.45E-014	no	no
Steelhead	-0-	5.45E-014	no	no
Pacific lamprey	-0-	5.45E-014	no	no
Applegate River				
Rainbow trout	-0-	3.22E-015	no	no
Daphnia magna	-0-	3.38E-015	no	no
Pacific chorus frog tadpole	-0-	3.38E-015	no	no
Coho salmon	-0-	3.55E-015	no	no
Chinook salmon	-0-	3.55E-015	no	no
Cutthroat trout	-0-	3.55E-015	no	no
Steelhead	-0-	3.55E-015	no	no
Pacific lamprey	-0-	3.55E-015	no	no

*HHW = Hydraulic Sprayer with Hand-Held Wand
**ND= No data

Table 9-40. Aquatic Risks from Picloram--HHW* or Backpack

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	-0-	no	no
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Williams Creek				
Rainbow trout	-0-	-0-	no	no
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no
Applegate River				
Rainbow trout	-0-	-0-	no	no
Daphnia magna	-0-	-0-	no	no
Pacific chorus frog tadpole	-0-	-0-	ND	ND
Coho salmon	-0-	-0-	no	no
Chinook salmon	-0-	-0-	no	no
Cutthroat trout	-0-	-0-	no	no
Steelhead	-0-	-0-	no	no
Pacific lamprey	-0-	-0-	no	no

*HHW = Hydraulic Sprayer with Hand-Held Wand
**ND= No data

Table 9-41. Aquatic Risks from Triclopyr--HHW* or Backpack

			Exceeds MATC?**	
Species	**Typical**	**Maximum**	**Typical**	**Maximum**
Irrigation Ditches				
Daphnia magna	-0-	9.70E-011	ND	ND
Pacific chorus frog tadpole	-0-	4.93E-011	ND	ND
Williams Creek				
Rainbow trout	-0-	5.50E-010	ND	ND
Coho salmon	-0-	3.44E-010	ND	ND
Chinook salmon	-0-	6.88E-011	ND	ND
Cutthroat trout	-0-	5.50E-010	ND	ND
Steelhead	-0-	5.50E-010	ND	ND
Pacific lamprey	-0-	5.50E-010	ND	ND
Applegate River				
Rainbow trout	-0-	2.14E-010	ND	ND
Daphnia magna	-0-	4.09E-011	ND	ND
Pacific chorus frog tadpole	-0-	2.08E-011	ND	ND
Coho salmon	-0-	1.34E-010	ND	ND
Chinook salmon	-0-	2.68E-011	ND	ND
Cutthroat trout	-0-	2.14E-010	ND	ND
Steelhead	-0-	2.14E-010	ND	ND
Pacific lamprey	-0-	2.14E-010	ND	ND

*HHW = Hydraulic Sprayer with Hand-Held Wand

**ND= No data

Table 9-42. Aquatic Risks from Fertilizers*

Species	Risk Quotient Typ	Risk Quotient Max
Irrigation Ditches		
Daphnia magna	-0-	1.68E-006
Pacific chorus frog tadpole	-0-	7.08E-005
Williams Creek		
Rainbow trout	-0-	7.66E-006
Coho salmon	-0-	8.15E-006
Chinook salmon	-0-	8.15E-006
Cutthroat trout	-0-	4.44E-006
Steelhead	-0-	8.15E-006
Pacific lamprey	-0-	8.15E-006
Applegate River		
Rainbow trout	-0-	3.61E-006
Daphnia magna	-0-	8.58E-008
Pacific chorus frog tadpole	-0-	3.61E-006
Coho salmon	-0-	3.63E-006
Chinook salmon	-0-	3.63E-006
Cutthroat trout	-0-	1.87E-006
Steelhead	-0-	3.63E-006
Pacific lamprey	-0-	3.63E-006
Exceeds Ammonia Ambient Water Quality Criteria?		
Irrigation ditches--no salmonids	no	no
Williams Creek--no salmonids	no	no
Williams Creek--salmonids present	no	no
Applegate River--salmonids present	no	no

*Includes additive risks from all fertilizers used.

