ROADSIDE VEGETATION MANAGEMENT

Environmental Impact Statement
Appendix B

Washington State
Department of Transportation

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Roadside Vegetation Management

Draft Environmental Impact Statement

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Appendix B - Contents

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Contents

Chapter 1 Fate, Transport and Metabolism of Herbicides, Adjuvants and Fertilizers

1.0 Introduction B1-1
- 1.1 Objectives B1-1
- 1.2 Techniques B1-1
- 1.3 Data Sets B1-1

2.0 Herbicides (active ingredients) B1-2
- 2.1 2,4-D B1-2
- 2.2 2,4-DP B1-3
- 2.3 Benefin B1-3
- 2.4 Bromacil B1-4
- 2.5 Chlorsulfuron B1-5
- 2.6 Chlorthal Dimethyl B1-6
- 2.7 Dicamba B1-7
- 2.8 Dichlobenil B1-8
- 2.9 Diuron B1-9
- 2.10 Fluazifop-butyl B1-9
- 2.11 Fosamine B1-10
- 2.12 Glyphosate B1-11
- 2.13 Imazapyr B1-12
- 2.14 Isoxaben B1-13
- 2.15 Mecoprop B1-13
- 2.16 Mefluidide B1-14
- 2.17 Metsulfuron Methyl B1-15
- 2.18 Napropamide B1-16
- 2.19 Oryzalin B1-17
- 2.20 Oxadiazon B1-18
- 2.21 Picloram B1-18
- 2.22 Pronamide B1-19
- 2.23 Sethoxydim B1-20
- 2.24 Sulfometuron Methyl B1-21
- 2.25 Triclopyr B1-22

3.0 Herbicides (inert ingredients) B1-22
- 3.1 Petroleum Distillates B1-22
- 3.2 Xylene B1-23

4.0 Adjuvants B1-24
- 4.1 Surfactant B1-24
- 4.2 Foam Suppressants B1-24

5.0 Fertilizers B1-24
- 5.1 Terrestrial Fate B1-25
- 5.2 Aquatic Fate B1-25

Chapter 2 Wildlife Risk Assessment

Toxicity Data

1.0 Introduction B2-1
1.1 Objectives B2-1
1.2 Techniques B2-1
2.0 Toxicological Issues B2-2
2.1 Herbicide Formulations B2-2
2.2 Synergistic Effects B2-2
2.3 Conflicting Data B2-3
3.0 Herbicides (active ingredients) B2-3
3.1 2,4-D B2-3
3.2 2,4-DP B2-4
3.3 Benefin B2-4
3.4 Bromacil B2-4
3.5 Chlorsulfuron B2-5
3.6 Chlorthal Dimethyl B2-5
3.7 Dicamba B2-5
3.8 Dichlobenil B2-6
3.9 Diuron B2-6
3.10 Fluazifop-butyl B2-6
3.11 Fosamine B2-7
3.12 Glyphosate B2-7
3.13 Imazapyr B2-7
3.14 Isoxaben B2-8
3.15 Mecoprop B2-8
3.16 Mefluidide B2-8
3.17 Metsulfuron methyl B2-9
3.18 Napropamide B2-9
3.19 Oryzalin B2-9
3.20 Oxadiazon B2-10
3.21 Picloram B2-10
3.22 Pronamide B2-11
3.23 Sethoxydim B2-11
3.24 Sulfometuron methyl B2-11
3.25 Triclopyr B2-12
4.0 Herbicides (inert ingredients) B2-12
4.1 Petroleum Distillates B2-12
4.2 Xylene B2-12
5.0 Adjuvants B2-12
5.1 Terrestrial Wildlife B2-12
6.0 Fertilizers B2-13
6.1 Terrestrial Wildlife B2-13
7.0 Uncertainty and Data Gaps B2-13

Wildlife Impacts

1.0 Introduction B2-14
2.0 Terrestrial Risk Assessment B2-14
2.1 2,4-D and 2,4-DP B2-16
2.2 Chlorthal Dimethyl B2-17
2.3 Dicamba B2-17
2.4 Diuron B2-18
2.5 Glyphosate B2-19
2.6 Mecoprop B2-19
2.7 Triclopyr B2-20
2.8 Petroleum Distillates and Xylene B2-21

Chapter 3 Aquatic Risk Assessment

1.0 Introduction B3-1
1.1 Objectives B3-1
1.2 Techniques B3-2
2.0 Toxicological and Impact Issues B3-3
2.1 Direct Effects B3-3
2.2 Indirect Effects of Pesticides B3-6
3.0 Effects of Herbicides by Active Ingredient B3-7
3.1 Aquatic Wildlife Toxicity B3-7
4.0 Inert Ingredients B3-12
4.1 Aquatic Wildlife Toxicity B3-12
5.0 Adjuvants B3-13
5.1 Aquatic Wildlife Toxicity B3-13
6.0 Fertilizers B3-13
6.1 Aquatic Wildlife B3-13
7.0 Uncertainty and Data Gaps B3-13
7.1 Toxicity Information B3-14
7.2 Estimated environmental Concentrations B3-14
7.3 Exposure Parameters B3-14
7.4 Sublethal Effects B3-15
7.5 Potential Hazards to Populations B3-15
8.0 Conclusions B3-15
8.1 Mitigating Measures B3-16

Chapter 4 Human Health Risk Assessment

1.0 Introduction B4-1
1.1 Purposes B4-1
1.2 Organization of This Chapter B4-1
1.3 What is a Risk Assessment? B4-1
1.4 Structure of the Risk Assessment B4-2
2.0 Hazard Identification B4-9
2.1 Introduction B4-9
2.2 Toxicology B4-12
2.3 Toxicology Summaries of the 25 Herbicides B4-20

3.0 Exposure Assessment B4-66
3.1 Introduction B4-66
3.2 Exposure Estimates B4-69
3.3 Summary of Exposure Equations, Assumptions, Acronyms and Examples B4-86
4.0 Dose-response Assessment B4-100
4.1 Introduction B4-100
4.2 Determination of Toxicity Values B4-101
5.0 Risk Characterization B4-102
5.1 Introduction B4-102
5.2 Risk Characterization Summary by Chemical B4-107
5.3 Synergistic, Additive and Antagonistic Effec6s B4-137
5.4 Quantitative Risk From Multiple Pathways B4-137
5.5 Quantitative Risk From Multiple Chemicals B4-138
5.6 Qualitative Human Health Risk Analysis B4-139
6.0 Risk Communication (Summary) B4-141
6.1 Understanding Risk B4-141
6.2 Uncertainties B4-142
6.3 Differences Between Risk Assessments B4-145

Chapter 5 Toxicological Data Summaries

1.0 Introduction B5-1

Chapter 6 Hazardous Materials and Waste

1.0 Federal Laws and Regulations B6-1
1.1 The Federal Insecticide, Fungicide, and Rodenticide Act B6-1
1.2 The Resource Conservation and Recovery Act B6-1
1.3 Other Federal Laws and Regulations B6-1
2.0 Washington State Laws and Regulations B6-2
2.1 Washington Department of Agriculture Regulations B6-2
2.2 Other Laws and Regulations Applicable to the WDOT Program B6-2
3.0 Hazardous Waste Recycling B6-5
3.1 Pesticide Formulation B6-6
3.2 Waste Volume B6-6
3.3 Use Equipment Modifications and Source Reduction Practices to Reduce Waste Volumes B6-7
3.4 Washwater from Rinsing and Cleaning Spray Equipment Exterior B6-7
3.5 Empty Pesticide Containers B6-8
3.6 Spill Cleanup Residues B6-8
4.0 Hazardous Materials and Waste B6-10
4.1 Criteria for Identifying the Characteristics of Hazardous Waste B6-10
4.2 Identifying and Handling Hazardous Waste B6-11

List of Figures

Figure 2-1 Relative Mammalian Toxicities of the Active Ingredients Found in the Herbicides to be Used by WSDOT for Roadside Vegetation Management B2-22
Figure 2-2 Relative Avian Toxicities (LD_{50}) of the Active Ingredients Found in the Herbicides to be Used by WSDOT for Roadside Vegetation Management B2-23
Figure 2-3 Relative Mammalian Toxicities (LC_{50}) of the Active Ingredients Found in the Herbicides to be Used by WSDOT for Roadside Vegetation Management B2-24
Figure 4-1 Components of a Risk Assessment B4-148
Figure 4-2 Diagram of Quantal Dose Response Relationship B4-149
Figure 4-3 Diagram of Dose Response Relationship B4-150
Figure 4-4 Dose Response Curve Resolved by Modeling B4-151
Figure 4-5 A Part of Hazard Identification for Developmental Toxicants . B4-152
Figure 4-6 Schematic Illustrating the Various Avenues for Degradation and Movement of Xenobiotics in the Environment B4-153

Lists of Tables

Table 1-1 Fertilizers to be Used by WSDOT for Roadside Vegetation Management. B1-26
Table 2-1 EPA Descriptive Classifications for Toxicity Data B2-25
Table 2-2 Data Gathered for Use in Assessing the Relative Impacts of Herbicides Used by WSDOT on Wildlife B2-26
Table 3-1 Relative Annual Precipitation and Relative WSDOT Road Densities by Phytogeographic Province B3-18
Table 3-2 Relative Persistence of WSDOT Applied Herbicides in Soil . . B3-19
Table 3-3 Relative Exposure of Aquatic Organisms To Herbicides By Phytogeographic Province B3-20
Table 3-4 Relative Risk To Fish From Exposure To Herbicides By Phytogeographic Province B3-22
Table 3-5 Relative Risk To Aquatic Invertebrates From Exposure To Herbicides By Phytogeographic Province B3-24
Table 3-6 Relative Acute Aquatic Toxicities Of the Herbicides Used by the WSDOT B3-26
Table 3-7 Quantities of Benefin, Diuron, and Oxadiazon Required to Achieve the Lowest LC_{50} Values for Fish and Invertebrates in a 100 Acre Lake Compared to the Quantities Applied in the Puget Trough Phytogeographic Province from July 1989 to June 1990 . . . . . B3-27
Table 4-1 Exposure Estimates for Workers Applying Herbicides with Truck Mounted Spray Equipment B4-154
Table 4-2 Exposure Estimates for Workers Mixing and Loading Herbicides into Truck Mounted Spray Equipment B4-155
Table 4-3 Exposure Estimates for Workers Applying, Mixing and Loading Herbicides Using Truck Mounted Spray Equipment . B4-156

Table 4-4 Exposure Estimates for Workers Using Backpack or Handheld Spray Equipment B4-157
Table 4-5 Exposure Estimates for the Public-Ingestion of Drift Contaminated Berries, Adults B4-158
Table 4-6 Exposure Estimates for the Public-Ingestion of Drift Contaminated Berries, Child B4-159
Table 4-7 Exposure Estimates for the Public-Ingestion of Drift Contaminated Vegetables, Lettuce B4-160
Table 4-8 Exposure Estimates for the Public-Ingestion of Drift Contaminated Vegetables, Beans B4-161
Table 4-9 Exposure Estimates for the Public-Dermal Exposure from Drift Contaminated Berries B4-162
Table 4-10 Exposure Estimates for the Public-Dermal Exposure from Contaminated Vegetation B4-163
Table 4-11 Critical Exposure Estimate Criteria B4-164
Table 4-12 List of Reference Doses B4-165
Table 4-13 Potential Carcinogens B4-168
Table 4-14 Reference Doses and Cancer Potency Factors Used in Risk Assessment B4-169
Table 4-15 Estimates of Non-Cancer and Cancer Risks for Workers Applying Herbicides Using Truck-Mounted Spray Equipment . B4-170
Table 4-16 Estimates of Non-Cancer and Cancer Risk for Workers Mixing and Loading Herbicides into Truck-Mounted Spray Equipment B4-171
Table 4-17 Estimates of Non-Cancer and Cancer Risks for Workers Applying, Mixing and Loading Using Truck-Mounted Spray Equipment B4-172
Table 4-18 Estimates of Non-Cancer and Cancer Risk for Workers Applying Herbicides Using Backpack or Hand-Held Spray Equipment B4-173
Table 4-19 Estimates of Non-Cancer and Cancer Risk from Ingestion of Drift Contaminated Berries, Adults B4-174
Table 4-20 Estimates of Non-Cancer and Cancer Risk from Ingestion of Drift Contaminated Berries, Child B4-175
Table 4-21 Estimates of Non-Cancer and Cancer Risk from Ingestion of Drift Contaminated Vegetables, Lettuce B4-176
Table 4-22 Estimates of Non-Cancer and Cancer Risk from Ingestion of Drift Contaminated Vegetables, Beans B4-177
Table 4-23 Estimates of Non-Cancer and Cancer Risk from Dermal Contact with Drift Contaminated Berries B4-178
Table 4-24 Estimates of Non-Cancer and Cancer Risk from Dermal Contact with Sprayed Vegetation B4-179
Table 4-25 Hiking Through Sprayed Vegetation to Pick Contaminated Blackberries Which Are Consumed the Same Day B4-180
Table 4-26 Ingestion of Contaminated Lettuce, Beans and Berries in the Same Day B4-181
Table 4-27 Hazard Indices for WSDOT Worker Risk From Multiple Chemicals B4-182

Table 4-28 Hazard Indices for Public Risk From Exposure to Multiple Chemicals B4-183
Table 4-29 Compounds of Negligible Risk B4-184
Table 4-30 Compounds of Low Risk B4-185
Table 4-31 Compounds of Moderate Risk B4-186
Table 4-32 Compounds of High Risk B4-187
Table 4-33 Toxicity Categories B4-188
Table 4-34 Precautionary Statements B4-189
Table 4-35 List of Classifications Set by EPA B4-190
Table 4-36 WSDOT's Average and Maximum Application Rates for the 25 Herbicides from July 1989 through June 1990 . . . . . B4-191

Table 5-1 Signal Words and Toxicity Categories for Each Active Ingredient and Formulated Product to be Used for Roadside Vegetation Management B5-3
Table 5-2 Amount of Active Ingredient in Each Formulation and the Maximum Label Rate for Each of the Products to be used for Roadside Vegetation Management B5-6
Table 5-3 Formulated Products, Manufactures for the Maximum Label Rate for Each of the Products to be Used for Roadside Vegetation Management B5-9
Table 5-4 Toxicity Data for the Active Ingredients to be Used by WSDOT for Roadside Vegetation Management B5-22
Table 5-5 Environmental Fate of Active Ingredients to be Used by WSDOT for Roadside Vegetation Management B5-35
Table 5-6 Wildlife Toxicity and Efficacy of Active Ingredients to be Used by WSDOT for Roadside Vegetation Management . . B5-48
Table 5-7 Physical Data, Toxicity Information, Environmental Fate and Wildlife Toxicity of the Inert Ingredients Found in the Herbicides to be Used by WSDOT for Roadside Vegetation Management B5-55
Table 5-8 Physical Data, Toxicity Information, Environmental Fate and Wildlife Toxicity Information for the Adjuvants Used for Roadside Vegetation Management B5-56
Tables 5-9 through 5-33 WSDOT's Herbicide Use Rates B5-62
References for Tables 5-3 through 5-8 . . . . . B5-87

Appendix B - Chapter 1

Draft EIS
Roadside Vegetation Management

amount of additional nutrients (fertilizers). This influx of nutrients in turn produces algal blooms that can deplete the oxygen in the water. This is most likely to occur in areas of heavy agricultural activity. Anaerobic waters may also be prevalent in the groundwater.

2.2 2,4-DP

2.2.1 Environmental Fate

Soil - Soil half life in sandy loam soil is 10 days (USFS, 1989). Metabolism involves the degradation of the side chain to 2,4-dichlorophenol followed by ring hydroxylation and ring opening (Hartley and Kidd, 1987).

Water - No data were found concerning 2,4-DP's aquatic environmental fate.

2.2.2 Transport

No data were found concerning leaching potential for 2,4-DP.

2.2.3 Metabolism

Mode of Action - 2,4-DP ia a systemic, hormone type herbicide which is absorbed by the leaves with translocation to the roots, following absorption. Its action is very similar to 2,4-D, in that it acts as a growth inhibitor (Hartley and Kidd, 1987).

Plant Metabolism - Metabolism is similar to 2,4-D (hydroxylation, conjugation and excretion). The parent compound is converted to 2,4-dichlorophenol in both susceptible and resistant plants (Menzie, 1980).

Animal Metabolism - Similar to that of 2,4-D.

2.2.4 Potential Regional Impacts

The regional impacts discussed for 2,4-D also apply to 2,4-DP.

2.3 Benefin

2.3.1 Environmental Fate

Soil - The aerobic half-life in sandy loam soil is approximately 1.7 months, while its anaerobic sandy loam soil half-life is about 15 days (Melichar, 1991). Other studies have determined that the half-life on bare soil in a field is 117-131 days while in turf the half-life drops to 70 days (Melichar, 1991).

The primary route of degradation in soils is via dealkylation, nitro group reduction, and oxidation of the alkylamine group and ring to form benzimidizoles. In aerobic soils, nine metabolites were identified (Melichar, 1991).

Water - These data are not required for registration of this product, therefore, no data were available concerning benefin's aquatic environmental fate.

2.3.2 Transport

Soil - Leaching and Mobility - Benefin has adsorption coefficients of 27 in sand and 117 in clay loam (Melichar, 1991). These high numbers indicate that benefin adsorbs to soil very well. As a result, benefin is considered to be immobile in soil (Melichar, 1991).

2.3.3 Metabolism

Mode of Action - Benefin is a preemergence herbicide with moderate residual activity. It works by interfering with tubulin synthesis which in turn inhibits root development (USFS, 1988).

Plant Metabolism - Very little uptake and metabolism of benefin occurs in plants. That which does get absorbed is generally metabolized to water soluble compounds indicating extensive metabolism has occurred. Very little unmetabolized benefin is detected in plant tissues (Melichar, 1991).

Animal Metabolism - Residues have not been found in milk or edible tissues when studies are conducted in meat or milk producing animals. This indicates that any benefin that is absorbed is rapidly metabolized or excreted (Melichar, 1991).

2.3.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.4 Bromacil

2.4.1 Environmental Fate

Soil - Bromacil's soil half-life ranges from 137 days on the west coast (California) to 165 days on the east coast (Delaware) (Wellings, 1991). No data were specific for Washington. The primary degradation metabolite is carbon dioxide (80%) with 5 other metabolites having been identified, each of them comprising less than 3% of the total metabolites (Wellings, 1991).

Water - In the absence of ultraviolet radiation, bromacil is stable (Wellings, 1991). In the presence of ultraviolet radiation it degrades primarily to a debrominated analog of bromacil (Wellings, 1991). Its photodegradation half-life ranges from 7 days at pH 9 to 102 days at pH 5 (Wellings, 1991).

2.4.2 Transport

Soil - Leaching and Mobility - Bromacil is considered mobile in low organic matter soils (Wellings, 1991) with adsorption coefficients from 18 ml/gm (Jones and Stokes, 1991) to 72 ml/gm (Kenaga and Gorung, 1978). The combination of bromacil's long half-life, its mobility within soils, and its stability in water indicates that this is a compound where the possiblity of groundwater contamination should be explored.

Appendix B - Chapter 1 Fate, Transport and Metabolism of Herbicides Adjuvants and Fertilizers

1.0 Introduction

1.1 Objectives

This chapter is presented as a technical review of the environmental fate of the herbicide active ingredients, inert ingredients, adjuvants and fertilizers. This review is necessary in order to understand how each of these compounds behaves once introduced into our environment. These data will help to assess regional differences in environmental fate as they relate to soil types, precipitation, use patterns, etc.

1.2 Techniques

A summary of the data presented in this chapter is presented in Chapter 4 of this appendix. This table also lists all references for the data presented in this chapter. The majority of the data on the active ingredients was collected directly from the manufacturers of the products or from other previously published EISs that reviewed the same products. As data gaps became evident the journal literature was searched for information to fill in those gaps.

1.3 Data Sets

There is a considerable volume of information for the active ingredients of pesticide formulations because the United States Environmental Protection Agencies (US EPA) requires tests on the technical grade product for registration of the end use products. Although some of these studies have been conducted by independent researchers, there simply is not the volume of information from these sources as there is from the manufacturers. In addition, many of the independent researchers are conducting their research with grants provided by the manufacturers so the manufacturers can use the data for registration purposes. Therefore, we felt it appropriate to go directly to the manufacturers first and then to the literature to fill in the data gaps. Data was also obtained from US EPA by a series of Freedom of Information Act requests.

Since the US EPA does not require the same level of testing for the inert ingredients, adjuvants and fertilizers as it does for the active ingredients, there are considerably less data for these compounds. The data presented here represents data available from manufacturers and, in some cases, from literature searches. These data will generally be less complete and may not be able to answer all of the questions that data for the active ingredients answer.

2.0 Herbicides (active ingredients)

2.1 2,4-D

2.1.1 Environmental Fate

Soil - 2,4-D has a half life in soil of less than 28 days in sandy loam soil (USFS, 1989) and less than 7 days in prairie soils (Menzie, 1980). On the average its soil half life ranges from 10 to 50 days (Howard, et al., 1991). Following application of two forms of 2,4-D (the dimethylamine salt and the octyl ester) to Naff silt loam soil, the ester hydrolyzed, and then both forms followed the basic metabolic pattern of carboxyl and ring degradation to carbon dioxide (Menzie, 1980).

Water - Studies preformed with 2,4-D (butoxyethanol ester), indicated that 50% of the 2,4-D was degraded in 12 hours under ultraviolet light. Degradation was greatest under high temperatures and high light levels (Menzie, 1980). The aquatic half-life ranges from 5-10 days in aerobic water and 4 weeks to 6 months in anaerobic waters (Howard, et al., 1991).

2.1.1.1 Transport

No data were found concerning leaching potential for 2,4-D.

2.1.1.2 Metabolism

Mode of Action - 2,4-D uptake is primarily through the foliage and stems (USFS, 1988). It interferes with cell wall development by acting as a plant growth hormone (Klassen, et al., 1986).

Plant Metabolism - Plant metabolism is quite variable depending on the plant species being studied. In general, the major pathway involves ring hydroxylation followed by sugar conjugation and then excretion (Menzie, 1980).

Animal Metabolism - 2,4-D is primarily (82.3%) excreted in the urine unchanged, but a small amount (12.8%) was an unidentified conjugate. In rats, the 2,4-D was converted to glycine and taurine conjugates prior to elimination (Menzie, 1980; USFS, 1989).

2.1.1.3 Potential Regional Impacts

With 2,4-D's high aquatic degradation rate in high temperatures and high light, the dissipation residues may be slower for cooler areas with extended periods of overcast weather. This would include most areas west of the Cascades.

Also, the high water solubility of the amine salts may present a potential for groundwater contamination for all regions. Information on leaching properties could confirm or refute this assumption.

2,4-D's high half-life in anaerobic waters is of concern in areas where anaerobic waters are found. These areas are typically small ponds or lakes that have received a high

2.4.3 Metabolism

Mode of Action - Bromacil is a preemergence, long residual, soil active herbicide. It is absorbed by the roots and translocated to the foliage where it inhibits photosynthesis within the chloroplasts (USFS, 1988).

Plant Metabolism - The primary metabolite of bromacil is 5-bromo-3-secbutyl-6-hydroxymethyluracil (Wellings, 1991).

Animal Metabolism - Same as plant metabolism (Wellings, 1991).

2.4.4 Potential Regional Impacts - There is concern that this compound may be a potential groundwater contaminant. The greatest concern would be for areas that have low organic matter such as the Okanogan Highlands and the Columbia Basin province. Typically, however, these regions have fairly deep water tables and low rainfall (or groundwater recharge rates).

2.5 Chlorsulfuron

2.5.1 Environmental Fate

Soil - The half-life of chlorsulfuron in silty loam soil at 25°C is approximately 1 to 2 weeks (Rapisarda, 1981). Its degradation in soils increases with decreasing pH values and decreases with increased soil clay content (Joshi and Van, 1984). Microbial degradation is temperature and moisture sensitive (Rapisarda, 1981).

Water - The aquatic half-life for chlorsulfuron ranges from 20-22 weeks in anaerobic environments and 7 to 8 weeks in flooded soils. Its degradation increases with pH and the amount of oxygen in the water. Its primary degradates include; 2-chlorobenzene sulfonamide; 2-chlorophenyl sulfonyl urea and 2-amino-4-methoxy-6-methyl-1,3,5-triazine (Chrzanowski, 1981).

2.5.2 Transport

Soil - Leaching and Mobility - Chlorsulfuron is weakly adsorbed to loam, silt loam and sandy loam soils. Its metabolites, however, are readily adsorbed to soil particles. Chlorsulfuron is listed (Jones and Stokes, 1991) as having a low likelihood of moving through the soil column.

2.5.3 Metabolism

Mode of Action - Chlorsulfuron is a systemic herbicide that is absorbed by the roots and the foliage. It interferes with plant cell division and growth by inhibiting the biosynthesis of valine and isoleucine (Denny, 1991).

Plant Metabolism - The primary metabolite of chlorsulfuron in wheat and barley is the 5-hydroxyphenyl analog of chlorsulfuron and its glucose conjugate. Following this it is further degraded to 2-chloro-5-hydroxybenzenesulfonamide and its glucose conjugate. The primary metabolite in broadleaved plants is 6-hydroxymethyl triazine (Denny, 1991).

Animal Metabolism - After 24 hours 78.5% of the chlorsulfuron remained intact after in vitro incubation in tissue cultures. In rats, 95% of the chlorsulfuron was eliminated in the urine, 4% in the feces and 1% remained in the body after 72 hours. The major urine component was the parent material (Denny, 1991), which means chlorsulfuron is essentially excreted without being metabolized.

2.5.4 Summary

Regional Impacts - Although chlorsulfuron is weakly adsorbed to soil particles, it presents a minor threat to ground water contamination or long term effects in the soil in any of the regions. Its soil half-life provides relatively rapid degradation after which the metabolites bind to the soil.

Chlorsulfuron does have a relatively long half-life (20-22 weeks) in anaerobic soils, which could be of concern in warm areas with heavy agricultural emphasis (such as the Columbia Basin) and which are flooded in the spring. Anaerobic soil conditions in culturally "active" surface soils are usual.

2.6 Chlorthal Dimethyl

2.6.1 Environmental Fate

Soil - Chlorthal dimethyl (dimethyl tetrachloroterephthalate) has a soil half-life of 18 to 92 days depending on environmental and soil conditions (Howard, et al., 1991). It degrades microbially to monomethyl tetrachloroterephthalate and 2,3,5,6-tetrachloroterephthalatic acid (Hartley and Kidd, 1987). It is assumed that conditions favorable for the growth of soil microflora would be conditions in which you would find the shorter half-lives, such as warmer temperatures and higher moisture content.

Water - The aquatic half-life is the same as that for soil, 18 to 92 days (Howard, et al., 1991).

2.6.2 Transport

Soil - Leaching and Mobility - No data were found concerning the leaching potential of chlorthal dimethyl.

2.6.3 Metabolism

Mode of Action - Chlorthal dimethyl is a non-systemic herbicide that works by killing germinating seedlings (Hartley and Kidd, 1987).

Plant Metabolism - No residues were found in carrots that were treated with and then analyzed for chlorthal dimethyl (Menzie, 1980). It is not metabolized by plants (Hartley and Kidd, 1987).

Animal Metabolism - Chlorthal dimethyl is metabolized to monomethyl tetrachloroterephthalate and 2,3,5,6-tetrachloroterephthalatic acid and eliminated in the urine (Hartley and Kidd, 1987).

2.6.4 Potential Regional Impacts - The data available do not indicate that there are any specific regional concerns for this compound.

2.7 Dicamba

2.7.1 Environmental Fate

Soil - One reference listed dicamba's soil half-life to be <25 days (Sandoz, 1992) while another listed a half-life range from 31 days in aerobic soils to 58 days in anaerobic soils (Krueger, et al., 1991). It is microbially degraded in the soil to 3,6-dichlorosalicylic acid and then to carbon dioxide (Sandoz, 1992).

Water - No data were found that indicated an aquatic half-life for dicamba or how dicamba is metabolized in an aquatic environment.

2.7.2 Transport

Soil - Leaching and Mobility - Dicamba has an adsorption coefficient of 0 to 0.9 (Gowanlock, 1991). This indicates that it adsorbs poorly to soils and leaches readily, except in soils with high organic matter content. The degradation product (3,6-dichlorosalicylic acid) adsorbs to the soil more readily and is less mobile. Dicamba is listed (Jones and Stokes, 1991) as having a "high" likelihood of migrating to groundwater. In addition, dicamba's resistance to hydrolysis contributes to its potential for groundwater contamination.

2.7.3 Metabolism

Mode of Action - Dicamba is a selective systemic herbicide which is absorbed by the leaves and roots and translocated throughout the plant. It functions as an auxin-like growth regulator (Gowanlock, 1991).

Plant Metabolism - Dicamba is metabolized by plants to 5-hydroxy-3,6-dichloro-o-ansinic acid. Other minor metabolites include; 3,6-dichlorosalicylic acid and 5-hydroxy-3,6-dichloro-2-methoxy-dichlorobenzoic acid (Gowanlock, 1991).

Animal Metabolism - Dicamba is readily absorbed in the gastro-intestinal tract and rapidly eliminated in the urine, mainly as dicamba and partly as a glucuronic acid conjugate of dicamba (Sandoz, 1992).

2.7.4 Potential Regional Impacts

The properties of the compound suggest a possibility of water or groundwater contamination in the vicinity of applications.

2.8 Dichlobenil

2.8.1 Environmental Fate

Soil - Dichlobenil's half-life ranges from two to five months depending on moisture temperature and soil type (Uniroyal, Undated(a)), and averages about 70 days (Nimmo, 1985; Walstra, et al., 1989). Initial metabolism of dichlobenil in soils is very rapid (Uniroyal, Undated(b)). It is primarily microbially degraded to 2,6-dichlorobenzamide. Following the formation of this initial metabolite, it is slowly converted to carbon dioxide (Nimmo, 1985; Walstra, et al., 1989).

Water - The aquatic half-life for dichlobenil is about 15-69 days (Jacobson, et al., 1991, FL; Jacobson, et al., 1991, OR). Metabolism in the aquatic environment is very similar to that in the soil environment (Dykstra, 1991).

2.8.2 Transport

Soil - Leaching and Mobility - Dichlobenil has an average adsorption coefficient ranging from 125 (Walstra, et al., 1988) to 890 (Jones and Stokes, 1991) indicating a very low soil mobility potential. The primary soil metabolite (2,6-dichlorobenzamide) has a greater soil mobility than its parent compound (Walstra, et al., 1988), but its potential to move through soil is still quite low. Dichlobenil's high adsorption coefficient prevents it from becoming a significant threat as a groundwater contaminant. Dichlobenil is considered (Jones and Stokes, 1991) among those chemicals least likely to move through the soil column.

2.8.3 Metabolism

Mode of Action - Dichlobenil is a broad spectrum preemergence herbicide that prevents germination and interferes with the development of actively dividing meristems by inhibiting the incorporation of glucose into cellulose, which is the main cell wall constituent (Uniroyal, 1991; Hogetsu, et al., 1974). It also inhibits germination and actively dividing meristems (Hogetsu, et al., 1974; Kawamura, et al., 1976; Umetsu, et al., 1976; Meyer and Herth, 1978 and Montezioos and Delmer, 1980).

Plant Metabolism - The major residue found in plant tissue is 2,6-dichlorobenzamide (Hubert, 1990, apples; Hubert, 1990, grapes). This metabolite is primarily found in the stem of treated plants (Menzie, 1980).

Animal Metabolism - Initial metabolism of dichlobenil involves hydroxylation followed by glucose conjugation and excretion. Within 168 hours, 90% of all dichlobenil is excreted, most of which is excreted within the first 24 hours (Koorn, et al., 1987; Kaldenberg and Lange, 1988; Cameron, et al., 1988; Vincent, et al., 1989; Lange, 1989; Hubert, 1990).

2.8.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.9 Diuron

2.9.1 Environmental Fate

Soil - Diuron is relatively (130 to 160 day half-life) persistent in soils, especially in soils with high clay or organic matter content. It has a reported half-life of 130 to 160 days (Hawaii) (Menzie, 1980). It is degraded primarily by microbial means (USFS, 1988) to monomethyl diuron and other conjugated residues (Menzie, 1980).

Water - Diuron has an aquatic half-life of greater than 500 days in the absence of ultraviolet radiation (Wellings, 1991). In the presence of light, its aquatic half-life drops to 43 days (Wellings, 1991). Otherwise, diuron is stable in aquatic environments.

2.9.2 Transport

Soil - Leaching and Mobility - Diuron will not leach into the soil greater than six inches in typical texture and moisture conditions (USFS, 1988; USDI, 1989). Although traces of diuron have been detected in small amounts in some groundwater samples, it is considered to be among those chemicals that have a low likelihood of moving through the soil column.

2.9.3 Metabolism

Mode of Action - Diuron is a medium to long residual preemergence herbicide. It is rapidly absorbed by the roots and translocated throughout the plant. It acts by inhibiting photosynthesis (USFS, 1988).

Plant Metabolism - The only available data on plant metabolism were for sugarcane. Although sugarcane is not a highway plant, it may provide insight into the plant metabolism of diuron. Sugarcane has been reported to have metabolized diuron to methyl diuron, 3,4-dichorophenol urea and glucose conjugates of N-hydroxy-methyl-diuron and desmethyl diuron (Menzie, 1980).

Animal Metabolism - No data were found concerning the metabolism of diuron by animals.

2.9.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.10 Fluazifop-butyl

2.10.1 Environmental Fate

Soil - The soil half-life for fluazifop-butyl ranges from 1 to 21 days (Hartley and Kidd, 1987; Kieft, 1991). In moist soils it is rapidly metabolized with a half-life of less than 1 week. The major degradation product is fluazifop which has a half-life of less than 3 weeks in moist soils (Hartley and Kidd, 1987).

Water - No data were available concerning the aquatic half-life and metabolism of fluazifop-butyl.

2.10.2 Transport

Soil - Leaching and Mobility - Fluazifop-butyl has an adsorption coefficient of 8.2 to 13.9 (Kieft, 1991). This would indicate that it has an affinity for soil particles and is not very mobile. Fluazifop-butyl is listed (Jones and Stokes, 1991) as a compound that is not likely to move through the soil column.

2.10.3 Metabolism

Mode of Action - Fluazifop-butyl is a systemic herbicide that is absorbed by the leaves, activated and then translocated to the meristematic tissue where it inhibits growth (Hartley and Kidd, 1987).

Plant Metabolism - Within the plant, fluazifop-butyl is rapidly hydrolyzed (activated) to fluazifop (Hartley and Kidd, 1987) which is converted to glutathione conjugates (Kieft,1991).

Animal Metabolism - No data were available for the mechanism of metabolism of fluazifop-butyl by animals.

2.10.4. Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.11 Fosamine

2.11.1 Environmental Fate

Soil - One study reported a soil half-life of less than 10 days in sandy loam soil (USFS, 1989) and another reported a half-life of 2 to 5 days (Eble, 1989). Fosamine is rapidly metabolized by soil microbes to carbamyl phosphonic acid (Han and Krause, undated).

Water - The data on aquatic half-life and metabolism in aquatic environments for fosamine are not required for its current label uses and are therefore not presented.

2.11.2 Transport

Soil - Leaching and Mobility - No adsorption coefficient is available for fosamine, but it is described as weakly to highly adsorbed, with variation due to different soil types. Fosamine is converted to carbamyl phosphonic acid, which does bind strongly to soils (Priester and Sheftic, 1987).

2.11.3 Metabolism

Mode of Action - Fosamine is a contact herbicide that inhibits or prevents bud formation (USFS, 1989).

Plant Metabolism - Fosamine is rapidly metabolized in pasture flora to carbamyl phosphonic acid with a half-life of 7 days (Freidman, 1991).

Animal Metabolism - Fosamine is totally eliminated from the body of rats in 72 hours. The only metabolites identified were carbamylphosphonate and fosamine (USFS, 1989).

2.11.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.12 Glyphosate

2.12.1 Environmental Fate

Soil - Glyphosate typically has a half-life of less than 60 days (Wilkerson and Kim, 1986). However, it is relatively persistent in sandy loam soils (USDI, 1989). Microbial degradation is the major decomposition path. The primary metabolite is aminomethylphosphonic acid. There are three other phosphonic acids that are usually produced. Further microbial activity breaks down the phosphonic acids to carbon dioxide (Menzie, 1980).

Water - Aquatic half-lives have been reported ranging from 7 weeks in aquatic sediment to 10 weeks in pond water. If glyphosate comes into contact with sediment in the water then it will be degraded rapidly, otherwise no significant chemical degradation or hydrolysis occurs.

2.12.2 Transport

Soil - Leaching and Mobility - Glyphosate has very little potential for mobility. It is rapidly and strongly adsorbed to soil, especially clay particles at lower pH values. Glyphosate is considered to have a low potential to move through the soil column (Jones and Stokes, 1991).

2.12.3 Metabolism

Mode of Action - Glyphosate is a non-selective, systemic herbicide that is absorbed by the foliage and rapidly translocated throughout the plant. It inhibits various enzyme systems, interfering with amino acid and other biosynthesis reactions (Merck Index, 1989).

Plant Metabolism - Glyphosate is not metabolized by plants (Menzie, 1980).

Animal Metabolism - Glyphosate is poorly absorbed across the GI tract. That which is absorbed is largely eliminated in the feces and some in the urine (Monsanto, 1990(c)).

2.12.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.13 Imazapyr

2.13.1 Environmental Fate

Soil - Imazapyr has a half-life of 27 days in sandy loam soils (USFS, 1989). Higher temperatures and high soil moisture may increase this degradation rate (Little, 1991). The main route of degradation in soils is by photodegradation. Therefore, since imazapyr is stable in light with the absence of water, it degrades very slowly in dry soils. Microbes play a minor role in the soil metabolism of imazapyr (Little, 1991).

Water - Imazapyr is very stable in dark conditions. Imazapyr will degrade very rapidly if exposed to light in an aqueous solution (Little, 1991).

2.13.2 Transport

Soil - Leaching and Mobility - The adsorption coefficients for imazapyr are 0, 0.07, 0.17 and 0.19 for clay, clay loam, loamy sand, and sandy clay loam, respectively. As a result, imazapyr is considered to be fairly mobile (Little, 1991). Although it has relatively low adsorption coefficients, imazapyr is not considered to be a potential groundwater contaminant because of its rapid degradation following irrigation or rain.

2.13.3 Metabolism

Mode of Action - Imazapyr is a non-selective systemic herbicide that is absorbed by the roots and foliage and rapidly translocated to the meristematic regions where it accumulates. It then interferes with cell growth by inhibiting the synthesis of specific amino acids (Willis, 1991; Little, 1991).

Plant Metabolism - Very few plant species metabolize imazapyr. Most of imazapyr is excreted through the roots as the unmetabolized acid. The plant half-life is approximately 12 to 40 days (Little, 1991).

Animal Metabolism - Approximately 87% of the imazapyr, when dosed to rats, is excreted in the urine and feces within 24 hours as the unmetabolized acid (American Cyanamid, 1986).

2.13.4 Potential Regional Impacts

The persistence, mobility and relatively slow degradation rate of imazapyr in dry soils would be expected to influence soil half-life in the drier areas east of the Cascades, especially in non-irrigated areas.

2.14 Isoxaben

2.14.1 Environmental Fate

Soil - Conflicting studies have reported soil half-lives of 34 days for turf and 146 for bare soil applications and up 5 to 6 months under other conditions. Isoxaben is degraded primarily by oxidation and dealkylation of the alkyl side chain, with metabolism of the isoxazole ring also occurring (Dow/Elanco, Undated(b); Jones, 1991).

Water - Isoxaben's aquatic half-life ranges from 3.9 to 65.3 days. Much of this degradation is a result of photodegradation. Isoxaben is not susceptible to hydrolysis (Dow/Elanco, Undated(b); Jones, 1991).

2.14.2 Transport

Soil - Leaching and Mobility - Isoxaben has an adsorption coefficient of 0.81 to 6.63 (Jones, 1991). The lower range of this coefficient indicates a tendency to move but the higher end of the coefficient range indicates that it has a high affinity for soil particles and organic matter. It also has a very low water solubility which helps prevent leaching. Isoxaben is considered to be unlikely to move through the soil column (Jones and Stokes, 1991).

2.14.3 Metabolism

Mode of Action - Isoxaben is a selective preemergence herbicide that is absorbed by the roots and translocated to the stem and leaf tissues where it disrupts leaf and shoot development. It must be activated by rainfall or irrigation (Dow/Elanco, Undated(b); Jones, 1991).

Plant Metabolism - Isoxaben is taken into the plant in very low levels. That which is absorbed is metabolized primarily through hydroxylation of the alkyl side chain (Jones, 1991; Hartley and Kidd, 1987).

Animal Metabolism - The only available information on animal metabolism indicates that isoxaben is metabolized and excreted very rapidly (Jones, 1991).

2.14.4 Potential Regional Impacts

The adsorption coefficient range indicates that isoxaben is more mobile in soils with less organic matter. This would include most of the regions east of the Cascades. However, its low water solubility significantly reduces the threat of leaching in these areas.

2.15 Mecoprop

2.15.1 Environmental Fate

Soil - The soil half-life data were derived from soils tested in Finland. In north Finland soils mecoprop had a half-life of 20 days while in southern Finland the half-life was 60 days (Menzie, 1980). The primary soil metabolic pathway involves degradation of the

side chain to 4-chloro-2-methylphenol followed by ring hydroxylation and ring opening (Hartley and Kidd, 1987).

Water - The aquatic half-life and metabolism is not required by the US EPA for registration under mecoprop's current label. No data were available.

2.15.2 Transport

Soil - Leaching and Mobility - Adsorption coefficients range from 0.19 in sand to 0.43 in silt loam indicating that mecoprop is relatively mobile (USDA, 1987).

2.15.3 Metabolism

Mode of Action - Mecoprop is a selective systemic herbicide that is absorbed by the leaves and translocated to the roots where it acts as a plant hormone to inhibit growth (Hartley and Kidd, 1987).

Plant Metabolism - Metabolism in plants is very similar to the metabolism in soil. Eighty-five percent of the material absorbed into the plant is hydroxylated at the methyl group. Some ring metabolism occurs but it is a relatively minor metabolic pathway (Leeming, 1991).

Animal Metabolism - There is very little information concerning the metabolism of mecoprop in animals. Personal communications with the manufacturer indicated that data are available to show that 80% of the mecoprop is excreted in the urine within 8 hours of exposure (Leeming, 1991).

2.15.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.16 Mefluidide

2.16.1 Environmental Fate

Soil - Information from the manufacturers of this product indicated that many of these studies are in progress at the time this draft was being written. Therefore, no information is available at this time concerning soil half-lives or metabolism (Armbruster, 1991).

Water - Aquatic half-lives and metabolism data are not required by the US EPA for registration of this product for current label uses. Therefore, no information is available for aquatic environmental fate (Armbruster, 1991).

2.16.2 Transport

Soil - Leaching and Mobility - Studies to determine the adsorption coefficients and movement potential of mefluidide in soils were in progress at the time this DEIS was

being written. Therefore, no data are available concerning leachability or movement of mefluidide (Armbruster, 1991). Mefluidide is considered to have a low probability of moving through the soil column (Jones and Stokes, 1991).

2.16.3 Metabolism

Mode of Action - Mefluidide inhibits the growth and development of meristematic regions of susceptible plant species (Armbruster, 1991).

Plant Metabolism - Information was available from one study and indicated that mefluidide was metabolized to 4,6-diamino-*m*-xylene, 6-acetamide-4-amino-*m*-xylene and other conjugates (Menzie, 1980).

Animal Metabolism - Studies designed to determine the metabolism of mefluidide in animals were in progress at the time this DEIS was being written (Armbruster, 1991).

2.16.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.17 Metsulfuron Methyl

2.17.1 Environmental Fate

Soil - Metsulfuron methyl has a half-life of approximately 8 months in silt loam soils at 25°C. The primary degradates include dihydroxy methyl triazine and carbon dioxide (Rhodes, 1986).

Water - The aquatic half-life for metsulfuron methyl ranges from 36 days to 139 days in sterile water. Anaerobic metabolism may produce the following metabolites; free acid and O-demethyl analogs; O-demethyl triazine amine; bis-O-demethyl analog; and dihydroxymethyl triazine. The final products of aquatic metabolism are carbon dioxide and methane (Swanson, 1988).

2.17.2 Transport

Soil - Leaching and Mobility - Metsulfuron methyl is poorly adsorbed on neutral sandy loam and slightly alkaline loam soils with adsorption coefficients (K_d) of 0.05 and 0.15 respectively. It is weakly adsorbed on slightly alkaline sandy clay loam and clay loam soils with K_d values of 0.3 and 0.6, respectively. It is also weakly adsorbed on acidic clay loam and sandy loam soils with K_d values of 0.3 and 4.9 respectively (Yang, 1987). Given the above information and its slow degradation rate, metsulfuron does have the potential to move through the soil column.

2.17.3 Metabolism

Mode of Action - Metsulfuron methyl is a systemic, pre- or post-emergence herbicide that is absorbed by the roots and foliage. It is translocated to the meristematic regions where it inhibits cellular division (Hartley and Kidd, 1987).

Plant Metabolism - A study performed in cereal/grain crops indicated that metsulfuron methyl was hydrolyzed rapidly to non-toxic metabolites. The specific metabolites were not identified (Hartley and Kidd, 1987).

Animal Metabolism - Metsulfuron methyl is rapidly eliminated once ingected by animals. One study indicated that 91% of the compound was eliminated within 96 hours of ingestion (USEPA, 1986).

2.17.4 Potential Regional Impacts

Since metsulfuron methyl is either poorly or weakly adsorbed to soils of varying organic content, and has a fairly long half-life in soil, consequently, this compound has a potential to move through the soil column.

2.18 Napropamide

2.18.1 Environmental Fate

Soil - There is conflicting data concerning the half-life of napropamide in soils. One source identified a half-life of 8 to 12 weeks (Hartley and Kidd, 1987). Another study identified the half-life from 9 to 10 days. The photodegradation of napropamide in soil results in a half-life of 28 days (Keift, 1991). Napropamide degrades primarily through photodegradation to the following metabolites; 2-(a-naphthoxy)-N-ethylpropionamide;2-(naphthoxy)propionamide-1-naphthol and 1,4-naphthoquinone (Hartley and Kidd, 1987).

Water - There were no data found on the aquatic half-life or metabolism of napropamide.

2.18.2 Transport

Soil - Leaching and Mobility - Napropamide has an adsorption coefficient of 3.4 to 14.8 indicating that it has an affinity to soil particles although it may not be especially strong (Keift, 1991). This K_d range does not present a significant risk to movement through the soils and into the groundwater. Napropamide is considered among the compounds that are unlikely to move through the soil column (Jones and Stokes, 1991).

2.18.3 Metabolism

Mode of Action - Napropamide is a systemic herbicide which is absorbed by the roots and translocated throughout the plant. It kills the plant by inhibiting root development and growth (Hartley and Kidd, 1987).

Plant Metabolism - Once absorbed into the plant, napropamide is rapidly metabolized to water soluble metabolites (Hartley and Kidd, 1987).

Animal Metabolism - After oral exposures to napropamide in animals, 98.6% is excreted within 96 hours (Hartley and Kidd, 1987).

2.18.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.19 Oryzalin

2.19.1 Environmental Fate

Soil - Oryzalin has a soil half-life of approximately 2.1 months (Blewett, 1991). The only information found concerning the soil metabolism of oryzalin indicated that it is rapidly degraded (Menzie, 1980).

Water - Data on aquatic half-lives and metabolism are not required by the EPA for registration of oryzalin under current label uses (Blewett, 1991). Therefore, no data are available.

2.19.2 Transport

Soil - Leaching and Mobility - Although no adsorption coefficients are presented, the available data identifies oryzalin as a compound that strongly adsorbs to soil particles and resists movement by water (Dow/Elanco, Undated(c)). It has not been detected below 6 inches of soil (Blewett, 1991). This would be supported by its very low water solubility (2.5 ppm) (Dow/Elanco, Undated(c); Farm Chemicals Handbook, 1991). Oryzalin is considered to be unlikely to move through the soil column (Jones and Stokes, 1991).

2.19.3 Metabolism

Mode of Action - Oryzalin is a preemergence herbicide with medium residual activity and inhibits growth by preventing seed germination and root development (Dow/Elanco, Undated(c)).

Plant Metabolism - Oryzalin is not readily absorbed by plants, therefore, unmetabolized oryzalin is rarely detected in plant tissues (Blewett, 1991).

Animal Metabolism - Oryzalin is rapidly metabolized and excreted in the urine and feces. In the rat, 97% of the oryzalin is excreted in 72 hours (Dow/Elanco, undated(c)). No residues have ever been found in milk or edible tissues of animals exposed to oryzalin (Blewett, 1991).

2.19.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.20 Oxadiazon

2.20.1 Environmental Fate

Soil - Two differing half-life ranges have been reported for oxadiazon. The first ranges from 180 to 365 days (Michaich, 1991) and the other from 3 to 6 months (Hartley and Kidd, 1987). The only soil data collected on soil metabolism indicate that oxadiazon is degraded slowly with the formation of some carbon dioxide (Menzie, 1980).

Water - There is no information available from the manufacturers concerning half-lives or metabolism in aquatic environments. This is most likely because oxadiazon is not registered for aquatic uses. However, some hydrolysis data are available. For pH 5, 7 and 9 the hydrolysis half-lives are 124, 114 and 14 days, respectively (Mihaich, 1991).

2.20.2 Transport

Soil - Leaching and Mobility - Oxadiazon is considered to be immobile in the soil (Mihaich, 1991). It is strongly absorbed to soil with very little movement or leaching (Hartley and Kidd, 1987). Oxadiazon is considered to be unlikely to move through the soil column (Jones and Stokes, 1991).

2.20.3 Metabolism

Mode of Action - Oxadiazon is a broad spectrum, preemergence herbicide (Mihaich, 1991). It kills young germinating seedlings as they come into contact with the herbicide.

Plant Metabolism - There are no data available on the metabolism of oxadiazon by plants. Oxadiazon is a contact and not a systemic herbicide (Hartley and Kidd, 1987). Therefore, metabolism of oxadiazon within the plant is unlikely.

Animal Metabolism - After an oral administration of oxadiazon, 93% of the chemical is eliminated within the first 72 hours (Menzie, 1980). Another study found that there were no residues in cows or quail after 12 days after oral exposure (Hartley and Kidd, 1987).

2.20.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.21 Picloram

2.21.1 Environmental Fate

Soil - Picloram is moderately to highly persistent in soils with half-lives ranging from one month in favorable conditions to greater than four years in arid regions (USFS, 1988). It is very slowly degraded by soil microbes, especially in cold climates (USFS, 1988) and microbial degradation ceases completely in anaerobic conditions (Mullison, Undated). It is primarily degraded through photodegradation with a half-life of 7 days (Mullison, Undated).

Water - Picloram's half-life in water is approximately 2.5 days (Karwal, 1991). It is primarily degraded by photodegradation, especially in clear shallow water. It is very resistent to hydrolysis with a hydrolysis half-life of 18 years (Mullison, Undated).

2.21.2 Transport

Soil - Leaching and Mobility - Picloram is considered to be fairly mobile especially the potassium salts and the acids (USFS, 1988). This combined with its slow degradation rate makes it a concern for movement through the soil column.

2.21.3 Metabolism

Mode of Action - Picloram is a pre- or postemergence systemic herbicide that is absorbed by the roots and foliage and translocated to the rest of the plant. It controls plant growth by acting as a plant growth regulator (USFS, 1988).

Plant Metabolism - Very little data were collected concerning plant metabolism of picloram, but the data collected do indicate that it accumulates in plant tissues (USFS, 1988), which would be expected in a systemic herbicide.

Animal Metabolism - Picloram is rapidly excreted, unmetabolized in the urine (USFS, 1988, 1989). One study found that cows excrete 97.7% of the picloram within the first five days after exposure (Mullison, Undated).

2.21.4 Potential Regional Impacts

Arid regions east of the Cascade mountains, especially the Columbia Basin are areas in which picloram may have extended half-lives. In addition, its movement within soils makes it a concern statewide. The Washington State Department of Agriculture has developed state restrictions on pichloram use in certain regions of the state.

2.22 Pronamide

2.22.1 Environmental Fate

Soil - The soil half-life depends on temperature, soil moisture content and pH and it generally ranges from one to three months (Hartley and Kidd, 1987). Increased temperature, moisture and pH all decease the half-life. In soils pronamide is primarily degraded by microbes to a final degradation product, carbon dioxide (Nelson, 1991).

Water - There were no aquatic half-life or metabolism data found for pronamide.

2.22.2 Transport

Soil - Leaching and Mobility - Pronamide is relatively non-mobile in soils with an adsorption coefficient of 3.15 in clay and 10.1 in loam (Nelson, 1991). Pronamide is considered among those chemicals that are unlikely to move through the soil column (Jones and Stokes, 1991).

2.22.3 Metabolism

Mode of Action - Pronamide is a preemergence medium to long residual, systemic herbicide that is absorbed by the roots and translocated throughout the rest of the plant (Hartley and Kidd, 1987). It kills the plants by inhibiting the photosynthetic process.