Table 9-43. Risks from Accidental Ingestion of Acephate Implant Capsules

Animal	Risk Quotient
General Terrestrial Species	
Deer	4.29E-002
Coyote	1.28E-001
Long-tailed vole	**5.87E+001**
Long-eared myotis	**1.04E+002**
Pocket gopher	**3.28E+001**
Raccoon	3.88E-001
Dog	7.08E-002
Black-capped chickadee	**1.00E+003**
Western bluebird	**3.80E+002**
Mallard duck	**2.20E+000**
Great blue heron	**4.94E+000**
Song sparrow	**5.50E+002**
Tree swallow	**5.50E+002**
Canada goose	**2.96E+000**
Common barn owl	**2.39E+001**
Osprey	**7.41E+000**
Pacific chorus frog	**4.38E+003**
Gopher snake	**2.58E+001**
Western fence lizard	**9.65E+002**
E&T and Sensitive Terrestrial Species	
Spotted owl	**1.80E+001**
Bald eagle	**2.54E+000**
Western pond turtle	**1.29E+001**
Common kingsnake	**1.34E+002**

Table 9-44. Risk from Concentrate Spill at Mixing Area

	Risk Quotient							
Chemical	**Rainbow Trout**	**Daphnia magna**	**Pacific chorus frog tadpole**	**Coho salmon**	**Chinook salmon**	**Cutthroat trout**	**Steelhead**	**Pacific lamprey**
Acephate	2.01E-008	5.19E-006	2.80E-009	2.01E-008	2.01E-008	2.01E-008	2.01E-008	2.01E-008
Chlorpyrifos	9.17E-003	**5.50E-001**	3.67E-005	9.17E-003	9.17E-003	2.05E-003	9.17E-003	9.17E-003
Diazinon	1.75E-003	3.94E-001	3.15E-003	1.75E-003	1.75E-003	9.26E-005	1.75E-003	1.75E-003
Dimethoate*	1.03E-004	4.90E-004	1.82E-004	1.03E-004	1.03E-004	1.03E-004	1.03E-004	1.03E-004
Esfenvalerate*	7.98E-002	1.54E-001	2.85E-003	**1.73E+001**	**1.73E+001**	**1.73E+001**	**2.36E-001**	**1.73E+001**
Horticultural Oil	1.13E-005	1.02E-003	5.11E-004	1.13E-005	1.13E-005	1.13E-005	1.13E-005	1.13E-005
Permethrin*	9.31E-002	1.59E-001	9.17E-004	**9.31E-002**	**9.31E-002**	**9.31E-002**	**9.31E-002**	**9.31E-002**
Propargite	2.54E-004	8.11E-004	8.11E-004	2.54E-004	2.54E-004	2.54E-004	2.54E-004	2.54E-004
Chlorothalonil	1.54E-002	1.91E-002	8.13E-003	1.54E-002	1.54E-002	1.54E-002	1.54E-002	1.54E-002
Dicamba	1.43E-005	1.56E-007	2.44E-006	1.43E-005	1.43E-005	2.50E-006	1.43E-005	1.43E-005
Glyphosate	6.25E-007	1.37E-006	1.68E-006	9.34E-006	1.12E-005	1.12E-005	1.12E-005	1.12E-005
Hexazinone	3.44E-006	3.62E-006	7.24E-006	3.79E-006	3.79E-006	3.79E-006	3.79E-006	3.79E-006
Picloram	8.06E-005	3.63E-006	2.08E-006	8.06E-005	8.06E-005	7.35E-005	8.06E-005	8.06E-005
Triclopyr butoxyethyl ester	5.38E-003	1.03E-003	5.23E-004	3.37E-003	6.73E-004	5.38E-003	5.38E-003	5.38E-003
Fertilizers*	2.54E-003	6.04E-004	2.54E-003	2.91E-002	2.91E-002	2.88E-002	2.91E-002	2.91E-002

*Includes additive risks from other ingredients in pesticide formulations or all fertilizers used in mixture.

Table 9-45. Risk from Mixture Spill into Bridge Point Ditch

		Risk Quotient							
Chemical	App Method	Rainbow Trout	Daphnia magna	Pacific chorus frog tadpole	Coho salmon	Chinook salmon	Cutthroat trout	Steelhead	Pacific lamprey
Chlorpyrifos	HPHS	**1.25E+000**	**7.50E+001**	5.00E-003	**1.25E+000**	**1.25E+000**	**2.80E-001**	**1.25E+000**	**1.25E+000**
Chlorpyrifos	HHW	1.00E-001	**6.00E+000**	4.00E-004	**1.00E-001**	**1.00E-001**	2.24E-002	**1.00E-001**	**1.00E-001**
Diazinon	HPHS	3.89E-002	**8.75E+000**	7.00E-002	3.89E-002	3.89E-002	2.06E-003	3.89E-002	3.89E-002
Diazinon	HHW	3.11E-003	**7.00E-001**	5.60E-003	3.11E-003	3.11E-003	1.65E-004	3.11E-003	3.11E-003
Dimethoate*	HHW	2.55E-004	1.19E-003	4.38E-004	2.55E-004	2.55E-004	2.55E-004	2.55E-004	2.55E-004
Dimethoate*	Backpack	6.38E-005	2.97E-004	1.09E-004	6.38E-005	6.38E-005	6.38E-005	6.38E-005	6.38E-005
Esfenvalerate*	HPHS	4.42E-001	**8.52E-001**	1.58E-002	**9.58E+001**	**9.58E+001**	**9.58E+001**	**1.31E+000**	**9.58E+001**
Esfenvalerate*	HHW	3.54E-002	6.81E-002	1.26E-003	**7.67E+000**	**7.67E+000**	**7.67E+000**	**1.05E-001**	**7.67E+000**
Esfenvalerate*	Backpack	8.85E-003	1.70E-002	3.16E-004	**1.92E+000**	**1.92E+000**	**1.92E+000**	2.61E-002	**1.92E+000**
Horticultural Oil	HPHS	2.15E-005	1.95E-003	9.77E-004	2.15E-005	2.15E-005	2.15E-005	2.15E-005	2.15E-005
Permethrin*	HPHS	4.19E-001	**7.15E-001**	4.13E-003	**4.19E-001**	**4.19E-001**	**4.19E-001**	**4.19E-001**	**4.19E-001**
Permethrin*	HHW	3.35E-002	5.72E-002	3.30E-004	3.35E-002	3.35E-002	3.35E-002	3.35E-002	3.35E-002
Permethrin*	Backpack	8.38E-003	1.43E-002	8.25E-005	8.38E-003	8.38E-003	8.38E-003	8.38E-003	8.38E-003
Propargite	HPHS	4.66E-002	1.49E-001	1.49E-001	4.66E-002	4.66E-002	4.66E-002	4.66E-002	4.66E-002
Propargite	HHW	3.73E-003	1.19E-002	1.19E-002	3.73E-003	3.73E-003	3.73E-003	3.73E-003	3.73E-003
Propargite	Backpack	9.32E-004	2.97E-003	2.97E-003	9.32E-004	9.32E-004	9.32E-004	9.32E-004	9.32E-004
Chlorothalonil	HPHS	2.30E-001	2.87E-001	1.22E-001	**2.30E-001**	**2.30E-001**	**2.30E-001**	**2.30E-001**	**2.30E-001**
Chlorothalonil	HHW	1.84E-002	2.29E-002	9.75E-003	1.84E-002	1.84E-002	1.84E-002	1.84E-002	1.84E-002
Dicamba	HHW	4.29E-004	4.69E-006	7.32E-005	4.29E-004	4.29E-004	7.50E-005	4.29E-004	4.29E-004
Dicamba	Backpack	1.07E-004	1.17E-006	1.83E-005	1.07E-004	1.07E-004	1.88E-005	1.07E-004	1.07E-004
Dicamba	Boom	2.14E-003	2.34E-005	3.66E-004	2.14E-003	2.14E-003	3.75E-004	2.14E-003	2.14E-003
Dicamba	Wick	1.07E-004	1.17E-006	1.83E-005	1.07E-004	1.07E-004	1.88E-005	1.07E-004	1.07E-004
Glyphosate	HHW	6.75E-006	1.48E-005	1.82E-005	1.01E-004	1.21E-004	1.21E-004	1.21E-004	1.21E-004
Glyphosate	Backpack	1.69E-006	3.69E-006	4.54E-006	2.52E-005	3.03E-005	3.03E-005	3.03E-005	3.03E-005
Glyphosate	Boom	2.70E-005	5.90E-005	7.26E-005	4.04E-004	4.84E-004	4.84E-004	4.84E-004	4.84E-004
Glyphosate	Wick	4.50E-007	9.84E-007	1.21E-006	6.73E-006	8.07E-006	8.07E-006	8.07E-006	8.07E-006
Hexazinone	HHW	6.56E-005	6.91E-005	1.38E-004	7.24E-005	7.24E-005	7.24E-005	7.24E-005	7.24E-005
Hexazinone	Backpack	1.64E-005	1.73E-005	3.45E-005	1.81E-005	1.81E-005	1.81E-005	1.81E-005	1.81E-005
Picloram	HHW	2.35E-003	1.06E-004	6.08E-005	2.35E-003	2.35E-003	2.15E-003	2.35E-003	2.35E-003
Picloram	Backpack	5.89E-004	2.65E-005	1.52E-005	5.89E-004	5.89E-004	5.37E-004	5.89E-004	5.89E-004
Triclopyr butoxyethyl ester	HHW	1.25E-001	2.38E-002	1.21E-002	**7.79E-002**	1.56E-002	**1.25E-001**	**1.25E-001**	**1.25E-001**
Triclopyr butoxyethyl ester	Backpack	3.12E-002	5.96E-003	3.03E-003	1.95E-002	3.89E-003	3.12E-002	3.12E-002	3.12E-002