Plant Metabolism - No data have been found for pronamide which elaborate on the mechanism of metabolism in plants.

Animal Metabolism - No data have been found.

2.22.4 Potential Regional Impacts

The data available do not indicate that there are any specific regional concerns for this compound.

2.23 Sethoxydim

2.23.1 Environmental Fate

Soil - The soil half-life for sethoxydim is 3 to 181 days in the field. Low soil moisture is the primary variable that increases the half-life (BASF, Undated(hh)). The aerobic half-life ranges from 0.9 days in sandy loam to 0.7 days in sandy clay loam (BASF, Undated(ff)). The anaerobic half-life ranges from 60 days in sandy loam and 30 days in sandy clay loam (BASF, Undated(gg)). Sethoxydim degrades primarily to a sulfoxide metabolite (BASF, Undated(dd)).

Water - Data for the half-life and metabolism of sethoxydim in aquatic environments were under development at the time this DEIS was written. Photodegradation has been shown to occur in water with a half-life of 5.5 days at pH 6.7 and 25C (BASF, Undated(ee)). Sethoxydim also has a hydrolysis half-life of 8.7, 155 and 284 days at pH 5, 7 and 9, respectively (BASF, Undated(cc)).

2.23.2 Transport

Soil - Leaching and Mobility - There is no evidence that sethoxydim is mobile within the soil or that it leaches. No residues have been found in soil samples greater than eight inches deep (BASF, Undated(hh)). Sethoxydim is considered to be among the chemicals that are unlikely to move through the soil column (Jones and Stokes, 1991).

2.23.3 Metabolism

Mode of Action - Sethoxydim is a systemic, broad-spectrum postemergence herbicide that is absorbed by the foliage and translocated to the root system where it inhibits plant growth (Hartley and Kidd, 1987).

Plant Metabolism - Once sethoxydim is absorbed into the plant it is rapidly oxidized and converted to sulfoxides and sulfones (BASF, Undated(kk)).

Animal Metabolism - In the rat, 98% of the sethoxydim is excreted as sulfoxide and sulfone metabolites following an oral dose. The remaining 2% remains in the body (liver, kidney) (BASF, Undated(y), Undated(nn), and Undated(mm)).

2.23.4 Potential Regional Impacts - The data available do not indicate that there are any specific regional concerns for this compound.

2.24 Sulfometuron Methyl

2.24.1 Environmental Fate

Soil - The soil half-life of sulfometuron methyl ranges from 10 days in sandy loam soil (USFS, 1989) to 34 days in other soils (Ryan and Atkins, 1986). It is primarily degraded by hydrolysis and microbial action (Anderson and Dulka, 1985). In addition to carbon dioxide and saccharin, at least three other major aromatic sulfonyl breakdown products have been identified (Harvey, Dulka and Anderson, 1985).

Water - There were no data available for aquatic half-lives and metabolism of sulfometuron methyl in aquatic environments. Photolysis, however, has been determined to play a minor role in the degradation of this product in water (Ryan and Atkins, 1986). In addition, hydrolysis does occur at pH 5 converting sulfometuron methyl to methyl-2-(aminosulfonyl)-benzoate and saccharin (Harvey, Dulka, and Anderson, 1985).

2.24.2 Transport

Soil - Leaching and Mobility - Very few data were available which described the leaching or mobility of sulfometuron methyl. Sulfometuron methyl is considered among those chemicals that are unlikely to move through the soil column (Jones and Stokes, 1991).

2.24.3 Metabolism

Mode of Action - Sulfometuron methyl is a broad-spectrum, systemic herbicide that is absorbed by the roots and foliage and translocated throughout the plant. It inhibits mitotic cell division in the growing tips of roots and plants (Hartley and Kidd, 1987).

Plant Metabolism - There were no data available on the metabolism of sulfometuron methyl by plants.

Animal Metabolism - No data available.

2.24.4 Potential - Regional Impacts Summary

There are no impacts of regional concern.

2.25 Triclopyr

2.25.1 Environmental Fate

Soil - The average soil half-life is approximately 30 days. This varies considerably with differences in soil moisture and temperature. Triclopyr can undergo rapid microbial breakdown in the soil (Dow/Elanco, Undated(d)).

Water - There was very little information collected concerning the half-lives and metabolism of triclopyr in aquatic environments. However, the rapid photodegradation of triclopyr along with the relatively rapid hydrolysis of triclopyr in aqueous solutions indicate a relatively short half-life in water (Dow/Elanco, Undated(d)).

2.25.2 Transport

Soil - Leaching and Mobility - The triclopyr acid has a very low leaching potential (Dow/Elanco, Undated(d)). Triclopyr is listed (Jones & Stokes, 1991) as one of those chemicals that is unlikely to move through the soil column.

1.2.25.3 Metabolism

Mode of Action - Triclopyr is a selective, systemic herbicide that is absorbed by the foliage and roots and translocated throughout the plant. Residues are eventually concentrated in the meristematic tissue. Triclopyr kills plants by inducing an auxin-like response (Dow/Elanco, Undated(d)).

Plant Metabolism - No data were found for the metabolism of triclopyr by plants.

Animal Metabolism - Triclopyr is rapidly excreted through the urine as unchanged triclopyr (Dow/Elanco, Undated(a)).

2.25.4 Potential Regional Impacts

There are no impacts of regional concern.

3.0 Herbicides (inert ingredients)

3.1 Petroleum Distillates (Naphthalene and related compounds)

3.1.1 Environmental Fate

Soil - Naphthalene is reported to have a soil half-life of 16.6 to 48 days (Howard, et al., 1991).

Water - In water, naphthalene has an aerobic half-life of approximately 20 days while its anaerobic half-life jumps to 25 to 258 days, depending on environmental conditions (Howard, et al., 1991).

3.1.2 Transport

Soil - Leaching and Mobility - No data were found concerning the leaching potential of petroleum distillates or their likelihood of contaminating groundwater. The upper end of the anaerobic aquatic half-life and the unknown mobility allow no conclusion about petroleum distillates with regard to movement through the soil column.

3.1.3 Metabolism

Plant Metabolism - No data found.

Animal Metabolism - No data found.

3.1.4 Potential Regional Impacts

No impacts of regional concern.

3.2 Petroleum Distillates (Xylene)

3.2.1 Environmental Fate

Soil - Xylene has a reported half-life in the soil of one to four weeks (Howard, et al., 1991).

Water - Xylene has an aerobic aquatic half-life of one to four weeks and an anaerobic aquatic half-life of six to twelve months (Howard, et al., 1991).

3.2.2 Transport

Soil - Leaching and Mobility - There were no data specific to xylene concerning its leaching potential or its likelihood of contaminating groundwater. The relatively short half-life of xylene in the soil would certainly help to reduce the likelihood of movement through the soil, but its long half-life in anaerobic waters could result in persistent residues in anaerobic aquatic systems.

3.2.3 Metabolism

Plant Metabolism - No data found.

Animal Metabolism - No data found.

3.2.4 Potential Regional Impacts

There are no impacts of regional concern.

4.0 Adjuvants

Adjuvants (or "additives") are a class of chemical used to enhance the action of an herbicide by improving the contact that an herbicide has with the target vegetation. This is generally achieved by lowering the surface tension of the spray solution or by increasing the wetability of the spray solution. These adjuvants are often referred to as surfactants, wetting agents or spreaders and include products such as Get Down, More, R-11, Sta-Put, Super Spred 200, X-77 and LI700. Adjuvants which contain only a surfactant and a petroleum distillate generally act by increasing the wetability of the herbicide. Examples of these adjuvants include Mor-Act and Nalcotrol. Silicone based adjuvants such as No Foam are generally used as foam suppressors. There are other adjuvants that color (Hi-Lite and Blazon) the tank mix so an applicator can see the spray pattern. Some adjuvants are used to mask the odors of some herbicides (Odor Mask).

The adjuvants described above are not regulated by the same criteria as are the active ingredients in the herbicides themselves. As a result there are significantly less data available for these products. Therefore, instead of addressing the environmental fate of each, this discussion will touch on the two general classes of adjuvant; surfactant, and foam suppressants.

4.1 Surfactants

Surfactants have been shown to adsorb to soil and particularly to the upper layer of soil (2cm). Wetting agents have also demonstrated an affinity for soil. However, as is the case with most chemicals, adsorption is dependent on concentrations applied and specific soil properties such as organic matter content and moisture. The nature of these products is to enhance adsorption or surface contact and binding.

Microbial degradation of surfactant may occur. If it occurs, it is generally related to the concentration applied and it is most prevalent when soil adsorption is low.

4.2 Foam Suppressants

No data were found concerning the environmental fate and metabolism of foam suppressants. Foam suppressants may be as simple as corn oil and are generally based on organic compounds which would be expected to serve as a carbon source for soil microflora.

5.0 Fertilizers

A list of fertilizers to be used by WSDOT is presented below in Table 1-1. Fertilizers are generally divided into primary nutrients (nitrogen, phosphorus, potassium), secondary nutrients (calcium, magnesium, sulfur), and micronutrients (boron, chlorine, cobalt, copper, iron, etc.). Dolomite, limestone, gypsum, and dolomitic limestone listed in Table 1-1 are intended to provide the secondary nutients calcium and magnesium in varying amounts. The remaining fertilizers in Table 1-1 are formulated to provide varying amounts of the primary nutrients.

The fertilizer grade is expressed as a set of three numbers that represent the percent of total nitrogen, available phosphate and soluble potash (potassium). For example, a fertilizer grade of 5-10-15 contains 5% nitrogen, 10% phosphorus, and 15% phosphate.

5.1 Terrestrial Fate

In the terrestrial environment, fertilizers are broken down, adsorbed to the soil (phosphorus) or absorbed by the plants relatively quickly and are generally not long lived in the soils. Part of the purpose of fertilization is to stimulate soil microflora and vegetative growth. Increased soil metabolism and increased plant growth promote the incorporation of nutrients into living systems.

5.2 Aquatic Fate

Of greatest concern to aquatic environments is the impact that excess fertilizers can have on aquatic systems. Direct losses of soil nitrates into water systems either dissolved in runoff or in sediments can cause an excess of nutrients to build in an aquatic system. These nutrients can then cause algae and other aquatic organisms to grow rapidly, resulting in water that is unfit or undesireable for drinking or recreation. In addition, the rapid decay of plant material in aquatic systems causes oxygen depletion which can be fatal to aquatic animals.

Phosphorus has the potential to create the same problems as does nitrogen. This depends on which of the nutrients are the limiting factors for growth in an aquatic system.

Table 1-1. Fertilizers To Be Used By WSDOT For Roadside Vegetation Management

Name	Ingredients	Formulation
Dolomite	60% $CaCO_3$, 40% $MgCO_3$	Dry material, course ground
Limestone	90% $CaCO_3$	Dry pelleted
Gypsum	60% $CaSO_4$	Dry pelleted
Dolomitic Limestone	50% $CaCO_3$, 40% $MgCO_3$	Dry pelleted
16-16-16	2% sulfur	Homogenous pellet
34-0-0	Ammonium nitrate	Homogenous pellet
8-10-8	50% nitrogen from urea formaldehyde	Blended granular material
15-5-7	17% sulfur	Homogenous pellet
38-0-0	Urea formaldehyde	Homogenous granule
10-20-20	Data Not Available	Blended granular material
24-4-12	45% N from urea formaldehyde	Data Not Available
18-5-11	Data Not Available	Data Not Available
6-2-0	4.5% iron	Data Not Available
16-20-02	Data Not Available	Homogenous pellet
21-3-5	Data Not Available	Homogenous pellet
16-16-16	0.5% zinc, 0.18% boron, 5% sulfur	Blended granular material
32.8-0-0	0.55% boron, 5% sulfur	Blended granular material
16-6-8	18% sulfur, 1.5% iron, 0.1% zinc	Homogenous pellet
21-10.5-10.5	Data Not Available	Homogenous pellet
18-3-18	15% ammonium sulfate, 30% potash sulfate, 5% sulfur, 0.25% iron, 0.05% zinc 0.05% manganese, 0.01% copper	Data Not Available

Appendix B - Chapter 2

Draft EIS
Roadside Vegetation Management

Toxicity Data

1.0 Introduction

1.1 Objectives

An ecological risk assessment is a qualitative and/or quantitative appraisal of the actual or potential effects of hazardous materials on animals and plants other than people and domesticated animals (USEPA, 1989). The introduction of contaminants into an ecosystem can cause direct harm to organisms, or may indirectly affect their ability to survive and reproduce. The results of contamination may be immediately apparent or may become noticeable only after considerable delay. The effects of contaminants on ecosystems are due in part to the physical and chemical properties of the chemicals themselves, but are also mediated by the unique combination of physical, chemical, and biological processes occurring in each ecosystem. In addition, populations of exposed organisms can differ in their response to contaminants depending on their natural tolerance to the chemical, their behavioral and life-history characteristics, the dose to which they are exposed, and the duration of exposure. Furthermore, responses may be transient (and therefore reversible) or permanent (irreversible) (USEPA, 1989).

Ecological assessment seeks to determine the nature, magnitude, and transience or permanence of observed or expected effects. This must be accomplished in an environment that is itself changing and causing change in the organisms and systems under study.

1.2 Techniques

The assessment presented here is a qualitative assessment of risk. It is a non-mathematical examination of the chemical properties (toxicity, fate, metabolism) that influence the impact that these chemicals will have on wildlife. The first section of this chapter reviews the available toxicity data for mammals, birds, insects, and aquatic species (fish, invertebrates). These data are also included in the toxicity tables in Chapter 4 of this appendix along with other toxicity values not discussed in this section. The relative toxicities will be graphically presented in Figures 2-1 through 2-3. These graphs will identify which of the 25 active ingredients and the two inert ingredients are considered to be practically non-toxic, slightly toxic, moderately toxic, highly toxic or very highly toxic according to the criteria presented in Table 2-1.

In the assessment of toxicity of pesticides, standardized tests are conducted and the results are statistically derived in "acute" (short term) or "chronic" (long term) studies. A tiered approach is used in the conduct of these studies so that resources and living organisms are spared needless use. Testing begins with "acute" (short term) studies, and if results indicate a need, testing progresses to "chronic" (long term) studies. In the assessment of WSDOT pesticide impact, it is more appropriate to utilize values derived from acute

studies, although chronic values, where available, will also be considered. Use of acute values parallels the "once a season per area" use pattern of WSDOT applications.

In Section II the toxicity information will be compared, qualitatively, with information presented in Appendix B, Chapter 1 (Fate, Transport and Metabolism of Herbicides, Adjuvants and Fertilizers). A review of the potential impacts of chemical vegetation management on wildlife is then presented with an emphasis on regional differences of effects between the phytogeographic regions.

Section I uses the descriptions of toxicity that are established by the Environmental Protection Agency (EPA). These descriptions vary slightly depending on the class of animal being tested (avian or mammalian) and the study being performed (acute oral LD_{50} or dietary LC_{50}). LD_{50} is the statistical derivation of a value shich is predicted to cause 50% mortality in the given population being tested. LC_{50} is a similar number, based on the concentration of a compound in feed, air or water. The criteria for these descriptions are presented below in Table 2-1.

2.0 Toxicological Issues

2.1 Herbicide Formulations

The toxicity of various formulations of the same active ingredient are not addressed in this risk assessment except in specific cases in which different formulations significantly reduce or increase the toxicity of an active ingredient. In these cases the various toxicities of that active ingredient will be addressed.

2.2 Synergistic Effects

Synergistic effects of chemicals are those that occur from exposure to two chemicals either simultaneously or within a relatively short period of time. Synergism occurs when the combined effects of two chemicals is greater than the sum of the effects of each agent individually (simple additive effect). Instances of chemical combinations that cause synergistic effects are relatively rare. Kociba and Mullison (1985, as cited in USDA, 1989), in describing toxicological interactions with agricultural chemicals state:

> "Our present scientific knowledge in toxicology indicates that an exposure to a mixture of pesticides is more likely to lead to additivity or antagonisms rather than synergism when considering the toxicological effects of such a combination. To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response."

In the case of registered pesticides, a great amount of toxicological information is developed during the research and development of each individual pesticide. In addition to this information on individual pesticides, short term toxicity studies are always done prior to the selling of a pesticide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two pesticides, it would be identified in such cases and would then be dealt with accordingly. In toxicological tests involving a combination of commercial pesticides, synergism has not generally been observed.

Synergistic toxic effects of herbicide combinations other than EPA-registered commercial mixtures are not normally studied. Time and money normally limit toxicity testing to the effects of the herbicides individually. Combinations that could be tested are too numerous to make that testing feasible. Based on the limited amount of data available on pesticide combinations, it is unlikely that synergistic effects could occur as a result of exposure to two or more of the herbicides considered in this analysis.

2.3 Conflicting Data

In cases where data collected for this risk assessment conflict, then data that were generated under generally accepted EPA guidelines will be used for this risk assessment. Data presented to the EPA for the purpose of registration of a product have been generated using standardized tests adopted by the EPA. In using this data it becomes possible to compare toxicity values from compound to compound and from species to species. Other data may not have been generated using a standardized method and, therefore, comparisons with data by other means is inappropriate. If there were no data available that have been generated under EPA guidelines, then the values used will be those that are the most conservative or would pose the greatest risk.

3.0 Herbicides (active ingredients)

3.1 2,4-D

3.1.1 Terrestrial Wildlife

Mammalian - 2,4-D is considered to be moderately toxic to mammals based on an acute oral LD_{50} of 375 mg/kg in male rats (USDI, 1989) and 370 mg/kg in female rats (USDHHS, 1980). Its acute dermal toxicity ranges from 1,400 mg/kg (USDHHS, 1980) to 3,980 mg/kg (USDI, 1989) indicating that it is slightly to practically non-toxic as a result of dermal exposures.

Avian - 2,4-D ranges from being moderately toxic to pheasants (LD_{50} = 472 mg/kg) (USDI, 1989) to practically non-toxic to mallards (LD_{50} > 2,000 mg/kg) (USDI, 1989; USFS, 1988). The dietary LD_{50} of >5,000 indicates that 2,4-D is practically non-toxic to birds when exposed through their diet (USDI, 1989).

Insects (bees) - The relatively high LD_{50} for bees of 11.5 - 105 μg/bee indicates that 2,4-D is essentially non-toxic to bees (USDI, 1989).

3.2 2,4-DP

3.2.1 Terrestrial Wildlife

Mammalian - The acute oral LD_{50} for rats of 500 - 800 mg/kg establishes 2,4-DP as being slightly toxic to mammals. Acute dermal LD_{50} values of 1,400 mg/kg for both the rat and mouse also indicate that 2,4-DP is only slightly toxic to mammals after dermal exposure (USDHHS, 1980).

Avian - No data were available concerning the acute LD_{50} for birds. However, the dietary LC_{50} has been reported to be > 20,000 ppm which indicates that 2,4-DP is practically non-toxic to birds exposed through their diet (Mihaich, 1991).

Insects (bees) - 2,4-DP is not considered to be toxic to bees (Hartley and Kidd, 1987).

3.3 Benefin

3.3.1 Terrestrial Wildlife

Mammalian - Benefin is considered to be practically non-toxic based on an acute LD_{50} of > 10,000 mg/kg in the rat (USDHHS, 1980). It is also supported by an acute oral LD_{50} of > 5,000 mg/kg in the mouse (Hartley and Kidd, 1987). Acute dermal LD_{50} values are also > 5,000 mg/kg (Melichar, 1991).

Avian - Benefin is practically non-toxic to birds based on an LD_{50} > 2,000 mg/kg for chickens, mallards, and quail. It is also practically nontoxic for birds exposed through their diet based on LC_{50} values of > 5,000 ppm for the northern bobwhite and the mallard (Melichar, 1991).

Insects (bees) - Benefin is considered to be essentially non-toxic to bees based on an acute contact LD_{50} of > 105 μg/bee (Melichar, 1991).

3.4 Bromacil

3.4.1 Terrestrial Wildlife

Mammalian - Bromacil's acute oral LD_{50} of 3,998 mg/kg establishes it as being practically non-toxic to mammals. An acute dermal LC_{50} of > 2,000 mg/kg for the rabbit also indicates a low toxicity to mammals (USDI, 1989).

Avian - Bromacil is considered to be practically non-toxic to birds based on an acute oral LD_{50} of 2,250 mg/kg for the northern bobwhite (Wellings, 1991). An avian dietary LC_{50} of 10,000 ppm in the mallard and northern bobwhite also establishes bromacil as being practically non-toxic when exposed through the diet (USDI, 1989; Hartley and Kidd 1987).

Insects (bees) - It has been established that bromacil is essentially non-toxic to honey bees (Wellings, 1991; Hartley and Kidd, 1987).

3.5 Chlorsulfuron

3.5.1 Terrestrial Wildlife

Mammalian - Chlorsulfuron is considered to be practically non-toxic to mammals based on acute oral LD_{50} values of 3,053 mg/kg for the male rat and 2,341 mg/kg for the female rat (Dupont,1989(b); Denny, 1991). This is supported by an acute dermal LD_{50} of > 2,000 mg/kg in the rabbit (Dupont,1989(b)).

Avian - Avian studies also indicate that chlorsulfuron is practically non-toxic based on an acute LD_{50} for the northern bobwhite and the mallard of > 5,000 mg/kg (USDI, 1989; Hinkle, 1980). The dietary LC_{50} of > 5,000 ppm also establishes chlorsulfuron as practically non-toxic to birds (USDI, 1989; Hinkle, 1979).

Insects (bees) - Chlorsulfuron is considered to be essentially non-toxic to bees based on an acute contact LD_{50} of > 25 μg/bee (Meade, 1983).

3.6 Chlorthal Dimethyl

3.6.1 Terrestrial Wildlife

Mammalian - Although differing LD_{50} values are presented in the toxicity tables, they both indicate the chlorthal dimethyl is practically non-toxic to mammals. This is also supported by an acute dermal LD_{50} for the rabbit of > 10,000 mg/kg (Hartley and Kidd, 1987).

Avian - There were no data available at the time the DEIS was prepared.

Insects (bees) - Chlorthal dimethyl is considered to be slightly toxic to bees (Hartley and Kidd, 1987).

3.7 Dicamba

3.7.1 Terrestrial Wildlife

Mammalian - Dicamba is considered to be slightly toxic to mammals based on an acute oral LD_{50} range from 757 - 1701 mg/kg for the rat (Sandoz, 1992). This is also supported by an acute dermal LD_{50} of > 2,000 mg/kg for the rabbit (Sandoz, 1992; IRDC, 1974).

Avian - Avian LD_{50} values of 637 mg/kg for the pheasant (USDI, 1989) and 2,000 mg/kg for the mallard (USDI, 1989; Sandoz, 1992) establishes dicamba as being moderately to slightly toxic to birds. However, the dietary LD_{50} for mallards and northern bobwhites is > 10,000 ppm indicating that dicamba is practically non-toxic when birds are exposed through their diet (USDI, 1989; Sandoz, 1992).

Insects (bees) - Dicamba is considered to be essentially non-toxic based on an acute contact LD_{50} of > 100 μg/bee (Stevenson, 1978).

3.8 Dichlobenil

3.8.1 Terrestrial Wildlife

Mammalian - Dichlobenil toxicity ranges from slightly toxic for the guinea pig (681 mg/kg) (USDHHS, 1980) to practically non-toxic for the mouse and rat (2,056 mg/kg (USDHHS, 1980; Uniroyal,Undated(a)) and > 3,160 mg/kg, respectively (Uniroyal, Undated(a)). This is supported with an acute dermal LC_{50} of 1,350 mg/kg for the rabbit (USDHHS, 1980; Uniroyal, Undated(a)).

Avian - Dichlobenil is considered to be moderately to slightly toxic based on acute LD_{50} values ranging from > 50 mg/kg to 1,000 mg/kg. The acute dietary LC_{50} values range from 1,500 mg/kg in the pheasant to > 5,200 mg/kg for the northern bobwhite and the mallard (Roberts and Hawkin, 1988(a)). These numbers establish Dichlobenil as being slightly to practically non-toxic to birds exposed to dichlobenil through their diet.

Insects (bees) - Dichlobenil is essentially non-toxic based on an acute contact LD_{50} of > 100 μg/bee (Atkins, et al., 1981).

3.9 Diuron

3.9.1 Terrestrial Wildlife

Mammalian - Diuron is practically non-toxic based on rat LD_{50} values ranging from 3,750 mg/kg (USDI, 1989) to 2,900 mg/kg (Dupont, 1989(a)). This is supported by an acute dermal LD_{50} for the rat of > 10,000 mg/kg (USDI, 1989), and an acute dermal LC_{50} for the rabbit of > 2,000 mg/kg (Dupont, 1989).

Avian - Diuron is practically non-toxic to birds based on an acute oral LD_{50} of > 2,000 mg/kg (USDI, 1989; USEPA, 1983). This is supported by an avian dietary LC_{50} of greater than 5,000 ppm (USDI, 1989) for the mallard and pheasant. The acute dietary LC_{50} for the northern bobwhite is considerably less (1,730 ppm) (USDI, 1989; USEPA, 1983).

Insects (bees) - Diuron is essentially non-toxic to bees (Wellings, 1991).

3.10 Fluazifop-butyl

3.10.1 Terrestrial Wildlife

Mammalian - Fluazifop-butyl is considered to be practically non-toxic based on an acute LD_{50} for rats of 3,328 mg/kg (Kieft, 1991; Hartley and Kidd, 1987; ICI Americas, 1990). Acute LD_{50} values for the rabbit and mouse indicate that for these species, fluazifop-butyl is slightly toxic. Acute dermal LD_{50} values for the rat also indicate the fluazifop-butyl is practically non-toxic to mammals (Hartley and Kidd, 1987).

Avian - Fluazifop-butyl is also practically non-toxic to birds based on an acute oral LD_{50} of > 17,000 mg/kg for the mallard and based on dietary LC_{50} of 18,500 ppm and 25,000 ppm for the pheasant and mallard, respectively (Hartley and Kidd, 1987).

Insects (bees) - An acute contact LD_{50} of > 200 μg/bee indicates that fluazifop-butyl is essentially non-toxic to bees (Worthing, 1991).

3.11 Fosamine

3.11.1 Terrestrial Wildlife

Mammalian - Fosamine is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of 24,400 mg/kg for the rat. This is supported by an acute oral LD_{50} for the guinea pig of 7,380 mg/kg (USFS, 1988).

Avian - An acute oral LD_{50} for the northern bobwhite and the mallard of > 5,000 (Reno, 1979) and a dietary LC_{50} for the same species of > 10,000 ppm establishes fosamine as being practically non-toxic to birds (USFS, 1989).

Insects (bees) - Fosamine is essentially non-toxic to bees based on a contact LD_{50} of > 200 μg/bee (USFS, 1989; Meade, 1983).

3.12 Glyphosate

3.12.1 Terrestrial Wildlife

Mammalian - An acute oral LD_{50} of 4,300 mg/kg to 5,600 mg/kg for rats establishes glyphosate as being practically non-toxic to mammals. This is supported by an acute oral LD_{50} for rabbits of 3,800 mg/kg and an acute dermal LD_{50} for the rabbit of > 5,010 mg/kg (USEPA, 1988).

Avian - Glyphosate is considered practically non-toxic to birds based on acute oral LD_{50} values of > 2,000 mg/kg for the mallard (USDI, 1989) and > 4,640 mg/kg for the northern bobwhite (Monsanto, 1990(c)). The dietary LC_{50} indicates that glyphosate is slightly toxic based on LC_{50} values of 4,640 mg/kg for both of these species (Monsanto, 1990(c)).

Insects (bees) - Glyphosate is essentially non-toxic to bees based on a contact LD_{50} of > 100 μg/bee (Monsanto, 1990(c)).

3.13 Imazapyr

3.13.1 Terrestrial Wildlife

Mammalian - Imazapyr is practically non-toxic based on a range of LD_{50} values from > 2,000 mg/kg for the mouse to > 5,000 mg/kg for the rat. This classification is supported by an acute oral LD_{50} of 4,800 mg/kg for the rabbit. Acute dermal LD_{50} values are also greater than 2,000 mg/kg for both rabbits and rats (American Cyanamid, 1986).

Avian - Imazapyr is also considered to be practically non-toxic to birds based on an LD_{50} value of > 2,150 mg/kg for the northern bobwhite and the mallard. It is also considered to be practically non-toxic based on a dietary LC_{50} > 5,000 ppm for the same species (American Cyanamid, 1986).

Insects (bees) - The contact LD_{50} for imazapyr is > 100 μg/bee which is essentially non-toxic to the bees (American Cyanamid, 1986).

3.14 Isoxaben

3.14.1 Terrestrial Wildlife

Mammalian - The acute oral LD_{50} values for isoxaben range from > 5,000 mg/kg for the dog to > 10,000 mg/kg for the mouse and rat (Hartley and Kidd, 1987). These values establish isoxaben as being practically non-toxic to mammals. This classification is supported by acute dermal LD_{50} values of > 2000 mg/kg) (Jones, 1991).

Avian - Isoxaben is practically non-toxic to birds based on an acute oral LD_{50} of > 2,000 mg/kg for the northern bobwhite. This is supported by the dietary LC_{50} of > 5,000 ppm for the northern bobwhite and the mallard (Jones, 1991).

Insects (bees) - Isoxaben is considered to be essentially non-toxic based on a contact LD_{50} of > 100 μg/bee (Jones, 1991).

3.15 Mecoprop

3.15.1 Terrestrial Wildlife

Mammalian - Mecoprop is slightly toxic to mammals based on an acute oral LD_{50} of 650 mg/kg for the rat (USDHHS, 1980) and mouse and slightly toxic to practically non-toxic based on an acute dermal LD_{50} of 900 mg/kg to 4,000 mg/kg for the rat and rabbit respectively (Worthing and Hance, 1991).

Avian - Acute oral LD_{50} values for birds range from moderately toxic for the mallard (> 486 mg/kg) to slightly toxic for the northern bobwhite. However, the dietary LC_{50} for the mallard indicates that mecoprop is practically non-toxic to birds when exposed through their diet (Leeming, 1991).

Insects (bees) - Mecoprop is not considered to be toxic to bees (Hartley and Kidd, 1987).

3.16 Mefluidide

3.16.1 Terrestrial Wildlife

Mammalian - Mefluidide is practically non-toxic to mammals based on an acute oral LD_{50} of 4,000 mg/kg for the rat (USDHHS, 1980). This is supported by an acute dermal LD_{50} of > 4,000 mg/kg in the rabbit (USDI, 1989; PBI Gordon, 1991(dd)). The acute LD_{50} of 1,920 mg/kg for the mouse indicates that mefluidide is slightly toxic to mammals (USDHHS, 1980).

Avian - Acute toxicities of > 2,000 mg/kg in the northern bobwhite (PBI Gordon, 1991(dd)) and > 4,640 mg/kg in the mallard (USDI, 1989) indicate that mefluidide is practically non-toxic to birds also. All dietary LC_{50} values are greater than 5,000 mg/kg

indicating that mefluidide is practically non-toxic when administered in the food (PBI Gordon, 1991(z,aa)).

Insects (bees) - Studies to produce this data were in progress at the time this DEIS was being prepared (Armbruster, 1991).

3.17 Metsulfuron methyl

3.17.1 Terrestrial Wildlife

Mammalian - Metsulfuron methyl is practically non-toxic to mammals based on an acute oral LD_{50} of > 5,000 mg/kg in the rat. This is supported by an acute dermal LD_{50} of > 2,000 mg/kg in the rabbit (USDI, 1989).

Avian - Metsulfuron is also considered to be practically non-toxic to birds based on an acute oral LD_{50} of > 2,510 mg/kg for the mallard (USDI, 1989; Wildlife International, 1981). The avian dietary LC_{50} for the mallard also establishes that metsulfuron methyl is practically non-toxic to birds (>5,620 ppm) (USDI, 1989; Wildlife International, 1980 & 1981).

Insects (bees) - The contact LD_{50} for metsulfuron methyl is > 25 μg/bee, which establishes it as being relatively non-toxicity to bees (Meade, 1984; USEPA, 1986).

3.18 Napropamide

3.18.1 Terrestrial Wildlife

Mammalian - Napropamide is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of 5,000 mg/kg for the rat (USDHHS, 1980). Dermal LD_{50} values of > 2,000 mg/kg and > 4,640 mg/kg for the guinea pig and rabbit, respectively, also indicate a relatively low toxicity to mammals (Hartley and Kidd, 1987; Kieft, 1991).

Avian - An LD_{50} of 486 mg/kg for the mallard establishes napropamide as being moderately toxic to birds (Keift, 1991). However, the dietary LC_{50}s indicate that napropamide is practically non-toxic to birds (> 5,600 for northern bobwhite and mallard) (Worthing and Hance, 1991).

Insects (bees) - Napropamide is considered to be essentially non-toxic to bees based on an acute contact LD_{50} of 121 μg/bee (Hartley and Kidd, 1987; Kieft, 1991; Worthing and Hance, 1991).

3.19 Oryzalin

3.19.1 Terrestrial Wildlife

Mammalian - Oryzalin is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of >10,000 mg/kg for the rat (USDHHS, 1980; Dow/Elanco, Undated(c)). Other values include an LD_{50} of >1,000 mg/kg for the dog which would be considered slightly toxic (Dow/Elanco, Undated(c)).

Avian - Oryzalin is considered to be slightly toxic to birds based on acute oral LD_{50} values of > 500 mg/kg in northern bobwhites and mallards and >1,000 mg/kg in chickens (Hartley and Kidd, 1987). This is supported by a high avian dietary LD_{50} of > 5,000 ppm (Blewett, 1991).

Insects (bees) - Oryzalin is not considered to be dangerous to bees when used according to label directions (Hartley and Kidd, 1987).

3.20 Oxadiazon

3.20.1 Terrestrial Wildlife

Mammalian - Oxadiazon is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of 3,500 mg/kg for the rat (USDHHS, 1980). This is supported by acute dermal LD_{50} values of > 2,000 mg/kg (Mihaich, 1991) and > 8,000 mg/kg for the rabbit and rat respectively (Hartley and Kidd, 1987).

Avian - Oxadiazon is considered to be slightly toxic to birds based on an acute oral LD_{50} of > 1,000 mg/kg in the mallard and practically non-toxic based on an acute LD_{50} of > 6,000 mg/kg in the northern bobwhite (Mihaich, 1991; Hartley and Kidd, 1987). An avian dietary NOEL has been established at 5,000 ppm for the northern bobwhite which is considered to be practically non-toxic.

Insects (bees) - Oxadiazon is considered to be toxic to bees (Hartley and Kidd, 1987).

3.21 Picloram

3.21.1 Terrestrial Wildlife

Mammalian - Picloram is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of 8,200 mg/kg for the rat (USFS, 1989). Other oral LD_{50} include 6,000 mg/kg for the chicken (USFS, 1989) and 2,000 to 4,000 mg/kg for the female mouse (Dow/Elanco, Undated(e)), all of which are considered to be practically non-toxic. The high acute dermal LD_{50} of > 3,980 mg/kg for the rabbit supports a low toxicity to mammals (Dow/Elanco, (e)).

Avian - Picloram has been established to be practically non-toxic based on an avian LD_{50} of > 2,000 mg/kg for both the mallard and pheasant (USFS, 1989). The avian dietary LC_{50} is > 5,000 ppm for the northern bobwhite, Japanese quail, pheasant and the mallard which establishes picloram as practically non-toxic to birds exposed through their diet (USFS, 1989; USEPA, 1988).

Insects (bees) - Picloram is considered to be relatively non-toxic to bees based on an LD_{50} of 14 μg/bee (USFS, 1989; USEPA, 1988).

3.22 Pronamide

3.22.1 Terrestrial Wildlife

Mammalian - Pronamide is considered to be practically non-toxic to mammals based on an acute oral LD_{50} of 5,620 mg/kg for the rat (USDHHS, 1980) and > 10,000 mg/kg for the dog (Hartley and Kidd, 1987). This is supported by a dermal LD_{50} of > 3,160 mg/kg for the rabbit (Hartley and Kidd, 1987).

Avian - Pronamide is practically non-toxic to birds based on acute avian LD_{50} values of >14,000 mg/kg for the mallard (Longacre, 1990) and 8,770 mg/kg for the Japanese quail. The avian dietary LD_{50} for both the northern bobwhite (Longacre, 1990) and the mallard is >20,000 mg/kg which is considered to be practically non-toxic (USEPA, 1986).

Insects (bees) - Although no LD_{50} value is reported here, pronamide is considered to be non-toxic to bees (Hartley and Kidd, 1987).

3.23 Sethoxydim

3.23.1 Terrestrial Wildlife

Mammalian - Sethoxydim is practically non-toxic to mammals based on LD_{50} values of 2,676 mg/kg for the female rat (BASF, Undated(oo)) and 3,125 mg/kg for the male rat (Hartley and Kidd, 1987). Relatively high acute dermal LD_{50} values (> 5,000 mg/kg for both the rabbit and the rat) also indicate a low toxicity to mammals (BASF, Undated(oo)).

Avian - Sethoxydim is considered to be practically non-toxic to birds based on an avian acute LD_{50} for the mallard of 5,620 mg/kg. Avian dietary LC_{50} values range from 5,620 mg/kg for the mallard to > 5,620 mg/kg for the northern bobwhite which also indicate that sethoxydim is practically non-toxic to birds (BASF, Undated(oo)).

Insects (bees) - Sethoxydim is considered to be non-toxic to bees based on an LD_{50} of > 10.0 mg/bee (BASF, Undated(oo)).

3.24 Sulfometuron methyl

3.24.1 Terrestrial Wildlife

Mammalian - Sulfometuron methyl is considered to be practically non-toxic to mammals based on an acute LD_{50} of > 5,000 mg/kg for the male rat. Acute dermal LD_{50} values range from > 8,000 mg/kg for the male rabbit to > 2,000 mg/kg for the female rabbit also indicate that sulfometuron methyl is of low toxicity to mammals (Dupont, Undated).

Avian - Sulfometuron methyl is considered to be practically non-toxic to birds based on an acute LD_{50} value of > 5,000 mg/kg for mallards (Hazelton Lab, 1981; USFS, 1989). Avian dietary LC_{50} values are reported at > 5,000 ppm (Dupont, Undated; Hazelton Labs, 1980; USFS, 1989) and >5,620 ppm (Dupont, Undated; Wildlife International,

1981; USFS, 1989) for mallards and northern bobwhite, respectively. These values also indicate that sulfometuron methyl is practically non-toxic to birds.

Insects (bees) - Sulfometuron methyl is considered to be moderately toxic to bees based on a contact LD_{50} of > 12.5 μg/bee (USFS, 1989).

3.25 Triclopyr

3.25.1 Terrestrial Wildlife

Mammalian - The mammalian acute oral LD_{50} values for triclopyr range from 630 to 729 mg/kg for rats (Dow/Elanco, Undated(d)) which establishes triclopyr as being slightly toxic to mammals.

Avian - Triclopyr is considered to be slightly toxic based on a reported LD_{50} of 1698 mg/kg for the mallard. However, avian dietary LC_{50} values for the mallard and the northern bobwhite are reported to be > 5,600 and 2935 ppm in the diet, respectively, which indicate that triclopyr is practically non-toxic to birds when they are exposed through their diet (Dow/Elanco, Undated(d)).

Insects (bees) - Triclopyr is considered to be relatively non-toxic based on an oral LD_{50} of > 60.4 μg/bee.

4.0 Herbicides (inert ingredients)

4.1 Petroleum Distillates

4.1.1 Terrestrial Wildlife

Petroleum distillates are considered as practically non-toxic to mammals based on acute LD_{50} values ranging from 28,000 mg/kg for kerosene and 9,000 mg/kg for diesel (Genium Publishing, 1990, 1982).

4.2 Xylene

4.2.1 Terrestrial Wildlife

Xylene is considered to be practically non-toxic based on an acute LD_{50} of 4,300 mg/kg for the rat (Genium Publishing, 1988). No data were found concerning the toxicity of xylene to other wildlife species.

5.0 Adjuvants

5.1 Terrestrial Wildlife

All of the adjuvants proposed for use by WSDOT for their roadside vegetation management program are considered to be practically non-toxic. This is based on acute oral LD_{50} values from 2,500 mg/kg to 10,000 mg/kg, with the exception of Odor Mask

which is considered to be slightly toxic based on an acute LD_{50} of 887 mg/kg (Monterey Chemical, 1989).

6.0 Fertilizers

A list of the fertilizers to be used by WSDOT is presented in Table 1-1 of Chapter 1 of this appendix. No information was found concerning the toxicity of these fertilizers to terrestrial or aquatic organisms.

6.1 Terrestrial Wildlife

There has been very little concern in the past that fertilizers such as these listed here are harmful to terrestrial wildlife species. The concern about fertilizers is that they may become groundwater contaminants that are detrimental to human health, and not that they are acutely or chronicly toxic to terrestrial wildlife. For the purposes of this review, fertilizers are not considered to pose a significant risk to terrestrial wildlife as a result of their use on roadside right-of-ways. On the contrary, the use of fertilizers could increase food sources and habitat for those animals likely to be inhabiting the roadside environment.

7.0 Uncertainty and Data Gaps

Uncertainty is inherent in risk assessments because a quantitation of risk is based on assumptions about the level and frequency of exposure. Such assumptions can be refined or limited by the use of even more data, but they can never be eliminated. Due to the programmatic nature of this impact evaluation and the fact that most WSDOT vegetation management tools are of low impact, a qualitative comparison, based on previously described parameters, has been provided. Its limitations are described below.

The estimates of exposure and the estimates of risk based on these exposures should not be taken out of context. They are based on available information and informed assumptions. When considering the risk, the bounds of this exposure assessment as well as the mitigating factors, such as the actual amount of herbicide used throughout the state or the likelihood of exposure, should be considered. This assessment is most useful for comparative purposes, that is, given the same set of exposure parameters and assumptions, the extent of exposure to each of the herbicides and the resulting estimates of risk can be compared. It is especially important to consider the uncertainty inherent in the assessment as well as any data gaps when evaluating these estimates of risk. Data gaps include actual roadside acreage, actual environmental conditions, and related information. Compound data is fairly complete for the determinations made here.

In this risk assessment, there is uncertainty associated with the estimated risks. The main sources of uncertainty occur in the evaluation of exposure for specific species and the estimation of potential hazards to populations.

Wildlife Impacts

1.0 Introduction

There are a number of the herbicides used by WSDOT which are considered to be practically non-toxic based on their acute LD_{50} values for mammals (rats) or their acute dietary LC_{50} values for birds (quail). These compounds are not considered to present a risk to wildlife statewide as a result of their use for roadside vegetation management and will not be discussed any further in this risk analysis. These chemicals include:

benefin	metsulfuron methyl
bromacil	napropamide
chlorsulfuron	oryzalin
dichlobenil	oxadiazon
fluazifop-butyl	picloram
fosamine	pronamide
imazapyr	sethoxydim
isoxaben	sulfometuron methyl
mefluidide	petroleum distillates and xylene

The remaining herbicides which will be considered in the following discussion of impacts on mammals includes: 2,4-D, 2,4-DP, dicamba, mecoprop, and triclopyr. For impacts on birds, the herbicides that will be discussed will be chlorthal dimethyl, diuron, glyphosate, and triclopyr.

Although there is very little environmental fate information for 2,4-DP, it is considered to behave very similarly as 2,4-D in the environment. This assumption will be adopted for this risk assessment.

2.0 Terrestrial Risk Assessment

This risk assessment is a qualitative assessment. Although it does involve some mathematical calculations, these calculations are not intended as a quantitative assessment of the impacts to wildlife. They are presented here as a means by which the toxicities of these herbicides can be placed into perspective.

In order to provide a basis by which the toxicity of these chemicals can be put into perspective, a number of assumptions must be made. First of all, one mammal (rat) and one bird (quail) are being discussed in this section. The mouse and the quail were chosen because they are relatively abundant in Washington and there are toxicity data available for these species and related species. Secondly, the route of exposure in this assessment is limited to oral exposure through consumption of grain treated with the chemical. Other routes of exposure (dermal) would have to be considered in order to conduct a complete quantitative risk assessement. However, this is not necessary for a qualitative risk assessement designed to provide a perspective for the toxicities. Thirdly, although the grains eaten by mammals and quail vary widely from region to region and season to season, an average estimate of the surface area and the weight of one grain of wheat are used as an estimate of the surface areas and weights of other grains. Fourthly, average

weights and average daily consumption for rats and quail were used to represent exposure. However, these numbers can exhibit a great deal of inter- and intra-species variation.

Finally, This discussion involves the consumption of wheat grain, but the environmental fate data that are generally referenced are the half-lives in soil. It should be noted that wheat grain is not likely to remain on the gound for the duration of most of these half-lives. The wheat will be eaten, or it may sprout. In either case, treated wheat will not likely be available for the length of the half-life. It is assumed that it will in this risk assessment to present a worst case scenario and this should be considered a mitigating factor.

The values which were used for the surface area and weight of one grain of wheat and the weights and daily food consumption for rats and quail are presented in Table 2-2.

This assessment of risk was performed using the data in Table 2-2 in the following manner:

1) The number of grains of wheat that would be needed to cover one acre, when laid out flat on the ground, were determined.

$$\text{No. of grains/acre} = 4047\ m^2/\text{acre} \div 1.0 \times 10^{-5}\ m^2$$

2) The number of mg of active ingredient per grain was then determined.

chemical/grain (mg) = (# lbs/acre ÷ # grains/acre) X 453592.4 mg/lb

3) The number of mg of each chemical that would be ingested for rats and quail given their daily food intake, the number of mg of chemical/grain and the weight of one grain was then calculated.

chemical ingested (mg) = (chemical/grain (mg) ÷ 1 grain (g)) X daily food intake (g)

4) Then the dose in mg of chemical/kg body weight was determined.

Dose (mg/kg) = chemical ingested (mg) ÷ rat/quail weight (kg)

5) The final step was then to compare the LD_{50}, LC_{50} and/or the NOEL with the calculated exposure.

The following discussion presents the results of this assessment.

2.1 2,4-D and 2,4-DP

2,4-D and 2,4-DP are practically non-toxic to birds based on acute feeding studies (LC_{50}). Therefore, this discussion of impacts will be limited to the effects of 2,4-D and 2,4-DP on mammals.

2.1.1 Chemical Impacts (mammals)

For mammals 2,4-D and 2,4-DP are among the most acutely toxic of the herbicides used by the Washington State Department of Transportation. They are considered to be moderately toxic to mammals with LD_{50} values for rats of 370 mg/kg (USDHHS, 1980) and 400 mg/kg (USFS, 1989) respectively . Short, acute exposures to these herbicides are not considered to be a significant risk to mammals when used according to label specifications. A rat would have to consume approximately 27 times and 17 times its normal daily intake of grain sprayed directly with the maximum application rate of 2,4-D and 2,4-DP, respectively, in order to ingest a lethal dose of these herbicides.

The EPA has established a sub-chronic no observable effect level (NOEL) for 2,4-D to be 1 mg/kg/day (USDI, 1989). For the rat description above, this would mean eating 0.6 grams of freshly sprayed grain a day for 90 consecutive days. This is approximately 4% of a mouse's normal daily food consumption. This presents a concern about the impact that long term exposures might have on the health of small mammals if one area were to be treated repeatedly and no degradation of the compound occurs. The same concern exists for 2,4-DP which has a sub-chronic NOEL of 5 mg/kg/day (USEPA, 1988). With this NOEL a rat would have to eat 3.2 grams of freshly sprayed grain a day for 90 consecutive days which is approximately 21% of a rats normal daily food consumption.

There are a number of factors that would mitigate the likelihood of these long term exposures. The soil half-life presented in the toxicity tables (Chapter 4 of this appendix) for 2,4-D and 2,4-DP indicate a range from 10 to 50 days (Howard, et al., 1991). This significantly reduces the possibility of 90 consecutive days of exposure. This difference is primarily a function of environmental conditions. In the case of 2,4-D and 2,4-DP, there are no hydrolyzable groups, so areas of high rainfall will not significantly increase the degradation rate although microbial degradation may increase in these areas. 2,4-D and 2,4-DP are primarily degraded in the environment by means of photooxidation (the half-life of 2,4-D in air exposed to light is 1.8 to 18 hours) and to a lesser extent, microbial degradation (Michalenko, et al., 1991). Therefore, 2,4-D is likely to have a half-life of closer to 10 days in the Okanogan Highland, Columbia Basin and Blue Mountains, on the eastern slopes of the Cascades in the Northern Cascades and the Southern Washington Cascades province where there is considerably more sunshine year round. Regions such as the Olympic Peninsula and the Coast Range provinces may have half-lives closer to 50 days due to overcast skies during a large portion of the year.

2.1.2 Mitigating Measures

The main mitigating measure would be to apply these compounds at the lowest concentrations possible to achieve the desired vegetation control, once per season on a given road right-of-way. Historical use of 2,4-D has indicated that WSDOT's average application rate is 13% of the maximum label application rate allowed. Historical use of 2,4-DP has indicated that WSDOT's average application rate is 7% of the maximum label application rate allowed. WSDOT's application rates significantly reduce the impact that this compound will have on the environment.

Additional mitigating measures would include avoiding applications to areas with high small mammal densities.

2.2 Chlorthal Dimethyl

Chlorthal dimethyl is considered to be practically non-toxic to mammals and is not considered to be a significant risk to mammals statewide. Therefore, this discussion will be limited to the impact of chlorthal dimethyl on birds.

2.2.1 Chemical Impacts (birds)

There is insufficient data to determine the potential impacts that chlorthal dimethyl might have on birds. However, the half-life of chlorthal dimethyl in soil is estimated to be 18 to 92 days with a photooxidation half-life in air of 6.1 to 61.1 days (Howard, et al., 1991) which would significantly reduce the likelihood of long term exposures. New data on the toxicity of this compound to birds should be evaluated as soon as it becomes available.

2.2.2 Mitigating Measures

A reduction in application concentrations would help prevent unnecessarily high exposures to wildlife species. WSDOT has a very limited history of use of this chemical, because it is a relatively newly registered product, but average applications rates are generally 85% of the maximum label rate.

2.3 Dicamba

Dicamba is considered to be practically non-toxic to birds and slightly toxic to mammals. Therefore, this discussion will be limited to dicamba's impacts on mammals since dicamba is not considered to be an environmental hazard to birds.

2.3.1 Chemical Impacts (mammals)

Although dicamba is slightly toxic to mammals based on its LD_{50} of 757 mg/kg (Sandoz, 1991), it is not considered to be a serious threat to wildlife on a statewide basis for the following reasons: (1) In order for a rat to ingest enough grain treated with dicamba to cause death, it would have to eat approximately 67 times its normal daily intake of treated grain; (2) dicamba has a subchronic (90 day) NOEL (No Observable Effect Level) for the rat of 250 mg/kg/day (Sandoz, 1992; IRDC, 1980). The same rat would have to eat roughly 22 times its normal daily intake of dicamba treated grain, every day, for 90 days, and it would still not exhibit any signs of toxicity; (3) dicamba's half-life in soils ranges from 25 to 58 days (Sandoz, 1991) which would make daily exposures for 90 days virtually impossible following a single application. In addition, dicamba's resistance to hydrolysis and its tendency towards photodegradation may increase its half-life (closer to 58 days) in cloudy (western Washington) portions of the state, but may produce half-lives closer to the 25 days in eastern Washington where it is sunny.

2.3.2 Mitigating Measures

Application concentrations below the maximum label rates would significantly reduce the risk of impacting wildlife species. WSDOT's historical use of dicamba indicates that applications rates are, on the average, 14% of the maximum label rate.

2.4 Diuron

Diuron is considered to be practically non-toxic to mammals, therefore this discussion will focus on diuron's environmental impacts on birds.

2.4.1 Chemical Impacts (birds)

Diuron has an acute toxicity (LD_{50}) of greater than 2,000 mg/kg for birds (USDI, 1989; USEPA, 1983) which establishes it as being practically non-toxic. However, its dietary toxicity (LC_{50}) of 1,730 ppm (USDI, 1989; USEPA, 1983) establishes it as being slightly toxic to birds after dietary exposures. This LC_{50} value is contrasted with the LC_{50}s for mallards and pheasant (>5,000 ppm) (USDI, 1989). No LD_{50} value was found for the quail.

Diuron is considered to be safe for wildlife as far as acute toxicity is concerned. A 200 gram quail would have to eat 14 times its normal daily intake of food (grain) sprayed directly with diuron, in a matter of minutes, in order to receive a lethal dose of diuron.

Diuron's risk to wildlife from eight day dietary exposures also supports the assessment that diuron is not a wildlife hazard. The eight-day dietary LC_{50} for the quail is 1730 ppm or mg/kg (USDI, 1989; USEPA, 1983). This is approximately 1.5 times the concentration that would be found on grain following an application at the maximum label rate. The environmental fate of diuron indicates that it is relatively persistent in the environment, especially in areas with high clay and/or organic matter (USDI, 1991). This would include the lowlands in the Olympic Peninsula province, the Coast Range province, Puget Trough, and some of the more agricultural areas in the Columbia Basin. This persistence could provide a continuous route of exposure for wildlife species, but at levels below the identified effect level.