*Includes risks from other ingredients in pesticide formulation.

Table 9-46. Risk from Mixture Spill into Williams Creek

		Risk Quotient							
Chemical	**App Method**	**Rainbow Trout**	**Daphnia magna**	**Pacific chorus frog tadpole**	**Coho salmon**	**Chinook salmon**	**Cutthroat trout**	**Steelhead**	**Pacific lamprey**
Chlorpyrifos	HPHS	**1.33E+000**	**8.00E+001**	5.33E-003	**1.33E+000**	**1.33E+000**	**2.99E-001**	**1.33E+000**	**1.33E+000**
Chlorpyrifos	HHW	1.07E-001	**6.40E+000**	4.27E-004	**1.07E-001**	**1.07E-001**	2.39E-002	**1.07E-001**	**1.07E-001**
Diazinon	HPHS	3.89E-002	**8.75E+000**	7.00E-002	3.89E-002	3.89E-002	2.06E-003	3.89E-002	3.89E-002
Diazinon	HHW	3.11E-003	**7.00E-001**	5.60E-003	3.11E-003	3.11E-003	1.65E-004	3.11E-003	3.11E-003
Dimethoate*	HHW	2.60E-004	1.23E-003	4.54E-004	2.60E-004	2.60E-004	2.60E-004	2.60E-004	2.60E-004
Dimethoate*	Backpack	6.49E-005	3.07E-004	1.14E-004	6.49E-005	6.49E-005	6.49E-005	6.49E-005	6.49E-005
Esfenvalerate*	HPHS	4.42E-001	**8.52E-001**	1.58E-002	**9.58E+001**	**9.58E+001**	**9.58E+001**	**1.31E+000**	**9.58E+001**
Esfenvalerate*	HHW	3.54E-002	6.81E-002	1.26E-003	**7.67E+000**	**7.67E+000**	**7.67E+000**	**1.05E-001**	**7.67E+000**
Esfenvalerate*	Backpack	8.85E-003	1.70E-002	3.16E-004	**1.92E+000**	**1.92E+000**	**1.92E+000**	2.61E-002	**1.92E+000**
Horticultural Oil	HPHS	2.20E-005	2.00E-003	1.00E-003	2.20E-005	2.20E-005	2.20E-005	2.20E-005	2.20E-005
Permethrin*	HPHS	4.19E-001	**7.15E-001**	4.13E-003	**4.19E-001**	**4.19E-001**	**4.19E-001**	**4.19E-001**	**4.19E-001**
Permethrin*	HHW	3.35E-002	5.72E-002	3.31E-004	3.35E-002	3.35E-002	3.35E-002	3.35E-002	3.35E-002
Permethrin*	Backpack	8.38E-003	1.43E-002	8.27E-005	8.38E-003	8.38E-003	8.38E-003	8.38E-003	8.38E-003
Propargite	HPHS	4.66E-002	1.49E-001	1.49E-001	4.66E-002	4.66E-002	4.66E-002	4.66E-002	4.66E-002