It is important to note that the dietary LC_{50} for mallards and pheasants is nearly 3 times higher (3 times less toxic) than for the quail. This information in addition to the fact that this analysis is considering grain that has been sprayed directly with diuron, can be viewed as a worst case scenario. Under normal label uses, this product will not cause wildlife mortalities. Its environmental fate and subacute toxicity data suggests that it has the potential to cause sublethal effects in wildlife species when used at maximum label rates and repeated applications.

2.4.2 Mitigating Measures

There are several measures that could be taken to reduce the impact the diuron might have on the wildlife. The reduction of applications to levels below the maximum allowed by the label will very significantly reduce wildlife impacts. Historical use of diuron by WSDOT indicates that average use rates are generally only 8% of the maximum labeled

rate. At these rates, no adverse impacts on wildlife are expected. In addition, the spot application of diuron as opposed to broadcast use would significantly reduce the likelihood of repeated exposures to wildlife.

2.5 Glyphosate

Glyphosate is practically non-toxic to mammals. Therefore, this discussion will concentrate on the impacts that glyphosate has on birds.

2.5.1 Chemical Impacts (birds)

Although glyphosate is considered to be practically non-toxic based on the avian LD_{50} values (>2000 mg/kg), it is considered to be slightly toxic based on its LC_{50} value of 4640 ppm for the mallard and quail (Monsanto, 1990(c)).

Glyphosate is not considered to be a risk to wildlife as a result of its use for roadside vegetation management. A 200 gram quail would have to eat approximately 14 times its normal daily intake of glyphosate-treated grain in a matter of minutes in order to ingest a lethal dose of glyphosate. This is assuming that glyphosate was treated at the maximum application rate. The 8-day dietary study (LC_{50}) has established a median lethal concentration in the food of 4640 mg/kg. The concentration on grain following an application of glyphosate at the maximum label rate would be approximately 15 times less than the LC_{50} concentration.

In addition, although some references indicate that glyphosate is persistent in sandy loam soils (USDI, 1991), other references have indicated that soil half lives for glyphosate are generally less than 60 days (Wilkerson and Kim, 1988). Rapid degradation would also help to reduce the risk of long term exposures to glyphosate.

2.5.2 Mitigating Measures

Again the primary mitigating measure would be a reduction in application rates. Historical use of glyphosate by WSDOT for roadside vegetation management has established that glyphosate's average application rate is approximately 15% of the maximum labeled rate.

2.6 Mecoprop

Mecoprop is listed as slightly toxic to birds based on an acute LD_{50} of 486 mg/kg (Leeming, 1991). However, its rapid metabolism and excretion by birds reduces its toxicity over an 8-day feeding study (LC_{50}) to 5620 ppm (Leeming, 1991) which is considered to be practically non-toxic. Therefore, mecoprop is not considered to be an environmental risk to birds and the remainder of this discussion will address the environmental impact that mecoprop might have on mammals.

2.6.1 Chemical Impacts (mammals)

Mecoprop is considered to be slightly toxic to mammals based on an acute oral LD_{50} value of 650 mg/kg (USDHHS, 1980). However, it is also not considered to be an

environmental risk to mammals primarily because of its low application rates. With a mammalian LD_{50} of 650 mg/kg (USDHHS, 1980) and a maximum application rate of 0.54 lbs/acre, a 300 gram rat would have to eat approximately 859 times its normal daily food intake of grain in order to ingest a lethal dose of mecoprop.

Long term exposures to mecoprop are also very unlikely given the relatively short half-life in soil (20 to 60 days) (Menzie, 1980). Subchronic studies on mammals generally last 90 days. The subchronic studies for mecoprop lasted from 3 weeks to 7 months and identified an subchronic NOEL of 3 mg/kg/day (USEPA, 1988; Leeming, 1991). To reach this level of exposure, a rat would have to eat nearly 4 times its normal daily intake of grain that has been treated with mecoprop for 21 consecutive days.

2.6.2 Mitigating Measures

Although mecoprop is not considered to be an environmental threat to wildlife, a reduction in application rates to levels that still provide desired vegetation control would futher reduce risk. WSDOT's historical use of mecoprop indicates that it is used at maximum label rates.

2.7 Triclopyr

Triclopyr is slightly toxic to both birds and mammals based on an avian dietary LC_{50} of 2935 ppm for the quail and a mammalian acute LD_{50} of 630 mg/kg for the rat (Dow/Elanco, Undated(d)). The acute LD_{50} for birds is 1698 mg/kg which is also considered to be slightly toxic. Therefore, the environmental impacts that triclopyr might have on birds and mammals will be discussed in this section.

2.7.1 Chemical Impacts (birds and mammals)

Short (acute) exposures to maximum label rates of triclopyr are not considered to be a risk to either birds or mammals. A 200 gram quail would have to eat 49 times its normal daily food intake of triclopyr-treated grain in a very short period in order to ingest a lethal dose of triclopyr. For mammals, a 300 gram rat would be required to eat almost 54 times its normal daily food intake of triclopyr-treated grain in order to ingest a lethal dose.

For dietary exposures to triclopyr for birds, an 8-day dietary LC_{50} of 2935 mg/kg in the food has been established (Dow/Elanco, Undated(d)). This is roughly 11 times the concentration that would be found on grain following an application of triclopyr at the maximum label rate.

Subchronic studies in the male rat have established an NOEL to be 30 mg/kg/day (Dow/Elanco, Undated(a)). These studies are generally 90 day exposures. In order to ingest enough grain treated with triclopyr to get to 30 mg/kg/day, a rat would have to eat over 2 times its normal daily intake of food for 90 consecutive days. In addition, the average half-life in soils for triclopyr is approximately 30 days (Dow/Elanco, Undated(d)). Its photo-degradation half-life is even less (8 to 10 hours). These low half-lifes would make it virtually impossible to achieve a 90 day repetitive exposure situation for wildlife from chemical applications of right-of-ways. This significantly reduces the

amount of triclopyr that is biologically available, especially in the provinces east of the Cascades where it is sunny much of the year.

2.7.2 Mitigating Measures

A reduction of the application rates to those below the maximum label rate would further reduce the impacts on the environment. Historical use of triclopyr by WSDOT for roadside vegetation management has established that its average application rate is approximately 18% of the maximum labeled rate. Spot application would also reduce the possibility of sublethal impacts on wildlife.

2.8 Petroleum Distillates and Xylene

2.8.1 Chemical Impacts (Mammals)

There is very little information on the toxicity of petroleum distillates (naphthalene, kerosene, and diesel) and Xylene. These compounds are considered to be practically non-toxic to mammals and they are not a risk to mammals statewide.

It is very difficult to assess the impact that these chemicals might have on birds because of the lack of information. Impacts to birds will primarily be based on the environmental fate of these compounds. Any new data that becomes available for tyes of products concerning avian toxicity should be reviewed.

Xylene has an estimated half-life of 1 to 4 weeks (Howard, et al., 1991) in the soil. This half-life significantly reduces the possibility of long term impacts on wildlife. Similarly, since the petroleum distillates are hydrocarbons, they can be expected to have relatively short half-lives in the environment.

Neither of these compounds are expected to significantly impact the wildlife in Washington.

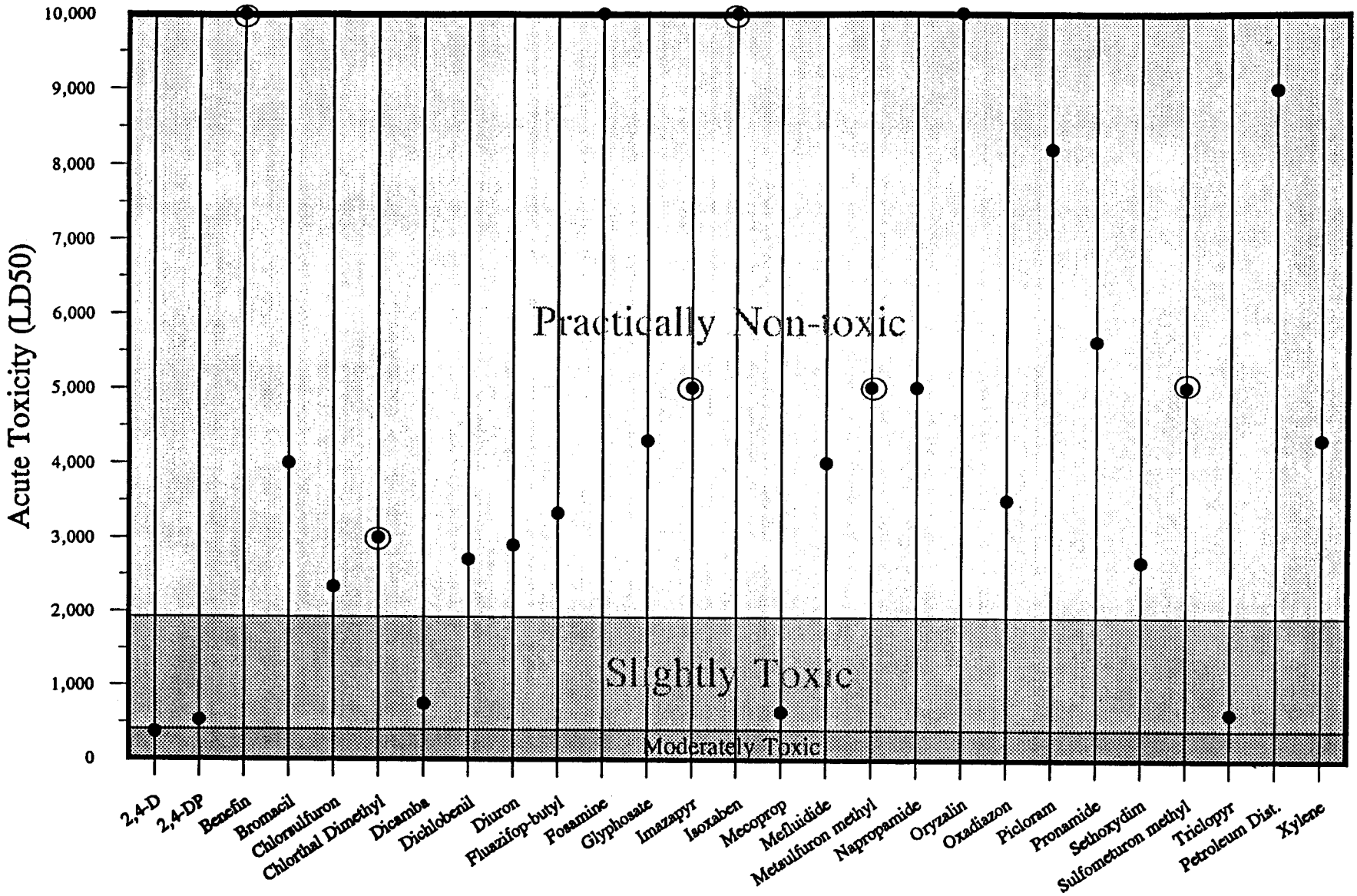


Figure 2-1 Relative mammalian toxicities of the active ingredients found in the herbicides to be used by WSDOT for roadside vegetation management. A circle around the point indicates that the value is reported as greater than (>) the LD50 indicated. For example: The benefin LD50 = >10,000 mg/kg. The fosamine LD50 is reported to be 24,000 mg/kg.

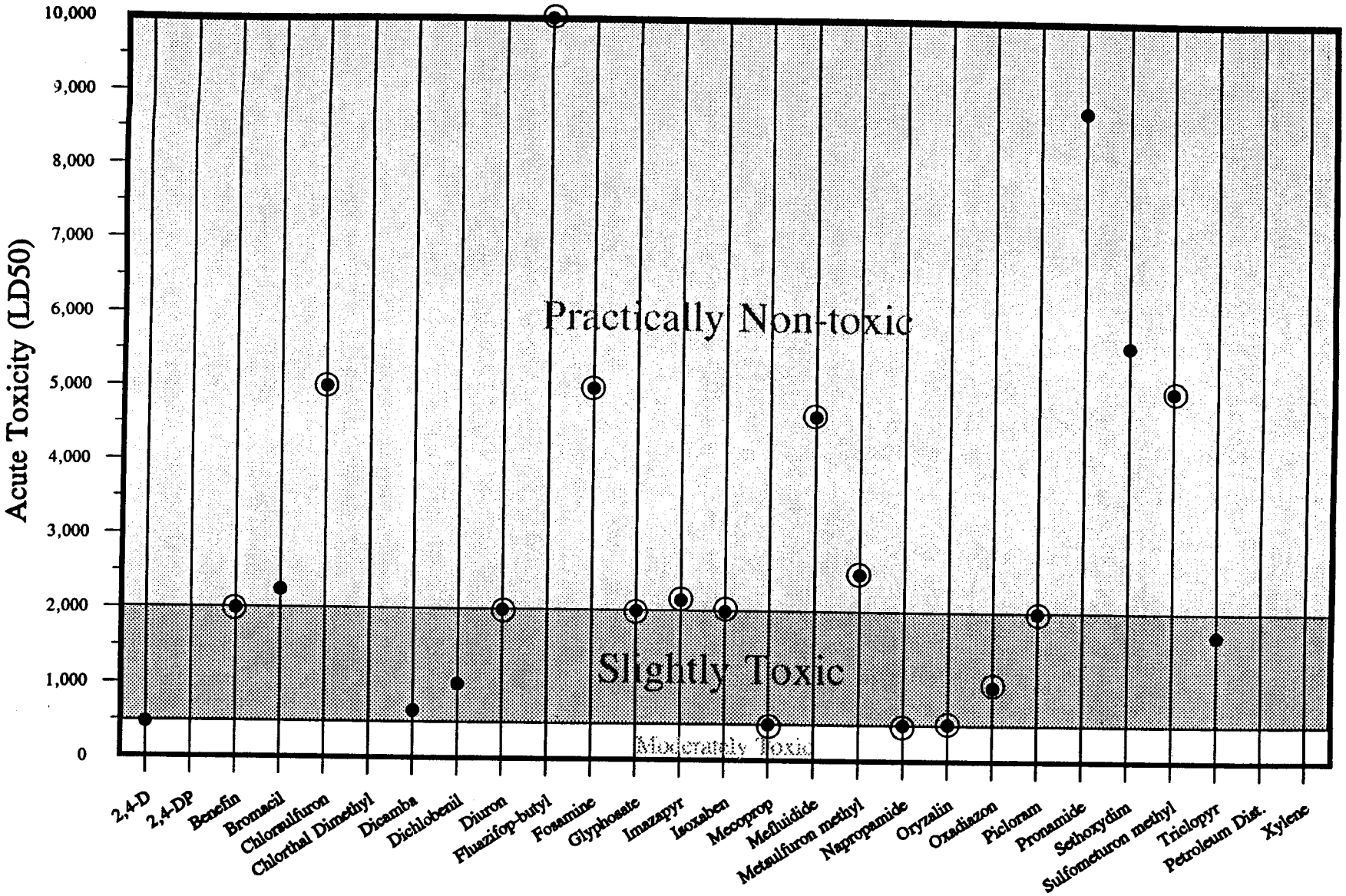


Figure 2-2 Relative avian toxicities of the active ingredients found in the herbicides to be used by WSDOT for roadside vegetation management. A circle around the point indicates that the value is reported as greater than (>) the LD50 indicated. For example: The benefin LD50 = >2,000 mg/kg. Fluazifop-butyl is reported as being. greater than 17,000 mg/kg

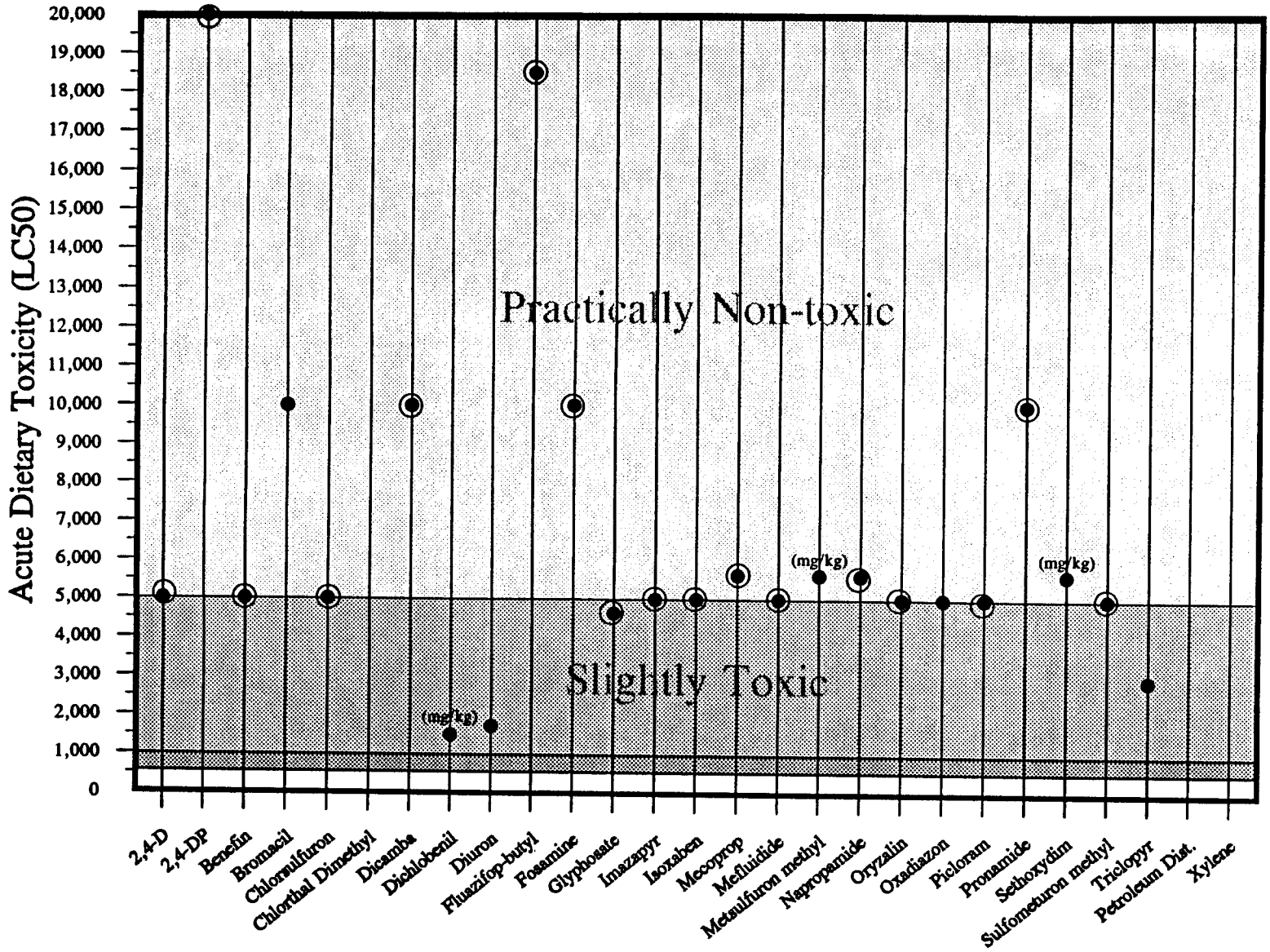


Figure 2-3 Relative avian toxicities of the active ingredients found in the herbicides to be used by WSDOT for roadside vegetation management. A circle around the point indicates that the value is reported as greater than (>) the LD50 indicated. For example: The 2,4-D LC50 = >5,000 ppm.

Table 2-1. EPA descriptive classifications for toxicity data.

	Very Highly Toxic	Highly Toxic	Moderately Toxic	Slightly Toxic	Practically Non-toxic
MAMMALS					
LD_{50} (mg/kg)	< 10	10-50	51-500	501-2000	> 2000
LC_{50} (ppm)	< 50	51-500	501-1000	1001-5000	> 5000
BIRDS					
LD_{50} (mg/kg)	< 10	10-50	51-500	501-2000	> 2000
LC_{50} (ppm)	< 50	51-500	501-1000	1001-5000	> 5000
AQUATIC					
LC_{50} (ppm)	< 0.1	0.1 - 1	>1 - 10	>10 - 100	> 100

Table 2-2. Data gathered for use in assessing the relative impacts of herbicides used by WSDOT on wildlife.

Mammals							
Chemical	Application Rate[a] (lbs/acre)	Grain Surface Area (sq. m)	Grain Weight[b] (g/grain)	Rat Weight[c] (g)	Daily Food Intake[c] (g)	Rat LD_{50}[a] (mg/kg)	Rat NOEL[a] (mg/kg/day)
2,4-D	16.65	1.0×10^{-5}	4.0×10^{-2}	300	15	639.00	1.00
2,4-DP	16.65	1.0×10^{-5}	4.0×10^{-2}	300	15	400.00	5.00
Dicamba	8.00	1.0×10^{-5}	4.0×10^{-2}	300	15	757.00	250.00
Mecoprop	0.54	1.0×10^{-5}	4.0×10^{-2}	300	15	650.00	3.00
Triclopyr	9.00	1.0×10^{-5}	4.0×10^{-2}	300	15	630.00	30.00
Birds							
Chemical	Application Rate[a] (lbs/acre)	Grain Surface Area (sq. m)	Grain Weight[b] (g/grain)	Quail Weight[d] (g)	Daily Food Intake[d] (g)	Quail LD_{50}[a] (mg/kg)	Quail LC_{50}[a] (mg/kg)
Chlorthal	15.00	1.0×10^{-5}	4.0×10^{-2}	200	25	N/A	N/A
Diuron	40.00	1.0×10^{-5}	4.0×10^{-2}	200	25	2000.00	1730.00
Glyphosate	10.60	1.0×10^{-5}	4.0×10^{-2}	200	25	2000.00	4640.00
Triclopyr	9.00	1.0×10^{-5}	4.0×10^{-2}	200	25	1698.00	2935.00

[a] Toxicity tables, Appendix B, Chapter 4
[b] Sunderman, 1991
[c] Konaga, 1972
[d] Beavers, 1991

Appendix B - Chapter 3

Draft EIS
Roadside Vegetation Management

1.0 Introduction

An ecological risk assessment is a qualitative or quantitative appraisal of the actual or potential effects of hazardous materials on animals and plants other than people and domesticated animals (USEPA, 1989). The introduction of contaminants into an ecosystem can cause direct harm to organisms, or may indirectly affect their ability to survive and reproduce. The results of contamination may be immediately apparent or may become noticeable only after considerable delay. The effects of contaminants on ecosystems are due in part to the physical and chemical properties of the chemicals themselves, but are also mediated by the unique combination of physical, chemical, and biological processes occurring in each ecosystem. In addition, populations of exposed organisms can differ in their response to contaminants depending on their natural tolerance to the chemical, their behavioral and life-history characteristics, the dose to which they are exposed, and the exposure time. Furthermore, responses may be transient (and therefore reversible) or permanent (irreversible) (USEPA, 1989).

Ecological assessment seeks to determine the nature, magnitude, and transience or permanence of observed or expected effects. This must be accomplished in an environment that is itself changing and causing change in the organisms and systems under study. Our goal here is to determine the potential for WSDOT's maintenance activities to adversely affect the aquatic system.

1.1 Objectives

The objectives of the aquatic section of the ecological risk assessment are as follows:

- To describe the toxicity of the selected herbicides to and marine aquatic organisms
- To identify sensitive habitats and species that may be impacted by the different vegetation management methods
- To characterize exposure by identifying actual and potential exposure pathways, taking into account environmental fate and transport through both physical and biological means
- To characterize the risk or threat to environmental receptors potentially exposed to the selected herbicides

The aspect of WSDOT's activities which is of the greatest concern to other agencies and the public is its use of herbicides. This assessment concentrates, therefore on herbicides use and impact.

1.2 Techniques

As stated above, an ecological risk assessment can be quantitative or qualitative, depending on the type and quality of data available. In the aquatic ecological risk assessment, as in the terrestrial ecological risk assessment, a qualitative rather than a quantitative assessment was conducted.

Determination of toxicity to aquatic species in a given system generally does not focus on a single receptor but on a summarization of toxicity data for several species within a genus or for several genera. Waters impacted by WSDOT activities will be immediately adjacent to or near roadsides. Due to the demands of road construction and road safety, some impact on aquatic systems adjacent to them is inevitable, whether it be from the impact of the road and traffic or maintenance activities. The potential for and degree of impact of vegetation managment techniques, in particular herbicide use, is our concern here. For this purpose, compound toxicity to freshwater and estuarine/marine fish and invertebrates was assessed. Brush removal, grading and invasion by undesirable plants can also have an impact on aquatic systems by increasing turbidity and sedimentation, increasing water temperature, and by bringing about habitat loss. These activities will be addressed in general.

In the assessment of toxicity of herbicides, standardized tests are conducted and the results are statistically derived in "acute" (short term) or "chronic" (long term) studies. A tiered approach is used in the conduct of these studies so that resources and living organisms are spared needless use. Testing begins with acute (short term) studies, and if results indicate a need, testing progresses to chronic (long term) studies. In the assessment of WSDOT herbicide impact, it is more appropriate to utilize values derived from acute studies, although chronic values, where available, will also be considered. Use of acute values parallels the "once a season per area" use pattern of WSDOT applications.

Results of chronic studies are typically expressed in terms of a no observed effect level (NOEL); that is the concentration at which no effect is observed. Studies determining the NOEL often also generate data on the lowest concentration causing an effect (the lowest observed effect level or LOEL). These values are important to use in comparison to the expected water concentrations which could be reached due to spraying activities. Chronic effects in aquatic organisms are usually based upon adverse reproductive or growth effects, such as decreased hatching success, decreased survival of larvae, decreased growth of larvae or juveniles, and decreased reproductive capability. For the majority of the compounds under consideration in this assessment, little or no chronic toxicity data were available because earlier tier acute testing did not indicate the need for further data development. In those instances where chronic studies are available, the species or the endpoints used were not consistent among studies, however, any available data will be related to potential impact.

Acute data (short term studies) are generated by the conduct of 2 to 4 day-long studies under carefully controlled conditions. The statistical values derived from these studies are expressed as "LC_{50}" or "EC_{50}" values. The LC_{50} value is the concentration which would be projected to cause 50% mortality in the exposed population. The "EC_{50}" value is the projected concentration which would be expected to elicit a given effect in an

exposed population. The LC_{50} and EC_{50} values are used for qualitative predictions of toxicity and for the establishment of requirements for product use limitation or further testing.

Assessing toxicity to aquatic species based on acute toxicity instead of chronic toxicity is reasonable for this investigation. It is unlikely that application practices employed by the WSDOT would result in sustained, relatively constant concentrations of these herbicides in aquatic environments; therefore, chronic toxicity of aquatic species is unlikely. Additionally, the compounds reviewed here generally fall into toxicity categories which do not trigger the need for further chronic testing.

In reviewing WSDOT application records for the 1989-90 spray season, it was apparent that the herbicide application practices would more likely result in intermittent discharges in surface runoff and in possible spray drift into roadside surface water bodies. Herbicides which reach receiving waters would most likely eventually dissipate through dilution, degradation, and sequestering by a variety of processes.

A wide range of fish and aquatic invertebrates, both freshwater and marine, occur in the surface waters of Washington. The types of species found depend upon such factors as water temperature, salinity, and flow conditions. Because of the diversity of aquatic habitats throughout the state no representative aquatic species were selected, rather fish and invertebrates are selected as animal groups to be evaluated. Where data are available, the impact of herbicides on aquatic plants is also discussed. Because most of the compounds used by WSDOT are herbicides, if their effective levels and physical and chemical properties allow, they may have some impact on non-target aquatic vegetation.

2.0 Toxicological and Impact Issues

2.1 Direct Effects

Of primary concern is the evaluation of LC_{50} and EC_{50} values to predict whether WSDOT's use of products will result in concentrations approaching lethal or adverse effect levels. Values for fish and invertebrates have been reported later in this appendix and comparison with use generally indicate there is a low potential for direct aquatic impact (that is, mortality or overt toxicity) due to WSDOT's activities. Details on compounds are presented in Sections 3, 4, and 5. These sections are equivalent to a "hazard assessment." (See Appendix B, Chapter 4 on Human Health Risk Assessement.)

In the aquatic environment, herbicides may be transported and accumulated in various media (sediment, water, plants, aquatic invertebrates, and fish) and/or degraded by a variety of biological, physical, and chemical mechanisms. The environmental fate of a chemical is an important consideration in assessing its relative safety and environmental suitability.

In order to determine direct effects, some calculation of exposure is needed. The degree of exposure is a subjective evaluation of use patterns, the physical and chemical properties of a given compound and assumptions about road density and location.

2.1.1 Exposure Assessment

Exposure assessment involves the estimation of the amount of a chemical that an individual or a population may potentially encounter, and considers the extent, frequency, and duration of such exposures. Typically this estimation is quantitative in nature. However, for the aquatic ecological risk assessment, a qualitative exposure assessment was performed. This was necessitated due to the many assumptions which must be made to describe, on a statewide basis, the herbicide application practices of the WSDOT throughout the eight physiographic provinces of the state.

The exposure of aquatic organisms was assumed to be primarily from surface runoff which results when storm events or irrigation follow closely the timing of the herbicide application. Other sources of the selected herbicides were not considered in estimating exposure and are expected to be minor in comparison. Many of the selected herbicides are commonly used in commercial agricultural applications as well as in private homes and gardens. The Washington State Department Agriculture does not maintain records of use or sales of herbicides for either agricultural or private use (Creiger, personal communication, 1991), so it was neither practical nor possible to determine to what extent exposure from these other sources might occur.

Even though spray records were available for WSDOT uses, a conservative approach was taken for the exposure assessment, and it was assumed that all of the herbicides are used throughout all of the physiographic provinces. This conservative approach will tend to over estimate the likelihood of impacts to both the aquatic wildlife and vegetation, especially in western Washington (Puget Trough) where the road density is high.

In order to estimate the exposure of aquatic organisms to those chemicals applied to WSDOT rights-of-way for vegetation management, four parameters were considered: annual rainfall, road density, maximum label application rate of the active ingredient, and the soil half life of each chemical. Ranges within each parameter were assigned a relative rating on an assumptive basis. For example, rainfall in each of the physiographic provinces was rated from a low value of 1 for those provinces whose upper limit of annual rainfall was less than 50 inches to a high of 3 for those regions whose upper limit of annual rainfall was 90 or more inches (Table 3-1). Road densities within each physiographic province were rated using a WSDOT road map showing WSDOT rights-of-way within each province. The Puget Trough Province was used as a reference against which road densities in the other provinces were judged (Table 3-1). Chemical half life in soil (Table 3-2) was also rated as follows:

(1) 0 to 3 months - low persistence

(2) 4 to 6 months - moderate persistence

(3) Greater than 6 months - high persistence.

Comparative application rates were classified by the maximum application rates of the active ingredients (pounds/acre - Chapter 5, Table 5-2) as follows:

(1) 0.08 to < 1.00 lbs/acre (low rate)

(2) 1.00 to < 10.00 lbs/acre (moderate rate)

(3) > 10.00 lbs/acre (high rate)

Using the assigned ratings for each of the four parameters, relative exposure was calculated as follows:

$$\frac{\text{Rainfall X Road Density X Persistence X Application Rate}}{12} = \text{Exposure}$$

In order to maintain the same rating schedule (i.e., 1 = low, 2 = moderate, and 3 = high) for relative exposure as for the parameters used to estimate it, the highest product obtained (36) by multiplying the relative ratings of rainfall, road density, application rate, and persistence was divided by a factor of 12 (the sum of the highest possible rating) so that a relative exposure rating of 3 would indicate a high potential for exposure of aquatic organisms to the selected herbicides. In those cases where division by a factor of twelve resulted in a fractional quotient, all values less than one were given a value of one. Values of 1.5 and 2.5 were given values of 2 and 3, respectively.

Relative exposure ratings for each of the herbicides in each physiographic province are summarized in Table 3-3. For the majority of the herbicides, the potential for exposure is low. However, in the Puget Trough Province, because of the high road density, there is a greater potential for exposure for a number of the herbicides, especially bromacil, diuron, oxadiazon and picloram, for which there is a high exposure potential.

2.1.2 Risk Characterization

This section discusses the estimated risks to aquatic wildlife receptors (that is, the exposed organism) that might be inadvertently exposed to herbicides, inert ingredients, and adjuvants as a result of WSDOT Vegetation Management program. Risk to ecological receptors from exposure to a potentially hazardous substance is determined by two equally important factors: duration of exposure and the concentration or dose of the chemical (which is a function of the potency or toxicity of the chemical).

The relative hazard of individual herbicides to aquatic receptors was determined qualitatively. WSDOT application records are kept in a site and occasion specific format and were summarized for this EIS. However, to derive estimated environmental concentrations of the herbicides, would require volumes of site specific precipitation, water, and land area data and is not practical. Therefore, the classification scheme developed by the U.S. EPA (1985) for herbicides which was discussed earlier was used as the basis for rating herbicide toxicity. Herbicides were assigned numerical ratings as follows:

(1) > 100 mg/l - practically non-toxic

(2) > 10 to ≤ 100 mg/l - slightly toxic

(3) > 1 to ≤ 10 mg/l - moderately toxic

(4) 0.1 to 1 mg/l - highly toxic

(5) <0.1 mg/l - very highly toxic

The toxicity rating for each herbicide was then multiplied by the exposure rating and the product divided by a factor of four:

$$\frac{\text{Exposure Rating X Toxicity Rating}}{4} = \text{Relative Risk}$$

A factor of four was used in order to maintain the same rating scheme as was used for estimating exposure. The highest product obtained (12) was divided by a factor that would result in the highest risk factor corresponding to a value of three. Moderate and low risk were rated as two and one, respectively.

Relative risk was estimated by physiographic province for fish and aquatic invertebrates for each of the herbicides and for petroleum distillates (Tables 3-4 and 3-5). Because insufficient toxicity or environmental fate data were available for the adjuvants utilized by WSDOT, no risk characterization was performed for these substances. The toxicity information for the adjuvants is listed in Chapter 5, Table 5-8 of this appendix, and suggests that the environmental risks associated with the use of these adjuvants are relatively low.

Tables 3-4 and 3-5 list the relative risk characterization factors for fish and aquatic invertebrates, respectively. The majority of the herbicides pose low relative risk to both fish and aquatic invertebrates; however, benefin and diuron pose a potentially high relative risk to fish species in the Puget Trough Province while aquatic invertebrates in the same province may be at a high relative risk from exposure to diuron and oxadiazon. The high risk factors for these three herbicides can be attributed primarily to their potency, application rates, and the high road density in the Puget Trough Province, which is assumed to be directly related to the volume of herbicide application. These risks assume that the maximum label rates are being used.

2.2 Indirect Effects of Herbicides

Indirect effects of herbicides or other herbicides would be expressed as habitat loss, changes in competition between species within an ecosystem, or other aquatic system variations not brought about due to direct toxicity. For example, changes in plant density or species distribution can result in loss of refuge for fish food organisms but can clear areas for feeding by fish. Conversely, changes in pH or water hardness could stress fish or invertebrate populations by enhancing diseases or overt stress.

Herbicides are not applied directly to waters along roadsides and therefore would not be expected to reach the extremely high concentrations which would be required to bring about changes in the physical parameters of the aquatic system (water hardness, dissolved oxygen, pH, temperature, suspended solids, etc.).

Most terrestrial herbicides are not effective weed control agents in aquatic systems due to system dynamics, mode of action and chemical properties. Glyphosate and 2,4-D are

registered for aquatic uses, but at rates much higher than those which would be expected to occur from indirect exposure resulting from roadside use. Therefore, herbicide use by WSDOT would not be expected to result in major habitat changes for fish or invertebrates unless an intentional and direct application were made to a significant portion of the water body in question.

The public has also expressed concern about the evaluation of the possible synergistic effects of compounds used in WSDOT's maintenance program. Synergistic effects of chemicals are those that occur from exposure to two chemicals either simultaneously or within a relatively short period of time. Synergism occurs when the combined effects of two chemicals are greater than the sum of the effects of each agent individually (simple additive effect). Instances of chemical combinations that cause synergistic effects are relatively rare. Kociba and Mullison (1985, as cited in USDA, 1989) in describing toxicological interactions with agricultural chemicals state:

> "Our present scientific knowledge in toxicology indicates that an exposure to a mixture of pesticides is more likely to lead to additivity or antagonisms rather than synergism when considering the toxicological effects of such a combination. To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response."

In the case of registered herbicides, a great amount of toxicological information has been developed during the research and development of each individual herbicide. In addition to this information on individual herbicides, short term toxicity studies are always done prior to the selling of a herbicide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two herbicides, it would be identified in such cases and would then be dealt with accordingly. In toxicological tests involving a combination of commercial herbicides, synergism has not generally been observed.

Synergistic toxic effects of herbicide combinations other than EPA-registered commercial mixtures are not normally studied. Time and money normally limit toxicity testing to the effects of the herbicides individually. Combinations that could be tested are too numerous to make that testing feasible. Additionally, combination testing which has been conducted for this purpose has not been fruitful in discovering anything other than additive effects. Generally, the potential for synergism can be predicted by mode of action and metabolism data. Based on the limited amount of data available on herbicide combinations, it is unlikely that synergistic effects would occur as a result of exposure to two or more of the herbicides considered in this analysis. Furthermore, WSDOT uses mixtures which are legally registered, and rapidly sequential exposures to a large number of different products would not be typical.

3.0 Effects of Herbicides by Active Ingredient

3.1 Aquatic Wildlife Toxicity

The toxicity of the herbicides and additives to aquatic species is summarized in this section. Information is presented on the acute toxicities of the herbicides to freshwater and marine fish and invertebrates, for compounds proposed for use in WSDOT's vegetation management program. The toxicity information presented in this section has

been developed from laboratory studies using laboratory-reared animal species, primarily rainbow trout (*Oncorhynchus mykiss*, a cold water fish species), bluegill (*Lepomis macrochirus*, a warm water fish species), and the water flea (*Daphnia magna* or *D. pulex*, an invertebrate). The results of the laboratory studies are considered to be representative of the effects that would occur in similar species in the wild.

The relative acute toxicities of the herbicides are classified according to a scheme designed by the U.S. EPA (1985) where LC_{50} values are described as follows:

- <0.1 ppm (mg/l) - very highly toxic
- 0.1 ppm to 1 ppm - highly toxic
- >1 ppm to ≤ 10 ppm - moderately toxic
- >10 ppm to $\leq$ 100 ppm - slightly toxic
- $>$ 100 ppm - practically non-toxic

These toxicity categories are used in the ecological toxicity profiles to qualitatively describe the acute toxicity of the chemicals (Table 3-6). Normally, concentrations of >1 ppm would not be expected to occur as a result of indirect exposure to biologically significant water bodies.

The terms listed below pertain to aquatic toxicology and are used frequently in this section:

LC_{50} - the calculated concentration of a toxicant in water that is predicted to be lethal to 50 percent of a population of test organisms within a specific period of time (usually reported for 96 hours).

EC_{50} - the calculated concentration of a toxicant in water that is predicted to have a specific effect on 50 percent of the test organisms. It is often used with animals where determining death is difficult, such as *Daphnia* sp.

Toxicity information on the selected herbicides, inert ingredients and adjuvants is summarized in Chapter 5 of this Appendix, and their general toxicity to aquatic organisms is discussed briefly below.

Bioaccumulation is determined or predicted by a number of means. Standard aquatic laboratory studies are conducted on compounds which, by other properties, are predicted to have some potential to bioaccumlate. Those "other properties" are the compound's environmental degradation rate, its octanol/water partition coefficient, and its metabolic half-life. Generally, an octanol/water partition coefficient of ≥ 1000 indicates a potential for bioaccumulation or <u>bioconcentration</u>. Similarly, a biological clearance rate which is slower than environmental breakdown may indicate a potential for <u>bioaccumulation</u>, a condition which exists only as long as exposure continues. None of the herbicides reviewed exhibit a potential to <u>bioconcentrate</u>; some have a low to moderate potential to bioaccumulate in certain organisms. Bioaccumulation in itself may not be a problem as

long as exposure does not span a period which would result in the accumulation of harmful levels of compound.

2,4-D

The toxicity of 2,4-D to aquatic organisms ranges from practically non-toxic to highly toxic. The herbicide 2,4-D amine is highly toxic to rainbow trout but practically non-toxic to bluegill, while the butoxyethanol ester is moderately toxic to both rainbow trout and bluegill. Both 2,4-D amine and 2,4-D butoxyethanol ester are moderately toxic to aquatic invertebrates. Esters are typically 100 times more toxic to aquatic organisms than their corresponding acids and most amine formulations, but in most instances the esters rapidly hydrolyze to corresponding acids (Ghassemi, et al., 1981). Bioaccumulation of 2,4-D is low, and it generally is rapidly excreted in the urine unchanged or as a conjugate (USDA, 1989).

2,4-DP

Very little information was found describing the toxicity of 2,4-DP to fish and no studies were found for aquatic invertebrates. A 48-hour LC_{50} of 1.1 ppm (isooctyl ester) has been reported for adult bluegill. This value compares closely with the 96-hour LC_{50} of 1.2 ppm for the butoxyethanol ester of 2,4-D for the same fish species. Because of the chemical similarity of the two herbicides, it is expected, but not proven, that their toxicities to aquatic organisms, as well as their metabolism and bioaccumulation potential, would be similar (USDA, 1989).

Benefin

The toxicity of benefin to fish ranges from very highly toxic in trout to moderately toxic in sheepshead minnow. In aquatic invertebrates, benefin toxicity ranges from slightly toxic in mysid shrimp to highly toxic in oysters.

No information was found describing the metabolism or bioaccumulation potential of benefin in aquatic animals, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Bromacil

Bromacil toxicity to fish and aquatic invertebrates varies from practically non-toxic to slightly toxic. Bromacil is metabolized in aquatic organisms to its debrominated analog. It does not biaccumulate in aquatic organisms.

Chlorsulfuron

Chlorsulfuron is practically non-toxic to the fish and aquatic invertebrates tested and does not tend to bioaccumulate.

Chlorthal Dimethyl

From the limited data available, chlorthal dimethyl has been found to be practically non-toxic to fish and highly toxic to aquatic invertebrates. No data were found describing its metabolism or bioaccumulation in aquatic organisms, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Dicamba

In the studies cited, dicamba is sightly to practically non-toxic to fish while being slightly to moderately toxic to aquatic invertebrates (USDA, 1989). The salts and free acid of dicamba are considered toxicologically equivalent because the salt hydrolyzes to the free acid in an aqueous environment (USEPA, 1983, as cited in USDI, 1991). Based on the available data, dicamba does not appear to bioaccumulate.

Dichlobenil

Dichlobenil is slightly to moderately toxic to fish and moderately to highly toxic to aquatic invertebrates (Kersting & van der Horning, 1981). From the limited data available, it does not appear that dichlobenil bioaccumulates to a great extent in fish.

Diuron

The toxicity data indicate that diuron is moderately to highly toxic to fish and aquatic invertebrates. Limited data are available describing the bioaccumulation of diuron in aquatic organisms. There is some evidence of limited bioaccumulation in carp and some aquatic invertebrates upon prolonged exposure.

Fluazifop-butyl

There is limited information available describing the toxicity of fluazifop-butyl to aquatic organisms. Fluazifop-butyl is considered moderately to highly toxic to fish. The only information found reporting the toxicity of fluazifop-butyl to aquatic invertebrates described its toxicity as "low." No data were found in the literature concerning the bioaccumulation potential of fluazifop-butyl. However, an empirical regression equation, based on the solubility of fluazifop-butyl, has been used to estimate a bioconcentration factor of 160 (Kenaga and Goring, 1978), indicating a low potential for bioaccumulation.

Fosamine

Fosamine is considered practically non-toxic to fish and aquatic invertebrates. The limited evidence available does not indicate that fosamine bioaccumulates.

Glyphosate

Glyphosate is practically non-toxic to moderately toxic to fish and slightly to practically non-toxic to aquatic invertebrates (USDA, 1988 & 1989). The available toxicity data do not indicate that it bioaccumulates.

Imazapyr

Technical imazapyr is demonstrated to be practically non-toxic to fish and aquatic invertebrates. The evidence available does not indicate that imazapyr bioaccumulates.

Isoxaben

Isoxaben is moderately toxic to both fish and aquatic invertebrates. Isoxaben has a low bioaccumulation potential.

Mecoprop

The only information describing the toxicity of mecoprop to aquatic organisms is that for fish, which indicates that mecoprop is practically non-toxic. Mecoprop accumulates slightly in bluegill sunfish.

Mefluidide

Mefluidide was slightly toxic to fish and practically non-toxic to aquatic invertebrates. Based on the limited information available, mefluidide does not appear to bioaccumulate in fish.

Metsulfuron Methyl

Metsulfuron methyl is practically non-toxic fish and aquatic invertebrates. No information was found describing its metabolism or bioaccumulation in aquatic species, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Napropamide

Napropamide is slightly toxic to moderately toxic to fish and slightly toxic to aquatic invertebrates. No information was found describing its metabolism or bioaccumulation in aquatic species, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Oryzalin

Oryzalin is moderately toxic to fish and slightly to highly toxic to aquatic invertebrates. No information was found describing its metabolism or bioaccumulation in aquatic species, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Oxadiazon

Oxadiazon is slightly to moderately toxic to fish and moderately to highly toxic to aquatic invertebrates. The potential for bioaccumulation of oxadiazon in aquatic organisms is low.

Picloram

Picloram is slightly to moderately toxic to fish and practically non-toxic to moderately toxic to aquatic invertebrates (Mayes & Dill, 1984; Sander, 1969). Picloram does not bioaccumulate.

Pronamide

Pronamide was slightly to practically non-toxic to fish and moderately toxic to aquatic invertebrates. No information was found describing its metabolism or bioaccumulation in aquatic species, but we can predict (based on metabolism and the octanol/water partition coefficient) that bioaccumulation is unlikely to occur.

Sethoxydim

Sethoxydim is moderately toxic to fish and moderately toxic to aquatic invertebrates. No data on the bioaccumulation potential of sethoxydim were found in the literature. Based on a solubility of 6,900 mg/l, the bioconcentration factor was estimated to be 4 (Kenaga and Goring, 1978). This would indicate that sethoxydim has little tendency to bioaccumulate.

Sulfometuron Methyl

Sulfometuron methyl was slightly toxic to fish and practically non-toxic to slightly toxic to aquatic invertebrates. Studies indicate that sulfometuron methyl does not bioaccumulate in aquatic species.

Triclopyr

The toxicity of triclopyr to fish ranges from practically non-toxic to highly toxic (USDA, 1984, as cited in USDA, 1989), whereas it is practically non-toxic to aquatic invertebrates. Rapid excretion of triclopyr suggests that there is low potential for bioaccumulation.

4.0 Inert Ingredients

4.1 Aquatic Wildlife Toxicity

Petroleum Distillates

The term petroleum distillate encompasses many chemical substances derived from the distillation of petroleum including gasoline, kerosene, jet fuels, fuel oils, xylenes, and other products. Because of the diversity of products and their range of chemical and physical properties, their toxicities are also far ranging. The toxicity of petroleum distillates to fish range from practically non-toxic to highly toxic (CIS, 1991; USDA, 1989) and their toxicities to aquatic invertebrates range from practically non-toxic to very highly toxic.

Because there are many forms of petroleum distillates, it is difficult to generalize about the ability of these products to bioaccumulate and bioconcentrate in aquatic organisms. Therefore, information is presented on the bioaccumulation and bioconcentration properties of xylene, a petroleum distillate that is used by the WSDOT.

The reported bioconcentration factors for xylene range from 45 to 105. Based on this and the half-life in water of 2.6 to 11 days, bioconcentration is not likely to occur. Bioaccumulation in aquatic in organisms is slight to moderate (USEPA, 1985).

5.0 Adjuvants

5.1 Aquatic Wildlife Toxicity

The adjuvants used by the WSDOT and their toxicities to aquatic organisms are listed in Chapter 5, Table 5-7 of this appendix. For the majority of the adjuvants listed, no toxicity data were found partly due to their biologically inactive structures or to lack of listing when not in the presence of an "active ingredient." For those compounds where toxicity data were available, the findings show them to be practically non-toxic to slightly toxic to aquatic species.

6.0 Fertilizers

6.1 Aquatic Wildlife

The primary concern with the use of fertilizers is the potential for nutrient loading to aquatic habitats via surface runoff. Elevated concentrations of phosphates and nitrates in lakes and estuaries can result in increased phytoplankton productivity, which if great enough and of sufficient duration, can result in degradation of water quality through oxygen depletion. Chronic nutrient enrichment of lakes can result in eutrophication.

Ammonia-based fertilizers can be toxic to aquatic organisms due to their high concentrations of ammonia. Ammonia toxicity is pH and temperature dependent. The chronic ambient water quality criteria for the protection of freshwater organisms developed by the U.S. Environmental Protection Agency for unionized and total ammonia are 2.9 and 2400 micrograms per liter, respectively, at a temperature of 10 degree centigrade and a pH of 7.0 (USEPA, 1986).

7.0 Uncertainty and Data Gaps

Uncertainty is inherent in risk assessment because a quantitation of risk is based on assumptions about the level and frequency of exposure. Such assumptions can be refined or limited by the use of ever more data, but they can never be eliminated. Due to the programmatic nature of this impact evaluation and the fact that most WSDOT vegetation management tools are of low aquatic impact, a qualitative comparison, based on previously described parameters, has been provided. Its limitations are described below.

The estimates of exposure and the estimates of risk based on these exposures should not be taken out of context. They are based on available information and informed

assumptions. When considering the risk to the identified aquatic receptors, the bounds of this exposure assessment, as well as the mitigating factors, such as the actual amount of herbicide used throughout the state or the likelihood of exposure should be considered. This assessment is most useful for comparative purposes, that is, given the same set of exposure parameters and assumptions, the extent of exposure to each of the herbicides and the resulting estimates of risk can be compared. It is especially important to consider the uncertainty inherent in the assessment as well as any data gaps when evaluating these estimates of risk. Data gaps include actual roadside acreage, actual environmental conditions, and related information. Compound data is fairly complete for the determinations made here.

In this risk assessment, there is uncertainty associated with the estimated risks to aquatic receptors. The main sources of uncertainty occur in the evaluation of exposure for specific species and the estimation of potential hazards to populations.

7.1 Toxicity Information

Information on the toxicity of the chemicals proposed for use in the WSDOT Roadside Vegetation Management Program is based exclusively on laboratory studies involving a limited number species of aquatic organisms. There is some uncertainty associated with using laboratory values to estimate responses in the field. There is also uncertainty in extrapolating laboratory data obtained from a particular life stage of one species to different species, life stages, or populations.

7.2 Estimated Environmental Concentrations

Because insufficient data were available for calculating an estimated environmental concentration and project time and funds were limited, a qualitative rather than a quantitative approach was adopted for evaluating exposure. Only rainfall, road density, and soil half life values for each of the herbicides were used in estimating exposure. Due to the lack of site-specific information, there is uncertainty associated with the relative exposure ratings developed for the herbicides in each of the physiographic provinces. Such factors as total area treated, concentrations of the herbicides used, and herbicide use by physiographic province were not considered due to the complexities of calculation and programmatic nature of this project. The use of specific information in a quantitative assessment may reduce some of the uncertainty, but would not be expected to alter conclusions.

7.3 Exposure Parameters

There is substantial uncertainty associated with the parameters used to estimate exposure. Surface runoff was the only exposure pathway considered in estimating exposure, but spray drift may also account for a certain amount of additional exposure. Surface runoff may be overestimated in low rainfall areas. Additionally, other sources of herbicide input were not considered when estimating exposure. One of the greatest sources of uncertainty is exposure duration, as mobile organisms may avoid or leave a treated area, and application may vary from once a year to once every 2-4 years per site.

7.4 Sublethal Effects

Potential hazards of adverse effects to aquatic organisms from exposure to the chemicals of concern were evaluated by selection of the lowest available acute toxicity value. Relatively little information is available describing chronic toxicity of the herbicides of concern to aquatic organisms. Conversely, chronic exposure potential is low based on WSDOT's application practices. Therefore, it may be assumed that sublethal effects, if they occur, would add to the uncertainty in estimating risk.

7.5 Potential Hazards to Populations

There is significant uncertainty as to the impact of these chemicals on populations or communities. Hypothetically, at the population level, deleterious effects may be manifested as a reduction in the number of animals in a given population. This reduction in the numbers of animals in an area could result from either lower survival (via mortality or diminished reproductive capacity) or relocation of individuals in the population to alternative habitats (displacement). The more dependent a species is on a specific geographic region or unique habitat, the greater the potential for impact may be.

8.0 Conclusions

The majority of the chemicals evaluated present low relative risk to aquatic species in the eight physiographic provinces in the State of Washington. Chlorosulfuron, 2,4-D, 2,4-DP, fluazifop-butyl, fosamine, imazapyr, metsulfuron methyl, napropamide, oryzalin, pronamide, sulfometuron methyl, triclopyr, and xylene are considered to present low relative risk to fish and aquatic invertebrates.

Bromacil, dichlobenil, glyphosate, isoxaben, mecoprop, mefluidide, oxadiazon, picloram, sethoxydim, and diuron are considered to present moderate relative risk to fish species within the Puget Trough Physiographic Province. Oxadiazon, picloram, and diuron are also considered to present moderate relative risk to fish in the Olympic Peninsula, Coast Ranges, and Northern and Southern Cascades Physiographic Provinces.

Benefin, bromacil, chlorthal dimethyl, dicamba, dichlobenil, isoxaben, mecoprop, picloram, and sethoxydim pose a potential moderate relative risk to aquatic invertebrates in the Puget Trough Physiographic Province while diuron and oxadiazon pose moderate risk to aquatic invertebrates in the Olympic Peninsula, Coast Ranges, and Northern and Southern Cascades Physiographic Provinces.

The Puget Trough Physiographic Province was the only region in which some of the chemicals were characterized as having a high relative risk. Diuron poses high relative risk to both fish and aquatic invertebrates while benefin and oxadiazon pose high relative risks to fish and aquatic invertebrates, respectively.

The high proportion of moderate and high relative risks of herbicides in the Puget Trough Physiographic Province is due primarily to the high road density and the relatively high rainfall in this region. Both of these factors increase the probability of surface runoff of herbicides to surface waters.

In order to illustrate the potential for achieving toxic concentrations of benefin, diuron, and oxadiazon in aquatic environments, a hypothetical scenario. In this scenario, the quantities of each of these herbicides necessary to achieve the lowest LC_{50} were calculated for fish and aquatic invertebrates in a 100 acre lake with a maximum depth of 20 feet. The volume of the lake is that described by a right circular cone with a height of twenty feet:

$$\text{Volume} = \frac{\pi(r^2)h}{3} = \frac{\pi(4{,}356{,}000 \text{ square feet})\ (20 \text{ feet})}{3}$$

The volume of the lake would be 26,000,000 cubic feet or 741,000,000 liters.

Because the WSDOT herbicide application records (Tables 5-9 to 5-33 of Chapter 5 of this appendix) listed herbicide application by WSDOT District rather than by physiographic province, it was necessary to approximate the quantities of benefin, diuron, and oxadiazon applied in the Puget Trough Physiographic Province. This was accomplished by taking the sum of the quantities of each herbicide applied in WSDOT districts 1, 3, and 4 (a portion of each district is located in the Puget Trough Physiographic Province) and then assuming that half of this sum was applied within the Puget Trough Physiographic Province. In the case of oxadiazon, which has not been used previously by WSDOT, it was assumed that future oxadiazon use would be similar to that of benefin, which has a maximum label rate of six pounds of active ingredient per acre while that of oxadiazon is four pounds of active ingredient per acre. The total quantity of benefin applied to WSDOT districts 1, 2, and 4 (148 pounds) was multiplied by the ratio of the maximum label rate of oxadiazon to that of benefin to arrive at the estimated total quantity of oxadiazon that would be used in Districts 1, 2, and 4:

$$4/6 \text{ X } (148 \text{ pounds}) = 99 \text{ pounds.}$$

The estimated quantities of benefin, diuron, and oxadiazon applied in the Puget Trough Physiographic Province are 74, 604, and 50 pounds, respectively (Table 3-7).

The quantities of the three herbicides necessary to achieve the lowest LC_{50} values for fish and invertebrates are listed in Table 3-7 with the estimated quantities of each herbicide applied in the Puget Trough Physiographic Province between July 1989 and June 1990.

Based upon the quantities of each of the herbicides that would be necessary to achieve the LC_{50} values listed in Table 3-7, it is highly unlikely that these quantities of herbicides would be carried to a single body of surface water via runoff or spray drift, based on the estimates of the quantities applied throughout the entire Puget Trough Physiographic Province. An accidental spill of a large quantity of one of these herbicides would be the most likely scenario for achieving concentrations that would threaten aquatic species. However, WSDOT does not transport in large single load quantities.

8.1 Mitigating Measures

Considering the relatively low usage of benefin and oxadiazon in the Puget Trough Physiographic Province by the WSDOT, it is unlikely that these herbicides will present a threat to aquatic organisms within this physiographic province. However, to avoid the

potential for the contamination of aquatic habitats, use adjacent to aquatic enironments should be avoided.

The application rates of benefin, diuron, and oxadiazon should be the lowest possible to provide effective vegetation control. The highest average application rates of benefin and diuron from WSDOT Districts 1, 2, and 4 were 3.5 and 3 pounds of active ingredient per acre, respectively. These rates represent 50 percent of the maximum label rate for benefin and 8.75 percent of that for diuron.

Benefin and diuron are moderately persistent in soils while oxadiazon is somewhat more persistent. The frequency of application for these herbicides should be kept to a minimum to prevent the accumulation of high soil residues.

Table 3-1 Relative Annual Precipitation and Relative WSDOT Road Densities by Physiographic Province

Physiographic Province	Annual Rainfall[a]	Relative Rainfall[b]	Road Density[b]
(1) Olympic Peninsula	>240"	3	1
(2) Coast Range	60-120"	3	1
(3) Puget Trough	15-60"	2	3
(4) Northern Cascades	50-180"	3	1
(5) Southern Cascades	50-140"	3	1
(6) Okanogan Highlands	12-45"	2	2
(7) Columbia Basin	7-20"	1	2
(8) Blue Mountains	18-40"	2	1

[a] Franklin and Dyrness, 1973

[b] A rating of 3 indicates high rainfall or road density whereas ratings of 2 and 1 correspond to moderate and low values, respectively.

Table 3-2 Relative Persistence of WSDOT Applied Herbicides in Soil

Chemical	Persistence[a]	Chemical	Persistence
2,4-D	1	Isoxaben	2
2,4-DP	1[b]	Mecoprop	2
Benefin	2	Mefluidide	2[c]
Bromacil	2	Metsulfuron methyl	3
Chlorsulfuron	1	Napropamide	1
Chlorthal dimethyl	1	Oryzalin	1
Dicamba	1	Oxadiazon	3
Dichlobenil	2	Picloram	3
Diuron	2	Pronamide	1
Fluazifop-butyl	1	Sethoxydim	2
Fosamine	1	Sulfometuron methyl	1
Glyphosate	2[d]	Triclopyr	1
Imazapyr	1	Petroleum distillate	1

[a] Persistence ratings are based on reported half life in soil (see Section 2.1.1, Chapter 3, Appendix A) as follows:

1: 0 to 3 months
2: 4 to 6 months
3: Greater than 6 months

[b] No information was found describing the half life of 2,4-DP in soil, but because it is chemically similar to 2,4-D, it was assumed for the purposes of this assessment that its half life in soil would be similar as well.

[c] No information was found describing the half life of mefluidide in soil so it was assumed for the purposes of this assessment that it was moderately persistence (4 to 6 months) and was therefore given a persistence rating of 2.

[d] This persistence rating is for sandy loam soils, in most soils glyphosate has a soil half life less than 60 days.

Table 3-3 Relative Exposure[a] of Aquatic Organisms To Herbicides By Physiographic Porvince

	Physiographic Province[b]							
Chemical	1	2	3	4	5	6	7	8
2,4-D	1	1	1	1	1	1	1	1
2,4-DP	1	1	1	1	1	1	1	1
Benefin	1	1	2	1	1	1	1	1
Bromacil	2	2	3	2	2	1	1	1
Chlorsulfuron	1	1	2	1	1	1	1	1
Chlorthal dimethyl	1	1	2	1	1	1	1	1
Dicamba	1	1	2	1	1	1	1	1
Dichlobenil	1	1	2	1	1	1	1	1
Diuron	2	2	3	2	2	1	1	1
Fluazifop-butyl	1	1	1	1	1	1	1	1
Fosamine	1	1	2	1	1	1	1	1
Glyphosate	1	1	2	1	1	1	1	1
Imazypyr	1	1	1	1	1	1	1	1
Isoxaben	1	1	2	1	1	1	1	1
Mecoprop	1	1	2	1	1	1	1	1
Mefluidide	1	1	2	1	1	1	1	1
Metsulfuron methyl	1	1	2	1	1	1	1	1
Napropamide	1	1	1	1	1	1	1	1
Oryzalin	1	1	1	1	1	1	1	1
Oxadiazon	2	2	3	2	2	1	1	1
Picloram	2	2	3	1	2	1	1	1
Pronamide	1	1	1	1	1	1	1	1
Sethoxydim	1	1	2	1	1	1	1	1
Sulfometuron methyl	1	1	1	1	1	1	1	1
Triclopyr	1	1	1	1	1	1	1	1
Petroleum distillate	1	1	1	1	1	1	1	1

Table 3-3 (Continued)

[a] Exposure is based upon average annual precipitation and road density in each of the physiographic provinces as well as the maximum application rate and half life of each chemical in soil. The relative exposure ratings are as follows:

1: Low exposure
2: Moderate exposure
3: High exposure

[b] Physiographic provinces:

(1) Olympic Peninsula
(2) Coast Ranges
(3) Puget Trough
(4) Northern Cascades
(5) Southern Cascades
(6) Okanogan Highlands
(7) Columbia Basin
(8) Blue Mountains

Table 3-4 Relative Risk[a] To Fish From Exposure To Herbicides By Physiographic Province

	Physiographic Province[b]							
Chemical	1	2	3	4	5	6	7	8
2,4-D	1	1	1	1	1	1	1	1
2,4-DP	1	1	1	1	1	1	1	1
Benefin	1	1	3	1	1	1	1	1
Bromacil	1	1	2	1	1	1	1	1
Chlorsulfuron	1	1	1	1	1	1	1	1
Chlorthal dimethyl	1	1	1	1	1	1	1	1
Dicamba	1	1	1	1	1	1	1	1
Dichlobenil	1	1	2	1	1	1	1	1
Diuron	2	2	3	2	2	1	1	1
Fluazifop-butyl	1	1	1	1	1	1	1	1
Fosamine	1	1	1	1	1	1	1	1
Glyphosate	1	1	2	1	1	1	1	1
Imazypyr	1	1	1	1	1	1	1	1
Isoxaben	1	1	2	1	1	1	1	1
Mecoprop	1	1	2	1	1	1	1	1
Mefluidide	1	1	2	1	1	1	1	1
Metsulfuron methyl	1	1	1	1	1	1	1	1
Napropamide	1	1	1	1	1	1	1	1
Oryzalin	1	1	1	1	1	1	1	1
Oxadiazon	2	2	2	2	2	1	1	1
Picloram	2	2	2	1	2	1	1	1
Pronamide	1	1	1	1	1	1	1	1
Sethoxydim	1	1	2	1	1	1	1	1
Sulfometuron methyl	1	1	1	1	1	1	1	1
Triclopyr	1	1	1	1	1	1	1	1
Petroleum distillate	1	1	1	1	1	1	1	1

Table 3-4 (Continued)

[a] Risk is characterized based upon likelyhood of exposure and the relative toxicities of the herbicides. The relative risk ratings are as follows:

1: Low risk
2: Moderate risk
3: High risk

[b] Physiographic provinces:

(1) Olympic Peninsula
(2) Coast Ranges
(3) Puget Trough
(4) Northern Cascades
(5) Southern Cascades
(6) Okanogan Highlands
(7) Columbia Basin
(8) Blue Mountains

Table 3-5 Relative Risk[a] To Aquatic Invertebrates From Exposure To Herbicides By Physiographic Province

	Physiographic Province[b]							
Chemical	1	2	3	4	5	6	7	8
2,4-D	1	1	1	1	1	1	1	1
2,4-DP	1	1	1	1	1	1	1	1
Benefin	1	1	2	1	1	1	1	1
Bromacil	1	1	2	1	1	1	1	1
Chlorsulfuron	1	1	1	1	1	1	1	1
Chlorthal dimethyl	1	1	2	1	1	1	1	1
Dicamba	1	1	2	1	1	1	1	1
Dichlobenil	1	1	2	1	1	1	1	1
Diuron	2	2	3	2	2	1	1	1
Fluazifop-butyl	1	1	1	1	1	1	1	1
Fosamine	1	1	1	1	1	1	1	1
Glyphosate	1	1	1	1	1	1	1	1
Imazypyr	1	1	1	1	1	1	1	1
Isoxaben	1	1	2	1	1	1	1	1
Mecoprop	1	1	2	1	1	1	1	1
Mefluidide	1	1	1	1	1	1	1	1
Metsulfuron methyl	1	1	1	1	1	1	1	1
Napropamide	1	1	1	1	1	1	1	1
Oryzalin	1	1	1	1	1	1	1	1
Oxadiazon	2	2	3	2	2	1	1	1
Picloram	1	1	2	1	1	1	1	1
Pronamide	1	1	1	1	1	1	1	1
Sethoxydim	1	1	2	1	1	1	1	1
Sulfometuron methyl	1	1	1	1	1	1	1	1
Triclopyr	1	1	1	1	1	1	1	1
Petroleum distillate	1	1	1	1	1	1	1	1

Table 3-5 (Continued)

[a] Risk is characterized based upon likelyhood of exposure and the relative toxicities of the herbicides. The relative risk ratings are as follows:

1: Low risk
2: Moderate risk
3: High risk

[b] Physiographic provinces:

(1) Olympic Peninsula
(2) Coast Ranges
(3) Puget Trough
(4) Northern Cascades
(5) Southern Cascades
(6) Okanogan Highlands
(7) Columbia Basin
(8) Blue Mountains

Table 3-6 Relative Acute Aquatic Toxicities Of the Herbicides Used by the WSDOT[a]

	Toxicity[b]			Toxicity	
Herbicide	F	I	Herbicide	F	I
2,4-D	3	3	Isoxaben	3	3
2,4-DP	3	3	Mecoprop	1	NA
Benefin	5	4	Mefluidide	2	1
Bromacil	2	2	Metsulfuron methyl	1	1
Chlorsulfuron	1	1	Napropamide	3	2
Chlorthal dimethyl	1	4	Oryzalin	3	4
Dicamba	2	3	Oxadiazon	3	4
Dichlobenil	3	4	Picloram	2	1
Diuron	4	4	Pronamide	2	3
Fluazifop-butyl	4	NA	Sethoxydim	3	2
Fosamine	1	1	Sulfometuron methyl	2	2
Glyphosate	3	2	Triclopyr	4	1
Imazapyr	1	1	Petroleum distillate	4	4

[a] Relative acute toxicities of the herbicides (USEPA, 1985):

LC_{50} or EC_{50} (mg/l)	Toxicity Category
<0.1	Very highly toxic (5)
0.1 to 1	Highly toxic (4)
>1 to ≤ 10	Moderately toxic (3)
>10 to ≤ 100	Slightly toxic (2)
>100	Practically non-toxic (1)

[b] F: Fish; I: Aquatic Invertebrates

Appendix B - Chapter 4

Draft EIS
Roadside Vegetation Management

Table 3-7 Quantities of Benefin, Diuron, and Oxadiazon required to achieve the lowest LC_{50} values for fish and invertebrates in a 100 acre lake and extrapolated to the Puget Trough Surface waters, which include approximately 1,347,030 acres (USDC,1990). These are compared to the total quantities applied in the Puget Trough Physiographic Province from July 1989 to June 1990.

Chemical	LC_{50} (mg/L)		Pounds of Herbicide				Pounds Applied in Puget Trough
			100 Acre Lake		Puget Sound Surface Water		
	Fish	Invert.	Fish	Invert.	Fish	Invert.	
Benefin	0.0008[a]	0.1[a]	1.3	163	17,511	2,195,610	220
Diuron	0.25[b]	0.16[c]	410	262	5,522,700	3,529,140	17,890
Oxadiazon	1.76[d]	0.5[d]	2,874	816	38,712,780	10,991,520	200

[a] Melichar, 1991
[b] Hartley and Kidd, 1987
[c] USDI, 1991
[d] Worthing and Hance, 1991

1.0 Introduction

1.1 Purpose

The purpose of this chapter is to examine the potential of adjuvants, inert ingredients, fertilizers and 25 herbicides to cause adverse health effects in humans. The information generated will be used to evaluate the risks to workers and the public verses the benefits of using herbicides to control vegetation on and near roadways in the state of Washington. Of the 25 herbicides selected by the Washington State Department of Transportation (WSDOT) for possible use in their vegetation management program, 23 have a history of usage. Two, fluazifop-butyl and oxadiazon, are being evaluated for future use. Unlike the United States Forestry Service (USFS) or United States Department of Interior Bureau of Land Management (USDI) Environmental Impact Statements (EIS), this document assesses the potential risk of herbicide treatment to only highway rights-of-way in Washington. The acreage sprayed and the populations which might conceivably be exposed are greatly decreased from the vegetation control programs analyzed in the previous EIS's. Also, the probability that the public will be exposed is reduced from those calculated for forestry uses since the methods used to apply herbicides to highway rights-of-way are less likely to cause drift, overspray or direct accidental exposure (i.e., aerial applications vs. truck and hand spraying).

1.2 Organization of This Chapter

This section presents the purpose and describes the structure and methodology used to perform the human health risk assessment. Section 2, Hazard Identification, gives an introduction to toxicology, summarizes the epidemiology and laboratory toxicology data available for each chemical and discusses data gaps and study acceptability. Section 3, Exposure Assessment, describes the populations at risk, identifies the exposure routes and pathways, and estimates the amount of active ingredient which the public or WSDOT employee might receive. Section 4, Dose-Response Assessment, identifies the critical systemic (noncancer) and carcinogenic (cancer) toxicity values are used to perform the risk assessment. Section 5, Risk Characterization, quantifies the risk to workers and the public from individual and multiple pathways of exposure and from individual herbicides and herbicides commonly used together. Section 6, Risk Communication, summarizes the findings in the form of risk tables, presents uncertainties and discusses conflicting data noted in the other EIS's.

1.3 What is a Risk Assessment?

1.3.1 Overview of the Risk Assessment:

The risk assessment technique provides a means of analyzing available relevant data and estimating possible adverse human health effects. Unfortunately it conveys a level of precision which is misleading due to the subjective nature of assumptions made,

regardless of how well documented these assumptions are. The risk assessment contained in this chapter uses assumptions and methodology generally accepted by the scientific community and regulatory agencies.

Single Day Doses (SDD), also referred to as the acceptable daily dose, were calculated for each chemical and Lifetime Average Daily Doses (LADD) were calculated for the six potential carcinogens identified. Four exposure scenarios were evaluated for the public and WSDOT employees using two sets of assumptions, average and maximum. The parameters used to calculate the average doses include the historical WSDOT average application rate for each herbicide or low label recommended application rate if historical data were not available, and average or reasonable estimates of duration of exposure, frequency of exposure, area exposed, amount of drift, number of berries consumed, etc. The parameters used to calculate the maximum dose levels include maximum recommended label rates and more conservative (higher) exposure assumptions. Methods and assumptions used to estimate human doses are presented in detail in Section 3 of this chapter.

This estimated human dose is then compared to the appropriate toxicity values, determined from animal laboratory testing, and an "uncertainty factor" is applied. For systemic and reproductive or developmental toxicity this analysis compares a single (or acute) human exposure to a chronic and sub-chronic animal exposure respectively. In addition, risk of carcinogenicity was evaluated for six herbicides (2,4-D, Bromacil, Oryzalin, Oxadiazon, Picloram, and Pronamide) and petroleum distillates, kerosene and diesel. The risk of mutagenicity was evaluated qualitatively based on the weight of evidence data available from genotoxicity (mutagenicity) testing. Exposures from multiple pathways and herbicide mixtures were also evaluated.

1.4 Structure of the Risk Assessment

This risk assessment is comprised of four analytical sections, as recommended by the United States Environmental Protection Agency (USEPA) and a summary: hazard identification, exposure assessment, dose-response assessment, and risk characterization and communication. The components of, and methodology utilized in each of these sections are described in the following paragraphs and illustrated in Figure 4-1.

1.4.1 Hazard Identification:

The first step in risk assessment, by any method, is hazard identification (or hazard analysis), which is presented in Section 2 of this chapter. The 25 herbicide active ingredients quantitatively evaluated were chosen by the WSDOT as vegetation control agents which could conceivably be used in their management program. They are:

1) 2,4-D
2) 2,4-DP
3) Benefin
4) Bromacil
5) Chlorsulfuron
6) Chlorthal dimethyl
7) Dicamba

8) Dichlobenil
9) Diuron
10) Fluazifop-butyl
11) Fosamine
12) Glyphosate
13) Imazapyr
14) Isoxaben
15) Mecoprop
16) Mefluidide
17) Metsulfuron methyl
18) Napropamide
19) Oryzalin
20) Oxadiazon
21) Picloram
22) Pronamide
23) Sethoxydim
24) Sulfometuron methyl
25) Triclopyr

Qualitative risk analysis discussions included cover:

1) Inert ingredients
2) Adjuvants
3) Fertilizers

Do these chemicals present an unreasonable health hazard for the public or worker safety in comparison to alternative methods of vegetation control available? To answer this question an indepth analysis of the available toxicology and epidemiology data must be performed. Ideally results from human studies would provide the best information on which to base a risk assessment. For obvious reasons, human toxicology data are normally not available and epidemiology data are usually qualitative, rendering it of limited use for a quantitative risk analysis. For most of the herbicides examined in this risk assessment, no human data are available.

In order to register a pesticide in the United States, The Environmental Protection Agency requires a battery of animal testing which usually is comprised of acute, subchronic, chronic, reproduction/developmental, carcinogenicity, mutagenicity, and ecotoxicity (aquatic, avian and wildlife) studies. The number and type of studies required is dictated by the use pattern of each individual chemical. For a non-food use pattern typical of most herbicides, the battery of tests required for registration is limited. Toxicity data is even more limited for adjuvants, inert ingredients, carriers and fertilizers since they are not pesticides and do not need to be registered by the Agency.

The hazard identification section begins with a overview of toxicology and summarizes the laboratory toxicity data generated for, and submitted to, regulatory agencies (EPA and CDFA) in support of pesticide registrations. For each chemical, it presents the following:

1) Brief discussions—
   a) Class of herbicide
   b) Physical and chemical properties
   c) Environmental fate
   d) Adverse effects in humans available from literature
   e) Data gaps noted

and

2) Summaries of laboratory data—
   a) Acute/Subacute
   b) Subchronic/Chronic
   c) Reproduction/Developmental
   d) Carcinogenicity/oncogenicity
   e) Mutagenicity

1.4.2 Exposure Assessment:

Assessment of the potential risk of adverse health effects associated with the use of herbicides in a vegetation management program requires evaluating the possible types of exposures resulting from the actual herbicide application and public activities in and around the sprayed site. Section 3, Exposure Assessment, estimates the amount, duration and frequency of exposure, combined with an evaluation of the bioavailability of each chemical. These values are then used to calculate the chemical intake.

A review of the herbicide application methods employed by the Washington State Department of Transportation, historical average WSDOT application rates for each herbicide, and maximum recommended label rates for each herbicide are discussed in detail in this section. Environmental fate is presented in Appendix B, Chapter 1, but is not taken into consideration for the quantitative portion of the risk analysis. This risk assessment assumes that 100 percent of the sprayed formulated product contacted is available for uptake, WSDOT average and maximum recommended label application rates, and no environmental degradation.

The exposure scenarios for two populations (the public and worker) are examined. Public dose estimates are based on routine activities performed at or near the treated site, through one or more of the following routes:

1) Oral ingestion of berries which have received spray drift, adults and children.

2) Oral ingestion of garden vegetables (lettuce and beans) which have received spray drift.

3) Dermal contact from picking berries which have received spray drift.

4) Dermal contact with vegetation which has been sprayed.

The following WSDOT worker exposure scenarios were examined in the risk assessment:

1) Workers applying herbicides using truck mounted spray equipment.

2) Workers mixing and loading herbicides into application equipment.

3) Workers mixing, loading and applying herbicides using truck mounted spray equipment.

4) Workers applying herbicides using backpack tanks or hand held spray equipment.

Average and maximum plausible case calculations were made for both public and worker exposure scenarios, by varying the parameters and assumptions used; such as application rates, amount of drift, area exposed and/or frequency and duration of exposure. Tables 4-1 through 4-10, present average and maximum estimates of the Single Day Dose and Lifetime Average Daily Dose for the public and worker exposure scenarios outlined above. Table 4-11 contains application rates, seasons applied, number of months sprayed and dermal bioavailability information used to estimate exposures.

The inhalation route was not considered since worker field studies have consistently shown inhalation to be an insignificant route of exposure. (Jones & Stokes, 1991; USDI, 1991)

The risk associated with drinking water contaminated by drift or runoff was not estimated. This route of exposure was evaluated in previously cited documents. Not one compound was found to be a cause for concern, from either systemic or cancer risk, and most roadsides in Washington are designed to minimize contamination of surface waters due to runoff.

Dermal exposure to and inadvertent ingestion of contaminated soil by children was not evaluated. For the sake of risk assessment, the scientific community considers children to be under 6 years of age. Children in this age range are not usually allowed to play on roads or highways. The majority of dermal exposure from rights-of-way spraying would be to older children or adults walking to school (school bus) or work, joggers, hikers, and berry pickers.

The risk associated with accidental exposure to herbicides was not examined. Testing, regulations and the product label are designed to mitigate adverse effects so when used according to label instructions and precautions, pesticides can be a benefit to society with minimal risk. In the case of an accidental exposure, there is a probability of the development of an adverse effect. It should be noted that the WSDOT usually mixes the concentrated product with its carrier (usually water) at their maintenance yard, away from the public, and their largest spray tank contains only 70 gallons of spray concentrate. Both these practices greatly reduce public risk from accidental exposure. Occasionally, when the distance to the spray site is great, WSDOT workers carry additional concentrate for mixing on site. (It is possible to carry two 70 gallon tanks and one 50 gallon tank for a maximum of 190 gallons of concentrate).

1.4.3 Dose-Response Assessment:

The dose-response assessment, Section 4, is a collection of tables which identify the critical toxicity and carcino genicity values which are used to calculate the hazard quotient and cancer risk probability. Summaries of all available laboratory toxicity data have been compiled for each compound and can be found in Chapter 5 of the appendix.

The methods used to evaluate systemic (noncancer) and cancer effects differ. Theoretically one molecule of a pesticide can cause injury to liver or kidney cells, etc., resulting in injury or death to a single cell, which would be biologically insignificant. The Lowest Observable Effect Level (LOEL) and the No Observable Effect Level (NOEL) are identified for systemic toxic effects (noncancer) in each animal study preformed. The NOEL is the highest dosage on a study which produces no adverse or toxic effect and the LOEL is the dose level in which the toxic effect first appears. After examination of the toxicity data the lowest NOEL is identified, usually from a chronic study, and divided by an "uncertainty factor" (UF) to arrive at the Reference Dose (RfD). The criteria used by the EPA for applying "uncertainty factors" are as follows:

The Uncertainty factors generally consist of multiples of 10 (although values of less than 10 are sometime used), with each factor representing a specific area of uncertainty inherent in the extrapolation from available data. The bases for application of different uncertainty factors are explained below.

- A UF of 10 is used to account for variation in the general population and is intended to protect sensitive subpopulations (e.g., elderly, children.)

- A UF of 10 is used when extrapolating from animals to humans. This factor is intended to account for the interspecies variability between humans and other mammals.

- A UF of 10 is used when a NOEL derived from a subchronic study is used as the basis for a chronic RfD.

- A UF of 10 is used when a LOEL is used instead of a NOEL. This factor is intended to account for the uncertainty associated with extrapolating from LOELs to NOELs.

Reference dose is defined as an estimate of the daily exposure to human populations (including sensitive subpopulations), measured in specific units, generally mg/kg/day, that reflects the level at which no adverse noncarcinogenic effects are expected should humans be chronically exposed to that level (Jones and Stokes, 1991). The EPA has set RfD values for many chemicals. For those herbicides which did not have a published RfD, one was assigned using the method of calculation employed by the EPA. A listing of RfD values can be found in Table 4-12 of this chapter. The uncertainty factor applied, level of confidence in the RfD value, NOEL with critical toxic effect and source references are also included in this table.

At present, it is generally accepted by the scientific community that any level of exposure to a potential carcinogen poses a risk. In contrast to systemic effects, a single molecule

of a potential carcinogen could cause alterations of the DNA in a single cell resulting in cloning of that cell, which in turn can lead to tumor formation and death. Therefore, an alternative approach to hazard evaluation of a potential carcinogen is necessary (Klassen, et al., 1986). Mathematical dose-response models are most often used to extrapolate the risk of getting cancer at very low doses (i.e., 1 in 100,000). Many of these models exist but the one most frequently used by regulatory agencies is the Linearized Multistage model. The slope (or Cancer Potency Factor) of the dose-response curve is calculated by the model.

Table 4-13 summarizes the critical carcinogenicity information including the Cancer Potency Factor (CPF). The CPF is expressed in (mg/kg/day) and is used to calculate the increased probability that an individual will develop a tumor due to exposure to a chemical. This risk assessment uses cancer potency values derived by the EPA or Office of Pesticide Products (OPP). Table 4-13 also includes the weight-of-evidence classification assigned by the EPA using the following guidelines:

For convenience Table 4-14 presents the RfD values for systemic or reproductive/developmental effects and CPF values for each herbicide.

1.4.4 Risk Characterization:

Risk characterization is the process of estimating the incidence of a health effect under the various conditions of human exposure described in the exposure assessment. It is performed by combining the information provided in the exposure and dose-response assessments (Paustenbach, 1989). The intent of the risk assessment is to develop a technically sound and useful portrayal of the potential human health risks associated with the WSDOT Vegetation Management program so that the risk managers can weigh risk and benefits and evaluate alternatives.

Noncancer effects are estimated by calculating a hazard quotient (HQ) for each pathway of exposure. This is the Single Day Dose (SDD) divided by the reference dose (RfD).

$$HQ = SDD \div RfD$$

Several simultaneous exposures to subthreshold levels could result in an adverse health effect. Therefore, the risk associated with multiple exposure pathways must also be estimated (i.e., dermal exposure picking berries and ingestion of the berries picked). Exposure to two different chemicals which have the same target organ may also cause an unexpected adverse effect. This risk assessment evaluates combined risks by calculating the hazard index (HI), which is the sum of hazard quotients for each pathway or the sum of the hazard quotients of herbicides frequently used together for one exposure pathway, respectively.

$$HI = HQ1 + HQ2 + \ldots HQX$$

Hazard quotient and hazard index values of one or less are typically considered to be of no concern. Values slightly larger than one are of limited concern, considering that the excepted practice of estimating the hazard quotient introduces a large degree of conservatism because it is calculated by comparing a single average acute exposure to a

value which represents a subchronic or chronic exposure plus "uncertainty" or "safety" factors.

Cancer risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen. Or, the increased probability of developing a tumor in a lifetime due to exposure from a potential carcinogen over the already existing probability that an individual will develop cancer in their lifetime.

The risk associated with exposure to a potential carcinogen is calculated by multiplying the lifetime average daily dose (LADD) by the cancer potency factor (CPF).

$$\text{RISK} = \text{LADD} \times \text{CPF}$$

In the past a risk of one in a million was considered to be an "acceptable" risk. Presently, regulatory agencies are taking exception to using the one in a million benchmark, especially since many common human activities entail greater risk. Examination of the risks of common human activities demonstrates a lifetime risk of one in 100,000 or more is within the realm of, or orders of magnitude below, everyday risks that generally do not cause undue concern. These are risks that people, while they are aware of them and may have some concern or fear over them, do not in general alter their behavior to avoid. (See page 102 which illustrates the risk from many activities which greatly exceed the level of 1 in 100,000). In comparison to the background risks of "everyday" activities, a lifetime risk of 1 in 100,000 is relatively small.

To put a 1 in 100,000 additional lifetime risk into perspective, consider that the actual lifetime risk of a person developing cancer in the United States is approximately 0.30 (e.g., about 30% of the population will contract cancer in a lifetime, although the percentage that actually die from cancer is somewhat less). Thus, an additional lifetime risk of 1 in 100,000 increases ones "risk" from 0.3 to 0.30001. (D. Eaton, 1992)

Regulatory action will not generally be justifiable unless risks are substantially higher than the 1 in 100,000 "benchmark" (Rodricks, et al., 1987). A retrospective examination of the level of risk which triggered regulatory action was done by Travis, et al., (1987) and summarized as follows:

1. Every chemical with an individual lifetime risk above 4×10^{-3} received regulation. Those with values below 1×10^{-6} remained unregulated.

2. For small populations, regulatory action never resulted for individual risks below 1×10^{-4}.

3. For effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action; above 3×10^{-4} it always triggered action.

This risk assessment considers a risk of one in 100,000 to be an "acceptable" risk.

Average and maximum hazard quotients and cancer risk probabilities for each herbicide are presented in Tables 4-15 through 4-24. Risks associated with multiple pathways of

exposure are presented in Tables 4-25 and 4-26, and multiple chemicals in Tables 4-27 and 4-28.

1.4.5 Risk Communication (Summary):

The risk values calculated in the previous section are used here to place each chemical, by exposure pathway, into four hazard tables:

1) Negligible Risk, Table 4-29
2) Low Risk, Table 4-30
3) Moderate Risk, Table 4-31
4) High Risk, Table 4-32

The criteria used to create and interpret these tables are defined.

Another important part of the risk characterization, presented here in Section 6, risk communication, is a discussion of and analysis of the uncertainties. By definition, uncertainties are inherent in all risk assessments. Due to the nature of risk assessments, and time and budget constraints of this project, there are five primary sources of uncertainty:

1. The risk assessment process itself
2. Toxicity values
3. Exposure analysis assumptions
4. Chemical fate and transport
5. Summing risks

Measures have been taken to minimize the effects of these uncertainties which are presented.

Lastly is a short discussion of the similarities, differences and errors found comparing this Environmental Impact Statement to The Bureau of Land Management EIS-13 Western States, the Forestry Service EIS- Pacific Northwest Region and the Caltrans EIR.

2.0 Hazard Identification

2.1 Introduction

The purpose of the hazard identification section is to:

- Identify all relevant chemicals.
- Review available epidemiology data related to these chemicals.

- Review all laboratory toxicology data for each active ingredient and formulation. Data include:

  * Acute toxicity
  * Sub-chronic/chronic toxicity
  * Reproductive/developmental toxicity
  * Carcinogenic/mutagenic potential

- Review the compound's physical and chemical properties.

- Review the environmental fate of each compound.

The first step in risk assessment, by any method, is hazard identification (or hazard analysis). Twenty-five herbicides were selected by the Washington Department of Transportation as chemicals which might be used to control vegetation on and near roadways in the State. Of the 25 herbicides selected, 23 have a history of usage in at least one district. Two, fluazifop-butyl and oxadiazon, are being evaluated for future use. The 25 active ingredients evaluated quantitatively for systemic or noncancer risks are:

1) 2,4-D
2) 2,4-DP
3) Benefin
4) Bromacil
5) Chlorsulfuron
6) Chlorthal dimethyl
7) Dicamba
8) Dichlobenil
9) Diuron
10) Fluazifop-butyl
11) Fosamine
12) Glyphosate
13) Imazapyr
14) Isoxaben
15) Mecoprop
16) Mefluidide
17) Metsulfuron methyl
18) Napropamide
19) Oryzalin
20) Oxadiazon
21) Picloram
22) Pronamide
23) Sethoxydim
24) Sulfometuron methyl
25) Triclopyr

The six herbicides evaluated for carcinogenic risk are:

1) 2,4-D
2) Bromacil

2.2 Toxicology

Paracelsus, who lived from 1493 - 1541, wrote: "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy." Water and the salt that is put on the table are capable of causing adverse health effects if taken in excessive quantities. The dose, or the amount of a substance that the body absorbs and the duration of exposure, determine whether a compound will be harmful.

In order to evaluate the potential of a chemical to cause adverse health effects one must understand the basic principals of toxicology and how to interpret results of these tests. Since human data are rarely available scientists rely on laboratory animal data to perform this evaluation. The basic types of toxicity studies are acute, subchronic, chronic, carcinogenicity (oncogenicity), reproductive, teratology (developmental) and genotoxic.

The Environmental Protection Agency (EPA) issues guidelines on how to conduct these studies in the interest of good science and in order to standardize them. Animal studies used for registration of pesticides must also conform to rigid laboratory "quality control" guidelines (GLPs) as mandated by the Good Laboratory Practices Act. The EPA is responsible for registering pesticides and dictates which data requirements (or studies) must be met to support registration, according to the particular compounds intended use pattern. Some herbicides have a terrestrial "non-food use" pattern which requires fewer studies to be submitted than a pesticide with a terrestrial "food use" pattern.

2.2.1 Acute Toxicity

An acute test is generally one that evaluates adverse effects from a single administered dose over a period of up to 2 weeks. This study is used to assess worker hazard or hazards from handling the concentrated materials. The most common measure of acute toxicity is the LD_{50} or "Lethal Dose, 50 percent." This is the calculated dose of a chemical which is expected to kill half of the animals tested. The actual value of the LD_{50} is expressed in milligrams of chemical for each kilogram of body weight (mg/kg). Oral and dermal exposure LD_{50}s are usually measured in rats and rabbits, respectively. Acute inhalation studies (usually 4 to 6 hours in duration) are routinely performed with rats and measure an LC_{50} value or "Lethal Concentration, 50 percent." The LC_{50} value is usually expressed as milligrams of chemical per liter or per cubic meter of air.

The LD_{50} (or LC_{50}) value is used to compare and classify toxicity among chemicals. The lower the LD_{50}, the more toxic the chemical. It does not, however, tell you anything about the dose response relationship or the sublethal effects. The following graphs in Figure 4-2 illustrate three different ways to represent the dose response curve. The curve is important in estimating the acute effects of a compound at less than its predicted "LD_{50}".

Figure 4-3 is a probit unit graph which shows the dose response curve for two chemicals, A and B, with the same LD_{50} value, 8 mg/kg. At one-half the LD_{50} (4 mg/kg) 20 percent of the animals given compound A would be expected to die while less than one percent of the animals given compound B would die.

3) Oryzalin
4) Oxadrozon
5) Picloran
6) Pronamide

Due to the lack of toxicity data a qualitative risk analysis was performed on the following compounds and presented in Section 5, risk characterization (these chemicals do not have any pesticidal activity):

1) Inerts
2) Adjuvants
3) Fertilizers

Do these chemicals present an unreasonable health hazard for the public or worker safety in comparison to alternative methods of vegetation control available? To answer this question an indepth analysis of the available toxicology and epidemiology data must be preformed. Ideally results from human studies would provide the best information on which to base a risk assessment. For obvious reasons, human toxicology data are normally not available and epidemiology data are usually qualitative, rendering these data of limited use for a quantitative risk analysis. For most of the herbicides examined in this risk assessment, no human data are available.

In order to register a pesticide in the United States, the Environmental Protection Agency requires a battery of animal testing which usually is comprised of acute, subchronic, chronic, reproduction/developmental, carcinogenicity, mutagenicity, and ecotoxicity (aquatic, avian and wildlife) studies. The number and type of studies required is dictated by the use pattern of each individual chemical. Some of the herbicides are registered for non-food use and the battery of tests required for registration is limited. Toxicity data is even more limited for adjuvants, inert ingredients, carriers and fertilizers since they are not pesticides and are not registered in the same manner.

The hazard identification section begins with an overview of toxicology and summarizes the laboratory toxicity data generated for, and submitted to, regulatory agencies (EPA and CDFA) in support of pesticide registrations. For each chemical, it presents the following:

1) Brief discussions—
   a) Class of herbicide
   b) Physical and chemical properties
   c) Environmental fate
   d) Adverse effects in humans available from literature
   e) Data gaps noted

   and

2) Summaries of laboratory data—
   a) Acute/Subacute
   b) Subchronic/Chronic
   c) Reproduction/Developmental
   d) Carcinogenicity/Oncogenicity
   e) Mutagenicity

2.2.2 Subchronic Studies

An organisms response to a single and overwhelming acute exposure frequently is quite different from the toxic effects produced by repeated lower exposures. An acute toxic exposure to ethanol causes central nervous system depression, while repeated daily exposure to non-toxic levels has been linked to cirrhosis of the liver. Certain types of compounds may accumulate over time if not totally metabolized or eliminated by each successive dose, causing unexpected toxic effects. Subchronic studies are performed on mammalian laboratory animals in order to assess these potential cumulative effects and others by using repeated daily exposure to a chemical.

The compound is given to animals by dietary mix, dermal application, orally (gavage/capsule) or occasionally by inhalation for a duration of 21 days (dermal rabbit studies), 90 days (rodent studies) or 6 months to a year for a dog study. The route is based upon the most critical or likely exposure pathway(s); duration of exposure is based on the expected interval over which accumulation would occur and is dictated by EPA guidelines. At least three dose groups and an untreated control are employed, with the highest dose ideally producing some toxic effects. Observations recorded include body weights, food consumption, systemic toxic signs and mortality. Urinalysis, hematology and blood chemistry measurements are usually taken at the end of the test period for all species and additionally at the beginning and midway for non-rodent species. At termination of the study the gross and microscopic condition of the organs is examined and selected organs are weighed.

Results are compared to control values (and within groups). Significance of the differences observed is then calculated. Systemic toxic effects frequently reported are decreased body weight gain, decreased food consumption, increased liver weights, increased plasma levels of liver enzymes and proteins, microscopic changes in the tissue (cell necrosis) and undesirable constituents in the urine. Subchronic toxicity studies not only characterize the dose response relationship of a test material following repeated administration but also provide data necessary for selection of doses for chronic exposure studies.

For each study a no observed effect level (NOEL) and a lowest observed effect level (LOEL) are identified. The NOEL is the highest dose level in which no dose related effects are observed. If no toxic effects are reported in a study, then the NOEL is said to be equal to or greater than the highest dose tested (HDT). The LOEL is the lowest dose at which the chemical has produced an effect of any kind. If toxic effects are observed in all compound treated groups, the LOEL is said to be less than the lowest dose group tested and no NOEL could be established. It is possible with systemic toxic effects to estimate the point at which the test animals system can no longer tolerate the chemical intrusion and begins to exhibit signs of toxicity. This point is called the toxic threshold and lies between the NOEL and the LOEL. Occasionally, in the following summaries, the terms no observed adverse effect level (NOAEL) and low observed adverse effect level (LOAEL) are used. If a compound tastes bad, all animals receiving the compound may salivate, or may not eat normally. This is a dose related effect but not a systemic toxic effect.

Most of the pesticides registered today are herbicides and, as a product group, have the lowest potential for detrimental effects on humans and animals. The acute oral LD_{50} values for aspirin and table salt are 1000 and 3000 mg/kg, respectively. This makes them more acutely toxic (or just about as acutely toxic) as 20 of the 25 herbicides evaluated in this document. Caffeine has an oral LD_{50} of 192 mg/kg, which makes it more acutely toxic than any of these chemicals reviewed here.

The following table will provide the layperson with the means to compare a rat LD_{50} to lethal human levels:

Oral LD_{50} in mg/kg	Degree of Toxicity	Probable Lethal Dose for 70 kg Man
<0.1	Extremely toxic	A taste
1 - 50	Highly toxic	A teaspoon
50 - 500	Moderately toxic	An ounce
500 - 5000	Slightly toxic	A pint
5000 - 15000	Practically non-toxic	A quart
>15000	Relatively harmless	More than a quart

Source: R.R. Levine. Pharmacology, Drug Actions and Reactions. p. 305. 1973

Other acute studies measure the local nonsystemic effects of exposure. The primary rabbit eye and skin irritation tests measure the local irritation effects and its reversibility, from exposure to compounds. Several different schemes exist which try to categorize a degree of toxicity in accordance with effects observed. This can cause confusion when trying to interpret results reported on Material Safety Data Sheets (MSDS) and summarizing this information here, since different standards may have been used. One well defined scheme was developed by the EPA and defined in 40 CFR 156.10. This federal regulation describes the EPA Toxicity Categories which are assigned to each pesticide product according to the results of each acute toxicity study (Table 4-33). It further mandates what precautionary statements are required on the label by toxicity category and the precise wording of these precautionary statements (Table 4-34). Where judgement was necessary for this review, a comparison of the precautionary statements on the label to the classification system and required label language published by the EPA was made.

Another study that is usually required in the acute battery of tests is the guinea pig sensitization study. There are various different testing methods but they all involve repeated dermal or intradermal exposure resulting in no response or an allergic (sensitization) reaction. Lastly, for chemical classes known to cause nervous system effects (i.e., cholinesterase inhibitors) an acute delayed hen neurotoxicity study is usually required. This test detects not only immediate nervous system disruption, but also the compound's potential to induce latent nervous system damage.

2.2.3 Chronic Toxicity and Oncogenicity Studies

Chronic studies are basically conducted in the same manner as subchronic studies except they are longer and use more animals. The compound is given to animals usually by dietary mix or orally (gavage/capsule) for a duration of two years for rodent studies or one year for a dog study. At least three dose groups and an untreated control are employed. Doses are selected based on the highest dose ideally producing some toxic effects and the low dose group producing no toxic effects. Observations recorded include body weights, food consumption, systemic toxic signs, and mortality. Urinalysis, hematology, and blood chemistry measurements are usually taken at 6-month intervals (beginning with pre-dose samples) and at the end of the test period. Ophthalmological exams are usually made in dog studies before administration of the test substance and prior to completion of the study. Upon termination, the gross and microscopic condition of the organs is examined and selected organs are weighed.

Results are compared to control values (and within groups) and the significance of differences noted are calculated. Systemic toxic effects frequently reported are decreased body weight gain, decreased food consumption, increased liver weights, increased plasma levels of liver enzymes and proteins, microscopic changes in the tissue (cell necrosis or in some cases neoplasia), and undesirable constituents in the urine.

The critical toxicity values (NOEL, LOEL or threshold level) calculated from a chronic study are indicative of the dose response relationship of a chemical in a test species upon repeated chronic or lifetime exposure. The reference dose (or acceptable daily intake) is calculated using the lowest NOEL identified in the data base. This NOEL frequently has been established for a chronic duration toxicity study, but can also be the NOEL established in a reproduction or teratology study.

Oncogenicity studies are performed on rats and mice and are basically the same as a chronic study. The study performed with rats is usually a combined chronic/oncogenicity study. The purpose of an oncogenicity study is to investigate the potential of a compound to cause tumors in animals when these animals are exposed to sublethal doses for a lifetime. Typically, a "background" incidence of tumors exists for each laboratory and strain of species used in testing. The tumors may be malignant (cancerous) or benign (noncancerous). Carcinogenicity refers to the ability to cause malignant tumors at an incidence rate above "background." Responses which indicate carcinogenic potential are 1) presence of tumor types not seen in controls, 2) increase in the incidence of the same tumor types occurring in controls, and 3) development of tumors earlier than in controls.

Emphasis is placed on the length of the study and whether or not a maximum tolerated dose (MTD) is attained when these studies are reviewed for scientific and regulatory purposes. The highest dose tested should not produce severe signs of toxicity or overt lethality, but should be a level the animals can tolerate over their lifetime. However, some toxic effects should be observed at this level so that there is some assurance that a "maximum tolerated dose" is achieved. Animals on an oncogenicity study are exposed to much higher doses of a chemical than would be experienced by humans, in order to exaggerate any potential oncogenic (or serious health) effect. To detect an incidence of cancer as low as 0.01% would represent 20,000 people in a population of 200 million and would be considered unacceptably high. To detect such a low cancer incidence in

experimental animals directly would require a minimum of 30,000 animals. To limit the over use of laboratory animals, there is no choice but to give large doses of a compound to a smaller number of animals and use mathematical models to predict risks at low doses.

Unlike systemic effects, cancer effects are assumed to have no threshold below which no effects occur. In other words, any dose of a carcinogen is assumed to cause an increased incidence of cancer. Using mathematical models, oncogenic potency is derived, or the cancer potency factor (CPF) expressed in $(mg/kg/day)^{-1}$. This is used with the lifetime average daily dose to estimate the extra lifetime cancer risk from exposure to a chemical. Figure 4-4 illustrates the portion of the dose response curve which is resolved by modeling.

2.2.4 Reproduction/Developmental

"The identification of a chemical which is likely to be a developmental or reproductive toxicant in humans is as complex as the identification of likely human carcinogens" (Paustenbach, 1989). Two types of studies are typically performed to evaluate compounds for these effects: 2 (or 3) generation reproduction studies and teratogenicity studies.

The multigeneration rat reproduction study provides information on fertility and fecundity by evaluating gonadal function, estrus cycles, mating behavior, conception, parturition, lactation, weaning, and the growth and development of the offspring. Male and female parental (P_1) animals are given the test substance orally (usually in the diet) prior to mating, through the pregnancy and through the weaning of the offspring (F_1). The chemical is then administered to F_1 offspring during their growth into adulthood, mating, through the pregnancy up until the offspring (F_2) are 21 days old.

Three dose groups and a control are typically employed, with the top dose group set at a level expected to induce some signs of toxicity. Observations recorded include parental weights and food intake, litter and pup weights, physical and behavioral abnormalities of the parents, litters and pups, signs of difficult or prolonged parturition, and mortality. All animals are examined upon termination of their portion of the study and a microscopic exam of selected organs is performed on the P_1 and F_1 parental animals. NOELs and LOELs are identified for reproductive effects as well as parental systemic toxic and/or developmental effects.

"...virtually every substance, when administered at the correct time to the mother, will induce some type of adverse developmental effect on the fetus if the dose is sufficiently high" (Paustenbach, 1989). Compounds which cause adverse effects on the fetus at levels that are easily tolerated by the mother are compounds of concern since in these cases the fetus is more sensitive than the parent. Paustenbach (1989) demonstrates this graphically in Figure 4-5.

Teratology studies are normally performed in two species, the rat and the rabbit, and are intended to determine the potential of a chemical to induce structural and/or other abnormalities to the fetus from exposure of the mother during pregnancy, in particularly during the period of embryonic development. These studies differ from multigeneration

studies in many ways, the most important for our purposes is that of higher dose levels and more detailed examination of the fetus.

Test animals are usually given the test material orally by gavage on days 6 to 15 of pregnancy for the rat and days 6 to 18 for the rabbit (this is the period of organogenesis). At least three dose levels and a control group should be used unless the material is relatively non-toxic then one dose of 1000 mg/kg/day and a control would suffice. The high dose animals must cause some evidence of maternal toxicity and the low dose should not exhibit any toxic effects. Observations are made daily for signs of toxicity, and body weights and food consumption should be recorded at frequent intervals. The study is terminated before birth and all animals are necropsied. The dams are checked for embryonic or fetal death, live fetuses, and the number of corpora lutea. Fetuses are sexed, weighed, and examined for visceral and skeletal anomalies.

NOELs and LOELs are established for maternal teratogenic (birth defects) and developmental effects. The latter includes fetotoxicity and delayed development. As with reproduction studies, a chemical that causes fetal effects at doses obviously toxic to the mother is generally not a compound of concern for the purpose of teratogenic evaluation. However, if fetal effects are observed at levels which are not toxic to the mother and these effects exceed the incidence in "historical" and concurrent controls, then the compound must be considered teratogenic or a developmental toxicant depending on the effects noted.

2.2.5 Genotoxicity (Mutagenicity) Studies

Results from mutagenicity tests are required by the EPA in order to:

1) To detect the capacity of a chemical to alter genetic material in cells.

2) Assess the potential of a chemical to cause heritable genetic alterations in humans.

3) Aid in the carcinogenicity weight-of-evidence assessment when results from a carcinogenicity test are being evaluated. Results from mutagenicity tests may also help select an appropriate risk extrapolation model. Mutagenicity may be functionally be related to carcinogenicity on a cellular level.

4) Provide the Agency with data to be used in the decision to require carcinogenicity testing on a chemical whose use pattern does not mandate submission of this study.

In order to support any pesticide registration the technical material must be tested in a minimum battery of three mutagenicity assays. These provide information on the chemical's ability to cause gene mutation, chromosome aberration and/or induce DNA damage and unscheduled repair. Other categories of assays exist, such as cell transformation, but are used to a lesser extent. The following is a list of commonly performed assays in each of the four categories and a brief description.

Gene Mutation

1) Ames *Salmonella*/microsome, *E. coli* and *B. subtilis* assays — bacteria assays testing for reverse mutations (base-pair substitution and frameshift mutagens).

2) *Saccharomyces cerevisiae* assay — eukaryotic microorganism (fungus) assays, which test for forward and reverse mutations (base-pair substitution and frameshift mutagens).

3) Mouse lymphoma L5178Y, V79 hamster cell HGPRT and Chinese Hamster Ovary (CHO) HGPRT assays — *in vitro*, mammalian cell assays testing for base-pair substitution and frameshift mutagens.

4) *Drosophila melanogaster* sex-linked recessive assay — insect assay using *Drosophila* tests for all classes of point mutations in germ cells.

5) Mouse specific locus assay — *in vivo*, mouse assay measures visible autosomal recessive mutations.

Chromosome Aberration

1) *Aspergillus nidulans* assay — eukaryotic microorganism (fungus) assay can detect chromosomal nondisjunction.

2) *Neurospora crassa* assay — eukaryotic microorganism (fungus) assay can detect multilocus chromosome deletions.

3) *In vitro* cytogenetics — mammalian cell assay using rat, CHO or human lymphocytes, detects chromosome breakage.

4) *Drosophila melanogaster* assays — insect assay using *Drosophila*, detects heritable chromosome effects, sex-chromosome loss and heritable translocations.

5) Bone marrow micronucleus assay — *in vivo*, mammalian assays using mice or rats, detects fragments of chromatin which may occur due to chromosome breakage or spindle dysfunction.