Propargite	HHW	3.73E-003	1.19E-002	1.19E-002	3.73E-003	3.73E-003	3.73E-003	3.73E-003	3.73E-003
Propargite	Backpack	9.32E-004	2.97E-003	2.97E-003	9.32E-004	9.32E-004	9.32E-004	9.32E-004	9.32E-004
Chlorothalonil	HPHS	2.30E-001	2.87E-001	1.22E-001	**2.30E-001**	**2.30E-001**	**2.30E-001**	**2.30E-001**	**2.30E-001**
Chlorothalonil	HHW	1.84E-002	2.29E-002	9.75E-003	1.84E-002	1.84E-002	1.84E-002	1.84E-002	1.84E-002
Dicamba	HHW	4.29E-004	4.69E-006	7.32E-005	4.29E-004	4.29E-004	7.50E-005	4.29E-004	4.29E-004
Dicamba	Backpack	1.07E-004	1.17E-006	1.83E-005	1.07E-004	1.07E-004	1.88E-005	1.07E-004	1.07E-004
Dicamba	Boom	2.14E-003	2.34E-005	3.66E-004	2.14E-003	2.14E-003	3.75E-004	2.14E-003	2.14E-003
Dicamba	Wick	1.07E-004	1.17E-006	1.83E-005	1.07E-004	1.07E-004	1.88E-005	1.07E-004	1.07E-004
Glyphosate	HHW	7.25E-006	1.58E-005	1.95E-005	1.08E-004	1.30E-004	1.30E-004	1.30E-004	1.30E-004
Glyphosate	Backpack	1.81E-006	3.96E-006	4.88E-006	2.71E-005	3.25E-005	3.25E-005	3.25E-005	3.25E-005
Glyphosate	Boom	2.90E-005	6.34E-005	7.80E-005	4.33E-004	5.20E-004	5.20E-004	5.20E-004	5.20E-004
Glyphosate	Wick	4.83E-007	1.06E-006	1.30E-006	7.22E-006	8.67E-006	8.67E-006	8.67E-006	8.67E-006
Hexazinone	HHW	6.56E-005	6.91E-005	1.38E-004	7.24E-005	7.24E-005	7.24E-005	7.24E-005	7.24E-005
Hexazinone	Backpack	1.64E-005	1.73E-005	3.45E-005	1.81E-005	1.81E-005	1.81E-005	1.81E-005	1.81E-005
Picloram	HHW	2.39E-003	1.08E-004	6.17E-005	2.39E-003	2.39E-003	2.18E-003	2.39E-003	2.39E-003
Picloram	Backpack	5.97E-004	2.69E-005	1.54E-005	5.97E-004	5.97E-004	5.44E-004	5.97E-004	5.97E-004
Triclopyr butoxyethyl ester	HHW	1.28E-001	2.44E-002	1.24E-002	**7.98E-002**	1.60E-002	**1.28E-001**	**1.28E-001**	**1.28E-001**
Triclopyr butoxyethyl ester	Backpack	3.19E-002	6.10E-003	3.10E-003	2.00E-002	3.99E-003	3.19E-002	3.19E-002	3.19E-002

*Includes risks from other ingredients in pesticide formulation.