6) *In vivo* cytogenetics assay — mammalian assays using rat and mice bone marrow, detects structural or numerical abnormalities of the chromosome.

7) Sister chromatid exchange — *in vivo* and *in vitro*, mammalian assays which can detect structural chromosome aberrations.

8) Dominant lethal assay — *in vivo*, mammalian assay, detects chromosome alterations manifested as a failure of the zygote to implant in the uterine wall or to survive to midpregnancy. Cytogenetic analysis can be done to confirm the mechanism.

9) Heritable translocation assay — *in vivo*, mammalian assay using mice which measures chromosomal translocations produced in germinal tissue that are capable of being transmitted to the next generation. Can also be performed using Drosophila.

10) Sex-chromosome loss assay — insect assay using *Drosophila*, detects sex chromosome nondisjunction.

DNA Damage and Repair

1) Differential lethality assays — bacteria assays using *E. coli*, *S. typhimurium* or *B. subtilis* measure the ability to repair DNA lesions due to an agents impact on the DNA.

2) Mitotic recombination assays — eukaryotic microorganisms (fungi) using *S. cerevisiae* or *A. nidulans* which measure DNA exchanges between segments of homologous chromatids.

3) Unscheduled DNA synthesis — *in vitro*, mammalian cell assay using primary rat hepatocytes, detects DNA repair in nonreplicating DNA.

4) Sister chromatid exchange — *in vivo and in vitro*, mammalian cell assay measures SCEs (in the DNA) caused by chromosome breaks and repair and is indicative of an effect on the DNA.

Oncogenic Transformation

1) Cell transformation assay — *in vitro*, mammalian cell assay using BALB/c-3T3 or C3H10T½ cells measures a chemicals ability to transform a normal cell to one with neoplastic properties.

When assessing the potential of a chemical to cause human heritable germ cell mutations a weight-of-evidence approach is utilized. One reason for this is that mutagenic assays are subject to both false positive and false negative results. Summarized briefly, more weight is placed on human data and the least on bacteria data.

Least	Plant and prokaryotic (bacteria) systems.
Least	Lower eukaryotic microorganisms and insect systems.
Least	Mammalian in vitro systems.
Least	Mammalian in vivo systems.
More	Human in vivo data

(Brusick, 1987)

2.3 Toxicology Summaries of the 25 Herbicides

2.3.1 2,4-D

2,4-D is a systemic postemergence chlorinated phenoxy herbicide which is absorbed by the foliage and stems of plants and translocated. It interferes with cell wall development, thus is acting as a plant growth regulator. The technical material comes in various physical solid forms (powder, flakes, and crystalline powder). It is white to tan with a phenolic or no odor. The acid has a low tendency towards volatilization, is unlikely to bioaccumulate based on a low K_{ow}, and would be expected to migrate through soils and sediment based on a low K_{oc}. 2,4-D has only slight solubility in water, is soluble in most organic solvents, and insoluble in benzene and petroleum oils. It is mobile to highly mobile in five soil types. However, the soil leaching potential is limited due to the rapid rate of degradation (USEPA, 1988(d)).

Human Data

The Washington Department of Transportation did not perform extensive literature searches, but instead used available information (i.e., from the USFS FEIS, USDI Bureau of Land Management DEIS, Jones & Stokes Caltrans DEIR, etc.) for the generation of this document. The Bureau of Land Management did an extensive literature search covering the effects of 2,4-D on humans. In order to provide these references and insure that the information attained from them is accurately relayed, the 2,4-D epidemiology summaries published in the USDI (1991) BLM EIS are reproduced here:

> "Peripheral neuropathy has been reported to result in humans from dermal exposure to 2,4-D. In one study, Goldstein, et al., (1959) reported three cases in agricultural workers following dermal exposure to 2,4-D. The neuropathy was characterized by progressive numbness, aching of the extremities, muscular fasciculation, enervation of muscles, and decreased conduction velocity in the ulnar nerve. The condition may be partially or totally reversible, depending on the dose level and the individual exposed (Todd, 1962; Berkeley and Magce, 1963; Wallis, et al., 1970). In one patient, only partial recovery was reported, even after 3 years of treatment (Goldstein, et al., 1959). His estimated exposure was 60 cubic centimeters of a 10-percent ester solution, approximately 60 mg/kg."
>
> "Several epidemiological investigations have been conducted to examine the link between human phenoxyacid herbicide exposure and cancer. In the mid and late 1970s, Hardell and colleagues (Hardell & Sandstrom, 1979; Eriksson, et al., 1981; Hardell, et al., 1981) conducted a series of case-control studies in rural Sweden. These studies found a significant increase of fivefold to sixfold in the relative risk of soft-tissue carcinomas, Hodgkin's disease, and non-Hodgkin's lymphoma among farmers using various herbicides. However, because of selection bias, observation bias, and uncontrolled confounding variables many experts have questioned the validity of these studies (Colton, 1986)."
>
> "In a Danish cohort study of workers manufacturing phenoxy herbicides, a significant increase in soft-tissue sarcoma (STS) was found, but no similar

increase in malignant lymphoma (Lynge, 1985). The cancer risk among persons manufacturing and packaging phenoxy herbicides was equivalent to the cancer risk in the Danish population (Lynge, 1985). A recent Swedish cohort study found no significantly increased relative risk (0.9) of STS in Swedish agricultural and forestry workers exposed to phenoxy acid herbicides (Wiklund and Holm, 1986). In addition, a case-control study conducted in New Zealand by Smith, et al., (1984) was negative for soft-tissue carcinomas, showing an estimated relative risk of 1.3."

"Recently, Hoar, et al., (1986) completed a case-control study in Kansas examining the risk of lymphoma and STS in men from agricultural herbicide exposure. The study found no association between exposure and soft-tissue carcinoma or Hodgkin's disease, but a significant association was observed for non-Hodgkin's lymphoma (NHL) and phenoxyacetic acid herbicide exposure, especially 2,4-dichlorophenoxy-acetic acid exposure. In addition, individuals exposed to herbicides for more than 20 days per year had a sixfold increase in NHL. Nonetheless, this study suffers from the same inherent limitations as the other case-control studies, mainly that it relies on the recall ability of the subject or their next of kin to determine their exposure status. If their recall is faulty, misclassification occurs. It is especially difficult to assess exposure-disease relationships in these types of epidemiological studies (NRC, 1986). For example, it is possible to have common exposures to other carcinogenic agents or other factors that result in disease but are not discovered in the interview and confound the results. Thus, uncontrolled confounding factors in observational epidemiological studies can be particularly troublesome in interpreting the results. However, the apparent dose-response relationship observed in the Hoar, et al., (1986) study for NHL is of public health concern and needs further examination. It should be noted that at least two additional studies are now underway that should be helpful in assessing risk to humans from the use of 2,4-D and other phenoxy herbicides (Colton, 1986)."

"In a recent review of the Hoar, et al., (1986) study conducted for the EPA, Brian MacMahon M.D., Ph.D., of the Havard School of Public Health, concluded:"

"In my opinion the weight-of-evidence does not support the conclusion that there is an association between exposure to 2,4-D and NHL. It is axiomatic that except when relative risks are very high--and sometimes even then--no single study will establish an association between an exposure and an outcome. The acceptance of an association depends on a number of studies showing consistent results across populations and across different epidemiological methods. The study of Hoar, et al., (1986) is a strong study--strong enough on its own to establish a hypothesis of relationship of exposure to 2,4-D with some small proportion of cases of NHL--a hypothesis that clearly deserves attempts at refutation or support in other populations. When one attempts to place the results of this study among the results of those published previously, the picture becomes very confusing--much more than if Hoar, et al., had been the only study published. Taken as a whole, I believe that the weight-of-evidence indicates that an association between 2,4-D and NHL remains a hypothesis that is still to be

tested. I am unwilling to speculate as to whether 2,4-D causes NHL (or some cases of NHL) until the evidence is clear that there is an association between them (MacMahon, 1986)."

"In a cohort study of forestry workers in Ontario, Canada, no evidence of increased mortality risk or cancer risk was observed in forestry workers after 15 or more years of exposure to phenoxy herbicide use (Green, 1986). The forestry workers had been employed by Ontario Hydro from 1950 through 1982."

"The Canadian Centre for Toxicology (1987) has reviewed recent case-control studies of phenoxy herbicides. A study conducted in western Washington State reported no overall increased risk associated with the past occupational exposure to phenoxy herbicides for STS or NHL (Woods, et al., 1987, as cited in Canadian Centre for Toxicology 1987). There was an elevated risk of NHL for men who had been farmers, forestry herbicide applicators, and those potentially exposed to phenoxy herbicides for 15 years or more during the 15-year period before cancer diagnosis. However, exposure to 2,4-D was not singled out."

"Another study reviewed by the Canadian Centre for Toxicology (1987) is being conducted by the National Cancer Institute in Iowa and Minnesota. Preliminary results indicate no overall increased risk for NHL associated with living or working on a farm and a slightly elevated (but not significant) risk in persons using 2,4-D (Cantor and Blair, 1986, as cited in Canadian Centre for Toxicology 1987). The investigators have decided to recontact subjects to gather more information on the number of days per year of pesticide use."

"After reviewing the 2,4-D epidemiology studies, the Canadian Centre for Toxicology (1987) concluded that there is limited evidence of carcinogenicity in humans from exposure to phenoxy herbicides, and there is inadequate evidence to classify 2,4-D as a carcinogen." (End of USDI BLM EIS discussion).

To date, whether or not 2,4-D is a carcinogen remains a controversy. The OPP has classified it as a Group "D" oncogen (not classifiable as to human carcinogenicity). The EPA is requiring additional data for further evaluation of 2,4-D.

Laboratory Generated Data

Acute Toxicity - 2,4-D exists in many forms depending upon its intended use. For the purpose of this discussion, the "acid" is considered to be the active ingredient, unless noted otherwise. 2,4-D is slightly to moderately toxic to laboratory animals. The acute oral LD_{50} is 639 and 764 mg/kg for male and female rats and the acute rat inhalation LC_{50} is 1.79 mg/l. Reported rabbit dermal LD_{50} values range from 1,400 to 3,980 mg/kg and a rat dermal LD_{50} was reported to be 1,500 mg/kg. It is negative for dermal sensitization in guinea pig studies. The acute toxicity of the manufacturing-use-product, diethanolamine salt, and two commonly used formulations, butoxyethyl ester and isopropyl ester, are presented in the tables at the end of this section. Primary eye and skin irritation were identified as data gaps for 2,4-D acid by the US EPA in 1988. (USEPA, 1988(d); USDI, 1991; USFS, 1988)

Subchronic Toxicity - 2,4-D was fed to rats for 91 days at 0, 1.0, 5.0, 15.0 or 45.0 mg/kg/day resulting in a NOEL of 1.0 mg/kg/day. Effects seen at the LOEL, 5.0 mg/kg/day, and higher were decreased hemoglobin, hematocrit and red blood cell levels (USEPA, 1990).

In a 91 day feeding study 2,4-D was given to mice at 0, 5.0, 15.0, 45.0 or 90.0 mg/kg/day. No NOEL was established; effects observed included increased adrenal weights in females at 5.0 mg/kg/day; altered organ weights and hematologic effects at 15 mg/kg/day and up (USEPA, 1990).

A 21-day dermal study was identified as a data gap (USEPA, 1988(d)).

Reproduction/Developmental Toxicity - Two rat reproduction studies have been performed using 2,4-D and have resulted in similar NOELs of 20 mg/kg/day and 500 ppm (25 mg/kg/day). Both studies indicate a possible adverse reproductive effect. The earlier study noted reduced pup survival and weanling weight at the top dose of 1500 ppm (75 mg/kg/day, LOEL) with no parental toxicity reported. The second study used the following levels 0, 5, 20 and 80 mg/kg/day. The top dose was discontinued due to severe prenatal and neonatal losses; some maternal body weight loss was also noted at this level (CDFA, 1989(b)). The EPA reports a NOEL for this reproduction study as 5 mg/kg/day, due to decreased maternal body weight gain and pup weights at 20 mg/kg/day (USEPA, 1988(d)).

In a teratology study rats were given 0, 8, 25 or 75 mg 2,4-D/kg/day resulting in delayed ossification at 75 mg/kg/day and apparently a questionable decrease in maternal body weight gains. The CDFA places the maternal and developmental NOEL at 25 mg/kg/day (CDFA, 1989(b)). The EPA placed the maternal NOEL at >75 mg/kg/day and the developmental NOEL at 25 mg/kg/day (USEPA, 1988(d)).

The rabbit teratology study has been identified as a data gap (USEPA, 1988(d)).

Chronic Toxicity/Oncogenicity - 2,4-D was fed to rats for 2 years at the levels of 0, 1, 5, 15 or 45 mg/kg/day resulting a NOEL of 1 mg/kg/day, based on kidney tubular cell pigmentation at 5 mg/kg/day and above. This NOEL was used by the US EPA to calculate the RfD value, using a 300 fold uncertainty factor due to the absence of a chronic dog study. There was also an increased incidence of astrocytomas (benign brain tumor) in high dose males. Several evaluations of the data could not provide strong statistical evidence that 2,4-D was oncogenic in the rat. The US EPA decided that a maximum tolerated dose (MTD) was not reached and that the rat oncogenicity study needs to be repeated. The EPA presently considers 2,4-D to be a Group "D" (formerly Group "C") oncogen (not classifiable as to human carcinogenicity). This risk assessment takes a conservative approach and considers 2,4-D to be a potential carcinogen using the cancer potency factor calculated by the US EPA, Office of Pesticide Programs (OPP) of 1.9×10^{-2} (CDFA, 1989(b)).

A chronic dog study has been identified as a data gap (USEPA, 1988(d)).

In a 2 year oncogenicity study, mice were fed 0, 1, 15 or 45 mg 2,4-D/kg/day resulting in a systemic NOEL of 1 mg/kg/day, based on reduction of cytoplasmic vacuoles in

males at 15 and 45 mg/kg/day. No oncogenic effects were reported. However, the EPA determined that an MTD was not reached and listed the mouse oncogenicity study as a data gap (CDFA, 1989(b)).

Mutagenicity - 2,4-D was negative for gene mutation when tested in the Ames assay. At the time of this report, results from no other validated genotoxicity studies were available from the US EPA or CDFA. The EPA has listed mutagenicity studies as a data gap. The USDI (1991) reports that 2,4-D tested negative in assays using *B. subtilis, E. coli, S. typhimurium, S. cerevisiae,* and Bacteriophage T4. Insect chromosomal studies with drosophila, cell culture studies and mammalian chromosomal studies were all predominately negative. 2,4-D also tested positive in the following assays; unscheduled DNA synthesis in human fibroblasts, arabain resistance in hamster lung cells, recessive lethal assay in drosophila, chromosome aberration in mouse bone marrow and sister chromatid exchange in human lymphocytes. They concluded from the information available that 2,4-D may be a weak mutagen, but that it is not likely to present a risk of human heritable mutations at the exposure levels that are predicted to occur in their vegetation management program.

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Diethanolamine salt	1,605- >2,000 (III)	>2,000 (III)	severe (I)	non-irritating (IV)	>3.8 (III)
Butoxyethyl ester	866 (III)	>2,000 (III)	mild irritant (III)	slight (III)	>4.6 (III)
Isopropyl ester	440-640 (II)	>2,000 (III)	slight (III)	non-irritating (IV)	>4.97 (III)
2,4-D Amine #4	370 (II)	1500 (rat) (II)	severe (I)	slight (III)*	(III)+
Crossbow	2,398-2,589 (III)	>2,000 (III)	moderate (II)*	slight (III)*	5 (III)
Envy 2,4-D	460 (II)	570 (II)	severe (I)	very slight (IV)	>2.1 (III)
Formula 40	1,391-2,525 (III)	>5,000 (III)	corrosive (I)	severe (I)	(IV)+
Tordon RTU	>5,000 (IV)	>3,980 (III)	moderate (II)	slight (III)*	(III)+
Tordon 101	2,598 (III)	>2,000 (III)	moderate (II)	moderate (II)*	(IV)+
Trimec Classic	1,500-2240 (III)	>2,010 (III)	corrosive (I)	moderate (II)	>2 (III)
Visko-Rhap LVE2D	900-1,200 (III)	>2,000 (III)	mild (III)	mild (III)	>5,000 (mg/kg)
Visko-Rhap A-3D	(III)+	(III)+	severe (I)t	(III)+	(III)+

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Weedmaster	>5,000 (IV)	>20,000 (IV)	minimal (III)	minimal (III)	>20.3 (IV)
Weedone 170	2,000 (III)	>2,000 (III)	minimal (III)	non irritant (IV)	140 (IV)
Weedar 64	1,150 (III)	1,530 (II)	severe (I)	minimal (IV)	(III)+
Weedone LV4	1,375 (III)	>2,000 (III)	mild (III)	mild (III)	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

t Description of irritation effects contradicts label language, "CAUTION" vs "May cause irreversible eye damage".

\+ No information available. Determination made according to label language.

2.3.2 2,4-DP

2,4-DP is a systemic postemergence chlorinated phenoxy herbicide, which is absorbed by the foliage and stems of plants and translocated. It interferes with cell wall development thus acting as a growth regulator hormone. The technical material is a white or tan solid with a slight phenolic odor. It is only slightly soluble in water but highly soluble in acetone, benzene, carbon tetrachloride, diesel oil and kerosene. The material is stable up to its melting point and has a negligible vapor pressure. It is unlikely to bioaccumulate based on a low K_{ow} and as a phenoxy herbicide has the potential to move through soil (limited due to the rapid rate of degradation). Concern about possible groundwater contamination exists for the family of 2,4-D compounds (2,4-D, 2,4-DB and 2,4-DP) due to mishandling at mixer/loader and disposal sites, US EPA (1988(e)).

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to 2,4-DP exposure.

Laboratory Generated Data

Acute Toxicity - 2,4-DP is slightly toxic with acute oral LD_{50}s of 500-620 mg/kg for mice and 500-700 mg/kg for rats. Acute dermal and inhalation, skin and eye irritation, and dermal sensitization studies have been identified as data gaps (USEPA, 1988(e)).

Subchronic Toxicity - Only supplementary data exists. The requirement for a subchronic rat study was waived due to the existence of an acceptable rat chronic study. A subchronic non-rodent study has been identified as a data gap (USEPA, 1988(e)).

Reproduction/Developmental Toxicity - A 3-generation reproduction study exists but was found to be supplementary, compound purity was not specified. The parental LOEL was 2000 ppm based on decreased body weight and food consumption. The NOEL was 1000

ppm, or approximately 50 mg/kg/day. A new study is not required for the registered use patterns (USEPA, 1988(e)).

A rat and rabbit teratology study were found to be unacceptable. The EPA has identified the rabbit teratology study as a data gap (USEPA, 1988(e)).

Chronic Toxicity/Oncogenicity - A rat chronic feeding study exists. Fisher 344 rats were maintained on 100, 300, 1000 and 3000 ppm for two years. Decreases in body weight and food efficiency, anemia, and kidney and liver effects were noted at the top dose. The LOEL was 300 ppm based on decreased urine specific gravity/protein and the NOEL was 100 ppm, or 4 mg/kg/day which was used to calculate the RfD (USEPA, 1988(e)).

A rat and mouse oncogenicity study were performed but found by the Agency to be invalid and supplementary respectively. No tumors were observed in the mouse study. A NOEL could not be identified, the LOEL for toxic effects was 25 mg/kg/day. The rat study could not be evaluated. A chronic/oncogenicity study is not required to support the registered use pattern for 2,4-DP, therefore this is not a data gap (USEPA, 1988(e)).

Mutagenicity - 2,4-DP was positive for gene mutation and DNA damage when tested in *S. cerevisiae* and *E. coli*. A mouse micronucleus and Ames assay were performed but found to be unacceptable. The US EPA has listed these studies as data gaps (USEPA, 1988(e)).

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Weedone 170	2,000 (III)	>2,000 (III)	minimal (III)	non-irritating (IV)	140 (IV)

2.3.3 Benefin

Benefin is a selective preemergence herbicide of the dinitroaniline family. It will not control established weeds but inhibits root development of emerging seedlings. The technical material is a yellow-orange crystalline solid. Benefin has a mild tendency to volatilize (based on the Henry's law constant) and bioaccumulate (based on a moderately high K_{ow} and Bioconcentration Factor). It would not be expected to migrate through the soil based on results of laboratory studies. Benefin is practically non-soluble in water (0.1 ppm) and readily soluble in organic solvents such as acetone and xylene.

Human Data

Benefin may cause skin sensitization reactions in sensitive individuals (Farm Chemicals Handbook, 1991).

Laboratory Generated Data

Acute Toxicity - Benefin is sightly toxic orally with LD_{50}s greater than 5,000 and 10,000 mg/kg for mice and rats, respectively. It is practically non-toxic orally to dogs and rabbits with LD_{50} values greater than 20,000 mg/kg. The low toxicity is supported by

an acute dermal LD_{50} of greater than 5,000 mg/kg in rabbits and an acute inhalation LC_{50} greater than 2.16 mg/l in rats (4 hour exposure). (Values reported are probably the highest dose tested and no conclusions should be drawn about species sensitivity). Benefin is irritating to the eyes and slightly irritating to the skin of rabbits. Dermal sensitization is identified as a data gap. (Hartley and Kidd, 1987; USDHHS, 1980; Melichar, 1991)

Subchronic Toxicity - A 90 day feeding study in rats was performed which had a NOEL of 5,000 ppm (250 mg/kg/day). A subchronic NOEL for dogs was established to be 12.5 mg/kg/day. Further details were not provided. (Melichar, 1991)

Reproduction/Developmental Toxicity - The potential of benefin to produce developmental effects was examined in rats and rabbits and found not to be teratogenic. A NOEL of 100 mg/kg/day was established for rabbits. Some reproductive problems were evident in rats at approximately 250 mg/kg/day, but not at 50 mg/kg/day (USEPA, 1990).

The compound was reported by DowElanco not to be a developmental toxin in rats but details of the study were not provided. (Melichar, 1991)

Chronic Toxicity/Oncogenicity - In addition to data collected from published and government sources, DowElanco provided information regarding chronic toxicity and oncogenicity of benefin. A two year rat feeding study established a NOEL of 1,000 ppm (50 mg/kg/day) and with no tumor formation. A chronic dog study was reported to have a NOEL of 5 mg/kg/day. Negative results were observed in a mouse oncogenicity study. (Melichar, 1991)

The EPA has set the RfD value using a no adverse effect level (NOAEL) of 25 mg/kg/day established for a chronic dog bioassay. The level was associated with a slight depression of erythrocyte counts with no reduction in blood hemoglobin concentrations or hematocrit. The low adverse effect level (LOAEL) was set at 125 mg/kg/day due to increased liver weights and depressed hemoglobin concentrations and hematocrit. (USEPA, 1990)

Mutagenicity - Published and government sources of information regarding the results of individual mutagenicity studies were not available. DowElanco provided a statement concerning the mutagenic potential of benefin, concluding that the compound was not mutagenic.

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
XL-2G	3,750 (III)	>2,000 (III)	irritant (III)*	slight (III)	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.4 Bromacil

Bromacil is a nonselective postenurgenic systemic herbicide of the uracil family. It is absorbed by the roots and inhibits photosynthesis within the chloroplasts. The technical material is a crystalline solid. Bromacil has a slight tendency toward volatilization and is unlikely to bioaccumumulate, based on a low Henry's law constant and low K_{ow}, laboratory studies indiate that it has a tendency to migrate through the soil and is relatively persistent. Bromacil has been detected in the groundwater in one county (Franklin) in Washington, probably due to agricultural use (Erickson and Norton, 1990). It is slightly soluble in water and moderately soluble in acetone.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to bromacil exposure.

Laboratory Generated Data

Acute Toxicity - Bromacil is very slightly to slightly toxic to rats with oral LD_{50} values ranging from 3,998 to 5,200 mg/kg, slightly toxic dermally to rabbits with an LD_{50} of greater than 2,000 mg/kg and practically non-toxic to rats via the inhalation route with an LD_{50} of greater than 57.6 mg/l (one hour). The technical material is a mild eye irritant and very slight skin irritant when tested in rabbits and did not cause dermal sensitization in guinea pigs or rabbits. (Jones & Stokes, 1991; USDI, 1991)

Subchronic Toxicity - A 90 day rat study was performed with 80% bromacil wettable powder in the diet at the following levels: 0, 25 and 125 mg/kg/day (the high dose was raised to 250 ppm on day 42). The NOEL was established at 25 mg/kg/day. High dose animals had decreased red blood cell counts, enlarged liver cells and increased thyroid activity (Jones & Stokes, 1991). A non-rodent subchronic toxicity study was not found in the available literature. This was not identified by the EPA as a data gap, probably due to the existence of a chronic dog study.

Reproduction/Developmental Toxicity - There were no developmental effects in rats given 75 mg/kg/day (NOEL), the maternal NOEL was 20 mg/kg/day. A second teratology study was performed in which rats were exposed to bromacil via the inhalation route at levels of 0, 38, 78, or 165 mg/m^3 for 1-3 hours/day. No adverse effects were noted. The developmental and maternal NOEL was greater than 165 mg/m^3. No adverse developmental effects were observed in a rabbit teratology study and the developmental and maternal NOEL was established at 100 mg/kg/day. (CDFA, 1991)

No adverse reproductive effects were observed in a 3 generation rat reproduction study (NOEL = 12.5 mg/kg) which was accepted by the EPA but rejected by the California Department of Food and Agriculture (CDFA) due to an inadequate number of animals tested. A new study is being conducted to support California registration. (CDFA, 1991)

Chronic Toxicity/Oncogenicity - Technical bromacil was administered to rats in the diet for two years at levels of 0, 50, 250 or 1250 ppm in order to determine chronic and oncogenic effects. Decreased body weight was observed down to 250 ppm (the LOEL)

which resulted in the establishment of a 50 ppm (or 2.5 mg/kg/day) NOEL. No Agency RfD was available. 50 ppm is the lowest NOEL for bromacil and was used to calculate the RfD. There was no treatment related oncogenicity noted. (CDFA, 1991)

Dogs were fed 0, 50, 250 or 1250 ppm bromacil for two years. Loss of body weight was seen at the top dose. The NOEL was 250 ppm or 6.25 mg/kg/day. (CDFA, 1991)

An oncogenicity study was conducted on mice which were maintained for 18 months on 0, 250, 1250, or 5000 ppm bromacil in the diet. An increased incidence of liver adenomas and carcinomas was observed in high dose males (CDFA, 1991). The EPA derived a cancer potency factor of 3.8×10^{-3} (USEPA, 1991(b)) for bromacil and assigned it to weight-of-evidence classification C, possible human carcinogen (Jones & Stokes, 1991).

Mutagenicity - Bromacil tested negative for gene mutation in the Ames assay and the CHO/HPRT assay but positive in the mouse lymphoma and Drosophila sex-linked recessive lethal assays. It was positive for chromosome aberration in human lymphocytes (*in vitro*) and negative for chromosome aberration in the mouse bone marrow micronucleus assay. Results of a sister chromatid exchange assay (in cultured chinese hamster ovary cells) and an *in vitro* unscheduled DNA synthesis (in rat primary hepatocytes) assay were negative for DNA damage. (CDFA, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Krovar I DF	>2,500 (III)	>2,000 (III)	mild (III)	irritant (III)*	(IV)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.5 Chlorsulfuron

Chlorsulfuron is a selective pre- and post-emergence systemic herbicide of the urea and triazine families. It is absorbed by broadleaf weeds and inhibits valine and isoleucine biosynthesis, thus inhibiting cell division and growth. The technical material is an odorless white crystalline solid with a high melting point. It has a low tendency toward volatilization and bioaccumulation, based on the Henry's law constant and K_{ow}, respectively. Laboratory data indicate that chlorsulfuron is not persistent, is weakly absorbed, and has a tendency to leach and migrate through the soil. Chlorsulfuron is slightly to non-soluble in water, moderately soluble in acetone and acetonitrile.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to chlorsulfuron exposure.

Laboratory Generated Data

Acute Toxicity - Chlorsulfuron is slightly toxic to laboratory animals. The acute oral LD_{50} in rats was 2,341 and 3,053 mg/kg, female and male respectively, the acute inhalation LC_{50} is reported to be greater than 5.9 mg/l (4 hour exposure). The low toxicity of chlorsulfuron is supported by an acute dermal LD_{50} in rabbits of greater than 2,000 mg/kg. The Telar material safety data sheet list chlorsulfuron as a mild eye irritant and not a skin irritant or sensitizer. (DuPont, 1989(b); USDI, 1989)

Subchronic Toxicity - Two rodent 90 day feeding studies were performed. Rats fed 500 ppm (25 mg/kg/day) exhibited decreased plasma creatinine levels and increased hematocrit. The NOEL was established at 100 ppm (5 mg/kg/day). Mice fed 650 mg/kg/day exhibited changes in plasma and blood parameters. The NOEL was set at 365 mg/kg/day. (USEPA, 1990; USDI, 1991)

Reproduction/Developmental Toxicity - In a three generation reproduction study rats were fed 0, 100, 500 or 2500 ppm chlorsulfuron per day. High dose animals exhibited decreased body weight establishing the maternal NOEL at 500 ppm (25 mg/kg/day). Decreased fertility indices were observed at the high dose establishing the reproductive NOEL at 500 ppm. (CDFA, 1989(c); USEPA, 1990)

Two teratology studies were reported in the literature, one each on rats and rabbits. The maternal and developmental NOEL in rats was established by the EPA at the highest dose tested, 2500 ppm (125 mg/kg/day). The maternal and developmental NOEL in the rabbit study was 25 mg/kg/day. Decreased maternal body weight gain, increased resorption and decreased viability were observed at the highest dose tested, 75 mg/kg/day. (CDFA, 1989(c); USEPA, 1990)

Chronic Toxicity/Oncogenicity - Chlorsulfuron was fed to rats at levels of 0, 100, 500 or 2500 ppm for two years in a combined chronic, oncogenicity and reproduction study. Mild to moderate reduction in body weights and weight gains were observed in males at the 500 and 2500 ppm levels. The NOEL was established at 100 ppm (5 mg/kg/day). This value was used by the EPA to calculate a RfD of $5x10^{-2}$ mg/kg/day. No oncogenic effects were observed. (USEPA, 1990)

Dogs fed 95% chlorsulfuron 0, 100, 500 or 2500 ppm for 6 months exhibited no adverse effects, establishing the NOEL at the highest dose tested (62.5 mg/kg/day). (CDFA, 1988(c); USEPA, 1990)

No oncogenic effects were reported in an 18 month mouse feeding study. Decreased body weights and food consumption were observed at the highest dose tested, 5000 ppm, establishing the chronic NOEL at 500 ppm (75 mg/kg/day). (CDFA, 1989(c); USEPA, 1990)

Mutagenicity - Chlorsulfuron was tested in a variety of *in vitro* assays. Results indicate that it does not cause gene mutation (Ames assay), chromosome aberration (cytogenetic assay in CHO cells) or DNA damage (DNA repair assay in rat hepatocytes). (CDFA, 1989(c))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Telar	2,341-3,053 (III)	>2,000 (III)	mild (III)	non-irritating (IV)	(III)*

*No information available. Determination made according to label language.

2.3.6 Chlorthal dimethyl

Chlorthal dimethyl is a selective preemergence herbicide of the phthalic family. It is a non-systemic herbicide and acts by killing germinating seeds. The technical material is a colorless crystalline solid. Chlorthal dimethyl has a slight tendency towards volatilization and bioaccumulation (based on the Henry's law constant and K_{ow}) and is not relatively persistent in the environment. Data regarding the leaching potential of chlorthal dimethyl were not available. However, due to its insolubility in water, high K_{ow} and rapid rate of degradation the likelihood that it would migrate through the soil is remote.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to chlorthal dimethyl exposure.

Laboratory Generated Data

Acute Toxicity - The material safety data sheets for two different formulations of chlorthal dimethyl report it to be very slightly to slightly toxic with oral rat LD_{50}s of 3,000 and 10,000 mg/kg/day. This low toxicity is supported by a rat inhalation LC_{50} of greater than 5.7 mg/l (4 hour exposure) and a rabbit dermal LD_{50} of 10,000 mg/kg. The technical material is reported to be a mild eye irritant and not irritating to the skin (Hartley and Kidd, 1987). Results of a dermal sensitization study were not located in the literature. If this study has not been submitted to the EPA it could be considered a data gap.

Subchronic Toxicity - One rat 90 day feeding study was reported in the literature. The NOEL was reported to be 10,000 ppm (500 mg/kg/day), the highest dose tested (USEPA, 1990).

Reproduction/Developmental Toxicity - A rat reproduction study was submitted to the EPA but was determined to be inadequate to support chlorthal dimethyl registration. It is listed as a data gap. The NOEL in the existing study and the highest dose tested is 10,000 ppm. (USEPA, 1990)

An acceptable rat teratology study exists which establishes the maternal and developmental NOEL equal to 2,000 mg/kg/day, the highest dose tested (USEPA, 1990).

A rabbit teratology study is identified by the EPA as a data gap (USEPA, 1990).

Chronic Toxicity/Oncogenicity - A rat chronic/oncogenicity study was submitted to the EPA and determined to be inadequate to support registration (classified as supplementary data). They have identified the rat chronic study to be a data gap but have used the existing study to calculate the RfD value. Rats were maintained on 0, 100, 1000 or 10,000 ppm chlorthal dimethyl in the diet for two years. Higher kidney weights in males and relative adrenal weights in females were observed at the high dose, establishing the NOEL equal to 1,000 ppm or 50 mg/kg/day. This value was used by the EPA to calculate an RfD. No oncogenic effects were reported. (USEPA, 1990)

An acceptable two year dog feeding study establishes a NOEL equal to 10,000 ppm (250 mg/kg/day), the highest dose tested. (USEPA, 1990)

Mutagenicity - No information on the mutagenic potential was located in the available literature.

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Dacthal 75W	>10,000 (IV)	>10,000 (III)	Slight (III)	non-irritating (IV)	>5.7 (III)

2.3.7 Dicamba

Dicamba is a selective postemergence systemic herbicide of the benzoic acid family. It is absorbed by the roots and leaves of broadleaf weeds and brush and acts as a growth regulator. The technical material is a brown crystalline solid. Dicamba has a low tendency towards volatilization and studies indicate that it will not bioaccumulate (based on the Henry's law constant and K_{ow}). The compound readily leaches in moist soils and would tend to migrate. Dicamba has not been detected in groundwater in California (Jones and Stokes, 1991). It is soluble in water, ethanol, acetone and other ketones and not relatively persistent.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to dicamba exposure.

Laboratory Generated Data

Acute Toxicity - Dicamba is slightly toxic to laboratory animals with a rat acute oral LD_{50} of 757 to 1,701 mg/kg. The relatively low toxicity is supported by a rat inhalation LC_{50} of 9.6 mg/l and an acute dermal LD_{50} of greater than 2,000 mg/kg. The technical material caused some transient low grade eye irritation and mild skin irritation. Dicamba is a moderate skin sensitizer in rabbits and guinea pigs. (Sandoz, 1992)

Subchronic Toxicity - One 90 day rat feeding study was reported in the literature. Effects observed at 10,000 ppm (500 mg/kg/day) include decreased body weight and food consumption and reduced cytoplasmic vacuolation of the hepatocytes. The NOEL was established at 5,000 ppm or 250 mg/kg/day. (USEPA, 1990; Sandoz, 1992)

A subchronic study in a nonrodent species apparently is not available. Due to the existence of a chronic dog study this is not considered to be a data gap.

Reproduction/Developmental Toxicity - No adverse effects were reported in a 3 generation reproduction study at the highest dose tested establishing a NOEL of 500 ppm or 25 mg/kg/day. (USEPA, 1990; Sandoz, 1992)

In a rat teratology study maternal effects of ataxia, salivation, decreased motor activity, increased mortality and decreased body weight were observed at the highest dose tested, 400 mg/kg/day. The maternal NOEL was established at 160 mg/kg/day and the fetotoxic NOEL at 400 mg/kg/day. Sketetal malformations (reduced ossification) were present in fetuses from all dose levels but were found not to be statistically significant and not attributable to treatment. (Sandoz, 1992; CDFA, 1989)

Dicamba was administered to pregnant rabbits at the levels of 0, 1.0, 3.0, or 10.0 mg/kg/day. Effects observed at the high dose level (LOEL) include lower maternal net weight gain, reduced fetal body weights, and increased post implantation loss. The maternal and developmental NOEL was 3.0 mg/kg/day, which the EPA used to calculate the RfD value. (USEPA, 1990; Sandoz, 1992)

Chronic Toxicity/Oncogenicity - In a combined chronic/oncogenicity study rats were fed 0, 50, 250, or 2500 ppm dicamba in the diet for two years. No significant systemic or oncogenic effects were observed at any dose level placing the systemic NOEL at 2,500 ppm or 125 mg/kg/day. (USEPA, 1990; Sandoz, 1992)

Dogs were fed dicamba at 0, 100, 500 or 2500 ppm for one year. No adverse effects were observed at any dose level placing the NOEL at 2500 ppm or 52 mg/kg/day. (USEPA, 1990; Sandoz, 1992)

Dicamba was fed to mice in the diet at 0, 50, 150, 1000, or 3000 ppm for 89 weeks. There was no significant biological evidence of oncogenicity. No treatment related systemic adverse effects were observed up to and including 3,000 ppm. An equivocal low effect level (LEL) for systemic toxicity, based on increased mortalities in males and decreased body weight in females, is 3000 ppm. The NOEL is 1,000 ppm (approximately 115 mg/kg/day) (USEPA, 1989(a)).

Mutagenicity - Dicamba was tested in a variety of *in vitro* assays. Results indicate that it does not cause gene mutation (Ames and sex-linked recessive assay), chromosome aberration (cytogenetic assay in CHO cells) or DNA damage (unscheduled DNA synthesis in human fibroblasts). (Sandoz, 1992)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Banvel SGF	6,764 (IV)	>20,000 (IV)	non-irritating (IV)	non-irritating (IV)	>20.2 (IV)
Weedmaster	>5,000 (IV)	>20,000 (IV)	minimal (III)	minimal (IV)	>20.3 (IV)
Trimec Classic	1,550-2,240 (III)	>2,010 (III)	corrosive (I)	slight (III)	>2 (III)

2.3.8 Dichlobenil

Dichlobenil is a selective pre- and post-emergence systemic herbicide of the benzonitrile family. It is absorbed by the plant and inhibits cellulose synthesis which is the main constituent of the cell wall. It must be activated by rainfall or irrigation. The technical material is a pale yellowish powder with a characteristic aromatic odor. Dichlobenil has a low tendency towards volatilization and bioaccumulation (based on a low Henry's law constant and K_{ow}). Dichlobenil leaches slowly in soil but has low mobility in soils with high organic content. The EPA listed dichlobenil as a chemical which could potentially reach the groundwater. It is practically nonsoluble in water and slightly soluble in xylene and ethanol. The parent compound is not very persistent in the environment. However, the major soil metabolite, dichlorobenzamide, is relatively persistent.

Human Data

One case study of human exposure was reported. Six men received an occupational exposure to dichlobenil which caused dermatitis in 1 week to 5 months. The authors (Hayes, 1982) reported that the dermatitis could possiblely have been due to contaminants. (Jones & Stokes, 1991)

Laboratory Generated Data

Acute Toxicity - Dichlobenil is slightly to moderately toxic orally to laboratory animals with LD_{50} values ranging from 681 mg/kg in the guinea pig to 4,250 mg/kg for the rat. It is slightly toxic via the inhalation route of exposure with an LC_{50} of greater than 5 mg/l and moderately toxic dermally to rabbits, LD_{50} equal to 1,350 mg/kg. The technical material is nonirritating to the skin and eyes of rabbits and is not a sensitizer. (Uniroyal undated(a); Uniroyal undated(b); USEPA, 1987(a); USDHHS, 1980)

Subchronic Toxicity - Three subchronic 90-day feeding studies in the rat, mouse and dog were reported in the literature. NOELs reported were 2.5, 1.25 and 1.25 mg/kg/day, respectively. Increased liver weight was the effect most often observed in these studies. (Esch & Arnoldussen, 1961; Uniroyal undated(a); Til, et al., 1967)

Reproduction/Developmental Toxicity - In a two generation rat reproduction study dichlobenil was fed in the diet at 0, 20, 50, and 100 ppm. Effects observed at the high dose (LOEL) include decreased body weight gain and infertility in the F_0 and F_1 females.

The NOEL was established at 50 ppm or 2.5 mg/kg/day. This study was found to be unacceptable due to multiple major deviations from EPA guidelines. (CDFA, 1989(d))

Dichlobenil administered in the diet to pregnant rats from days 6 to 16 of gestation resulted in no maternal or developmental adverse effects at 20 mg/kg/day (NOEL). Effects noted at 60 mg/kg/day and above were decreased food consumption, food efficiency, and weight gain, and increased visceral variants. Increased skeletal variants were also noted at a 180 mg/kg/day dose. (CDFA, 1989(d))

A rabbit teratology and new reproduction study were identified as a data gaps. (Jones & Stokes, 1991)

Chronic Toxicity/Oncogenicity - Rats received dichlobenil in the diet at 0, 50, 400, or 3200 ppm for two years in a combined chronic/oncogenicity study. Systemic effects observed at 400 ppm and above include decreased body weights, changes in hematology and urinalysis parameters, and an increase in liver and kidney weights. The systemic NOEL was 50 ppm, or 2.5 mg/kg/day. There were no significant increases in total tumor or malignant tumor incidences. However, Peto analysis indicates an increase in tumorgenicity at the top dose suggesting a possible adverse effect. (CDFA, 1989(d)) A second study has been requested in order to determine the oncogenic potential of dichlobenil (USEPA, 1987(a)).

Dichlobenil was administered to dogs in the diet at 0, 20, 50, or 350 ppm for two years. No adverse effects were observed at 50 ppm (1.25 mg/kg/day), at 350 ppm observations included increased organ weights, increased levels of liver enzymes, increased hepatic glycogen storage accompanied by enlarged centrolobular hepatocytes and a slight hepatitis. The dose of 1.25 mg/kg/day was identified to be the lowest NOEL and was used to calculate the RfD value. (CDFA, 1989(d))

The mouse oncogenicity study is considered a data gap by the US EPA (Jones & Stokes, 1991).

Mutagenicity - Dichlobenil was tested in a variety of *in vitro* assays. Results indicate that it does not cause gene mutation (Ames and mitotic gene conversion in *S. cerevisiae*), chromosome aberration (chromosome aberration in human lymphocytes and a mouse bone marrow micronucleus assay) or DNA damage (DNA repair in mammalian cells, transformation and a Rec-assay). A possible increase in mutations was reported in a mouse lymphoma L518Y cell mutation assay. (CDFA, 1989(d))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Casoron 4G	(III)+	(IV)+	(III)+	(III)+	(III)+
Casoron 50W	(III)+	(IV)+	(III)+	(III)+	(III)+
Norosac 10G	>3,160 (III)	1,350 (II)	slight (III)	slight (III)	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.
+ No information available. Determination made according to label language.

2.3.9 Diuron

Diuron is a nonselective pre- and post-emergence systemic herbicide of the substituted urea family. It is rapidly absorbed by the roots of young plants and seedlings and translocated throughout the plant where it inhibits photosynthesis. The technical material is a white crystalline odorless solid. It is unlikely to volatilize or bioaccumulate (based on a small Henry's law constant and K_{ow}). Diuron has a high affinity for soil and does not leach more than six inches in soils of typical texture and moisture conditions. It is practically nonsoluble in water and has very low solubility in hydrocarbon solvents. Diuron is relatively persistent in the terrestrial environment and stable in the aquatic environment.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to diuron exposure.

Laboratory Generated Data

Acute Toxicity - Diuron is slightly toxic acutely to laboratory animals by all dose routes. The acute oral LD_{50} is 3,400 mg/kg for the rat. Dermal LD_{50} values range from 10,000 mg/kg for the rat to 20,000 mg for the rabbit. An inhalation LC_{50} was reported to be less than 200 mg/l for a one hour exposure. The technical material is reported to be nonirritating to the eyes, slightly irritating to the skin, and nonsensitizing. (USEPA, 1983(b); Jones & Stokes, 1991; DuPont, 1989(a))

Subchronic Toxicity - In a 90 day oral dosing study in rats, no effects were noted at 2.5 mg/kg/day (NOEL) but growth retardation and blood abnormalities occurred at doses of 25 (LOEL) and 250 mg/kg/day. (Jones & Stokes, 1991)

A six month feeding study was performed in conjunction with a chronic/onco-genicity study. Levels tested were 0, 4, 10, and 25 ppm. Minor hematological changes were observed at the highest dose tested establishing the NOEL at 10 ppm or 0.5 mg/kg/day. (CDFA, 1990(d))

Reproduction/Developmental Toxicity - In a three generation reproduction study, rats were fed 125 ppm of diuron in the diet. No reproductive, fetotoxic or teratogenic effects were observed. The NOEL was established by the EPA as greater than 125 ppm or 6.25 mg/kg/day. (CDFA, 1990(d))

Diuron was administered to rats by gavage at 0, 16, 80 or 400 mg/kg/day on days 6 through 15 of gestation. Maternal toxic effects observed at doses of 80 and 400 mg/kg/day include reduced food consumption, body weight, and body weight gain. Reduced fetal body weight and delayed ossification occurred at 400 mg/kg/day only. The developmental and maternal NOELs were established at 80 and 16 mg/kg/day, respectively. (CDFA, 1990(d))

Diuron was administered to rabbits by gavage at 0, 2, 10, or 50 mg/kg/day on days 7 through 19 of gestation. Maternal toxicity was observed at 50 mg/kg/day only and included decreased food consumption, decreased body weight gain, and 1 abortion. No developmental effects were noted. The developmental and maternal NOELs were established at greater than 50 and 10 mg/kg/day, respectively. (CDFA, 1990(d))

Chronic Toxicity/Oncogenicity - Diuron was fed in the diet of rats for two years in a chronic/oncogenicity study at levels of 1, 25, 250, and 2,500 ppm. The systemic NOEL was set by the EPA at 25 ppm or 1.25 mg/kg/day. Systemic effects observed at 250 ppm or higher include slight anemia, enlarged spleens, increased erythrogenic activity in bone marrow, and abnormal pigments in the blood. Increased tumorgenicity was identified by the CDFA as a possible adverse effect but was not noted by the EPA. (CDFA, 1990(d))

A two year dog feeding study reported no significant effects at 25 ppm (or 0.625 mg/kg/day). At levels of 125 ppm and higher (1250 ppm, HDT) animals had abnormal blood pigments. The EPA used the NOEL for this study to calculate the RfD. (CDFA, 1990(d); USEPA, 1990)

A mouse oncogenicity study was identified as a data gap in the 1983 EPA Reregistration Guidance Document.

Mutagenicity - Diuron was tested in a variety of *in vitro* assays. It did not cause gene mutation (Ames and CHO/HGPRT assay) or DNA damage (unscheduled DNA synthesis in primary rat hepatocytes). The average number of chromosome aberrations increased at the 48-hour sampling in an *in vivo* assay of diuron for chromosome aberrations in rat bone marrow. (CDFA, 1990(d))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Karmex DF	2,900 (III)	>2,000 (III)	moderate (II)	non-irritating (IV)	(III)+
Direx 4L	(III)+	(III)+	(III)+	(III)+	(III)+
Krovar I DF	>2,500 (III)	>2,000 (III)	mild (III)	irritant (III)*	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.10 Fluazifop-butyl

Fluazifop-butyl is selective postemergence systemic herbicide of the phenoxy propionate family. It is absorbed by the leaves, hydrolyzed to fluazifop, translocated to the meristematic tissue where it interferes with ATP production. The technical material is a light yellowish colored liquid. Fluazifop-butyl has a slight potential to volatilize based on the Henry's law constant and has a tendency to bioaccumulate based on the K_{ow} and a calculated Bioconcentration Factor of 120 (Jones & Stokes, 1991). Laboratory studies indicate that fluazifop-butyl is rapidly degraded, readily adsorbs to soil particles and would not tend to migrate. Fluazifop-butyl is practically nonsoluble in water and soluble in most solvents.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to fluazifop-butyl exposure.

Laboratory Generated Data

Acute Toxicity - Fluazifop-butyl is slightly toxic to laboratory animals. Reported acute oral LD_{50} values are 621, 1,630, 2,659 and 3,328 mg/kg for rabbits, mice, guinea pigs and rats, respectively. Dermal LD_{50} range from greater than 2,420 mg/kg for rabbits to greater than 6,050 mg/kg for rats. This low toxicity is supported by an acute inhalation LC_{50} of greater than 5.2 mg/l (4 hour exposure). The technical material is practically nonirritating to slightly irritating to the eyes of rabbits, mildly irritating to the skin, and not a sensitizer in the guinea pig. (Hartley and Kidd, 1987)

An acute delayed neurotoxicity study was performed in hens at levels up to 15,000 mg/kg. Fluazifop-butyl did not cause acute delayed neurotoxicity. (Jones & Stokes, 1991)

Subchronic Toxicity - Rats and dogs were fed fluazifop-butyl for 90 days. In the rat study no adverse effects were observed at 10 ppm or 0.5 mg/kg/day (NOEL), at 100 ppm (5 mg/kg/day, LOEL) observations included decreased body weight, alterations of clinical chemistry parameters, and liver hypertrophy. The NOEL established in the dog study was 25 mg/kg/day. Toxic effects noted at the LOEL of 125 mg/kg/day were decreased

body weight, decreased platlets, and increased levels of liver enzymes. (Jones & Stokes, 1991; Kieft, 1991)

Reproduction/Developmental Toxicity - In a two generation rat reproduction study no effects on reproduction indices were noted at 80 ppm or 4 mg/kg/day. At 250 ppm or 12.5 mg/kg/day observations included reduced litter size and fetal viability and alterations in the reproductive organs of the males. (Jones & Stokes, 1991; Kieft, 1991)

Two rat teratology studies were performed which administered fluazifop-butyl at 0, 1, 5, 10, and 200 mg/kg/day. The maternal toxic level in both studies was 200 mg/kg/day, the NOEL being 10 mg/kg/day. The fetotoxic effect observed was delayed ossification at 5 mg/kg/day and higher. The fetotoxic NOEL in both studies was 1 mg/kg/day. One rat study produced a teratogenic effect of diaphragmatic hernias at the top dose. Therefore the teratogenic NOEL for one study was 10 mg/kg/day and for the other it was greater than 200 mg/kg/day. (Jones & Stokes, 1991; Kieft, 1991)

In a rabbit teratology study fetotoxicity, manifested as delayed ossification and eye opacities, was observed at 30 mg/kg/day while maternal toxicity (increased abortion rate) was reported at 90 mg/kg/day. NOELs 10, 30 and greater than 90 mg/kg/day were established for fetotoxicity, maternal toxicity and teratogenicity, respectively. (Jones & Stokes, 1991; Kieft, 1991)

Chronic Toxicity/Oncogenicity - Three chronic (or oncogenicity) studies were reported in the literature. However, details of these studies were not available. In a two year rat chronic/oncogenicity study increased mortality was observed at 3 mg/kg/day establishing the NOEL at 1 mg/kg/day (used by the EPA to calculate the RfD value as reported by Jones & Stokes, 1991). No oncogenic effects were noted. Dogs were maintained on fluazifop-butyl in the diet for one year. Alterations were seen in liver enzyme levels in the blood at 25 mg/kg/day establishing the NOEL at 5 mg/kg/day. Mice were fed fluazifop-butyl for 18 months in the diet. No oncogenic effects were reported. The NOEL for systemic effects was 1 mg/kg/day, 3 mg/kg/day produced enlarged liver cells. (Jones & Stokes, 1991; Kieft, 1991)

Mutagenicity - The mutagenic potential of fluazifop-butyl was tested in several *in vitro* and *in vivo* assays. It was negative for gene mutation (Ames assay), chromosome aberration (*in vivo* dominant lethal assays) and DNA damage (transformation assays). However it produced chromosomal gaps in an *in vivo* rat assay and increased levels of micronuclei in an *in vivo* mouse micronucleus assay. (Jones & Stokes, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Fusilade 2000	2,980-4,930 (III)	>2,000 (III)	corrosive (I)[t]	corrosive (I)[t]	(III)[+]

+ No information available. Determination made according to label language.

t Toxicity data contradicts label language, A similar formulation of this material induced chemical burns on rabbit skin and in the rabbit eye test vs "caution" causes eye irritation.

2.3.11 Fosamine

Fosamine ammonium is a systemic postemergence herbicide of the carbamate family. It is absorbed by the buds, stem, and foliage and inhibits or prevents bud formation. The technical material is a white crystalline solid. It has very little tendency to volatilize or bioaccumulate (based on the small Henry's law constant and K_{ow}). Fosamine is weakly to highly adsorbed onto different soil types where it is converted to carbamyl phosphonic acid that binds strongly to soils. It is highly soluble in water, has low solubility in methanol, and is slightly soluble in most organic solvents. Laboratory studies indicate that fosamine ammonium is rapidly degraded in the environment.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to fosamine ammonium exposure.

Laboratory Generated Data

Acute Toxicity - Fosamine ammonium is very slightly toxic to moderately toxic depending on the route. Oral LD_{50}'s range from 7,380 mg/kg for the guinea pig to 24,000 mg/kg for the rat. However its moderately toxic to rabbits by the dermal route of exposure with an LD_{50} greater than 1,683 mg/kg and slightly toxic when inhaled by rats with an LD_{50} of 3 mg/kg. The technical material is not irritating to the eyes, but caused mild to moderate skin irritation, and was not a skin sensitizer. (USFS, 1988)

Subchronic Toxicity - A systemic NOEL of 5,000/10,000 ppm or 250/500 mg/kg/day was established for a rat 90 day feeding study, which was the highest dose tested. (USFS, 1988)

Dogs fed fosamine in the diet for six months exhibited increased stomach weights at the LOEL (value not reported) which established the NOEL at 1,000 ppm or 25 mg/kg/day. This NOEL was identified to be the lowest in the toxicology data package and was used to calculate the RfD value. (USFS, 1988)

Reproduction/Developmental Toxicity - No adverse reproductive effects were reported in the rat at the highest dose tested, 5,000/10,000 ppm (250/500 mg/kg). This report was found to be unacceptable to the CDFA. (USFS, 1988)

A teratogenic effect of hydronephrosis was reported in a rat teratology study at 10,000 ppm (500 mg/kg/day). One female from the mid and high dose had litters which were completely resorbed. No NOEL was reported. Study was found unacceptable by the CDFA. (USFS, 1988)

Chronic Toxicity/Oncogenicity - No chronic or oncogenicity studies are available for fosamine ammonium and are not required to support its terrestrial non-food use registration.

Mutagenicity - Fosamine was tested in a variety of *in vitro* assays and one *in vivo* assay. Results indicate that it does not cause gene mutation (Ames and CHO assays), chromosome aberration (*in vivo* cytogenetic assay) or DNA damage (unscheduled DNA synthesis using rat hepatocytes). In an *in vitro* chromosome aberration assay (in CHO cells), fosamine tested positive both with and without activation. (USFS, 1988)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Krenite S	>5,000 (IV)	>5,000 (III)	moderate (II)	non-irritating (IV)	2.75-3.3 (III)

2.3.12 Glyphosate

Glyphosate is a nonselective postemergence systemic herbicide of the phosphonic acid family. It is absorbed by the plant foliage and rapidly translocated throughout the plant where it interferes with amino acid synthesis and other biosynthesis reactions. The technical material is a colorless crystalline solid. Glyphosate has little potential to volatilize or bioaccumulate (based on a low Henry's law constant and K_{ow}). Glyphosate technical has low solubility in water and is insoluble in common organic solvents. The alkali metal and amine salts are readily soluble in water. Glyphosate has very little potential to leach (nonmobil). It rapidly and strongly adsorbs to soil, particularly to clay particles at lower pH values. It is not very persistent in the terrestrial or aquatic environments.

Human Data

Roundup, a formulation of glyphosate, was reported to cause irritation of the mouth, nausea, intestinal discomfort, vomiting and diarrhea when ingested by humans. Ingestion of large quantities has been reported to result in hypotension and pulmonary edema (Jones & Stokes, 1991). A 6.4% aqueous solution of Roundup on the skin of humans did not cause irritation or sensitization.

Laboratory Generated Data

Acute Toxicity - Glyphosate is slightly toxic to laboratory animals. Acute oral LD_{50} values range from 4,320 for rats to 3,800 mg/kg for rabbits. This low toxicity is supported by an acute dermal LD_{50} of greater than 5,000 mg/kg in rabbits and a four hour inhalation exposure LC_{50} of greater than 12.2 mg/l in rats. The technical material is slightly irritating to the eyes of rabbits, nonirritating to the skin and not a sensitizer in guinea pig studies. (USEPA, 1988(f); Monsanto, 1990)

An acute delayed neurotoxicity study in hens was performed but results were inconclusive. Results from a delayed neurotoxicity study are not required at this time. (CDFA, 1988(a))

Subchronic Toxicity - Two subchronic glyphosate feeding studies were reported one each in the rat and dog. No adverse effects were reported up to and including the highest dose

tested. The NOEL for the rat study was greater than 2,000 ppm (100 mg/kg/day) and for the dog study greater than 2,000 ppm (50 mg/kg/day). (Jones & Stokes, 1991)

Reproduction/Developmental Toxicity - In a three generation reproduction study rats were fed glyphosate in the diet at 0, 3, 10, and 30 mg/kg/day (diet adjusted weekly). No treatment related effects on fertility were noted, nor were any systemic effects apparent. An increased incidence of unilateral renal tubular dilation in male F3b pups was observed at 30 mg/kg/day (LEL). This established the NOEL at 10 mg/kg/day and was used by the EPA to calculate the RfD value. (USEPA, 1990; CDFA, 1988(a))

Glyphosate was administered to rats at 0, 300, 1000 and 3500 mg/kg/day by gavage on days 6 through 19 of gestation. Maternal and fetal toxic effects reported at the highest dose tested were decreased body weight, an increase in the number of early resorptions and post implantation losses, and a decrease of total implantations, viable fetuses, fetal body weight and corpora lutea. An increase in the number of litters and fetuses with unossified sternebrae was also noted at 3500 mg/kg/day. The maternal and developmental NOEL was established at 1,000 mg/kg/day. (USEPA, 1990; CDFA, 1988(a))

In a rabbit teratology study animals were dosed at 0, 75, 175, and 350 mg glyphosate/kg/day. An increased incidence of soft stool, diarrhea and nasal discharge was observed at 350 mg/kg/day. No developmental were noted at any dose level. The maternal NOEL was established at 175 mg/kg/day and the developmental NOEL was greater than 350 mg/kg/day. (USEPA, 1990; CDFA, 1988(a))

Chronic Toxicity/Oncogenicity - In a chronic/oncogenicity study rats were fed dietary levels of 0, 30, 100, and 300 ppm glyphosate for two years. No adverse systemic effect was observed at any level tested. The systemic NOEL is greater than 300 ppm (33 mg/kg/day, actual average). No significant increase in tumor incidence was noted. Since this study did not have a maximum tolerated dose (MTD) it was not acceptable as an oncogenicity study and is being repeated. (USEPA, 1990; CDFA, 1988(a))

Dogs were maintained on dietary levels of glyphosate for one year, 0, 20, 100, and 500 mg/kg/day. Decreased absolute and relative pituitary weights were noted at 100 mg/kg/day and above. The NOEL was established at 20 mg/kg/day. (USEPA, 1990; CDFA, 1988(a))

Glyphosate was feed to mice for two years at 0, 1000, 5000 and 30,000 ppm. Systemic toxic effects of the liver and kidney were observed upon histopathological evaluation at the high dose, establishing the NOEL for systemic effects at 5000 ppm (710 mg/kg/day). An increased incidence of renal tubular adenomas and carcinomas was observed in males at the highest dose tested. Glyphosate was originally classified as a Group "C" carcinogen and a cancer potency factor was published. Following an independent review of the slides this classification was changed to "D" on the basis of a lack of statistical significance. The EPA has requested that a repeat study be performed. (USEPA, 1990; CDFA, 1988(a))

Mutagenicity - Glyphosate was tested in a variety of *in vitro* and *in vivo* assays. Results indicate that it does not cause gene mutation (Ames and CHO/HGPRT assays),

chromosome aberration (*in vivo* rat bone marrow cytogenetic assay and *in vivo* mouse dominant lethal assay) or DNA damage (unscheduled DNA synthesis using rat hepatocytes and DNA damage assay in *B. subtilis*). (CDFA, 1988(a))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Roundup	5,000 (III)	>5,000 (III)	moderate (II)	slightly (III)	3.18 (III)
Rodeo	>5,000 (IV)	>5,000 (III)	non-irritating (IV)	very slight (IV)	>1.3 (II)

2.3.13 Imazapyr

Imazapyr is a nonselective systemic herbicide of the imidazolinone family. It is absorbed by roots and foliage and rapidly translocated to the meristematic regions where it inhibits synthesis of branched chain amino acids thus interfering with cell growth. The technical material is a colorless crystalline solid. It has little potential to volatilize or bioaccumulate (based on a low Henry's law constant and K_{ow}). Laboratory studies indicate that imazapyr would tend to leach and migrate through the soil. It is soluble in water and has a soil half life.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to imazapyr exposure.

Laboratory Generated Data

Acute Toxicity - The acute toxicity of imazapyr to laboratory animals is low. Acute oral LD_{50} values reported are >5,000 mg/kg for the rat, > 2,000 mg/kg for female mice and 4,800 mg/kg for the rabbit. This low order of toxicity is supported by an acute dermal LD_{50} of >2,000 mg/kg for both rabbit and rat and an inhalation LC_{50} >5.1 mg/liter. The technical material is irritating to the eyes of rabbits, mildly irritating to the skin and not a sensitizer in guinea pig studies. A 21 day dermal study in rabbits showed no signs of systemic toxicity at 400 mg/kg/day (USDI, 1991). (American Cyanamid, 1986)

Subchronic Toxicity - One subchronic toxicity study was reported in the literature which established the NOEL to be greater than 10,000 ppm in the diet (500 mg/kg/day), the highest dose tested (USDI, 1991). Data from subchronic studies in other species is not required by the EPA for imazapyr at this time.

Reproduction/Developmental Toxicity - No developmental effects were observed in a rat or rabbit teratology study at the highest dose tested. Maternal and developmental NOELs in the rat study were 300 mg/kg/day and >1,000 mg/kg/day, respectively. The NOEL of 300 mg/kg/day established in this study was identified to be the lowest in the data available and was used to calculate the RfD value. The maternal and developmental

NOEL established for the rabbit teratology study was greater than 400 mg/kg/day. A three generation reproduction study appears to be a data gap. (USDI, 1991)

Chronic Toxicity/Oncogenicity - Studies evaluating the chronic toxicity and oncogenic potential of imazapyr are currently under review at the US EPA. No evidence of carcinogenicity was observed in rats after 12 months maintenance on 500 mg/kg/day imazapyr in the diet.

Mutagenicity - Imazapyr was tested in a variety of *in vitro* assays and one *in vivo* assay. Results indicate that it does not cause gene mutation (Ames and CHO HGPRT assays), chromosome aberration (*in vivo* mouse dominant lethal and CHO cytogenetics assay) or DNA damage (unscheduled DNA synthesis). (USDI, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Arsenal	>5,000 (IV)	>2,148 (III)	irritating (III)*	mildly (III)	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.14 Isoxaben

Isoxaben is a selective preemergence systemic herbicide of the isoxazole family. It is absorbed by the roots, translocated to the stem and leaf tissues where it disrupts leaf and shoot development in grass and broadleaf weeds. It must be activated by rainfall or irrigation. The technical material is a white crystalline solid. It has a low tendency toward volatilization and a low potential to bioaccumulate based on the Henry's law constant and K_{ow}, respectively. Isoxaben technical has very low solubility in water but is moderately soluble in methanol. It has a very high affinity for soil particles and organic matter. Based on this, its relative insolubility in water and the high K_d values range it would not tend to leach or migrate through the soil. Isoxaben is relatively non-persistent in the terrestrial and aquatic environments.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to isoxaben exposure.

Laboratory Generated Data

Acute Toxicity - The acute toxicity of isoxaben to laboratory animals is very low. Acute oral LD_{50} values range from >10,000 mg/kg for the rat and mouse to >5,000 mg/kg for the dog. This low order of toxicity is supported by an acute rat inhalation LC_{50} of >1.99 mg/l. An acute dermal LD_{50} of >200 mg/kg was reported which would indicate that isoxaben is moderately toxic via the dermal route. However, this is unlikely given the oral and inhalation values and details of the study were not available. Isoxaben

technical is slightly irritating to both the eyes and skin of laboratory rabbits and it was not a sensitizer in guinea pig studies. (Hartley and Kidd, 1987; Jones, 1991)

Subchronic Toxicity - The manufacturer reported that a maximum dose of 50,000 mg/kg in rats and 1,000 mg/kg in dogs produced no serious effects (whether it was in the diet or not and duration were not specified). Dogs feed isoxaben in the diet for 90 days exhibited increased liver weights at 500 mg/kg/day, establishing a NOEL at 110 mg/kg/day. (Jones, 1991)

Reproduction/Developmental Toxicity - No adverse reproductive effects were observed when isoxaben was tested in a three generation rat reproduction study which established the parental and developmental NOELs at 40 and 200 mg/kg/day, respectively. (Jones, 1991)

In a rat teratology study developmental effects reported at the highest dose tested include increases in preimplantation loss, small litter size, resorptions and the number of runt fetuses. The maternal NOEL was established at 1,000 mg/kg/day and the developmental at 320 mg/kg/day. (Jones & Stokes, 1991; Jones, 1991)

No adverse developmental effects were observed in rabbits treated with 1,000 mg isoxaben/kg/day, the highest dose tested (NOEL > 1,000 mg/kg/day). Maternal effects were observed at this high level establishing the NOEL at 320 mg/kg/day. (Jones, 1991)

Chronic Toxicity/Oncogenicity - Reported NOEL values for a rat two year feeding study range from 5 to 6.6 mg/kg/day (the discrepancy probably due to conversion of ppm in the diet to mg/kg body weight). Effects observed at 50.7 mg/kg/day include induction of liver enzymes, decreased food consumption and increased organ-to-body weight ratios. The dose of 5 mg/kg/day reported for this study was identified to be the lowest NOEL in the isoxaben data base and was used to calculate the RfD value. (Hartley and Kidd, 1987; Jones & Stokes, 1991; Jones, 1991)

Dogs and rats maintained for one year on isoxaben were reported to exhibit similar effects as those described above at levels of 100 and 62.5 mg/kg/day. NOELs were established at 10 mg/kg/day for dogs and 6.25 mg/kg/day for rats. (Jones, 1991)

Isoxaben was fed to mice for two years. Hepatocellular hyperplasia, decreased body weight, and cytomegaly were reportedly observed at the 143 mg/kg/day dose level, establishing a NOEL at 12 mg/kg/day. The EPA, Health Effects Division of the OPP (1991), reports isoxaben to be a Group "C" Carcinogen (possible human carcinogen) and that calculation of a cancer potency factor is not applicable to this compound. (Jones & Stokes, 1991; Jones, 1991)

Mutagenicity - The manufacturer reported isoxaben to be not mutagenic in genotoxicity tests conducted. (Jones, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Gallery	>5,000 (IV)	>5,000 (III)	irritant (III)*	irritant (III)*	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.15 Mecoprop

Mecoprop is a selective systemic postemergence broadleaf herbicide of the chlorinated phenoxy family. It is a hormone type herbicide which is absorbed by the leaves and the roots and is translocated throughout the plant where it either stimulates or inhibits cell division. The technical material is a white to light brown odorless crystalline solid. Mecoprop has a low tendency toward volatilization and a slight potential to bioaccumulate based on the Henry's law constant and laboratory studies, respectively. Mecoprop has very low solubility in water but is readily soluble in acetone, benzene, alcohol and chlorinated hydrocarbons. Laboratory studies indicate that it is very mobile in soils (with a higher affinity for soils high in organic content) and has the potential to leach and migrate. Mecoprop is not persistent in the environment with a half-life of 60 days or less.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to mecoprop exposure.

Laboratory Generated Data

Acute Toxicity - Mecoprop is slightly toxic to laboratory animals. Reported acute oral LD_{50} values range from 558 and 650 mg/kg for the rat and 650 mg/kg for the mouse. This low order of toxicity is supported by acute dermal LD_{50}s of >2,000 mg/kg (rabbit) and >4,000 mg/kg (rat) and an acute rat inhalation LC_{50} of >12.5 mg/l. One source reported a dermal LD_{50} for rabbits of 900 mg/kg which is unlikely when compared to the other reported values. Mecoprop is reported to be severely irritating to the eyes of rabbits, moderately irritating to the skin, and not a sensitizer in guinea pig studies. (USDHHS, 1980; Leeming, 1991; USEPA, 1988(b); Worthing and Hance, 1990)

Subchronic Toxicity - Rats were maintained for 90 days on 0, 50, 150 or 450 ppm mecoprop in the diet. Increased relative and absolute kidney weights were reported at 150 ppm and above establishing the NOEL at 50 ppm or 3 mg/kg/day. (USEPA, 1988(b); Leeming, 1991)

The manufacturer reported a NOEL for dogs at 114 ppm (2.9 mg/kg/day). (Leeming, 1991)

Reproduction/Developmental Toxicity - The EPA Science Chapter on mecoprop summarizes a rat teratology study in which animals were given 0, 20, 50 or 125 mg/kg/day. No maternal toxic effects were seen. However, developmental effects were observed at 125 mg/kg/day. These included increased intra-uterine deaths, decreased crown/rump lengths and an increase in the incidence of delayed or absent ossification of the sternebra. The maternal NOEL was > 125 mg/kg/day and the developmental NOEL = 50 mg/kg/day (USEPA, 1988(b)). The agency concluded that exposure from the use of mecoprop containing products falls well below the 100 margin of safety level for developmental effects. The manufacturer reported mecoprop to be not teratogenic or embryotoxic at doses up to and including 0.25 mg/kg/day (no details of the study were given). (Leeming, 1991)

Rabbits were treated with 0, 12, 30 or 75 mg mecoprop/kg/day. A slight reduction in body weights was observed in dams at 75 mg/kg/day but no developmental effects were seen at any dose level. The maternal and developmental NOELs were 30 and >75 mg/kg/day, respectively. The manufacturer reports that mecoprop was not teratogenic or embryotoxic at doses up to and including 7.5 mg/kg/day. (USEPA, 1988(b); Leeming, 1991)

The US EPA has not required a reproduction study on mecoprop based on its non-food use patterns.

Chronic Toxicity/Oncogenicity - The results of a two year rat study reported a NOEL of 20 ppm (1 mg/kg/day). (Worthing and Hance, 1990; Leeming, 1991)

The EPA has not required additional chronic or oncogenicity studies on mecoprop based on its non-food use patterns.

Mutagenicity - Mecoprop was negative for gene mutation in an Ames assay. In an *in vivo* sister chromatid exchange it reportedly caused a significant dose related increase in SCEs (DNA damage and repair). A study evaluating the potential of mecoprop to cause chromosome aberration was identified as a data gap (USEPA, 1988(b)). The manufacturer simply reports mecoprop to be not mutagenic (Leeming, 1991).

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Trimec Classic	>2,240 (III)	>2,010 (III)	corrosive (I)*	moderate (II)	>2 (III)

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

2.3.16 Mefluidide

Mefluidide is a plant growth regulator herbicide of the trifluoromethyl, acetamide family. It inhibits growth and development of meristematic regions of primarily perennial grasses. The technical material is a colorless crystalline solid. Mefluidide has a low tendency toward volatilization based on the Henry's law constant. The K_{ow} indicates that

it has a slight potential to bioaccumulate, however, no bioaccumulation was observed in catfish exposed to radiolabeled mefluidide applied to soil. It has low solubility in water but is moderately soluble in acetone and methanol. Adsorption of mefluidide to soil after a 3-hour contact period is negligible (Jones & Stokes, 1991). The K_{oc} value suggests that mefluidide may leach and migrate in the soil but movement would be limited due to it is rapid rate of degradation in the environment (Jones & Stokes, 1991).

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to mefluidide exposure.

Laboratory Generated Data

Acute Toxicity - The acute toxicity data suggests that mefluidide is only slightly toxic to laboratory animals. Acute oral LD_{50} values range from 4,000 mg/kg for rats, and 1,920 mg/kg for mice. This low order of toxicity is supportd by a dermal LD_{50} >4000 mg/kg for rabbits and a rat inhalation LC_{50} >5.4 mg/l (rat inhalation). The technical material is minimally irritating to the eyes of rabbits, non-irritating to the skin, and not a sensitizer in guinea pig studies. (USDI, 1991; USDHHS, 1980; PBI Gordon, 1991(u); PBI Gordon, 1991(w))

Subchronic Toxicity - The NOEL established in a 90 day rat feeding study was reported to be 6,000 ppm or 300 mg/kg/day. Higher doses produced body weight loss. Details of the study were not available. (USDI, 1991)

Dogs were fed mefluidide in the diet for 90 days at 0, 300, 1000 or 6000 ppm. Effects noted at 6000 ppm included decreased RBC count, hemoglobin and hematocrit levels (NOEL = 1000 ppm or 25 mg/kg/day). (USDI, 1991)

Reproduction/Developmental Toxicity - Teratology studies were performed with mefluidide on both rats and rabbits. No significant dose related maternal or developmental effects were reported up to and including the highest dose tested establishing NOELs of >60 mg/kg/day. (USDI, 1991)

In a three generation reproduction study, rats were fed mefluidide at 0, 600, 1800 or 6000 ppm resulting in no adverse effects being reported in either parents or offspring at 1800 ppm (90 mg/kg/day). Effects observed at 6000 ppm (300 mg/kg/day) include decreased parental and neonatal body weight, decreased liver weights in male neonates, and decreased spleen weights in female neonates. (USDI, 1991; Jones & Stokes, 1991)

Chronic Toxicity/Oncogenicity - At least one two year rat study was performed evaluating chronic effects of mefluidide in the diet. The only systemic effect noted in this study was body weight loss. A variety of chronic NOELs have been identified from 10, 30 and 100 (2000 ppm) mg/kg/day reported by Jones & Stokes (1991), BLM FEIS (USDI, 1989) and in 3M Report #177, respectively. No evidence of oncogenicity was reported.

Dogs were fed 0, 60, 600 or 6000 ppm mefluidide in the diet for one year. Effects observed at the top two dose levels were mild to marked body weight loss; kidney

toxicity was reported in the highest dose tested. The NOEL for body weight loss is 60 ppm or 1.5 mg/kg/day. This value was identified as the lowest NOEL in the mefluidide data base and was used to calculate the RfD value. (USDI, 1991; Jones & Stokes, 1991)

In a 18 month oncogenicity study mice were fed 0, 600, 1800 or 6000 ppm mefluidide in the diet resulting in no adverse effects at 600 ppm (90 mg/kg/day). Hyperplastic nodules of the liver and regeneration was observed at the highest dose tested. This was not considered to be an oncogenic response by a panel of three independent board-certified veterinary pathologists. (Jones & Stokes, 1991)

Mutagenicity - Mefluidide was tested in a variety of *in vitro* assays. It did not cause gene mutation (Ames and mouse lymphoma assays), chromosome aberration (sister chromatid exchange assay) or DNA damage/repair. (Jones & Stokes, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Embark 2-S	>5,000 (IV)	>20,000 (IV)	minimal (III)	minimal (IV)	>8.5 (III)

2.3.17 Metsulfuron methyl

Metsulfuron methyl is systemic postemergence herbicide of the sulfonylurea family. It is absorbed by the roots and foliage, translocated throughout the plant where it inhibits cellular division in the meristems. The technical material is a colorless crystalline solid. Metsulfuron methyl has a low tendency towards volatilization and will not bioaccumulate based on the Henry's law constant and K_{ow}, respectively. It is poorly to weakly adsorbed to soils and has low solubility in water and most organic solvents. Based on the water solubility and its poor affinity for soil, metsulfuron methyl would have a tendency to leach and migrate. It is not very persistent in the aquatic environment, but relatively persistent in soil.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to metsulfuron methyl exposure.

Laboratory Generated Data

Acute Toxicity - Metsulfuron methyl has a low order of acute toxicity to laboratory animals with an oral rat LD_{50} of >5,000 mg/kg/day, dermal LD_{50} values of >2,000 and >4,640 mg/kg for guinea pigs and rabbits respectively and a rat four hour inhalation LC_{50} of >5.0 mg/l. The technical material is reported to be severely irritating (but not corrosive) to the eyes of laboratory rabbits, moderately irritating to the skin and not a sensitizer in guinea pig studies. (USDHHS, 1990; USDI, 1991)

Subchronic Toxicity - In a subchronic study rats were fed metsulfuron methyl for 90 days resulting in no effects at 1,000 ppm (50 mg/kg/day, NOEL). At 7,500 ppm (187.5 mg/kg/day) effects observed include decreased body weight gain and serum protein. (USDI, 1991)

Dogs were fed metsulfuron methyl for 90 days, no treatment related effects were observed up to and including 5,000 ppm (NOEL >125 mg/kg/day), the highest dose tested. (USDI, 1991)

Reproduction/Developmental Toxicity - In a two generation reproduction study rats were fed 0, 25, 500 or 5000 ppm metsulfuron methyl in the diet. Maternal body weight gains were reduced at 5000 ppm, establishing the maternal NOEL at 500 ppm or 25 mg/kg/day. No effects were reported for any reproductive or fetal parameters, thus the NOEL >5000 ppm or 250 mg/kg/day. (USEPA, 1990; USDI, 1991)

Two teratology studies were performed administering metsulfuron methyl to rats and rabbits. No fetotoxic or teratogenic effects were observed up to and including the highest dose tested, establishing NOELs at 1,000 and 700 mg/kg/day for rats and rabbits, respectively. The rat maternal NOEL was <40 mg/kg/day due to hyperactivity and ungroomed coat observed at all dose levels. The rabbit maternal NOEL was established at 25 mg/kg/day based on decreased weight gain and death observed at the top three dose levels (100, 300 and 700 mg/kg/day). (USEPA, 1990; USDI, 1991)

Chronic Toxicity/Oncogenicity - Metsulfuron methyl was fed in the diet to rats for two years at levels of 0, 5, 25, 500 and 5000 ppm. Decreased body weight gain was noted at the top dose, establishing the systemic NOEL at 500 ppm or 25 mg/kg/day. This NOEL (25 mg/kg/day) was used by the EPA to calculate the RfD value. No treatment related oncogenic effects were observed. (USEPA, 1990; USDI, 1991)

Dogs were fed 0, 50, 500 or 5000 ppm metsulfuron methyl for one year. No dose related effects were observed up to and including 5000 ppm establishing the NOEL to be greater than 125 mg/kg/day (5,000 ppm). (USEPA, 1990; USEPA, 1986(a); USDI, 1991)

In an 18 month feeding study mice were fed metsulfuron methyl at the dietary levels of 0, 5, 25, 500 or 5000 ppm. No dose related systemic or oncogenic effects were reported up to and including 5000 ppm, therefore the NOEL is reported as greater than 5,000 ppm (750 mg/kg/day). This study was rated supplemental due to the lack of an MTD (maximum tolerated dose). (USEPA, 1990; USEPA, 1986(a); USDI, 1991)

Mutagenicity - Metsulfuron methyl was tested in a variety of *in vitro* assays and one *in vivo* assay. According to the results reported in these assays gene mutation (Ames and CHO assays), chromosome aberration (*in vivo* rat bone marrow cytogenetic assay) or DNA damage (unscheduled DNA synthesis using rat hepatocytes) does not occur. In an *in vitro* cytogenetic assay (in CHO cells), metsulfuron methyl tested positive. Since it tested negative for chromosome aberration in an *in vivo* assay, metsulfuron methyl is reportedly not mutagenic. (USEPA, 1986(a))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Escort	>5,000 (IV)	>2,000 (III)	slight (III)	non-irritating (IV)	(III)+

+ No information available. Determination made according to label language.

2.3.18 Napropamide

Napropamide is a selective systemic preemergence herbicide of the amide; naphthalene family. It is absorbed by the roots, translocated and inhibits root development and growth in annual grasses and broadleaf weeds (requires rainfall or irrigation). The technical material is a brown solid. Napropamide has a low tendency toward volatilization and a moderate potential to bioaccumulate based on the Henry's law constant and K_{ow}, respectively. It is not very soluble in water and is miscible in acetone and ethanol. Based on the low solubility in water and high affinity for soil and organic carbon (high K_d and K_{oc}) this compound would not tend to leach or migrate. Napropamide is not persistent in the environment.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to napropamide exposure.

Laboratory Generated Data

Acute Toxicity - According to the studies available for this report napropamide is only slightly toxic to laboratory animals. The acute oral LD_{50} in rats is greater than 5,000 mg/kg. This low order of toxicity is supported by dermal LD_{50} values ranging from >4,640 mg/kg for rabbits to >2,000 for guinea pigs, and an inhalation LC_{50} greater than 5.1 ml/l. The technical material is reportedly non to mildly irritating to the eyes of rabbits, non to mildly irritating to the skin and not a sensitizer in guinea pig studies. (Hartley and Kidd, 1987; Kieft, 1991)

Subchronic Toxicity - Napropamide was fed to rats for 13 weeks resulting in decreased mean relative uterine weights in females at the top dose of 50 mg/kg/day (LOEL). The amount of 25 mg/kg/day was identified by the EPA as the NOEL. (USEPA, 1990)

Dogs were fed napropamide in the diet for 13 weeks. Increased mean absolute and relative body weights were observed in males at the top dose of 100 mg/kg/day (LOEL). The amount of 40 mg/kg/day was identified by the EPA as the NOEL for this study. (USEPA, 1990)

Reproduction/Developmental Toxicity - In a three generation reproduction study rats were given 0, 10, 30 or 100 mg napropamide/kg/day. Effects observed at the highest dose tested include reduced pup weight and decreased weight gain in the parents. No dose related toxic effects were reported for reproductive parameters. The maternal and fetotoxic NOEL was established at 30 mg/kg/day, and the reproductive NOEL was

greater than 100 mg/kg/day. The 30 mg/kg/day NOEL was used by the EPA to calculate the RfD value. (USEPA, 1990)

Teratogenicity tests with napropamide were carried out recently on rats and rabbits. In a personal communication of proprietary information from the manufacturer, ICI Americas Inc. reported that no fetotoxic or embryotoxic effects were observed at 100 mg/kg/day. No information regarding maternal toxicity was volunteered. (Kieft, 1991)

A rat teratology study performed in 1982 (rated core grade minimum by the EPA) established a developmental NOEL >400 mg/kg/day and a maternal NOEL of 110 mg/kg/day, based on decreased body weight gain during gestation days 6 through 9. (Kieft, 1991)

Chronic Toxicity/Oncogenicity - A 24 month feeding study in rats was carried out with napropamide at the following levels; 0, 10, 30 and 100 mg/kg/day. The systemic NOEL was established at 30 mg/kg/day based on significantly lower body weight in females at 100 mg/kg/day. No significant dose related oncogenic effects were reported. (USEPA, 1990; Kieft, 1991)

A NOEL of 10 mg/kg/day was recently established for chronic exposure of dogs to napropamide (Kieft, 1991).

Mice were exposed chronically in the diet to napropamide resulting in no treatment related oncogenic effects and a systemic NOEL of 450 ppm (ca 67.5 mg/kg/day) (Kieft, 1991).

Mutagenicity - Napropamide was tested in a variety of *in vitro* and *in vivo* assays. Reported results were negative for gene mutation in the Ames and CHO HGPRT forward mutation assays, but was positive in the mouse lymphoma L5178Y forward mutation assay. Napropamide tested negative for chromosome aberration in the *in vivo* rat bone marrow cytogenetic assay and the *in vitro* mouse lymphoma cytogenetic assay. It also tested negative for DNA damage in the *in vivo* unscheduled DNA synthesis assay using rat hepatocytes and in an *in vitro* human fibroblast DNA assay. (CDFA, 1990)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Devrinol 50-WP	>4,640 (III)	>4,640 (III)	mildly (III)	mildly (III)	(III)+

+ No information available. Determination made according to label language.

2.3.19 Oryzalin

Oryzalin is a selective preemergence nitroaniline herbicide. It is incorporated into the soil where it effects seed germination and prevents weed growth by inhibition of root development. The technical material is a yellow-orange crystalline solid. Oryzalin has a low tendency towards volatilization and a moderate potential to bioaccumulate based on the Henry's law constant and K_{ow}, respectively. It has a very low solubility in water,

is readily soluble in polar organic solvents and insoluble in hexane. Oryzalin has a high affinity for the soil, is strongly adsorbed, and resists movement by water. Studies have shown that it does not leach below 6 inches. The compound is relatively nonpersistent in the environment.

Human Data

The most commonly reported effect of exposure is contact dermatitis, or rash. Some cases were reported during manufacture and may be related to an intermediate chemical, but other reports have involved finished products of oryzalin. Eye irritation may also occur. One instance of ingestion was reportedly uneventful (Dow/Elanco, undated(c)).

Laboratory Generated Data

Acute Toxicity - Oryzalin is very slightly to slightly toxic to laboratory animals with acute oral LD_{50} values ranging from >1,000 mg/kg for the dog and chicken to >10,000 mg/kg in the rat. This low order to toxicity is supported by reported dermal LD_{50} values of >2000 and >5000 mg/kg for rabbits and an acute rate inhalation LC_{50} >3, 56 mg/l. The technical material is reported to be slightly irritating to the eyes and skin of laboratory rabbits and was positive as a sensitizer in guinea pig studies. (DowElanco, undated(c); USDHHS, 1990; Blewett, 1991; USEPA, 1987(b))

Subchronic Toxicity - Rats were fed 0, 300, 900, or 2700 ppm oryzalin in the diet for one year. Effects reported at 900 ppm or higher were decreased body weight gain and increased organ weights, establishing a NOEL of 300 ppm (15 mg/kg/day). (USEPA, 1990; CDFA, 1989(a))

Dogs were maintained on dietary levels of oryzalin for 90 days resulting in alterations of hematology and blood chemistry values, hyperplastic bone marrow, hepatic and spleen changes, and anemia at the 56.25 mg/kg/day level. Based on these findings the NOEL was established at 18.75 mg/kg/day. (USEPA, 1990)

A NOEL of 1,000 mg/kg/day was listed by the US EPA for a 21 day dermal study in rabbits, the highest dose tested. (USEPA, 1987(b))

Reproduction/Developmental Toxicity - In a three generation reproduction study rats were fed 0, 250, 750, or 2250 ppm in the diet resulting in decreased body weight gain in pups at 750 ppm (LOEL, 37.5 mg/kg/day) and up. No dose related reproductive effects were observed at any dose level. The fetotoxic NOEL is 250 ppm (12.5 mg/kg/day) and the reproductive NOEL is greater than 2250 ppm (112.5 mg/kg/day). (USEPA, 1990)

Several rat teratology studies have been performed using oryzalin. In one submitted to the EPA animals were given 0, 25, 75, or 225 mg/kg/day which resulted in no dose related developmental or maternal effects, establishing the NOEL as greater than 225 mg/kg/day. In a second rat teratology study submitted to the CDFA animals were given 0, 50, 225, and 1000 mg/kg/day. Reported results include delayed ossification at all dose levels, decreased fetal weight at the mid and high dose and runts and diaphragmatic hernias at the high dose. Signs of maternal toxicity (decreased body weight gain and alopecia) were observed at the mid and high dose. Effect levels identified by the CDFA

were; developmental NOEL <50 mg/kg/day, developmental NOAEL = 50 mg/kg/day and maternal NOEL = 50 mg/kg/day. (USEPA, 1990; CFDA, 1989(a))

Several rabbit teratology studies were carried out with oryzalin. In one study animals were given 0, 25, 55, or 125 mg/kg/day resulting in maternal (decreased food consumption and body weights) and fetotoxic (increased resorptions and decreased litter size and body weights) effects the mid and high dose levels. The maternal and fetotoxic NOEL = 25 mg/kg/day and the teratogenic NOEL > 125 mg/kg/day. In a repeat study at the same dose levels no effects were reported at any dose level. The maternal, fetotoxic and teratogenic NOELs were greater than 125 mg/kg/day. (USEPA, 1990)

Chronic Toxicity/Oncogenicity - Oryzalin was fed to rats for two years at the dietary levels of 0, 300, 900, and 2700 ppm. The NOEL was established at 300 ppm (15 mg/kg/day) based on decreased hematologic parameters, increased liver and kidney weights, decreased survival, inhibition of growth and increased incidence of thyroid gland and skin tumors in both sexes and mammary tumors in females. The EPA has assigned oryzalin to weight-of-evidence classification "C" (possible human carcinogen) and calculated a cancer potency factor of 3.4×10^{-2} based on the skin tumors. Recently (5/22/91) the EPA, Office of Pesticides and Toxic Substances, Health Effects Division issued a new list of chemicals evaluated for carcinogenic potential where they reported a new cancer potency factor of 1.3×10^{-1} based on the incidence of mammary tumors. The latter value was used in this risk assessment. (USEPA, 1990; USEPA, 1987(b))

Dogs were orally dosed with oryzalin for one year at 0, 1.5, 5, 50, and 500 mg/kg/day resulting in no reported effects at the low dose. Effects reported at the mid and high dose include increased serum cholesterol, decreased levels of adrenal weights, increased relative liver and kidney weights, and decreased alanine transaminase. The NOEL and LOEL were 5 and 50 mg/kg/day, respectively. The 5 mg/kg/day value was used by the EPA to calculate the RfD value. (USEPA, 1990)

Mice were fed 0, 500, 1350, or 3650 ppm oryzalin per day for two years. Effects observed at the mid and high dose groups include decreased weight of uterus and ovaries. The systemic NOEL was 500 ppm or 75 mg/kg/day. No treatment related oncogenic effects were reported at any dose level. (USEPA, 1990; USEPA, 1987(b))

Mutagenicity - Oryzalin was tested in a variety of *in vitro* assays and *in vivo* assays. Results indicate that it does not cause gene mutation (Ames assay) or DNA damage (unscheduled DNA synthesis using rat hepatocytes). In an *in vivo* sister chromatid exchange in bone marrow of chinese hamsters oryzalin tested positive by the intraperitoneal route but negative by the oral route. Oryzalin also tested negative for chromosome aberration in an *in vivo* dominant lethal assay. Oryzalin was considered not to be mutagenic in this assessment since the i.p. route is an unlikely route of exposure. (USEPA, 1990; CDFA, 1989(a))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
XL-2G	3,750 (III)	>2,000 (III)	irritant (III)*	slight (III)	(III)+
Surflan	>595 (III)	>2,380 (III)	slight (III)	slight (III)	>2.83 (1 hour) (III)

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.20 Oxadiazon

Oxadiazon is a selective pre- and early post-emergence contact herbicide of the oxadiazole family. It controls many annual and broadleaf weeds by killing the young weed seedlings as they come into contact with the herbicide during germination. The technical material is a colorless crystalline solid. Oxadiazon has a low tendency towards volatilization and has a moderate potential to bioaccumulate based on the Henry's law constant and large K_{ow}, respectively. It has very low solubility in water and is moderately soluble in ethanol and acetone. Oxadiazon is strongly adsorbed by soil colloids and humus with very little leaching. Based on its immobility in soil, low water solubility, and high organic carbon partition coefficient, oxadiazon would not have a tendency to migrate. It is relatively persistent in the environment with a soil half-life of up to one year.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to oxadiazon exposure.

Laboratory Generated Data

Acute Toxicity - The acute toxicity of Oxadiazon appears to be relatively low. Acute oral LD_{50} values reported range from 3,500 (rat) to 8,000 mg/kg. This is further supported by acute dermal LD_{50}s of >2,000 (rabbit) and >8,000 (rat) mg/kg. The Caltrans DEIR reports rat acute inhalation LC_{50}s for technical grade Oxadiazon to be 10 mg/l for mice and 37 mg/l in rats. The technical material is a mild/slight eye an skin irritant and is reported not to be a sensitizer in guinea pig studies. (Hartley and Kidd, 1987; Jones & Stokes, 1991)

Subchronic Toxicity - Two subchronic studies were reported in the literature. Rats exposed to 25, 100, or 1000 mg/kg/day all exhibited signs of systemic toxicity. Observations included organ weight variations, decreased body weight, pathology of the liver and kidney, and alterations hematology parameters. Dogs exposed for 90 days reportedly had increased liver and thyroid weight at 250 mg/kg/day; while no effects were observed at 100 mg/kg/day. (Jones & Stokes, 1991)

Reproduction/Developmental Toxicity - Two reproduction studies were carried out in rats using oxadiazon. In the first study performed fetal development was adversely effected at 200 ppm (10 mg/kg/day, decreased litter size and increased stillborns and post-natal mortality) while parental toxicity was noted at 400 ppm. NOELs were 100 ppm (5 mg/kg/day) for reproductive effects and 200 ppm for parental toxic effects. In the second study, two generations of rats were exposed to oxadiazon levels up to 200 ppm. No treatment related effects were observed at any dose level, NOEL >200 ppm (10 mg/kg/day). (USEPA, 1990; CFDA, 1990(a))

Three rat teratology studies were performed. All exhibited developmental effects at levels below maternal toxic levels. In the most recent study rats received 0, 3, 12 or 40 mg/kg/day resulting in increased post implantation loss, decreased fetal weight and increased incidence of delayed ossification at the top dose, no maternal toxicity was reported. The developmental NOEL = 12 mg/kg/day and the maternal NOEL >40 mg/kg/day. (USEPA, 1990; CDFA, 1990(a))

Rabbits were given 0, 20, 60, or 180 mg oxadiazon/kg/day resulting in maternal decreased weight gain and increased postimplantation loss at the top two doses. The maternal and developmental NOEL = 20 mg/kg/day. (CDFA, 1990(a))

Chronic Toxicity/Oncogenicity - Rats were fed dietary levels of 0, 10, 100, 1000, or 3000 ppm oxadiazon for two years resulting in increased liver weight and serum proteins at 100 (5 mg/kg/day); effects observed at 1000 ppm were hepatotoxicity, hemolytic anemia, body weight decrease, decreased food consumption, increased kidney weight, and pigment nephrosis. The systemic NOEL established for this study and used by the EPA to calculate the RfD is 10 ppm or 0.5 mg/kg/day. Liver neoplasia was reported down to 1000 ppm. (USEPA, 1990; CDFA, 1990(a))

A second chronic/oncogenicity study was conducted in which rats were fed 0, 3, 10, 100, or 1000 ppm. The chronic NOEL was 100 ppm (5 mg/kg/day) based on decreased body gain and increased liver weights at 1000 ppm. Benign neoplastic hepatic nodules were observed in males in a dose related manner at 100 ppm and above and hepatocellular carcinomas were observed at 1000 ppm in both sexes. (CDFA, 1990(a))

Dogs were given 0,5, 20, 60, or 200 mg oxadiazon/kg/day for one year. The NOEL was established at 20 mg/kg/day based on increased liver weights at 60 mg/kg/day and above. (CDFA, 1990(a))

Three mouse oncogenicity studies were performed. The first maintained mice for two years on 0, 300, 1000, or 2000 ppm oxadiazon. Liver neoplasms were observed in all dose levels. Adverse effects of the eye vasculature, blood, and liver were also observed at all treatment levels. The second study used the same dose levels with the exception of the addition of 100 ppm as the lowest level. Results were the same as previously stated with the systemic and oncogenic NOEL <100 ppm (15 mg/kg/day). (CDFA, 1990(a))

The third mouse study used levels of 0, 3, 10, 100, and 1000 ppm. Systemic toxic effects observed at 100 ppm or higher included increased organ weights, anemia, liver lesions, and hepatocellular necrosis. Hepatocellular adenomas and carcinomas were

observed in males at ≥ 100 ppm and in females at 1000 ppm. The systemic and oncogenic NOELs equal 10 ppm (1.5 mg/kg/day).

The EPA presently considers oxadiazon to be a class "C" (formerly Group "B2") oncogen (possible human carcinogen) and has calculated a cancer potency factor of 1.4×10^{-1} based on the male and female mouse liver tumors. (US EPA, 1991(a); CDFA, 1990(a))

Mutagenicity - Oxadiazon was tested in many *in vitro* and *in vivo* assays. Reported results indicate that the pure (>95.5%) technical material does not cause gene mutation (Ames and mouse lymphoma assays), chromosome aberration (*in vitro* CHO cytogenetic assay), or DNA damage (*E. coli* and unscheduled DNA synthesis using rat hepatocytes). However, oxadiazon impurities and 90% technical material do cause gene mutation in the Ames assay, do not cause chromosome aberration in an *in vivo* mouse micronucleus assay, do not cause DNA damage in *E. coli* or yeast but do cause DNA damage in the cell transformation assay. (CDFA, 1990(a))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Ronstar G	>5,000 (IV)	>2,000 (III)	mild (III)	moderate (II)	>200 (nominal) (IV)
Ronstar 50WP	>5,000 (IV)	>2,000 (III)	irritant (II)*	severe (II)	>1.8 (II)

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

2.3.21 Picloram

Picloram is selective systemic postemergence herbicide of the picolinic acid family which controls woody plants and broadleaf weeds. It is absorbed by the roots and foliage, translocated to the rest of the plant where it acts as a growth regulator. The technical material is a colorless powder with a chlorine-like odor and decomposes at 215° without melting. Picloram has a low potential to volatilize or bioaccumulate based on Henry's law constant and the K_{ow}, respectively. It is slightly soluble in water and of low solubility in ethanol. Picloram is a relatively mobile herbicide and given the physical data would tend to leach and migrate through the soil. It is relatively non to highly persistent in soils depending on the moisture content, but degrades rapidly in water.

Human Data

In volunteer studies, exposure during applications of picloram are reportedly extremely low and far below the reference dose. The dermal penetration rate for a 12 hour exposure has been determined to be 0.2%. Following oral exposure, picloram is reported to be excreted unchanged within two to three days (Dow Chemical, undated(f)).

Laboratory Generated Data

Acute Toxicity - Technical picloram (acid) is only slightly toxic to laboratory animals by the oral and dermal routes but severely toxic via the inhalator route of exposure. Acute oral LD_{50} values vary from 2,000 mg/kg (rabbits), 2,000-4,000 (mice), 3,000 mg/kg (guinea pig), 4,012 to >5,000 mg/kg (female and male rats) to 6,000 mg/kg (chicks). An acute rabbit dermal LD_{50} was reported of >2,000 mg/kg. The acid is slightly irritating to the eyes of laboratory rabbits, essentially non-irritating to the skin, and not a sensitizer in guinea pig studies. Results of an acute inhalation would place the acid form of picloram in Toxicity Category I (severely toxic) with an acute rat LC_{50} of >0.035 mg/l. (USEPA, 1988(g); Blewett, 1991)

The potassium salt is also slightly toxic by the oral and dermal routes (skin and eye irritation) but moderately toxic via the inhalation route. The acute oral rat LD50 is >5,000 mg/kg (males) and 3,536 mg/kg (females). The rabbit dermal LD_{50} is >2,000 mg/kg and the inhalation LC_{50} is >1.5 mg/l. It is slightly irritating to the eyes of laboratory rabbits, non-irritating to the skin but was positive as a skin sensitizer in guinea pig studies. (USEPA, 1988(g) and Blewett, 1991)

Subchronic Toxicity - Two rat 90 day feeding studies were performed one in 1962 and the second in 1982. Both provided similar results; slight liver effects at 150 mg/kg/day and above and a NOEL of 50 mg/kg/day. (USEPA, 1990; CDFA, 1987)

A six month dog feeding study is available for picloram which establishes a NOEL at 7 mg/kg/day, based on increased liver weight at 35 mg/kg/day and above. This value was used by the EPA to calculate the RfD value. (USEPA, 1990; Blewett, 1991)

Reproduction/Developmental Toxicity - A three generation reproduction study resulted in a NOEL of 50 mg/kg/day, based on reduced fertility at the highest dose tested of 150 mg/kg/day (USDI, 1991).

Some developmental effects were observed in a rat teratology study but at doses which were also maternally toxic, 750 mg/kg/day. No fetotoxic NOEL level was determined and this study was originally classified by the EPA as supplemental. The IRIS printout shows that this has been upgraded to core grade minimum. (USEPA, 1990; CDFA, 1987)

In a rabbit teratology study a small number of fetuses showed abnormalities such as missing ribs, omphalocele, and hypoplastic tail. Historical control data are required to evaluate the observed abnormalities (USEPA, 1988(g)). The rabbit teratology study is considered to be a data gap.

Chronic Toxicity/Oncogenicity - Two rat chronic/oncogenicity studies were performed using picloram. One performed by the NTP was determined to be to short, no maximum tolerated dose (MTD), only two dose levels, etc. However, the study was rated as weakly positive in females for liver oncogenic effects at the high dose. A second study tested rats for two years at the levels of 0, 20, 60, and 200 mg/kg/day resulting in toxic changes in the liver at 60 mg/kg/day, NOEL = 20 mg/kg/day. No treatment related oncogenic effects were observed. The EPA has classified picloram as a Group "D" oncogen (not classifiable as to human carcinogenicity) and is scheduled for evaluation of

oncogenic potential. A cancer potency factor of $3x10^{-3}$, reported by the USDI (1991), was used in this risk assessment. (USEPA, 1988(g))

The oncogenic potential of picloram was tested in mice by the NTP. No oncogenic effects were reported. This study also as no MTD and has only two dose levels. The Agency is requiring that both the mouse and the rat study be repeated. (USEPA, 1988(g))

Mutagenicity - Picloram was tested in a variety of *in vitro* assays and *in vivo* assays. Results reportedly indicate that it does not cause gene mutation in the Ames, point mutation with *A. nidulans*, CHO forward mutation or *in vivo* drosophila assays but was positive in the "disk" assay with *S. coelicolor* (an unvalidated assay). Picloram caused chromosome aberrations in a CHO cytogenetics assay but was negative in an *in vivo* bone marrow cytogenetics assay. It was also reported to be positive for DNA damage in an *in vitro* sister chromatid exchange in CHO cells and negative in a mitotic disjunction assay with *A. nidulans*. (CDFA, 1987; USEPA, 1988(g))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Tordon K	(IV)+	>2,000 (III)	moderate (II)	moderate (II)	(III)+
Tordon 22K	(IV)+	>2,000 (III)	moderate (II)	moderate (II)	(III)+
Tordon RTU	>5,000 (IV)	>3,980 (III)	moderate (II)	mild (III)*	(III)+
Tordon 101R	2,598 (III)	>2,000 (III)	moderate (II)	moderate (II)	(IV)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.22 Pronamide

Pronamide is a selective pre- and post-emergence herbicide of the benzamide family. It is absorbed by the roots of certain broadleaf weeds and grasses and translocated throughout the plant where it inhibits photosynthesis. The technical material is a colorless odorless powder. Pronamide has a low tendency towards volatilization and a slight potential to bioaccumulate based on the Henry's law constant and K_{ow}, respectively. It has low water solubility and is soluble in aliphatic and aromatic solvents. Laboratory studies indicate that pronamide would be considered relatively immobile and not prone to leach or migrate. Pronamide is not very persistent in the environment with a soil half-life of 30 days.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to pronamide exposure.

Laboratory Generated Data

Acute Toxicity - Pronamide is only slightly toxic to laboratory animals. Acute oral LD_{50} values range from 5,620 to 16,000 mg/kg for the rat and > 10,000 mg/kg for the dog. This low order of toxicity is supported reported by rabbit dermal LD_{50} values of >3,160 and > 10,000 mg/kg and an inhalation LD_{50} of >3.2 mg/l. The technical material is reported to be only slightly irritating to the eyes and skin of laboratory rabbits and nonsensitizing to guinea pigs. (USEPA, 1986(b); USDHHS, 1980; Hartley and Kidd, 1987)

Subchronic Toxicity - In a 90 day rat feeding study the NOEL was established at 2.5 mg/kg/day based on reported increased liver/body weight ratios in males and increased liver weights in females at 7.5 mg/kg/day (LOEL). (USEPA, 1990)

A 90 day dog study was conducted using pronamide resulting in increased liver weights at the LOEL of 101.25 mg/kg/day; establishing the NOEL at 33.7 mg/kg/day. (USEPA, 1990; USEPA, 1986(b))

Reproduction/Developmental Toxicity - No parental or reproductive effects were observed in a three generation reproduction study up to and including 15 mg/kg/day, the highest dose tested (NOEL > 15 mg/kg/day). This study has been identified by the EPA as a data gap. (USEPA, 1990)

Results from an acceptable rat teratology study were not available and has been declared a data gap by the EPA. (USEPA, 1986(b))

The only effects observed in a rabbit teratology study were anorexia and punctate vacuolation of the hepatocytes at 20 mg/kg/day (LOEL) establishing the maternal and developmental NOELs at 5/mg/kg/day. (USEPA, 1990; USEPA, 1986(b))

Chronic Toxicity/Oncogenicity - In a chronic feeding study rats fed 0, 1.5, 5, or 15 mg/kg/day exhibited no chronic or oncogenic effects. This study was classified as core grade supplementary due to lack of an MTD. A chronic/oncogenicity rat feeding study has been identified by the US EPA as a data gap in the pronamide registration package. (USEPA, 1990; USEPA, 1986(b))

Pronamide was administered to dogs in the diet for two years at the levels of 0, 30, 100, or 300 ppm resulting in no observed treatment related effects of any kind (NOEL >300 ppm or 7.5 mg/kg/day). The EPA selected this NOEL to calculate the RfD value. (USEPA, 1990)

Two chronic mouse studies have been performed. In the first, mice were exposed to 0, 130 or 260 mg/kg/day for 18 months reportedly resulting in a statistically significant increase in the incidence of hepatocellular carcinomas in males of both dose groups. In the second study male mice were fed 0, 2.6, 13, 65, or 325 mg/kg/day for 2 years also resulting in a significant dose related increase in the incidence of hepatocellular carcinomas. The EPA classifies pronamide as a Group "C" oncogen (possible human carcinogen) and has calculated a cancer potency factor of 1.6×10^{-2}. (CDFA, 1990(b); USEPA, 1986(b))

Mutagenicity - Pronamide was tested in a variety of *in vitro* assays and one *in vivo* assay. Results reported indicate that it does not cause gene mutation (Ames and CHO assays), chromosome aberration (*in vivo* mouse cytogenetic assay) or DNA damage (B. subtilis Rec and unscheduled DNA synthesis using rat hepatocytes). (CDFA, 1990(b))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Kerb 50W	(III)+	(IV)+	slight (III)	irritating (III)*	(III)+

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

2.3.23 Sethoxydim

Sethoxydim is a selective systemic postemergence herbicide of the cyclohexene or oxime family. It is absorbed by plant foliage and translocated to the roots in annual and perennial grasses. The technical material is an amber oily liquid with a boiling point >90°C. Sethoxydim has low tendency towards volatilization and a slight potential to bioaccumulate based on the Henry's law constant and the K_{ow}, respectively. Sethoxydim has low water solubility but is readily soluble in organic solvents. Based on the low organic carbon partition coefficient (K_{oc}) and water solubility it would be expected to leach and migrate in the soil. However, in studies performed with sethoxydim there was no evidence of leaching (or its metabolites). No residues were found past 8 inches. It is not very persistent in environments high in moisture. Persistence increases in drier environments.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to sethoxydim exposure.