9.3 References

Caux, P.-Y., R.A. Kent, M. Taché, C. Grande, G.T. Fan, and D.D. MacDonald. 1993. Environmental fate and effects of dicamba: A Canadian perspective. Reviews of Environmental Contamination and Toxicology 133:1-59.

Eisler, R. 1992. Fenvalerate hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 2. U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC.

EPA. See U.S. Environmental Protection Agency.

Roberts, B.L. and H.W. Dorough. 1985. Hazards of chemicals to earthworms. Environmental Toxicology and Chemistry 4:307-323.

Stratton, G.W., and C.T. Corke. 1982. Toxicity of the insecticide permethrin and some degradation products towards algae and cyanobacteria. Environmental Pollution (A) 29:71-80.

U.S. Environmental Protection Agency. Undated. EFED acephate RED chapter. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 1984. Health and environmental effects profile for dimethoate. Office of Research and Development. Cincinnati, OH.

U.S. Environmental Protection Agency. 1993. Reregistration eligibility decision: Glyphosate. EPA 738-R-93-014. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 1994. Reregistration eligibility decision (RED): Hexazinone. EPA 738-R-94-022. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 1995. Reregistration eligibility decision (RED): Picloram. EPA 738-R95-019. Office of Prevention, Pesticides and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 1998. Reregistration eligibility decision (RED): Triclopyr. EPA 738-R-98-011. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 1999. Reregistration eligibility decision (RED): Chlorothalonil. EPA 738-R-99-004. Office of Prevention, Pesticides, and Toxic Substances. Washington, DC.

U.S. Environmental Protection Agency. 2000a. Reregistration eligibility science chapter for chlorpyrifos: Fate and environmental risk assessment chapter. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2000b. Environmental Fate and Effects Division science chapter for Reregistration Eligibility Document for propargite. Office of Pesticide Programs. Washington, DC.

U.S. Environmental Protection Agency. 2001. Environmental risk assessment for diazinon. Office of Pesticide Programs. Washington, DC.

10.0 GLOSSARY AND ACRONYMS

Note: All definitions are specific to the terms as they are used in this risk assessment.

AChE. acetylcholinesterase.

acute. single-dose toxicity study. May also refer to adverse effects which exhibit a short and relatively severe course.

a.i. active ingredient.

analysis. the second step of an ecological risk assessment, which examines the two primary components of risk–exposure and effects–and the relationships between each other and ecosystem characteristics.

assessment endpoint. an environmental value that is to be protected, defined by an ecological entity and its attributes. For example, salmon are valued ecological entities; reproduction is an attribute. Together, "salmon reproduction" represents an assessment endpoint.

BCF. bioconcentration factor.

bioconcentration factor (BCF). a parameter that represents the uptake and retention of a chemical in the tissues of an aquatic species in relation to the chemical's concentration in water, expressed in mg/kg per mg/L.

cancer slope factor. represents the probability that a 1-mg/kg/day chronic dose of a chemical will result in formation of a tumor. Expressed as a probability, in units of "per mg/kg/day" or $(mg/kg/day)^{-1}$.

chronic. long-term, usually lifetime or near lifetime in duration.

conceptual model. a written description and visual representation of predicted relationships between ecological entities and the stressors to which they may be exposed.

EC_{50}. median effective concentration.

ED_{50}. median effective dose.

exposure assessment. the second step in human health risk assessment, involving estimation of doses from various scenarios and routes of exposure.

FIFRA. Federal Insecticide, Fungicide, and Rodenticide Act.