Laboratory Generated Data

Acute Toxicity - Sethoxydim is only slightly toxic to laboratory animals with acute oral LD_{50} values of 2,676 and 3,125 mg/kg/for female and male rats respectively. This low order of toxicity is supported by a rabbit dermal LD_{50} of 5,000 mg/kg, a rat dermal LD_{50} of >5,000 mg/kg and an acute rat inhalation LC_{50} of >6 mg/l. The technical material is reported to be nonirritating to the eyes and skin of laboratory rabbits and was not a sensitizer in guinea pig studies. (Hartley and Kidd, 1987; BASF undated(m-r))

Subchronic Toxicity - Two 90 day rodent studies were performed resulting in reports of liver effects at 45 and 135 mg/kg/day (the LOEL for rats and mice, respectively). The NOEL for rats was established at 15 mg/kg/day (300 ppm) and for mice, 45 mg/kg/day (300 ppm). (Jones & Stokes, 1991; BASF undated(s))

Dogs were fed sethoxydim for 6 months at the following dietary levels; 0, 2, 20, or 200 mg/kg/day. Liver toxicity and nonspecific anemia were observed at the top dose. No treatment related effects were reported in the 20 mg/kg/day dosage group. (USEPA, 1990; Jones & Stokes, 1991)

Reproduction/Developmental Toxicity - In a two generation reproduction study parental effects reported at the high dose (162 mg/kg/day, raised from 54 mg/kg/day) were decreased body weight gain, increased male thyroid weights and increased female adrenal weights. There were no effects on reproductive parameters at any dose level. The parental NOEL was 18 mg/kg/day and the reproductive NOEL >162 mg/kg/day. (USEPA, 1990; BASF, Undated(aa); Jones & Stokes, 1991)

No developmental effects were observed in a rat teratology study at any dose level (0, 40, 100, and 250 mg/kg/day). Maternal toxic effects of reduced adrenal weights were reported in the top two dose groups. NOELs were 40 mg/kg/day (maternal) and >250 mg/kg/day (developmental). (USEPA, 1990; BASF, Undated(w); Jones & Stokes, 1991)

Sethoxydim did not produce any developmental or maternal toxic effects in a rabbit teratology study at 160 mg/kg/day (NOEL). Maternal and fetotoxic effects observed at the LOEL of 480 mg/kg/day were severe weight loss, mortality, increased abortion rate and reduction in the number of litters and viable fetuses. Developmental effects noted at 480 mg/kg/day include an increased number of skeletal and visceral abnormalities and reduced fetal weights. (USEPA, 1990; BASF, Undated(x); Jones & Stokes, 1991)

Chronic Toxicity/Oncogenicity - No oncogenic or systemic toxic effects were observed in rats fed up to 360 ppm (18 mg sethoxydim/kg/day) in the diet for two years. (USEPA, 1990; BASF, Undated(t))

Observations reported in a one year dog study identify a systemic NOEL of 300 ppm (average dose 9 mg/kg/day) based on mild anemia observed in males at 600 and 3600 ppm. The target organs were bone marrow and the liver. The NOEL from this study was used by the EPA to calculate the RfD value. (USEPA, 1990; BASF, Undated(u))

Mice exposed to sethoxydim in the diet for two years were reported to have developed non-neoplastic liver lesions at 54 mg/kg/day; the systemic NOEL = 18 mg/kg/day. No evidence of a treatment related oncogenic effect was observed at any dose level. (USEPA, 1990; BASF, Undated(v); Jones & Stokes, 1991)

Mutagenicity - Sethoxydim was tested in a variety of *in vitro* assays. Results reported indicate that it does not have any mutagenic activity in *E. coli, B. subtilis, S. typhimurium* or in a host mediated study in mice with *S. typhimurium* (Jones & Stokes, 1991; BASF, Undated(i)).

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Poast	4,900 (III)	>4,000 (III)	moderate (II)	moderate (II)	>7.6 (III)

2.3.24 Sulfometuron methyl

Sulfometuron methyl is a broad-spectrum systemic pre- or post-emergence herbicide of the sulfonylurea family. It is absorbed by the foliage, translocated throughout the plant where it stops cell division in the growing tips by mitotic inhibition. The technical material is a colorless odorless solid. Sulfometuron methyl has a low potential for volatilization and bioaccumulation based on the Henry's law constant and K_{ow}, respectively. It has low solubility in water and ethanol but is slightly soluble in acetone. It is a relatively mobile compound and would tend to leach and migrate through the soil. Sulfometuron methyl is not very persistent in the environment with a soil half-life of approximately 30 days and an aquatic half-life estimated to be a couple of days.

Human Data

No studies were located in the literature which reported adverse or the absence of effects in humans due to sulfometuron methyl exposure.

Laboratory Generated Data

Acute Toxicity - The following acute toxicity data is for Oust® by DuPont, a 75% sulfometuron methyl formulation which is only slightly toxic to laboratory animals with an acute oral rat LD_{50} of >5,000 mg/kg. This low order of toxicity is also supported by dermal LD_{50} values of >2,000 and >8,000 for female and male rabbits and an acute rat inhalation LC_{50} >5 mg/l. The material is reported to be slightly irritating to the eyes of laboratory rabbits, nonirritating to the skin, and not a sensitizer in guinea pig studies. (DuPont, Undated)

Subchronic Toxicity - Rats were fed 75% sulfometuron methyl for 90 days resulting in a NOEL of 50 mg/kg/day based on elevated white blood cell counts at 250 mg/kg/day, the highest dose tested and the LOEL. (Jones & Stokes, 1991)

No toxicity was observed in rabbits following dermal application of up to 2,000 mg/kg/day of the formulated product for 21 days (Jones & Stokes, 1991).

Reproduction/Developmental Toxicity - In a two generation reproduction test, no reproductive effects were observed in rats receiving sulfometuron methyl in their diet at 500 ppm (25 mg/kg/day). Maternal toxicity and a decreased number of viable pups were reported at 5,000 ppm (LOEL, 250 mg/kg/day). (DuPont, Undated; Jones & Stokes, 1991)

No maternal or fetotoxic effects were observed in a rat dietary teratology study at the 50 mg/kg/day (NOEL) level. Effects observed at the LOEL of 250 mg/kg/day were decreased maternal and fetal body weight gain. No teratogenic effects were seen at any dose level. (Jones & Stokes, 1991)

The maternal, fetotoxic and teratogenic NOEL in a rabbit teratology study was 300 mg/kg/day, the highest dose tested. (Jones & Stokes, 1991)

Chronic Toxicity/Oncogenicity - A NOEL was reported of 50 ppm sulfometuron methyl (2.5 mg/kg/day) for a 2 year dietary exposure in rats. The Oust MSDS reports that results of animal tests were negative for carcinogenicity.

Dogs were fed 75% sulfometuron methyl in the diet for one year at 0, 5, 25, and 125 mg/kg/day resulting in reports of decreased red blood cell counts, hemoglobin and hematocrit, and an increase in liver weights at 25 mg/kg/day (LOEL). The NOEL was established at 5 mg/kg/day. The rat study has the lowest NOEL but the reference is obscure. 5 mg/kg/day was used to calculate the RfD instead of the 2.5 mg/kg/day value reported for the rat study. (Jones & Stokes, 1991)

Mutagenicity - Very little information was available on the genotoxicity of sulfometuron methyl. It was tested in two *in vitro* assays. Reported results indicate that it does not cause gene mutation in the Ames or CHO assays. (Jones & Stokes, 1991)

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Oust	>5,000 (IV)	>8,000 (males) >2,000 (females) (III)	slight (III)	non-irritating (IV)	5 (III)

2.3.25 Triclopyr

Triclopyr is a selective systemic postemergence herbicide of the pyridine family used to control woody plants and broadleaf weeds. It is absorbed by the roots and foliage, translocated throughout the plant and concentrated in the meristematic tissue, where it induces auxin type responses in growing plants. The technical material is a fluffy colorless solid. Triclopyr has a very low tendency toward volatilization or bioaccumulation based on the Henry's law constant and K_{ow}, respectively. It is only slightly soluble in water and benzene but very soluble in ethanol. The compound is relatively mobile in soil and may be easily removed by runoff due to the rapid rate of desorption (Jones & Stokes, 1991). Field studies show that triclopyr is fairly rapidly degraded and that remaining residues are located within 12 inches of the surface. Triclopyr is not persistent in the environment with the soil half-life averaging 30 days and the water half-life expected to be short due to rapid hydrolysis.

Human Data

Reportedly more than 600 individuals in six European countries and the USA have been exposed to Garlon 3A during manufacture, formulation, and mixing/spraying in field tests. The only incidents reported involving irritation were two men in France who had slight and transient eye irritation from direct contact with the spray mist. One mishandled the product and had a mild skin irritation. (Dow/Elanco, undated (d))

No adverse effects were observed in humans following a single oral exposure to 0.5 mg/kg (Jones & Stokes, 1991).

Laboratory Generated Data

Acute Toxicity - Triclopyr is slightly toxic to laboratory animals with acute oral rat LD_{50} values of 630 to 730 mg/kg, females and males respectively. This low order of toxicity is supported by an acute dermal rabbit LD_{50} >2,000 mg/kg. The technical material is slightly irritating to the eyes of laboratory rabbits, nonirritating to the skin. No information was available on the acute effects of inhalation exposure to the active ingredient triclopyr or skin sensitization. (Blewett, 1991)

Subchronic Toxicity - In a 90 day rat feeding study with triclopyr, males reportedly exhibited decreased food consumption, body weight gain and absolute liver weight at 100 mg/kg/day (LOEL), establishing a NOEL of 30 mg/kg/day. (Jones & Stokes, 1991)

Dogs were fed triclopyr for six months at 0, 0.1, 0.5, or 2.5 mg/kg/day resulting in a NOEL of 0.5 mg/kg/day based on a slight decrease in phenolsulfon-phthalein excretion rate at 2.5 mg/kg/day. (CDFA, 1990(f))

Triclopyr was administered to monkeys via nasogastric intubation for 28 days at 0, 10, 20, or 30 mg/kg/day resulting in no reports of significant effects at any dose level. (CDFA, 1990(f))

Reproduction/Developmental Toxicity - A three generation rat reproduction study was performed using 0, 3, 10, and 30 mg triclopyr/kg/day. No parental or reproductive effects were reportedly observed up to and including 30 mg/kg/day, highest dose tested. (CDFA, 1990(f))

Triclopyr was tested for teratogenic effects in rats at 0, 50, 100, or 200 mg/kg/day resulting in a decrease in maternal body weight gain and food consumption at 100 mg/kg/day, retarded ossification of skull bones at 100 mg/kg/day, and no teratogenic effects at any dose level. The established NOEL's are 50 mg/kg/day for maternal and fetotoxic effects and >200 mg/kg/day teratogenic effects. (CDFA, 1990(f))

A rabbit teratology study was performed using 0, 10, 25, and 75 mg/kg/day resulting in maternal mortality at 75 mg/kg/day (NOEL = 25 mg/kg/day), no developmental effects were observed at any dose level (NOEL >75 mg/kg/day). (CDFA, 1990(f))

Chronic Toxicity/Oncogenicity - Rats were fed triclopyr in the diet for two years at 0, 3, 12, or 36 mg/kg/day resulting in a systemic NOEL of 3 mg/kg/day based on increased kidney weights in males. In a previous study no adverse effects were reported at levels up to and including 30 mg/kg/day (HTD). No evidence of oncogenic effects were reported for either study. (CDFA, 1990(f))

Triclopyr was fed to dogs for one year at 0, 0.5, 2.5, or 5 mg/kg/day establishing a NOEL of 2.5 mg/kg/day. Effects noted at 5 mg/kg/day (LOEL) were decreased phenolsulfonphthalein excretion, minimal increases in serum nitrogen and creatine and microscopic pigment deposits in the proximal tubular epithelial cells. The NOEL for this study was used to calculate the RfD value. While the NOEL for the six month dog study was lower, it was not used because the six month study is supplemental data used as a pilot for the one year study. (CDFA, 1990(f))

In an early mouse oncogenicity study all triclopyr fed mice developed lung tumors (240 ppm, HDT). The significance of this finding and its relationship to triclopyr exposure reportedly could not be decided. A second study was conducted at levels of 0, 50, 250, and 1250 ppm for 22 months. Reported results were negative for oncogenic effects. Systemic toxic effects observed were decreased body weight gain, increased water consumption, decreased specific gravity of urine at the 1250 ppm level, and enlarged thymus in males at 250 and 1250 ppm. The NOEL for systemic effects equals 50 ppm (5.55 mg/kg/day). (CDFA, 1990(f))

Mutagenicity - Triclopyr was tested in a variety of *in vitro* assays and *in vivo* assays. Results reported indicate that it does not cause gene mutation (Ames and *in vivo* host-mediated assays), chromosome aberration (*in vivo* mouse dominant lethal, *in vivo* rat cytogenetics, *in vivo* mouse micronucleus assay), or DNA damage (unscheduled DNA synthesis and *B subtilis* Rec assay). Observations reported indicate triclopyr produced a weak positive for chromosome mutation in a rat dominant lethal assay. Considering the negative results from all the other tests, the most that can be said is triclopyr may induce a possible adverse effect for chromosome mutation. (CDFA, 1990(f))

Formulation Toxicity

Formulation	Rat Oral LD_{50} (mg/kg)	Rabbit Dermal LD_{50} (mg/kg)	Eye Irritation	Skin Irritation	Rat Inhalation LC_{50} (mg/l)
Garlon 3A	1,847-2,574 (III)	>3,980 (III)	corrosive (I)	severe (I)	(IV)+
Garlon 4	1,338-1,581 (III)	>2,000 (III)	slight (III)	slight (III)	(III)+
Crossbow	2,398-2,589 (III)	>2,000 (III)	moderate (II)	slight (III)*	5 (III)

* Toxicology information alone insufficient to assign to a Toxicity Category. Determination made according to label language.

\+ No information available. Determination made according to label language.

3.0 Exposure Assessment

3.1 Introduction

Exposure scenarios for two populations were examined, WSDOT workers and the public. Public dose estimates are based on recreational activities performed at or near the treated site and residential exposure. Three routes of exposure were initially considered for each exposure pathway, inhalation, dermal, and oral. Those exposure routes or pathways which were deemed to have a very low potential for exposure or an insignificant amount of exposure were not considered in this risk assessment. Inhalation has been shown to be an insignificant route of exposure under conditions similar to those expected here (Oshita, et al., 1988; Jones & Stokes, 1991; USDI, 1991; USFS 1988). Dermal exposure to children, and inadvertent ingestion of contaminated soil by children was not evaluated. There is low potential for exposure via these pathways because young children are unlikely to play along roadsides.

The risk associated with accidental exposure to concentrated solutions of herbicides was also not examined. The purpose of this risk assessment is to analyze the potential for a chemical to cause a harmful effect under normal usage by WSDOT when being used to control unwanted vegetation along roadsides. Routine WSDOT practices greatly minimize the chance of the public being exposed. WSDOT usually mixes the concentrate with its carrier at the maintenance yard, their largest spray tank contains only 70 gallons and all workers are trained. Occasionally, when the distance to the spray site is a great distance, WSDOT workers carry additional concentrate for mixing on site. (It is possible to carry two 70 gallon tanks and one 50 gallon tank for a maximum of 190 gallons of concentrate.) These precautions lower the potential for accidental exposure to highly concentrated solutions.

Each exposure scenario has a set of parameters which uniquely characterizes that scenario. For instance:

1) The frequency of exposure is dependent upon the activity. WSDOT workers can be exposed to herbicides as often as 14 to 16 days every month all year while the berry picker is exposed maybe 2 to 3 days per month only during the months berries are ripe.

2) The duration of exposure is dependent upon the activity. WSDOT spray operators work an average of 4 to 10 years before advancing to a supervisory level, where they will not spray or handle material. The berry picker is assumed to be a resident who lives near the berry patch from 9 to 30 years.

3) The dose rate, calculated from field monitoring studies, is different for each WSDOT worker exposure scenario.

4) WSDOT workers are usually wearing more protective clothing than the passing berry picker but are exposed to a higher concentration of herbicide than the berry picker, who may be exposed as a result of drift.

5) The residue loading rate, calculated from field studies, is different for lettuce, beans, and berries, as is the ingestion rate.

These parameters are used to estimate the amount of each herbicide a person might be exposed to in a single day (single day dose, SDD) and the dose received over a lifetime (lifetime average daily dose, LADD).

An average and maximum SDD was calculated for each of the 25 herbicides in question. An average and maximum LADD was calculated for the six potential carcinogens (2,4-D, bromacil, oryzalin, oxadiazon, picloram, and pronamide). The SDD will be used to characterize the risk of potential systemic or non-cancer effects. The LADD will be used to characterize the lifetime risk of potential cancer effects. Due to the limited data, a quantitative risk assessment was not done on adjuvants, inerts, and fertilizers. (A qualitative analysis of risk is presented for adjuvants, inerts, and fertilizers in the risk characterization section of this chapter.)

The parameters used to calculate the average doses include the historical WSDOT average application rate for each herbicide or low label rate if historical data were not available,

and average or reasonable estimates of duration of exposure, frequency of exposure, area exposed, amount of drift, number of berries consumed, etc. The parameters used to calculate the maximum dose levels include, the maximum recommended label rates and more conservative exposure assumptions. All assumptions made for each exposure scenario are clearly defined in the following pathway assessments.

Without the luxury of having measured environmental concentrations of the 25 herbicides under evaluation, the human dose must be calculated using exposure levels estimated by means of mathematical modeling. An ideal model is a study, usually chosen from the literature, which measures pesticide levels on the same or similar population, performing the same or similar activity. A sufficient number of parameters need to be monitored and adequately reported in order to normalize the results to one pound active ingredient per acre. (The active ingredient is the compound in the formulated product which has herbicidal activity.) The resulting equation is used to estimate environmental concentrations or actual dose levels, for populations performing similar tasks using herbicides at different application rates.

Each of the models used in this risk assessment is thoroughly explained in the following pages. From this available information, models were chosen for use in this assessment. (USDI, 1991; USFS, 1988; Jones and Stokes, 1991)

Bioavailability:

Most herbicides are poorly (less than 10%) absorbed through the skin. This is not hard to understand when you consider that one major purpose of the skin is to act as a barrier. Absorption depends on the fat-solubility of the chemical:

- Water-soluble compounds typically do not cross to a significant degree.
- Fat-soluble compounds can cross intact skin by passive diffusion to produce toxic effects.

The percent of herbicide absorbed through the skin was taken from dermal absorption studies when available. Otherwise, a dermal absorption rate was estimated using the physical and chemical characteristics of each chemical.

The percent of 2,4-D absorbed by the skin was reported by Feldman and Maibach to average 5.8% on the forearm and to be 2 to 6 times higher on the face, scalp and neck in a study in which the chemical was applied in acetone and left on the skin for 24 hours. Shipp derived an average dermal absorption rate of 10% for 2,4-D using the data of Feldman and Maibach and assuming that two-thirds of the exposed skin will absorb at the 5.8% rate and one third at a rate 3 times higher, 17.4%. (Jones & Stokes, 1991)

Shipp estimated an average dermal absorption rate for glyphosate of 3.5% based on unpublished data from Monsanto Company, 1984 (Jones & Stokes, 1991).

Carmichael measured dermal absorption of Garlon 4 (triclopyr) in human volunteers. The herbicide was applied undiluted to the skin and left in place for 8 hours. Recovery in the urine indicated that dermal absorption was 1.65% (Jones & Stokes, 1991).

Lavy reported a dermal penetration rate of 0.48% for picloram and Draper and Street reported a 5% dermal absorption rate for dicamba (USDI, 1991).

Lavy reported a dermal absorption rate of 6.4% for 2,4-DP (USFS, 1988).

Jones & Stokes (1991) estimated the following dermal absorption rates:

- Herbicides with low potential for dermal absorption were assumed to absorb at a rate of 1%. These herbicides were: bromacil, chlorsulfuron, dicamba, mefluidide, sethoxydim, and sulfometuron methyl.

- Low to moderate dermal absorption rates of 5% were assumed for dichlobenil, diuron, fluazifop-butyl, isoxaben, napropamide, oryzalin, and pronamide.

A dermal absorption rate of 10%, commonly used for herbicides, was assumed for the remaining compounds, benefin, chlorthal dimethyl, imazapyr, mecoprop, metsulfuron methyl, and oxadiazon.

It was assumed for ingestion pathways that 100% of the herbicide residue eaten was absorbed by the body as parent compound (i.e., that no environmental degradation, metabolism, or excretion occurred). The purpose of the gastrointestinal (GI) tract is to absorb nutrients. A compound entering the GI tract must pass through a highly acidic environment, the stomach, and a neutral environment, the intestines. The lipid solubility can differ markedly in these two environments thus increasing the likelihood that some or all of a toxicant will be absorbed. The capacity of the intestine to absorb toxicants is even further magnified because of the very large surface provided by the villi and microvilli, approximately 600-fold. (Klassen, et al., 1986)

3.2 Exposure Estimates

3.2.1 Worker Exposure Estimates

Four exposure scenarios were examined for WSDOT workers during performance of their routine herbicide treatment activities. These scenarios and the activities necessary to complete each task are defined below.

1) Applicator - Applies the herbicide using a truck or tractor mounted spray equipment. Drives the vehicle, operates the spray boom, adjusts the spray nozzles as necessary, and handles equipment breakdown.

2) Mixer/Loader - Transfers the herbicide concentrate from the product container to the mixing tank. Calculates amount of carrier needed (usually water), adds carrier and adjuvants, and operates, cleans, and repairs mixing equipment.

3) Applicator/Mixer/Loader - preforms all of the activities described in 1 and 2 above.

4) Hand Sprayers- Uses a backpack or handheld spray equipment to perform spot spraying or to apply herbicides where the truck mounted equipment is inadequate. Mixes concentrate, carriers, and adjuvants and cleans equipment.

3.2.1.1 Exposure Estimates for WSDOT Workers Using Truck or Tractor Mounted Spray Equipment

Exposure estimates to workers applying herbicides using truck or tractor spray equipment were modeled using the 2,4-D worker exposure study by Nash, et al., (1982). Nash measured the urinary excretion of 2,4-D in 26 ground applicators (10 applicators, 7 mixer/loaders and 9 applicator/mixer/loaders) after a single day exposure applying the herbicide to wheat. Excretion rates were monitored for six days after exposure. Pounds of active ingredient utilized, hours exposed, daily excretion of 2,4-D (in mg/kg) and total exposure (in mg/kg) were reported.

The total exposure was divided by the number of pounds of active ingredient each individual was exposed to, in order to calculate the expected dose received from exposure to one pound of active ingredient, referred to as the dose rate (DR) and reported as mg/kg/lb ai (mg of herbicide per kg of body weight per pound of active ingredient applied). An average of the individual data was used to calculate the average single day doses of applicators, mixer/loaders, and applicator/mixer/loaders applying herbicide using ground (truck or tractor mounted) spray equipment. A value representing the 95.5% upper limit, or two standard deviations, was used to calculate the maximum single day dose.

It should be noted that the doses calculated for these workers represent the absorbed dose resulting from exposure via the oral, inhalation and dermal routes. The actual dose rates (DR) used are presented below:

Urinary Excretion Dose Rates

	Applicator (mg/kg/lb ai)	Mixer/Loader (mg/kg/lb ai)	Applicator/ Mixer/Loader (mg/kg/lb ai)
Average	6.0×10^{-5}	1.8×10^{-4}	1.8×10^{-4}
Maximum	2.4×10^{-4}	4.4×10^{-4}	3.7×10^{-4}

The average single day doses were calculated for each of the three scenarios using the following equation:

$$SDD = DR \times AR \times AC \times DBCF$$

Where: SDD = single day dose.

DR = the dose rate calculated from the 2,4-D field study

AR = the WSDOT average application rate, Table 4-11.

AC	=	the average number of acres a ground crew is capable of spraying in one day, 25, reported by BLM, 1989.
DBCF	=	the dermal bioavailability correction factor (the dermal bioavailability factor of each herbicide divided by the dermal bioavailability factor of the surrogate chemical 2,4- D), Table 4-11.

The maximum SDD's were calculated using the same formula with the following changes:

DR	=	the 95.5% upper limit dose rate calculated from the 2,4-D field study
AR	=	the maximum recommended label application rate, Table 4-11.
AC	=	the maximum number of acres a ground crew is capable of spraying in one day, 50, reported by USDI (1991).

The lifetime average daily dose (LADD) was calculated for the six potential carcinogens using assumptions specific for WSDOT workers. Spray operators remain at the level 1 and 2 positions for an average of 5 years and no longer than 10 years, when they are promoted to a supervisory level and no longer handle material (Berger, 1991). Crews normally spray six to eight months a year, fourteen to sixteen days per month and six to seven hours per day.

The equation used to calculate LADD's for ground mechanical workers:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

Where:	LADD	=	lifetime average daily dose, in mg/kg/day.
	SDD	=	single day dose, avg. and max. as appropriate.
	D	=	number of days per month worker is exposed to herbicide, 14 average and 16 maximum.
	M	=	number of months per year herbicide is applied. Picloram and 2,4-D can be applied all year long but WSDOT workers normally spray 6 to 8 months of each year. Therefore, 6 months was used for the average calculation and 8 months for the maximum. For the remaining four chemicals, the number of months applied was taken from Table 4-11.

Y	=	the number of years a WSDOT worker applies herbicide, 5 for the average and 10 for the maximum.
DY	=	days per year, 365.
YL	=	years per lifetime, 70 years, USEPA (1989(c)).

Tables 4-1 through 4-3 list the average and maximum SDD's and LADD's for WSDOT Applicators, Mixer/Loaders and Applicator/ Mixer/Loaders applying herbicide using a truck or tractor mounted spray equipment. All assumptions made, equations and sample calculations can be found later in this section.

3.2.1.2 Exposure Estimates to WSDOT Workers Using Backpack and Handheld Spray Equipment

Exposure to WSDOT workers applying herbicide to rights-of-way using backpack or handheld spray equipment was estimated using the Harris, article reported by Jones & Stokes (1991). This article, "Exposure of Homeowners, Professional Applicators, and Bystanders to 2,4-D" prepared for the Ministry of Environment in Canada, was apparently not yet published and consequently was not available for review. The following summary of the article was taken from the Caltrans document.

> "Harris, et al., (1990) measured total absorbed dose (by measuring urinary excretion), received by home applicators and professional applicators applying 2,4-D and bystanders living within the household but not applying the pesticide. Home applicators were divided into protective and non-protective apparel groups that applied a liquid formulation of 2,4-D. Protective apparel consisted of clean overalls, gloves, and rubber boots. Of the 11 individuals wearing protective clothing, only two excreted measurable amounts of 2,4-D. One of these individuals had removed his gloves. Urinary excretion of the professional applicators was also measured. However, these individuals were not wearing protective apparel similar to that used by [WSDOT] and total exposure was poorly correlated with total amount of herbicide applied. Therefore, the data on home applicators wearing protective apparel were assumed to be more applicable for this analysis".
>
> "....Of the 11 applicators, only two excreted measurable amounts of 2,4-D. For the nine others with no detectable levels, levels at one-half the detection limit were assumed for this analysis and the mean of all 11 measurements was derived for use as the average dose from one day exposure. The mean dose, normalized to a 1 lb. ai application rate, was estimated as 0.0015 mg/kg/day. It was assumed that the average time of exposure would be 4 hours. The 95% upper bound of the mean, 0.0065 mg/kg/lb ai applied, was used as an estimate of the maximum plausible exposure and it was assumed that this exposure would occur over 6 hours of spraying".

The average SDD's were calculated for WSDOT workers applying herbicides with backpack or handheld spray equipment, using the following equation:

$$SDD = HDR \times HR \times AR \times DBCF$$

Where:

SDD	=	single day dose.
HDR	=	hourly dose rate. Using the value of 0.0015 mg/kg/lb ai calculated from the Harris et al. study and dividing it by an assumed exposure duration of four hours to arrive at 0.00038 mg/kg/hr/lb ai/acre.
HR	=	the number of hours spent applying herbicides per day, 6 hours from a pole of WSDOT workers.
AR	=	the WSDOT average application rate, from Table 4-11.
DBCF	=	the dermal bioavailability correction factor (the dermal bioavailability factor of each herbicide divided by the dermal bioavailability factor of the surrogate chemical 2,4- D), Table 4-11.

The maximum SDD's were calculated using the same equation with the following differences:

HDR	=	hourly dose rate. Using the value of 0.0065 (95% upper bound of the mean) and dividing it by an assumed exposure duration of six hours to arrive at 0.0011 mg/kg/hr/lb ai/acre.
HR	=	the number of hours spent applying herbicides per day, 8 hours from a pole of WSDOT workers.
AR	=	the maximum recommended label application rate, from Table 4-11.

The lifetime average daily doses were calculated using the following equation:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

Where:

LADD	=	lifetime average daily dose.
SDD	=	single day dose, avg. and max. as appropriate.
D	=	days per month the WSDOT workers apply herbicide, 14 days average and 16 days maximum.

M	=	the number of months WSDOT workers apply that individual herbicide per year.
Y	=	the number of years a WSDOT worker sprays herbicides, 5 average and 10 maximum.
DY	=	days per year, 365.
YL	=	years per lifetime, 70 years, US EPA (1989(c)).

Table 4-4 lists the average and maximum SDD's and LADD's for WSDOT workers applying herbicides using backpack or handheld spray equipment. All assumptions made, equations and sample calculations can be found later in this section.

3.2.2 Public Exposure Estimates

Four exposure scenarios were evaluated for the public based on routine activities at or near the treated site. Although WSDOT policies, equipment, and training programs are geared toward minimizing public exposure to herbicides, the potential for direct contact to foliar residues or exposure due to drift does exist. The following pathways represent the most likely routes of public exposure:

1) Ingestion of berries which have received spray drift (adults and children).

2) Ingestion of garden vegetables (lettuce and beans) which have received inadvertant spray drift.

3) Dermal contact from picking berries which have received spray drift.

4) Dermal contact with vegetation which has been sprayed.

The inhalation route was not considered because numerous worker and public exposure studies have consistently shown inhalation to be an insignificant route of exposure. This decision is supported by Oshita (1988), Jones & Stokes (1991), USDI (1991), and USFS (1988).

The risk associated with drinking water contaminated by drift or runoff was not estimated. These routes of exposure were evaluated by Jones & Stokes, USDI, and the USFS. No compound was found to be a cause for concern in any of these three human health risk evaluations, from either systemic or cancer effects. Further more, most roadsides in Washington State are designed to minimize contamination of surface waters by channeling runoff away from sensitive areas. Finally, the corridor nature of highways and their subsequent maintenance reduces impact to any single receiving water. Consequently, the potential for contamination of drinking water by WSDOT vegetation management practices was assumed to be insignificant. Performing a drinking water analysis using WSDOT specific parameters would not be expected to change the findings in the previous environmental impact documents.

Dermal exposure to and inadvertent ingestion of contaminated soil by children was not evaluated. The scientific community considers a child to be under six years of age. Children in this age range are not usually allowed to play along roads or highways. The population with the largest potential for dermal exposure from right-of-way spraying would be older children or adults walking to and from school (school bus) or work, joggers, hikers, and berry pickers.

Ingestion of garden vegetables was not evaluated for children. For this risk assessment, it was assumed that adults consume more vegetables and are at greater risk of potentially receiving a significant exposure to herbicides via this pathway.

Dermal exposure estimates for the public are modeled after worker exposure studies, due to the lack of public right-of-way exposure studies found in the literature. In these studies cloth patches and gloves are used to measure the actual amount of pesticide, per hour, workers are exposed to when in contact with foliar residues. In order to calculate the estimated dose, dermal bioavailability, exposed and covered skin surface area, number of hours exposed, drift fraction (if applicable), and application rates must be factored in.

Oral exposure estimates were modeled using a data compilation article which reports the pesticide loading rates, investigated by numerous authors, onto various types of fruits and vegetables (Hoerger and Kenaga, 1972). In order to calculate the estimated dose the following must be known: serving size, drift fraction (percentage of herbicide deposited on fruit or vegetable due to drift), body weight, and application rate. Bioavailability is assumed to be 100% for oral exposure.

3.2.2.1 Exposure Estimates for the Public Ingesting Drift Contaminated Berries and Garden Vegetables (Lettuce and Beans).

Hoerger and Kenaga (1972) compiled literature residue and application rate data for more than 20 pesticides on more than 60 crops. They correlated this information by calculating RUD (Residue from a Unit Dosage) values. An RUD is derived by dividing the actual residue (in ppm) by the application rate (lbs ai per acre).

$$\text{RUD} = \frac{\text{actual residue}}{\text{treatment rate}} = \frac{\text{ppm}}{\text{lb ai/acre}}$$

This value is called the loading rate (LR) in this risk assessment and is used to estimate crop residues by multiplying it by the application rate and drift fraction. To calculate the daily dose, multiply the residue by the amount of crop eaten and divide by the body weight.

The effects of herbicide drift on wild plants adjacent to treated land were investigated by Marrs, et al. (1989) They discovered that drift from most herbicides fell within 2 meters of the sprayed area. From other studies conducted on drift they expected a deposition of 1.5% within 2 meters, and from 0.5 to 6% deposition within 5 meters. For this risk assessment it was assumed that contaminated berry bushes and gardens are within 2 to

5 meters of the treated area and the fraction of herbicide drift to be .015 for average calculations and .06 for maximum estimates.

Berries:

It is the policy of WSDOT not to spray roadsides or bushes when wild berries are bearing fruit or in bloom. Therefore berries would not become directly contaminated and most probably would not become contaminated from herbicide drift. However, in the interest of safety and public concern, the potential risk associated with ingesting drift contaminated berries was evaluated.

It is estimated that a person would ingest from 25 to 50 berries (½ to 1 cup). Assuming a berry weighs approximately 4 grams, consumption would be equivalent to 100 and 200 grams average and maximum values, respectively. Berries are assumed to be unwashed and frozen with no degradation. It was further assumed that an individual will ingest 30 to 60 servings of berries per year.(Jones & Stokes, 1991)

The equation and assumptions used to calculate the average SDD's for adults and children ingesting drift contaminated berries follows:

$$SDD = \frac{BLR \times AR \times DF \times IR \times CF}{BW}$$

Where:	SDD	=	single day dose.
	BLR	=	the berry residue loading rate, 1.85 μg/g berry/lb ai/A
	AR	=	the WSDOT average application rate, Table 4-11.
	DF	=	drift fraction, 0.015 (assuming fruit laden bushes are contaminated at all).
	IR	=	ingestion rate, 100 grams per day, reported by Jones & Stokes (1991).
	CF	=	conversion factor, 0.001 mg/μg.
	BW	=	body weight, 70 kg for an adult and 18 kg for a child, USEPA (1989(c)).

The same equation was used to calculate the maximum SDD's with the following changes:

BLR	=	the berry residue loading rate, 4.25 μg/g berry/lb ai/A
AR	=	the maximum recommended label application rate, from Table 4-11.
DF	=	drift fraction, 0.06.

IR	=	ingestion rate, 200 grams per day, reported by Jones & Stokes (1991).

The child SDD's were not used when calculating the lifetime average daily dose since the childhood years, 0-6, are already calculated into the equation.

The equation and assumptions used to calculate the average and maximum lifetime average daily dose follows:

$$LADD = \frac{SDD \times D \times ED}{DY \times YL}$$

Where:	LADD	=	lifetime average daily dose.
	SDD	=	single day dose, avg. and max. as appropriate.
	D	=	number of days per year berry eaters eat berries, 30 average and 60 maximum reported by Jones & Stokes (1991).
	ED	=	exposure duration, 9 average and 30 maximum (50th and 90th percentiles for years people stay at one residence) reported by the USEPA (1989(c)).
	DY	=	days in a year, 365.
	YL	=	years in a lifetime, 70 years, USEPA (1989(c))

Lettuce:

For this risk assessment it was assumed that the lettuce was eaten fresh and unwashed. Consumption rates were taken from a three day dietary recall by Pao, et al. (1982) as reported by the USEPA (1989(c)). The average consumption value representing the 50th percentile is 31 grams and the maximum value representing the 95th percentile is 113 grams. Only 4% of all lettuce eaten is homegrown. It was assumed that lettuce or salad is eaten an average of 200 days out of every year.

It is the general policy of WSDOT to spray an area only once per season. Hoerger and Kenaga (1972) reported residue levels on cabbage decreased approximately 75% after 7 days and almost twice as long for vegetables with a waxy cuticle, like string beans. It was assumed that herbicide residues remain on lettuce for an average of 7 days and twice that for a maximum of 14 days after drift contamination from a single spraying event. This represents a fraction of 0.019 (7 ÷ 365) and 0.038 (14 ÷ 365) of the year that homegrown lettuce could be contaminated.

The equation and assumptions used to estimate the average exposure due to ingestion of drift contaminated lettuce follows:

$$SDD = \frac{LLR \times DF \times IR \times AR}{BW}$$

Where:

SDD	=	single day dose.
LLR	=	lettuce residue loading rate, 19.4 mg/kg lettuce/lb ai/A
DF	=	drift fraction, 0.015.
IR	=	ingestion rate, 0.031 kg.
AR	=	the WSDOT average application rate, from Table 4-11.
BW	=	body weight, 70 kg reported by the USEPA (1989(c)).

The maximum SDD's were calculated using the same equation with the following changes in assumptions:

LLR	=	lettuce residue loading rate, 61.6 mg/kg lettuce/lb ai/A
DF	=	drift fraction, 0.06.
IR	=	ingestion rate, 0.110 kg.
AR	=	the maximum recommended label application rate, from Table 4-11.

The equation and assumptions used to calculate the average and maximum lifetime average daily dose follows:

$$LADD = \frac{SDD \times FHG \times FYC \times FL \times ED}{DY \times YL}$$

Where:

LADD	=	lifetime average daily dose, in mg/kg/day.
SDD	=	single day dose, avg. or max. as appropriate.
FHG	=	fraction of the total lettuce consumption which is homegrown, 0.04.
FYC	=	fraction of a year which homegrown lettuce could be contaminated, 0.019 and 0.038.
FL	=	frequency of eating lettuce or salad, 200 days/year.

ED	=	residential exposure duration, 9 and 30 years (USEPA, 1989(c))
DY	=	number of days per year, 365 days.
YL	=	number of years per lifetime, 70 years (USEPA, 1989(c)).

Beans:

As with the lettuce, this risk assessment assumes that beans are eaten fresh and unwashed. Consumption rates were taken from a three day dietary recall by Pao, et al. (1982) as reported by the USEPA (1989(c)). The average consumption value representing the 50th percentile is 23 grams and the maximum value representing the 95th percentile is 93 grams. Approximately 66% of all beans eaten are homegrown and it was assumed that they are eaten an average of 150 days out of every year.

Hoerger and Kenaga (1972) reported residue levels on cabbage decreased approximately 75% after 7 days and almost twice as long for vegetables with a waxy cuticle, like string beans. It was assumed that herbicide residues remain on beans for an average of 14 days and twice that for a maximum of 28 days after drift contamination from a single spraying event. This represents a fraction of 0.038 (14 ÷ 365) and 0.077 (28 ÷ 365) of the year that homegrown beans could be contaminated.

The equation and assumptions used to estimate the average exposure due to ingestion of drift contaminated beans follows:

$$SDD = \frac{BLR \times DF \times IR \times AR}{BW}$$

Where:	SDD	=	single day dose.
	BLR	=	bean residue loading rate, 4.5 mg/kg beans/lb ai/A
	DF	=	drift fraction, 0.015.
	IR	=	ingestion rate, 0.023 kg.
	AR	=	the WSDOT average application rate, from Table 4-11.
	BW	=	body weight, 70 kg reported by the USEPA (1989(c)).

The maximum SDD's were calculated using the same equation with the following changes in assumptions:

BLR	=	bean residue loading rate, 11.1 mg/kg beans/lb ai/A

DF	=	drift fraction, 0.06.
IR	=	ingestion rate, 0.93 kg.
AR	=	the maximum recommended label application rate, from Table 4-11.

The equation and assumptions used to calculate the average and maximum lifetime average daily dose follows:

$$LADD = \frac{SDD \times FHG \times FYC \times FL \times ED}{DY \times YL}$$

Where:	LADD	=	lifetime average daily dose, in mg/kg/day.
	SDD	=	single day dose, avg. or max. as appropriate.
	FHG	=	fraction of the total bean consumption which is homegrown, 0.66.
	FYC	=	fraction of a year which homegrown beans could be contaminated, 0.038 and 0.077.
	FL	=	frequency of eating wax or string, 150 days/year.
	ED	=	residential exposure duration, 9 and 30 years (USEPA, 1989(c))
	DY	=	number of days per year, 365 days.
	YL	=	number of years per lifetime, 70 years (USEPA, 1989(c)).

3.2.2.2 Exposure Estimates for the Public Picking Drift Contaminated Berries

It is the policy of WSDOT not to directly spray berry plants when in bloom or bearing fruit. Therefore, berries would not become directly contaminated and most probably would not become contaminated by herbicide drift. In the interest of safety and public concern, the potential risk associated with dermal exposure from drift contaminated blackberry plants was evaluated, blackberries are the most common berry producing plant along Washington highways.

Estimating dermal exposure from picking berries is a slightly more confusing process than previous calculations. The percentage of the body covered by clothing, and exposed, must first be estimated. For the average case we assumed that the hands, forearms and ½ the upper arm are exposed when picking berries. This is equivalent to 17.2% of the total body surface area (82.8% of the body being covered). For the

maximum case we assumed that the hands, forearms, ½ upper arms and calves are exposed or 30.7% of the total body surface area (69.3% unexposed), Paustenbach (1989).

Approximately ten percent of the dislodged residue which contacts clothing penetrates to the skin and is available for absorption (Ross, 1991). Residue contacting the skin is absorbed according to the individual dermal bioavailability factors found on Table 4-11.

The amount of dislodgeable foliar residue and the total body transfer coefficient were estimated from the study published by Zweig, et al,. (1985). They measured pesticide levels on cloth patches and gloves worn by strawberry harvesters and compared these to residue levels measured on circular leaf punches. The transfer coefficient used for this risk analysis is 5.5×10^3 cm^2/hr (which is the average of all values reported on Zweig's Table IX (Zweig, et al,. (1985)). This value was used for both average and maximum estimates. The average dislodgeable foliar residue is 1.6 $\mu g/cm^2/lb$ ai/A (mean, days 1-14) and the maximum value is 4.0 $\mu g/cm^2/lb$ ai/A (day 1 normalized to 1 lb ai per acre).

As stated previously for the ingestion of contaminated food stuffs pathways, the amount of drift was assumed to be 0.015 and 0.06, Marrs, et al. (1989)

It was assumed that people pick berries for two hours at a time, Jones & Stokes (1991).

For the lifetime average daily dose calculation Jones & Stokes (1991) assumed that people picked berries 2 to 3 times per month for three months which approximates the berry picking activities in Washington.

The equation and assumptions used to calculate dermal exposure to the public from picking drift contaminated blackberries follows:

$$SDD = \frac{[(TC \times DFR \times AR \times HR) \times (CS \times CPF + EPS)] \times DBF \times DF \times CF}{BW}$$

Where:

SDD	=	single day dose, in mg/kg.
TC	=	transfer coefficient, 5.5×10^3 cm^2/hr.
DFR	=	dislodgeable foliar residue, 1.6 $\mu g/cm^2/lb$ ai/A.
AR	=	the WSDOT average application rate, Table 4-11.
HR	=	the number of hours people pick berries, 2 hours.
CS	=	fraction of the total body surface area that is clothed, 0.828.
CPF	=	the clothing penetration factor, 0.10.
EPS	=	the fraction of the total body surface area that is exposed skin, 0.172.

DBF	=	the dermal bioavailability factor, from Table 4-11.
DF	=	the drift fraction, 0.015 (assuming the fruit laden bushes are contaminated at all).
CF	=	a conversion factor, 0.001mg/μg.
BW	=	body weight, 70 kg (USEPA, 1989(c)).

The same equation was used to calculate the maximum exposure with the following assumption changes:

DRF	=	dislodgeable foliar residue, 4.0 $\mu g/cm^2$/lb ai/A.
AR	=	the maximum recommended label rate, from Table 4-11.
CS	=	fraction of covered skin, 0.693.
EPS	=	fraction of exposed skin, 0.307.
DF	=	drift fraction, 0.06.

The equation and assumptions used to calculate the average and maximum lifetime average daily dose due to dermal exposure to contaminated berry bushes follows:

$$LADD = \frac{SDD \times DM \times MY \times ED}{DY \times YL}$$

Where:	LADD	=	lifetime average daily dose.
	SDD	=	single day dose, avg. and max. as appropriate.
	DM	=	days per month people pick berries, 2 average and 3 maximum.
	MY	=	months per year people can pick berries, 3 months.
	ED	=	residential exposure duration, 9 years average and 30 years maximum (USEPA, 1989(c)).
	DY	=	number of days per year, 365 days.
	YL	=	number of years per lifetime, 70 years (USEPA, 1989(c)).

3.2.2.3 Exposure Estimates for the Public Walking, Hiking or Jogging Through Sprayed Vegetation

Assumptions made to calculate the single day dose for dermal exposure to sprayed vegetation are the same as for dermal exposure to berry pickers with the following exceptions:

- The vegetation is not contaminated due to drift but sprayed directly.
- People may routinely use the roadsides to walk to work, the school bus, school, etc., or for recreational activities and exercise. Walking, jogging, and hiking represent the activities which would have the highest potential for exposure.
- The amount of exposed and covered skin is different. It was assumed for the average calculation that the hands only are exposed (cool months), representing 5.6% of the total body surface area (94.4% of the area is covered). The maximum calculation was assumed to be warm months when the hands, calves, ½ thighs, and the forearms are exposed, representing 34.8 percent of the total body surface area (65.2% of the area is covered).

The Worker Health and Safety Branch of the California Department of Food and Agriculture routinely use a dislodgeable foliar residue value of 1 $\mu g/cm^2/lb$ ai/A to calculate the risk associated with dermal exposure to herbicides. Although the values used in the previous pathway were calculated from a study performed on strawberry harvesters (Zweig, et al., 1985), it was decided that these values were a conservative estimate that adequately reflects the amount of dislodgeable herbicide residue expected from dermal contact with sprayed roadside vegetation.

The equation and assumptions used to calculate the average SDD's from dermal exposure to sprayed vegetation follow:

$$SDD = \frac{[(TC \times DFR \times AR \times HR) \times (CS \times CPF + EPS)] \times DBF \times CF}{BW}$$

Where:

SDD	=	single day dose, in mg/kg.
TC	=	transfer coefficient, 5.5×10^3 cm^2/hr.
DFR	=	dislodgeable foliar residue, 1.6 $\mu g/cm^2/lb$ ai/A.
AR	=	the WSDOT average application rate, Table 4-11.
HR	=	the number of hours people spend along the roadside, 0.5 hours.
CS	=	fraction of the total body surface area that is clothed, 0.944.
CPF	=	the clothing penetration factor, 0.10.

EPS	=	the fraction of the total body surface area that is exposed skin, 0.056.
DBF	=	the dermal bioavailability factor, from Table 4-11.
CF	=	a conversion factor, 0.001mg/μg.
BW	=	body weight, 70 kg (USEPA, 1989(c)).

The same equation was used to calculate the maximum exposure with the following assumption changes:

DRF	=	dislodgeable foliar residue, 4.0 $\mu g/cm^2$/lb ai/A.
HR	=	the number of hours people spend along the roadside, 1 hour.
AR	=	the maximum recommended label rate, from Table 4-11
CS	=	fraction of covered skin, 0.652.
EPS	=	fraction of exposed skin, 0.348.

In order to estimate the lifetime average daily dose for exposure to sprayed vegetation, the number of days per year the public walks through potentially sprayed areas must be calculated. Naturally people will use the rights-of-way more during the summer months than in the winter. For compounds that are sprayed all year (2,4-D and Picloram) a maximum value of 3.5 days per week (182 days per year) was calculated. This figure was arrived at by analyzing behavior patterns. For example:

- The person going to school walks along the roadway a maximum of 5 days per week, 12 weeks per quarter for 3 quarters or 180 days.
- The person walking to work will usually not do so in the winter and probably only occasionally during the spring and fall. But for this risk analysis it was assumed that people walk to work a maximum of 5 days per week, 4 weeks per month for 9 months, or 180 days.
- Most joggers only run 3 to 4 days per week, weather permitting.
- Hikers normally chose a more natural setting in which to walk and use the roadsides only as an access.

The remaining compounds are sprayed 1 to 2 months in the spring or fall. People in eastern Washington could probably walk to work or school 5 days a week. However, the majority of Washington's population lives in the west where it rains frequently. It was assumed for these compounds that people are potentially exposed to sprayed vegetation an average of 2 days per week and a maximum of 5 days per week for 1 to

2 months out of the year. An average value of 2 days per week was also assumed for 2,4-D and Picloram.

The equation and assumptions used to calculate the average and maximum lifetime average daily dose due to dermal exposure to directly sprayed vegetation follows:

2,4-D and Picloram:

$$LADD = \frac{SDD \times DW \times WY \times ED}{DY \times YL}$$

Bromacil, Oryzalin, Oxadiazon, and Pronamide:

$$LADD = \frac{SDD \times DW \times WM \times MY \times ED}{DY \times YL}$$

Where:	LADD	=	lifetime average daily dose.
	SDD	=	single daily dose, avg. and max. as appropriate.
	DW	=	number of days per week people use the roadsides. Average 2 days, maximum 3.5 days for 2,4-D and Picloram and 5 days for the remaining potentially carcinogenic herbicides.
	WY	=	number of weeks per year, 52 weeks.
	WM	=	number of weeks in a month, 4 weeks.
	MY	=	number of months the herbicide is sprayed per year.
	ED	=	residential exposure duration, avg. 9 and max. 30 years (EPA, 1989).
	DY	=	number of days per year, 365 days.
	YL	=	number of years per lifetime, 70 years (USEPA, 1989(c)).