GLEAMS. Groundwater Loading Effects of Agricultural Management Systems, a computer-based model for predicting the fate and transport of agricultural pesticides and fertilizers.

half-life. the time required for a chemical to degrade to 50% of its original concentration.

hazard assessment. the first step in human health risk assessment, in which each chemical's toxic properties and dose-response relationship are identified.

hazard index (HI). an indicator of risk to human health, representing the ratio of the estimated dose to the reference dose. A hazard index of 1 or less usually indicates negligible risk to human health.

HI. hazard index.

in vitro. "in glass". Refers to a laboratory study conducted in a test tube, petri dish, or other artificial environment.

in vivo. "in body". Refers to a laboratory study conducted in a living body.

isomer. a chemical compound with the same molecular formula as another compound, but different chemical and physical properties as a result of structural or conformational differences.

K_{oc}. organic carbon partition coefficient.

K_{ow}. octanol-water partition coefficient.

LC_{50}. median lethal concentration.

LD_{50}. median lethal dose.

LOEC. lowest-observed-effect concentration.

LOEL. lowest-observed-effect level.

lowest-observed-effect concentration (LOEC). the lowest chemical concentration in water at which adverse effects are observed in an aquatic toxicity study.

lowest-observed-effect level (LOEL). the lowest dose at which adverse effects are observed in a laboratory animal toxicity study.

MATC. maximum acceptable toxicant concentration.

maximum acceptable toxicant concentration (MATC). the geometric mean of the no-observed-effect concentration and the lowest-observed-effect concentration, representing a concentration in water that is expected to be tolerated by the test species.

median effective concentration (EC_{50}). the water concentration at which an effect other than mortality is observed in 50% of the test organisms.

median effective dose (ED_{50}). the dose level at which an effect other than mortality is observed in 50% of the test animals.

median lethal concentration (LC_{50}). the water concentration that is lethal to 50% of the test organisms.

median lethal dose (LD_{50}). the dose that is lethal to 50% of the test animals.

mg/kg. milligrams per kilogram, usually indicating a dose level in terms of milligrams intake of a substance per kilogram of body weight.

mg/kg/day. milligrams per kilogram per day, usually indicating a daily dose level in terms of milligrams intake of a substance per kilogram of body weight per day.

mg/L. milligrams per liter, usually indicating a concentration of a substance in water.

no-observed-effect concentration (NOEC). the highest water concentration at which no adverse effects are observed in an aquatic toxicity study.

no-observed-effect level (NOEL). the highest dose at which no adverse effects are observed in a laboratory toxicity study.

NOEC. no-observed-effect concentration.

NOEL. no-observed-effect level.

octanol-water partition coefficient (K_{ow}). the ratio of a chemical's concentration in the octanol phase to its concentration in the aqueous phase of a two-phase octanol/water system. The octanol-water partition coefficient is relevant to properties such as solubility, bioconcentration, and soil/sediment adsorption.

organic carbon partition coefficient (K_{oc}). the ratio of the amount of a chemical adsorbed to soil or sediment per unit weight of the organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium. The organic carbon partition coefficient represents the ability of an organic chemical to partition itself between the solid and solution phases of a water-saturated or unsaturated soil, or between runoff water and sediment.

ppm. parts per million, usually indicating milligrams of a substance per kilogram of food.

problem formulation. the first step in an ecological risk assessment, in which the purpose of the assessment is provided, the problem is defined, and a plan for analyzing and characterizing risk is determined.

Q. quotient.

quotient (Q). the ratio of a non-target species dose or exposure level to the median lethal dose or exposure level. Section 9.1 provides information for interpretation of quotients.

receptor. an ecological entity that is exposed to a stressor.

reference dose (RfD). an estimate of the highest possible daily dose of a chemical that will pose no appreciable risk of deleterious effects to a human during his or her lifetime.

RfD. reference dose.

risk characterization. the third step in both human health and ecological risk assessment, in which estimated doses are compared to a chemical's toxic properties to predict the potential for adverse effects under the given conditions of exposure.

stressor. any physical, chemical, or biological entity that can induce an adverse response.

subacute. refers to a short (few days to several weeks) exposure.

subchronic. refers to a medium-term (few weeks to several months) exposure.