3.3 Summary of Exposure Equations, Assumptions, Acronyms and Examples

3.3.1 WSDOT Worker Exposure - Applicators Using Truck or Tractor Mounted Spray Equipment

3.3.1.1 Equations

Single Day Dose:

$$SDD = DR \times AR \times AC \times DBCF$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

	Acronym	Average	Maximum
Urinary excretion dose rate (mg/kg/lb ai)	DR	0.00006	0.00024
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Acres a ground crew can cover in 1 day (Acres)	AC	25	50
Dermal bioavailability correction factor (unitless)	DBCF	Table 4-11	Table 4-11
Days per month applicator is exposed (days)	D	14	16
Months per year herbicide is applied (months)	M	Table 4-11 (6 and 8 for 2,4-D & Picloram)	Table 4-11
Years WSDOT workerapplies herbicide (years)	Y	5	10
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.1.2 Example Calculations (Picloram)

Average SDD = 0.00006 x 0.88 x 25 x 0.048

= 0.0000634 mg/kg/day

Maximum SDD = 0.00024 x 2.16 x 50 x 0.048

= 0.00124 mg/kg/day

Average LADD = $\frac{0.0000634 \times 14 \times 6 \times 5}{365 \times 70}$

= 0.00000104 mg/kg/day

Maximum LADD = $\frac{0.00124 \times 16 \times 8 \times 10}{365 \times 70}$

= 0.0000623 mg/kg/day

3.3.2 WSDOT Worker Exposure - Mixer/Loaders Using Truck or Tractor Mounted Spray Equipment

3.3.2.1 Equations

Single Day Dose:

$$SDD = DR \times AR \times AC \times DBCF$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

	Acronym	Average	Maximum
Urinary excretion dose rate (mg/kg/lb ai)	DR	0.00018	0.00044
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Acres a ground crew can cover in 1 day (Acres)	AC	25	50
Dermal bioavailability correction factor (unitless)	DBCF	Table 4-11	Table 4-11
Days per month applicator is exposed (days)	D	14	16
Months per year herbicide is applied (months)	M	Table 4-11 (6 and 8 for 2,4-D & Picloram)	Table 4-11
Years WSDOT worker applies herbicide (years)	Y	5	10
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.2.2 Example Calculations (Pronamide)

Average SDD = 0.00018 x 2 x 25 x 0.5

= 0.0045 mg/kg/day

Maximum SDD = 0.00044 x 4 x 50 x 0.5

= 0.044 mg/kg/day

Average LADD = $\frac{0.0045 \times 14 \times 1 \times 5}{365 \times 70}$

= 0.0000123 mg/kg/day

Maximum LADD = $\frac{0.044 \times 16 \times 1 \times 10}{365 \times 70}$

= 0.000276 mg/kg/day

3.3.3 WSDOT Worker Exposure - Applicator/Mixer/Loaders Using Truck or Tractor Mounted Spray Equipment

3.3.3.1 Equations

Single Day Dose:

$$SDD = DR \times AR \times AC \times DBCF$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

	Acronym	Average	Maximum
Urinary excretion dose rate (mg/kg/lb ai)	DR	0.00018	0.00037
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Acres a ground crew can cover in 1 day (Acres)	AC	25	50
Dermal bioavailability correction factor (unitless)	DBCF	Table 4-11	Table 4-11
Days per month applicator is exposed (days)	D	14	16
Months per year herbicide is applied (months)	M	Table 4-11 (6 and 8 for 2,4-D & Picloram)	Table 4-11
Years WSDOT worker applies herbicide (years)	Y	5	10
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.3.2 Example Calculations (Bromacil)

Average SDD = 0.00018 x 2.04 x 25 x 0.1

= 0.000918 mg/kg/day

Maximum SDD = 0.00037 x 12 x 50 x 0.1

= 0.0222 mg/kg/day

$$\text{Average LADD} = \frac{0.000918 \times 14 \times 2 \times 5}{365 \times 70}$$

$$= 0.00000503 \text{ mg/kg/day}$$

$$\text{Maximum LADD} = \frac{0.0222 \times 16 \times 2 \times 10}{365 \times 70}$$

$$= 0.000278 \text{ mg/kg/day}$$

3.3.4 WSDOT Worker Exposure - Applicators Using Backpack or Handheld Spray Equipment

3.3.4.1 Equations

Single Day Dose:

$$SDD = HDR \times HR \times AR \times DBCF$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times D \times M \times Y}{DY \times YL}$$

	Acronym	Average	Maximum
Urinary excretion dose rate (mg/kg/hr/lb ai/acre)	HDR	0.00038	0.0011
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Hours crews spray per day (hours/day)	HR	6	7
Dermal bioavailability correction factor (unitless)	DBCF	Table 4-11	Table 4-11
Days per month applicatoris exposed (days)	D	14	16
Months per year herbicide is applied (months)	M	Table 4-11 (6 and 8 for 2,4-D & Picloram)	Table 4-11
Years WSDOT worker applies herbicide (years)	Y	5	10
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.4.2 Example Calculations (Oryzalin)

Average SDD = 0.00038 x 6 x 3.12 x 0.5

= 0.00356 mg/kg/day

Maximum SDD = 0.0011 x 7 x 6 x 0.5

= 0.0231 mg/kg/day

Average LADD = $\frac{0.00356 \times 14 \times 2 \times 5}{365 \times 70}$

= 0.0000195 mg/kg/day

Maximum LADD = $\frac{0.0231 \times 16 \times 2 \times 10}{365 \times 70}$

= 0.000289 mg/kg/day

3.3.5 Public Exposure - Ingestion of Drift Contaminated Berries

3.3.5.1 Equations

Single Day Dose:

$$SDD = \frac{BLR \times AR \times DF \times IR \times CF}{BW}$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times D \times ED}{DY \times YL}$$

	Acronym	Average	Maximum
Berry residue loading rate (μg/g berry/lb ai/A)	BLR	1.85	4.25
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Drift fraction (unitless)	DF	0.015	0.06
Ingestion rate (grams/day)	IR	100	200
Conversion factor (mg/μg)	CF	0.001	0.001
Body weight (kg)	BW	70 (adult) 18 (child)	70 (adult) 18 (child)
Days per year people eat berries (days/year)	D	30	60
Residential exposure duration (years)	ED	9	30
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.5.2 Example Calculations (2,4-D)

Average SDD $= \frac{1.85 \times 2.2 \times 0.015 \times 100 \times 0.001}{70}$ (adult)

$= 0.0000872$ mg/kg/day

Maximum SDD $= \frac{4.25 \times 16.63 \times 0.06 \times 200 \times 0.001}{70}$ (adult)

$= 0.0121$ mg/kg/day

Average LADD $= \frac{0.0000872 \times 30 \times 9}{365 \times 70}$ (adult)

$= 0.00000092$ mg/kg/day

$$\text{Maximum LADD} = \frac{0.0121 \times 60 \times 30}{365 \times 70} \text{ (adult)}$$

$$= 0.000855 \text{ mg/kg/day}$$

3.3.6 Public Exposure - Ingestion of Drift Contaminated Lettuce

3.3.6.1 Equations

Single Day Dose:

$$SDD = \frac{LLR \times AR \times DF \times IR}{BW}$$

Lifetime Average Daily Dose;

$$LADD = \frac{SDD \times FHG \times FYC \times FL \times ED}{DY \times YL}$$

	Acronym	Average	Maximum
Lettuce residue loading rate (mg/kg lettuce/lb ai/A)	LLR	19.4	61.6
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Drift fraction (unitless)	DF	0.015	0.06
Ingestion rate (kgs/day)	IR	0.031	0.11
Body weight (kg)	BW	70	70
Fraction of total lettuce consumption which is homegrown (unitless)	FHG	0.04	0.04
Fraction of a year which lettuce could be contaminated (unitless)	FYC	0.019	0.038
Frequency people eat lettuce (days/year)	FL	200	200
Residential exposure duration (years)	ED	9	30
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.6.2 Example Calculations (Oxadiazon)

Average SDD $= \frac{19.4 \times 2 \times 0.015 \times 0.031}{70}$

$= 0.000258$ mg/kg/day

Maximum SDD $= \frac{61.6 \times 4 \times 0.06 \times 0.11}{70}$

$= 0.0232$ mg/kg/day

Average LADD $= \frac{0.000258 \times 0.04 \times 0.019 \times 200 \times 9}{365 \times 70}$

$= 0.0000000138$ mg/kg/day

Maximum LADD $= \frac{0.0232 \times 0.04 \times 0.038 \times 200 \times 30}{365 \times 70}$

$= 0.00000829$

3.3.7 Public Exposure- Ingestion of Drift Contaminated Beans

3.3.7.1 Equations

Single Day Dose:

$$SDD = \frac{BLR \times AR \times DF \times IR}{BW}$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times FHG \times FYC \times FB \times ED}{DY \times YL}$$

	Acronym	Average	Maximum
Bean residue loading rate (mg/kg beans/lb ai/A)	BLR	4.5	11.1
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Drift fraction (unitless)	DF	0.015	0.06
Ingestion rate (kgs/day)	IR	0.023	0.093
Body weight (kg)	BW	70	70
Fraction of total bean consumption which is homegrown (unitless)	FHG	0.66	0.66
Fraction of a year which beans could be contaminated (unitless)	FYC	0.038	0.077
Frequency people eat beans (days/year)	FB	150	150
Residential exposure duration (years)	ED	9	30
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.7.2 Example Calculations (Picloram)

Average SDD $= \frac{4.5 \times 0.88 \times 0.015 \times 0.02331}{70}$

$= 0.0000195$ mg/kg/day

Maximum SDD $= \frac{11.1 \times 2.16 \times 0.06 \times 0.093}{70}$

$= 0.00191$ mg/kg/day

Average LADD $= \frac{0.0000195 \times 0.66 \times 0.038 \times 150 \times 9}{365 \times 70}$

$= 0.0000000259$ mg/kg/day

$$\text{Maximum LADD} = \frac{0.00191 \times 0.66 \times 0.077 \times 150 \times 30}{365 \times 70}$$

$$= 0.0000171 \text{ mg/kg/day}$$

3.3.8 Public Exposure - Dermal Exposure From Drift Contaminated Berries

3.3.8.1 Equations

Single Day Dose:

$$SDD = \frac{[(TC \times DRF \times AR \times HR) \times (CS \times CPF + EPS)] \times DBF \times DF \times CF}{BW}$$

Lifetime Average Daily Dose:

$$LADD = \frac{SDD \times DM \times MY \times ED}{DY \times YL}$$

	Acronym	Average	Maximum
Transfer coefficient (cm^2/hr)	TC	5500	5500
Dislodgeable foliar residue ($\mu g/cm^2/lb$ ai A)	DFR	1.6	4.0
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Hours people pick berries (hours)	HR	2	2
Fraction of the total body surface area that is clothed (unitless)	CS	0.828	0.693
Clothing penetration factor (unitless)	CPF	0.10	0.10
Fraction of the total body surface area that is exposed skin (unitless)	EPS	0.172	0.307
Dermal bioavailability factor (unitless)	DBF	Table 4-11	Table 4-11
Drift fraction (unitless)	DF	0.015	0.06
Conversion factor (unitless)	CF	0.001	0.001
Body weight (kg)	BW	70	70
Days per month people pick berries (days/mo)	DM	2	3
Months per year people pick berries (months/yr)	MY	3	3
Residential exposure duration (years)	ED	9	30
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.8.2 Example Calculations (Oryzalin)

$$\text{Average SDD} = \frac{[(5500 \times 1.6 \times 3.12 \times 2)(.828 \times .10 + .172)](.05 \times .015 \times .001)}{70}$$

$$= 0.00015 \text{ mg/kg/day}$$

$$\text{Maximum SDD} = \frac{[(5500 \times 4.0 \times 6 \times 2)(.693 \times .10 + .307)](.05 \times .06 \times .001)}{7}$$

$$= 0.00426 \text{ mg/kg/day}$$

$$\text{Average LADD} = \frac{0.00015 \times 2 \times 3 \times 9}{365 \times 70}$$

$$= 0.000000317 \text{ mg/kg/day}$$

$$\text{Maximum LADD} = \frac{0.00426 \times 3 \times 3 \times 30}{365 \times 70}$$

$$= 0.000045 \text{ mg/kg/day}$$

3.3.9 Public Exposure - Dermal Exposure From Directly Sprayed Vegetation

3.3.9.1 Equations

Single Day Dose:

$$\text{SDD} = \frac{[(\text{TC} \times \text{DRF} \times \text{AR} \times \text{HR}) \times (\text{CS} \times \text{CPF} + \text{EPS})] \times \text{DBF} \times \text{CF}}{\text{BW}}$$

Lifetime Average Daily Dose:

2,4-D and Picloram:

$$\text{LADD} = \frac{\text{SDD} \times \text{DW} \times \text{WY} \times \text{ED}}{\text{DY} \times \text{YL}}$$

Bromacil, Oryzalin, Oxadiazon, and Pronamide:

$$\text{LADD} = \frac{\text{SDD} \times \text{DW} \times \text{WM} \times \text{MY} \times \text{ED}}{\text{DY} \times \text{YL}}$$

	Acronym	Average	Maximum
Transfer coefficient	TC	5500	(cm^2/hr)
Dislodgeable foliar residue ($\mu g/cm^2$/lb ai A)	DFR	1.6	4.0
Application rate (lb ai/acre)	AR	Table 4-11	Table 4-11
Hours spent along the roadside (hours)	HR	0.5	1
Fraction of the total body surface area that is clothed (unitless)	CS	0.944	0.652
Clothing penetration factor (unitless)	CPF	0.10	0.10
Fraction of the total body surface area that is exposed skin (unitless)	EPS	0.056	0.348
Dermal bioavailability factor (unitless)	DBF	Table 4-11	Table 4-11
Conversion factor (unitless)	CF	0.001	0.001
Body weight (kg)	BW	70	70
Days per week people use the roadsides (Days/wk)	DW	2	3.5 or 5
Weeks per year (weeks/yr)	WY	52	52
Weeks in a month (week/mo)	WM	4	4
Number of months the herbicide is sprayed (mo/yr)	MY	Table 4-11	Table 4 -11
Residential exposure duration (years)	ED	9	30
Days per year (days)	DY	365	365
Years per lifetime (years)	YL	70	70

3.3.9.2 Example Calculations (Bromacil)

$$\text{Average SDD} = \frac{[(5500 \times 1.6 \times 2.04 \times 0.5)(.944 \times .10 + .056)](.01 \times .001)}{70}$$

$$= 0.000193 \text{ mg/kg/day}$$

$$\text{Maximum SDD} = \frac{[(5500 \times 4.0 \times 12 \times 1)(.652 \times .10 + .348)](.01 \times .001)}{70}$$

$$= 0.0156 \text{ mg/kg/day}$$

$$\text{Average LADD} = \frac{0.000193 \times 2 \times 4 \times 2 \times 9}{365 \times 70}$$

$$= 0.00000109 \text{ mg/kg/day}$$

$$\text{Maximum LADD} = \frac{0.0156 \times 5 \times 4 \times 3 \times 30}{365 \times 70}$$

$$= 0.0011 \text{ mg/kg/day}$$

4.0 Dose-response Assessment

4.1 Introduction

The purpose of the dose-response assessment is to:

- Identify the lowest NOAELs (No Observable Adverse Effect Levels) for chronic systemic effects and for reproductive/developmental effects.
- Identify reference doses (or acceptable daily intake) available from the EPA.
- Calculate reference doses, when not available, using the appropriate NOAEL and an uncertainty factor.
- Identify cancer potency factors.

The dose-response assessment is a collection of tables which identify the critical toxicity and carcinogenicity values used to calculate the hazard quotient and cancer risk probability, respectively. Section 4.2 provides narrative summaries of the available toxicity data for each herbicide. Tabular summaries of all available laboratory and field data have been compiled for each compound and can be found in Chapter 5.

4.2 Determination of Toxicity Values

4.2.1 Systemic or Non-Cancer Effects

The methods used to evaluate systemic (noncancer) and cancer effects differ. Theoretically one molecule of a forgeign substance can cause injury to liver or kidney cells, etc., resulting in injury or death to a single cell, which would be biologically insignificant. The Lowest Observable Adverse Effect Level (LOAEL) and the No Observable Adverse Effect Level (NOAEL) are identified for systemic toxic effects (noncancer) in each animal study performed. The NOAEL is the highest dosage on a study which produces no adverse or toxic effect and the LOAEL is the dose level in which the toxic effect first appears. After examination of the toxicity data the lowest NOAEL is identified, usually from a chronic study, and divided by an "uncertainty factor" to arrive at the Reference Dose (RfD). The criteria used by the US EPA for applying "uncertainty factors" are as follows:

The uncertainty factors (UF) generally consist of multiples of 10 (although values of less than 10 are sometime used), with each factor representing a specific area of uncertainty inherent in the extrapolation from available data. The bases for application of different uncertainty factors are explained below.

- A UF of 10 is used to account for variation in the general population and is intended to protect sensitive subpopulations (e.g., elderly, children.)

- A UF of 10 is used when extrapolating from animals to humans. This factor is intended to account for the interspecies variability between humans and other mammals.

- A UF of 10 is used when a NOAEL derived from a subchronic study is used as the basis for calculating a chronic RfD.

- A UF of 10 is used when a LOAEL is used instead of a NOAEL. This factor is intended to account for the uncertainty associated with extrapolating from LOAEL's to NOAEL's.

Reference dose is defined as an estimate of the daily exposure to human populations (including sensitive subpopulations), measured in specific units, generally mg/kg/day, that reflects the level at which no adverse noncarcinogenic effects are expected should humans be chronically exposed to that level (Jones & Stokes, 1991). The US EPA has set RfD values for many chemicals. For those herbicides which did not have a published RfD, one was assigned using the method of calculation employed by the US EPA. A listing of RfD values can be found in Table 4-12 of this section. The uncertainty factor applied, level of confidence in the RfD value, NOAEL with critical toxic effect and source references are also included in this table.

4.2.2 Carcinogenic Effects

At present, it is generally accepted by the scientific community that any level of exposure to a potential carcinogen poses a risk. In contrast to systemic effects, a single molecule

of a potential carcinogen could cause alterations of the DNA in a single cell resulting in cloning of that cell, which in turn can lead to tumor formation and death. Therefore, an alternative approach to hazard evaluation of a potential carcinogen is necessary (Klassen, et al., 1986).

The dose-response for carcinogenic effects can be plotted at very high exposure levels. However, computerized mathematical dose-response models are most often used to extrapolate the risk of getting cancer at very low doses (i.e., 1 in 100,000). Many of these models exist but the one most frequently used by regulatory agencies is the Linearized Multistage model. The slope (or cancer potency factor) of the dose-response curve is calculated by the model and used to estimate the incremental lifetime risk from exposure to a potential carcinogen.

Table 4-13 summarizes the critical carcinogenicity information including the cancer potency factor (CPF). The CPF is expressed in (mg/kg/day)-1 and is used to calculate the increased probability that an individual will develop a tumor due to exposure to a chemical. This risk assessment uses cancer potency values derived by the US EPA (1991). Table 4-13 also includes the weight-of-evidence classification assigned by the US EPA using the following guidelines (Table 4-35).

For convenience Table 4-14 presents the RfD values for systemic or reproductive/ developmental effects and the cancer potency factors for each herbicide.

5.0 Risk Characterization

5.1 Introduction

Risk characterization is the process of estimating the incidence of a health effect under the various conditions of human exposure described in the exposure assessment. It is performed by combining the information provided in the exposure and dose-response assessments (Paustenbach, 1989). The intent of the current quantitative estimate of risk is to evaluate the economic and societal risk/benefits of herbicides in the WSDOT vegetation management program.

5.1.1 Noncancer (Systemic) Risk Characterization

Noncancer effects are estimated by calculating a hazard quotient (HQ) for each pathway of exposure, for each herbicide. This is the Single Day Dose (SDD) calculated in Section 3 of this chapter divided by the Reference Dose (RfD), identified in Section 4.

$$HQ = SDD \div RfD$$

Hazard quotient values of one or less are typically considered to be of no concern. Values slightly larger than one are of limited concern, considering that the hazard quotient is calculated by comparing a potential single acute exposure to a reference dose (RfD) value, which represents the NOAEL from a chronic exposure study plus "uncertainty" or "safety" factors. The table in section 6.1 of this chapter (understanding risk) will help the layperson put the risk numbers into perspective.

Tables 4-15 through 4-24, present the average and maximum hazard quotient values for each herbicide, per pathway.

5.1.2 Cancer Risk Characterization

Cancer risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen. Or, the increased probability of developing a tumor in a lifetime due to exposure from a potential carcinogen over the already existing probability that an individual will develop cancer in their lifetime.

The risk associated with exposure to a potential carcinogen is calculated by multiplying the lifetime average daily dose (LADD) by the cancer potency factor (CPF), identified in Section 4 of this chapter.

$$RISK = LADD \times CPF$$

In the past a lifetime incremental risk of one in a million was considered to be an "acceptable" risk. Presently, regulatory agencies are taking exception to using the one in a million benchmark, especially since many common human activities entail greater risk. "Examination of the risks of common human activities demonstrates...a lifetime risk of one in 100,000 or more is within the realm of, or orders of magnitude below, everyday risks that generally do not cause undue concern, see annual cancer risks below. These are risks that people, while they are aware of them and may have some concern or fear over them, do not in general alter their behavior to avoid. As the following table illustrates, the risk from many activities greatly exceed the level of 1 in 100,000. In comparison to these background risks of 'everyday' activities, a lifetime risk of 1 in 100,000 is relatively small. Accordingly, regulatory action will not generally be justifiable unless risks are substantially higher than this 1 in 100,000 'benchmark'." (Rodricks, et al., 1987)

Annual Cancer Risks For Everyday Living

Potential Cause	Risk (Chance/Million)
Smoker	8,400/100,000
Alcohol (liver cirrhosis)	350/100,000
Natural Background Radiation (sea level)	140/100,000
Diagnostic Medical X-Rays	140/100,000
One Diet Drink/Day (saccharin)	70/100,000
Flying 4 Hours/week (cosmic radiation)	70/100,000
Sharing Room with Smoker	70/100,000
4 Tablespoons Peanut Butter/Day (Aflatoxin)	56/100,000
Living in Colorado vs New York (cosmic radiation)	56/100,000
Living in Stone or Brick Building	42/100,000
Average Saccharin Consumption	14/100,000
One Pint Milk/Day (Aflatoxin)	14/100,000
1/2 lb Charcoal Broiled Steak/Week (Benzopyrene)	2/100,000
1 Transcontinental Flight/Year	0.7/100,000

(Source: County Sanitation Districts of Los Angeles County)

A retrospective examination of the level of risk which triggered regulatory action was done by Travis, et al., (1987) and summarized as follows:

1. Every chemical with an individual lifetime risk above 4×10^{-3} received regulation. Those with values below 1×10^{-6} remained unregulated.

2. For small populations, regulatory action never resulted for individual risks below 1×10^{-4}.

3. For effects resulting from exposures to the entire U.S. population, a risk level below 1×10^{-6} never triggered action; above 3×10^{-4} it always triggered action.

This risk assessment considers a risk of one in 100,000 to be an "acceptable" risk. Tables 4-29 through 4-32 of this chapter will help the layperson put the risk numbers into perspective.

Average and maximum hazard quotients and cancer risk probabilities for each herbicide are presented in Tables 4-15 through 4-24.

5.1.3 Sample Risk Calculations

The equation used to estimate both worker and public risk due to herbicide exposure from the WSDOT rights-of-way vegetation management program is:

$$HQ = SDD \div RfD$$

WSDOT Worker Risk- Applicators Using Truck or Tractor Mounted Spray Equipment (Picloram):

$$\text{Average HQ} = \frac{6.34\text{x}10^{-5}\ \text{mg/kg/day}}{7.0\text{x}10^{-2}\ \text{mg/kg/day}} = 9.06\text{x}10^{-4}$$

$$\text{Maximum HQ} = \frac{1.24\text{x}10^{-3}\ \text{mg/kg/day}}{7.0\text{x}10^{-2}\ \text{mg/kg/day}} = 1.77\text{x}10^{-2}$$

$$\text{Average Cancer Risk} = 1.04\text{x}10^{-6}\ \text{mg/kg/day} \times 3.0\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 3.12\text{x}10^{-9}$$

$$\text{Maximum Cancer Risk} = 6.23\text{x}10^{-5}\ \text{mg/kg/day} \times 3.0\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 1.87\text{x}10^{-7}$$

WSDOT Worker Risk- Mixer/Loaders Using Truck or Tractor Mounted Spray Equipment (Pronamide)

$$\text{Average HQ} = \frac{4.50\text{x}10^{-3}\ \text{mg/kg/day}}{7.5\text{x}10^{-2}\ \text{mg/kg/day}} = 6.00\text{x}10^{-2}$$

$$\text{Maximum HQ} = \frac{4.40\text{x}10^{-2}\ \text{mg/kg/day}}{7.5\text{x}10^{-2}\ \text{mg/kg/day}} = 5.87\text{x}10^{-1}$$

$$\text{Average Cancer Risk} = 1.23\text{x}10^{-5}\ \text{mg/kg/day} \times 1.6\text{x}10^{-2}\ (\text{mg/kg/day})^{-1}$$

$$= 1.97\text{x}10^{-7}$$

$$\text{Maximum Cancer Risk} = 2.76\text{x}10^{-4}\ \text{mg/kg/day} \times 1.6\text{x}10^{-2}\ (\text{mg/kg/day})^{-1}$$

$$= 4.42\text{x}10^{-6}$$

WSDOT Worker Risk- Applicator/Mixer/Loaders Using Truck or Tractor Mounted Spray Equipment (Bromacil)

$$\text{Average HQ} = \frac{9.18\text{x}10^{-4}\ \text{mg/kg/day}}{2.5\text{x}10^{-2}\ \text{mg/kg/day}} = 3.67\text{x}10^{-2}$$

$$\text{Maximum HQ} = \frac{2.22\text{x}10^{-2}\ \text{mg/kg/day}}{2.5\text{x}10^{-2}\ \text{mg/kg/day}} = 8.88\text{x}10^{-1}$$

$$\text{Average Cancer Risk} = 5.03\text{x}10^{-6}\ \text{mg/kg/day} \times 3.8\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 1.91\text{x}10^{-8}$$

$$\text{Maximum Cancer Risk} = 2.78\text{x}10^{-4}\ \text{mg/kg/day} \times 3.8\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 1.06\text{x}10^{-6}$$

WSDOT Worker Risk- Applicators Using Backpack or Handheld Spray Equipment (Oryzalin)

$$\text{Average HQ} = \frac{3.56\text{x}10^{-3}\ \text{mg/kg/day}}{5.0\text{x}10^{-2}\ \text{mg/kg/day}} = 7.12\text{x}10^{-2}$$

$$\text{Maximum HQ} = \frac{2.31\text{x}10^{-2}\ \text{mg/kg/day}}{5.0\text{x}10^{-2}\ \text{mg/kg/day}} = 4.62\text{x}10^{-1}$$

$$\text{Average Cancer Risk} = 1.95\text{x}10^{-5}\ \text{mg/kg/day} \times 1.3\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 2.54\text{x}10^{-6}$$

$$\text{Maximum Cancer Risk} = 2.89\text{x}10^{-4}\ \text{mg/kg/day} \times 1.3\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 3.76\text{x}10^{-5}$$

Public Risk- Ingestion of Drift Contaminated Berries (2,4-D)

$$\text{Average HQ} = \frac{8.72\text{x}10^{-5}\ \text{mg/kg/day}}{1.0\text{x}10^{-2}\ \text{mg/kg/day}} = 8.72\text{x}10^{-3}$$

$$\text{Maximum HQ} = \frac{1.21\text{x}10^{-2}\ \text{mg/kg/day}}{1.0\text{x}10^{-2}\ \text{mg/kg/day}} = 1.21\text{x}10^{0}$$

$$\text{Average Cancer Risk} = 9.92\text{x}10^{-7}\ \text{mg/kg/day x } 1.9\text{x}10^{-2}\ (\text{mg/kg/day})^{-1}$$

$$= 1.75\text{x}10^{-8}$$

$$\text{Maximum Cancer Risk} = 8.55\text{x}10^{-4}\ \text{mg/kg/day x } 1.9\text{x}10^{-2}\ (\text{mg/kg/day})^{-1}$$

$$= 1.62\text{x}10^{-5}$$

Public Risk- Ingestion of Drift Contaminated Lettuce (Oxadiazon)

$$\text{Average HQ} = \frac{2.58\text{x}10^{-4}\ \text{mg/kg/day}}{5.0\text{x}10^{-3}\ \text{mg/kg/day}} = 5.16\text{x}10^{-2}$$

$$\text{Maximum HQ} = \frac{2.32\text{x}10^{-2}\ \text{mg/kg/day}}{5.0\text{x}10^{-3}\ \text{mg/kg/day}} = 4.64\text{x}10^{0}$$

$$\text{Average Cancer Risk} = 1.38\text{x}10^{-8}\ \text{mg/kg/day x } 1.4\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 1.93\text{x}10^{-9}$$

$$\text{Maximum Cancer Risk} = 8.29\text{x}10^{-6}\ \text{mg/kg/day x } 1.4\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 1.16\text{x}10^{-6}$$

Public Risk- Ingestion of Drift Contaminated Beans (Picloram)

$$\text{Average HQ} = \frac{1.95\text{x}10^{-5}\ \text{mg/kg/day}}{7.0\text{x}10^{-2}\ \text{mg/kg/day}} = 2.79\text{x}10^{-4}$$

$$\text{Maximum HQ} = \frac{1.91\text{x}10^{-3}\ \text{mg/kg/day}}{7.0\text{x}10^{-2}\ \text{mg/kg/day}} = 2.73\text{x}10^{-2}$$

$$\text{Average Cancer Risk} = 2.59\text{x}10^{-8}\ \text{mg/kg/day x } 3.0\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 7.77\text{x}10^{-11}$$

$$\text{Maximum Cancer Risk} = 1.71\text{x}10^{-5}\ \text{mg/kg/day x } 3.0\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 5.13\text{x}10^{-8}$$

Public Risk- Dermal Exposure From Drift Contaminated Berries (Oryzalin)

$$\text{Average HQ} = \frac{1.50\text{x}10^{-4}\ \text{mg/kg/day}}{5.0\text{x}10^{-2}\ \text{mg/kg/day}} = 3.00\text{x}10^{-3}$$

$$\text{Maximum HQ} = \frac{4.26\text{x}10^{-3}\ \text{mg/kg/day}}{5.0\text{x}10^{-2}\ \text{mg/kg/day}} = 8.52\text{x}10^{-2}$$

$$\text{Average Cancer Risk} = 3.17\text{x}10^{-7}\ \text{mg/kg/day} \times 1.3\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 4.12\text{x}10^{-8}$$

$$\text{Maximum Cancer Risk} = 4.50\text{x}10^{-5}\ \text{mg/kg/day} \times 1.3\text{x}10^{-1}\ (\text{mg/kg/day})^{-1}$$

$$= 5.85\text{x}10^{-6}$$

Public Risk- Dermal Exposure From Directly Sprayed Vegetation (Bromacil)

$$\text{Average HQ} = \frac{1.93\text{x}10^{-4}\ \text{mg/kg/day}}{2.5\text{x}10^{-2}\ \text{mg/kg/day}} = 7.72\text{x}10^{-3}$$

$$\text{Maximum HQ} = \frac{1.56\text{x}10^{-2}\ \text{mg/kg/day}}{2.5\text{x}10^{-2}\ \text{mg/kg/day}} = 6.24\text{x}10^{-1}$$

$$\text{Average Cancer Risk} = 1.09\text{x}10^{-6}\ \text{mg/kg/day} \times 3.8\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 4.14\text{x}10^{-9}$$

$$\text{Maximum Cancer Risk} = 1.10\text{x}10^{-3}\ \text{mg/kg/day} \times 3.8\text{x}10^{-3}\ (\text{mg/kg/day})^{-1}$$

$$= 4.18\text{x}10^{-6}$$

5.2 Risk Characterization Summary by Chemical

5.2.1 2,4-D

5.2.1.1 Systemic Effects - Average

2,4-D is not expected to cause adverse health effects to WSDOT workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than 1, which indicates that the expected human doses are less than the RfD.

5.2.1.2 Systemic Effects - Maximum

Under the conditions specified for the maximum case exposure pathways, 2,4-D might be expected to pose a hazard to the public. All pathways evaluated have hazard quotients over one. Values range from 1.2 for adults ingesting drift contaminated berries to 22 for contact with directly sprayed vegetation. 2,4-D is sprayed all year round therefore it is quite possible that berries, lettuce and beans could become contaminated. (However, it

is the policy of the WSDOT not to spray near ripe fruit or vegetables and, therefore, it would be highly unlikely that they would become contaminated.) It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100.

Workers could be at risk from 2,4-D given the maximum case assumptions. Hazard quotient values range from 13 for handheld or backpack sprayers to 37 for mixer/loaders. Exposure can be reduced by wearing protective clothing during all phases of herbicide usage and reducing application rates.

5.2.1.3 Cancer Effects - Average

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (1 in one hundred thousand). The estimated cancer risks for worker average case scenarios are less than this benchmark, the highest risk being $3.1x10^{-6}$ (3.1 in one million) calculated for mixer/loaders and applicator/mixer/loaders to $1.0x10^{-6}$ (1.0 in one million) for applicators. Average cancer risk estimates for the public are also below the level of concern. Values range from $2.9x10^{-10}$ (2.9 in ten billion) for ingestion of drift contaminated lettuce to $1.5x10^{-6}$ (1.5 in one million) for dermal contact with directly sprayed vegetation.

5.2.1.4 Cancer Effects - Maximum

All maximum worker cancer risk estimates exceed $1x10^{-5}$, values range from $1.2x10^{-4}$ (1.2 in ten thousand) for handheld spray applicators to $3.5x10^{-4}$ (3.5 in ten thousand) for mixer/loaders. WSDOT workers can reduce exposure by wearing appropriate protective clothing when spraying 2,4-D and keeping application rates below maximum recommended label rates. Maximum cancer risk values for the public ingesting contaminated berries and dermal contact of directly sprayed vegetation exceed the $1x10^{-5}$ acceptable benchmark with values of $1.6x10^{-5}$ (1.6 in one hundred thousand) and $8.8x10^{-4}$ (8.8 in ten thousand), respectively.

Average exposure risk estimates indicate that no adverse systemic effects would be expected from use of 2,4-D in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, 2,4-D would have negligible to moderate potential for causing acute systemic effects. The carcinogenic risks for average exposure assumptions are below the level of concern and for maximum case exposure are negligible to moderate.

For worker and public safety 2,4-D application rates should not approach maximum recommended label rates. Historical records of 2,4-D use indicates that the WSDOT average application rate is approximately 13 percent of the maximum application rate allowed.

2,4-D Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$3.3x10^{-1}$	$2.0x10^{1}$
Mixer/Loader	$9.9x10^{-1}$	$3.7x10^{1}$
Applicator/Mixer/Loader	$9.9x10^{-1}$	$3.1x10^{1}$
Handheld Spray Applicator	$5.0x10^{-1}$	$1.3x10^{1}$

2,4-D Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$8.7x10^{-3}$	$1.2x10^{0}$
Ingestion of Drift Contaminated Berries (child)	$3.4x10^{-2}$	$4.7x10^{0}$
Ingestion of Drift Contaminated Lettuce	$2.8x10^{-2}$	$9.7x10^{0}$
Ingestion of Drift Contaminated Beans	$4.9x10^{-3}$	$1.5x10^{0}$
Dermal Contact With Drift Contaminated Berries	$2.1x10^{-2}$	$2.4x10^{0}$
Dermal Contact With Directly Sprayed Vegetation	$2.1x10^{-1}$	$2.2x10^{1}$

2,4-D Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.0x10^{-6}$	$1.9x10^{-4}$
Mixer/Loader	$3.1x10^{-6}$	$3.5x10^{-4}$
Applicator/Mixer/Loader	$3.1x10^{-6}$	$2.9x10^{-4}$
Handheld Spray Applicator	$1.6x10^{-6}$	$1.2x10^{-4}$

2,4-D Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.8x10^{-8}$	$1.6x10^{-5}$
Ingestion of Drift Contaminated Lettuce	$2.9x10^{-10}$	$6.6x10^{-7}$
Ingestion of Drift Contaminated Beans	$1.2x10^{-9}$	$2.5x10^{-6}$
Dermal Contact With Drift Contaminated Berries	$8.5x10^{-9}$	$4.8x10^{-6}$
Dermal Contact With Directly Sprayed Vegetation	$1.5x10^{-6}$	$8.8x10^{-4}$

5.2.2 2,4-DP

5.2.2.1 Systemic Effects - Average

2,4-DP is not expected to cause adverse health effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.2.2 Systemic Effects - Maximum

Under the conditions specified for the maximum case exposure scenarios, 2,4-D might be expected to pose a hazard to the public. Three pathways evaluated have hazard quotients over one. Values range from 0.3 for adults ingesting drift contaminated berries to 3.5 for dermal contact with directly sprayed vegetation. However, 2,4-DP is only sprayed in the winter months which greatly reduces 1) the likelihood that the public would exposed by walking along the roadside and 2) the likelihood that anyone would be wearing shorts and a T-shirt as is assumed for the maximum case exposure scenario. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100.

Workers could be at risk from 2,4-D given the maximum case assumptions. Hazard quotient values range from 2.1 to 5.9. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The average exposure risk estimates indicate that no adverse health effects would be expected from use of 2,4-DP in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, 2,4-DP would have a low potential for causing acute systemic effects in humans.

2,4-DP Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	2.9×10^{-2}	3.2×10^{0}
Mixer/Loader	8.7×10^{-2}	5.9×10^{0}
Applicator/Mixer/Loader	8.7×10^{-2}	4.9×10^{0}
Handheld Spray Applicator	4.4×10^{-2}	2.1×10^{0}

2,4-DP Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	1.2×10^{-3}	3.0×10^{-1}
Ingestion of Drift Contaminated Berries (child)	4.6×10^{-3}	1.2×10^{0}
Ingestion of Drift Contaminated Lettuce	3.9×10^{-3}	2.4×10^{0}
Ingestion of Drift Contaminated Beans	6.7×10^{-4}	3.7×10^{-1}
Dermal Contact With Drift Contaminated Berries	1.9×10^{-3}	3.8×10^{-1}
Dermal Contact With Directly Sprayed Vegetation	1.8×10^{-2}	3.5×10^{0}

5.2.3 Benefin

5.2.3.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to benefin are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of benefin in the WSDOT vegetation management program.

Benefin Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.0x10^{-2}$	$1.2x10^{-1}$
Mixer/Loader	$3.1x10^{-2}$	$2.2x10^{-1}$
Applicator/Mixer/Loader	$3.1x10^{-2}$	$1.9x10^{-1}$
Handheld Spray Applicator	$1.6x10^{-2}$	$7.7x10^{-2}$

Benefin Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$2.7x10^{-4}$	$7.3x10^{-3}$
Ingestion of Drift Contaminated Berries (child)	$1.1x10^{-3}$	$2.8x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$8.8x10^{-4}$	$5.8x10^{-2}$
Ingestion of Drift Contaminated Beans	$1.5x10^{-4}$	$8.8x10^{-3}$
Dermal Contact With Drift Contaminated Berries	$6.6x10^{-4}$	$1.4x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$6.5x10^{-3}$	$1.3x10^{-1}$

5.2.4 Bromacil

5.2.4.1 Systemic Effects - Average

There are no potential risks to workers and the public associated with any average case exposure pathways evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.4.2 Systemic Effects - Maximum

Bromacil is not expected to pose a hazard to the public by the maximum case exposure pathways. Ingestion of drift contaminated berries (by children) and lettuce are approaching a level of concern with hazard quotient values of 1.4 and 2.8, respectively. (It should be noted that the RfD is estimated using the NOAEL from a subchronic or

chronic study and applying an "uncertainty" or "safety" factor of 100). However, bromacil is only sprayed in the fall or winter months which greatly reduces the likelihood that ripe berries or lettuce could possibly be contaminated.

Hazard quotient values for maximum case worker exposure scenarios range from 0.37 to 1.1 (for mixer/loaders), approaching a level of limited concern. Bromacil may present some risk to mixer/loaders given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

5.2.4.3 Cancer Effects - Average

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (one in one hundred thousand). The estimated cancer risks for both worker and public average case scenarios are less than $1.9x10^{-8}$ (1.9 in one hundred million) calculated for mixer/loaders and applicator/mixer/loaders.

5.2.4.4 Cancer Effects - Maximum

The estimated cancer risks for both workers and the public using maximum exposure assumptions are less than $4.2x10^{-6}$ (4.2 in one million) calculated for dermal contact to directly sprayed vegetation by the public.

The average exposure risk estimates indicate that no systemic adverse health effects would be expected from use of bromacil in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, bromacil would have negligible to low potential for causing acute systemic effects in humans. According to the lifetime cancer risk estimated from average and maximum exposure scenarios and the very low carcinogenic potency of bromacil, 0.0038 $(mg/kg/day)^{-1}$, the risks associated with use of bromacil in the WSDOT vegetation management program are consider to be acceptable.

Bromacil Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.2x10^{-2}$	$5.8x10^{-1}$
Mixer/Loader	$3.7x10^{-2}$	$1.1x10^{0}$
Applicator/Mixer/Loader	$3.7x10^{-2}$	$8.9x10^{-1}$
Handheld Spray Applicator	$1.9x10^{-2}$	$3.7x10^{-1}$

Bromacil Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$3.2x10^{-3}$	$3.5x10^{-1}$
Ingestion of Drift Contaminated Berries (child)	$1.3x10^{-2}$	$1.4x10^{0}$
Ingestion of Drift Contaminated Lettuce	$1.1x10^{-2}$	$2.8x10^{0}$
Ingestion of Drift Contaminated Beans	$1.8x10^{-3}$	$4.2x10^{-1}$
Dermal Contact With Drift Contaminated Berries	$7.8x10^{-4}$	$6.8x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$7.7x10^{-3}$	$6.2x10^{-1}$

Bromacil Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$6.4x10^{-9}$	$6.8x10^{-7}$
Mixer/Loader	$1.9x10^{-8}$	$1.3x10^{-6}$
Applicator/Mixer/Loader	$1.9x10^{-8}$	$1.1x10^{-6}$
Handheld Spray Applicator	$9.7x10^{-9}$	$4.4x10^{-7}$

Bromacil Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$3.3x10^{-9}$	$2.3x10^{-6}$
Ingestion of Drift Contaminated Lettuce	$5.4x10^{-11}$	$9.5x10^{-8}$
Ingestion of Drift Contaminated Beans	$2.3x10^{-10}$	$3.6x10^{-7}$
Dermal Contact With Drift Contaminated Berries	$1.6x10^{-10}$	$6.8x10^{-8}$
Dermal Contact With Directly Sprayed Vegetation	$4.1x10^{-9}$	$4.2x10^{-6}$

5.2.5 Chlorsulfuron

5.2.5.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to chlorsulfuron are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of chlorsulfuron in the WSDOT vegetation management program.

Chlorsulfuron Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$2.9x10^{-4}$	$3.4x10^{-3}$
Mixer/Loader	$8.6x10^{-4}$	$6.2x10^{-3}$
Applicator/Mixer/Loader	$8.6x10^{-4}$	$5.2x10^{-3}$
Handheld Spray Applicator	$4.3x10^{-4}$	$2.2x10^{-3}$

Chlorsulfuron Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$7.5x10^{-5}$	$2.0x10^{-3}$
Ingestion of Drift Contaminated Berries (child)	$2.9x10^{-4}$	$7.9x10^{-3}$
Ingestion of Drift Contaminated Lettuce	$2.4x10^{-4}$	$1.6x10^{-2}$
Ingestion of Drift Contaminated Beans	$4.2x10^{-5}$	$2.5x10^{-3}$
Dermal Contact With Drift Contaminated Berries	$1.8x10^{-5}$	$4.0x10^{-4}$
Dermal Contact With Directly Sprayed Vegetation	$1.8x10^{-4}$	$3.6x10^{-3}$

5.2.6 Chlorthal Dimethyl

5.2.6.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to chlorthal dimethyl are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of chlorthal dimethyl in the WSDOT vegetation management program.

Chlorthal Dimethyl Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$3.8x10^{-2}$	$3.6x10^{-1}$
Mixer/Loader	$1.2x10^{-1}$	$6.6x10^{-1}$
Applicator/Mixer/Loader	$1.2x10^{-1}$	$5.6x10^{-1}$
Handheld Spray Applicator	$5.8x10^{-2}$	$2.3x10^{-1}$

Chlorthal Dimethyl Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.0x10^{-3}$	$2.2x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$3.9x10^{-3}$	$8.5x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$3.3x10^{-3}$	$1.7x10^{-1}$
Ingestion of Drift Contaminated Beans	$5.7x10^{-4}$	$2.7x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$2.5x10^{-3}$	$4.3x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$2.4x10^{-2}$	$3.9x10^{-1}$

5.2.7 Dicamba

5.2.7.1 Systemic Effects - Average

Dicamba is not expected to cause adverse systemic effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.7.2 Systemic Effects - Maximum

Under the conditions specified for the maximum case exposure scenarios, dicamba is not expected to pose a hazard to the public. Ingestion of drift contaminated lettuce and contact with directly sprayed vegetation are approaching the level of concern with a hazard quotient values of 1.6 and 1.7, respectively. When estimating the potential risk associated with eating contaminated lettuce we assumed that the lettuce was not washed. It is highly unlikely that herbicide levels used to calculate the maximum case exposure scenario will be reached.

Hazard quotient values for maximum case worker exposure scenarios range from 1.0 to 2.9 for mixer/loaders. While this does not automatically indicate that toxicity will occur for handheld or backpack sprayers, dicamba may present some risk given the maximum case assumptions. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The average exposure risk estimates indicate that no systemic adverse health effects would be expected from use of dicamba in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, dicamba would have negligible to low potential for causing acute systemic effects in humans.

Dicamba Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	2.8×10^{-2}	1.6×10^{0}
Mixer/Loader	8.4×10^{-2}	2.9×10^{0}
Applicator/Mixer/Loader	8.4×10^{-2}	2.5×10^{0}
Handheld Spray Applicator	4.3×10^{-2}	1.0×10^{0}

Dicamba Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	1.5×10^{-3}	1.9×10^{-1}
Ingestion of Drift Contaminated Berries (child)	5.8×10^{-3}	7.6×10^{-1}
Ingestion of Drift Contaminated Lettuce	4.8×10^{-3}	1.6×10^{0}
Ingestion of Drift Contaminated Beans	8.3×10^{-4}	2.4×10^{-1}
Dermal Contact With Drift Contaminated Berries	1.8×10^{-3}	1.9×10^{-1}
Dermal Contact With Directly Sprayed Vegetation	1.8×10^{-2}	1.7×10^{0}

5.2.8 Dichlobenil

5.2.8.1 Systemic Effects - Average

Dichlobenil is not expected to cause adverse systemic effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.8.2 Systemic Effects - Maximum

Risk estimates for all public exposure scenarios exceeded one. However, it should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100. Furthermore, dichlobenil is sprayed only one month in the winter. The chance that ripe berries or lettuce could become contaminated due to right-of-way spraying by the WSDOT is very remote. The likelihood that the public would be exposed to directly sprayed vegetation is also greatly reduced since people do not tend to walk to school or work much in the winter and they are usually not wearing shorts when they do (as is assumed for the maximum exposure scenario).

Hazard quotient values for maximum case worker exposure scenarios range from 1.9 to 5.3. While this does not automatically indicate that toxicity will occur, dichlobenil may present some risk given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The average exposure risk estimates indicate that no systemic adverse health effects would be expected from use of dichlobenil in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, dichlobenil would have negligible to moderate potential for causing acute systemic effects in humans.

The dichlobenil product primarily used by the WSDOT is Casaron, a dry granular formulation which is activated by water. The exposure scenarios developed for this risk assessment assume that the formulated material is liquid. Broadcast application of a granular product would result in negligible drift to nearby fruit or vegetables which may be present. Exposure to WSDOT workers would also be greatly reduced in comparison to applying a liquid formulation. Dichlobenil residues on sprayed vegetation would be more sparse and less uniform than a with a liquid formulation reducing exposure. Once the material is activated by sweat or precipitation it behaves more similar to a liquid.

Dichlobenil Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	3.0×10^{-1}	9.6×10^{0}
Mixer/Loader	8.9×10^{-1}	1.8×10^{1}
Applicator/Mixer/Loader	8.9×10^{-1}	1.5×10^{1}
Handheld Spray Applicator	4.5×10^{-1}	6.2×10^{0}

Dichlobenil Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	1.6×10^{-2}	1.2×10^{0}
Ingestion of Drift Contaminated Berries (child)	6.1×10^{-2}	4.5×10^{0}
Ingestion of Drift Contaminated Lettuce	5.1×10^{-2}	9.3×10^{0}
Ingestion of Drift Contaminated Beans	8.7×10^{-3}	1.4×10^{0}
Dermal Contact With Drift Contaminated Berries	1.9×10^{-2}	1.1×10^{0}
Dermal Contact With Directly Sprayed Vegetation	1.9×10^{-1}	1.0×10^{1}

5.2.9 Diuron

5.2.9.1 Systemic Effects - Average

Diuron is not expected to cause adverse health effects to the pubic under average conditions. All hazard quotients calculated for average case exposure scenarios were less than one, indicating that the expected acute dose for the public is less than the RfD.

Given the average case worker assumptions, diuron would have a low potential to cause acute systemic effects. Average worker exposure scenarios range from 1.3 for applicators to 3.8 for mixer/loaders and applicators/mixer/loaders. Hazard quotients

around one indicate that the expected acute human dose is approximately equal to the RfD. A hazard quotient greater than one does not automatically mean that an adverse effect will occur. Workers can reduce exposure by the using precautionary measures when spraying diuron such as using protective clothing and reducing application rates.

5.2.9.2 Systemic Effects - Maximum

Under the conditions specified for the maximum case exposure scenarios, diuron might be expected to pose a hazard to the public by any of the maximum case exposure pathways. All pathways evaluated have hazard quotients over one. Values range from 14 for dermal contact from drift contaminated berries to 130 for contact with directly sprayed vegetation. Diuron is sprayed two months in the spring, thus, the chances of contaminating ripe berries, lettuce, or beans are very remote. Public exposure to directly sprayed vegetation is also greatly reduced since people tend to walk to school or work less in the spring time than estimated for the maximum exposure scenario. Shorts are less frequently worn in the cool spring months than in the summer, as is assumed for the maximum exposure scenario.

Diuron could present some risk to workers given the maximum case assumptions. Hazard quotient values range from 77 for handheld or backpack sprayers to 220 for mixer/loaders. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 300. Diuron has a relatively small RfD value in comparison to acute toxicity values indicating a tendency towards slow clearance rates from the mammalian system. The risk associated with a single exposure could be less than indicated by these risk estimates. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and keeping application rates well below maximum recommended label rates.

Under conditions specified by the maximum case exposure assumptions, duiron would have moderate to high potential for causing acute systemic effects. For worker and public safety diuron application rates should not approach maximum recommended label rates. Historical use of diuron has indicated that average WSDOT rates are only eight percent of the maximum application rate allowed.

The use of diuron at the maximum label rate of 40 lb ai/acre is very rare. At this rate it is used as a soil sterilant. Examples of diuron use at this rate include small spot applications around road signs and barriers and in railroad applications where it is necessary to "knock back vegetation for a couple of years." Diuron is not normally use as a soil sterilant since other products are better suited for this purpose. (Bone, 1992)

Diuron Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.3x10^0$	$1.2x10^2$
Mixer/Loader	$3.8x10^0$	$2.2x10^2$
Applicator/Mixer/Loader	$3.8x10^0$	$1.9x10^2$
Handheld Spray Applicator	$1.9x10^0$	$7.7x10^1$

Diuron Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$6.6x10^{-2}$	$1.5x10^1$
Ingestion of Drift Contaminated Berries (child)	$2.6x10^{-1}$	5.7x1
Ingestion of Drift Contaminated Lettuce	$2.2x10^{-1}$	$1.2x10^2$
Ingestion of Drift Contaminated Beans	$3.7x10^{-2}$	$1.8x10^1$
Dermal Contact With Drift Contaminated Berries	$8.0x10^{-2}$	$1.4x10^1$
Dermal Contact With Directly Sprayed Vegetation	$7.9x10^{-1}$	$1.3x10^2$

5.2.10 Fluazifop-butyl

5.2.10.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to fluazifop-butyl are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of fluazifop butyl in the WSDOT vegetation management program.

Fluazifop Butyl Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$2.0x10^{-2}$	$2.3x10^{-1}$
Mixer/Loader	$5.9x10^{-2}$	$4.2x10^{-1}$
Applicator/Mixer/Loader	$5.9x10^{-2}$	$3.5x10^{-1}$
Handheld Spray Applicator	$3.0x10^{-2}$	$1.5x10^{-1}$

Fluazifop Butyl Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	1.0×10^{-3}	2.8×10^{-2}
Ingestion of Drift Contaminated Berries (child)	4.0×10^{-3}	1.1×10^{-1}
Ingestion of Drift Contaminated Lettuce	3.4×10^{-3}	2.2×10^{-1}
Ingestion of Drift Contaminated Beans	5.8×10^{-4}	3.4×10^{-2}
Dermal Contact With Drift Contaminated Berries	1.3×10^{-3}	2.7×10^{-2}
Dermal Contact With Directly Sprayed Vegetation	1.2×10^{-2}	2.5×10^{-1}

5.2.11 Fosamine

5.2.11.1 Systemic Effects - Average

Fosamine is not expected to cause adverse systemic effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.11.2 Systemic Effects - Maximum

Fosamine could potentially cause adverse health effects to the public under the conditions assumed for three of the maximum case exposure scenarios. Ingestion of drift contaminated berries (by children) and lettuce and contact with directly sprayed vegetation are approaching a level of concern with hazard quotient values of 2.7, 5.6 and 1.3, respectively. However, it should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 1000. Furthermore, fosamine is sprayed two months in the spring and has a relatively short half-life of less than 10 days. The chance of ripe berries or lettuce becoming contaminated due to right-of-way spraying by the WSDOT is unlikely. Fosamine is used to control woody plants such as blackberries, poison oak, evergreens, maple etc., not the type of vegetation hikers choose to walk through. Public exposure to directly sprayed vegetation is also greatly reduced since people tend to walk less to school or work in the spring time than estimated for the maximum exposure scenario. Shorts are worn less frequently in the cool spring months than in the summer, as is assumed for the maximum exposure scenario.

Hazard quotient values for maximum case worker exposure scenarios range from 0.74 for handheld or backpack sprayers to 2.1 for mixers/loaders. While this does not automatically indicate that toxicity will occur, fosamine may present some risk given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The average exposure risk estimates indicate that no systemic adverse health effects would be expected from use of fosamine in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates,

fosamine would have negligible to low potential for causing acute systemic effects in humans.

Fosamine Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$4.4x10^{-2}$	$1.2x10^{0}$
Mixer/Loader	$1.3x10^{-1}$	$2.1x10^{0}$
Applicator/Mixer/Loader	$1.3x10^{-1}$	$1.8x10^{0}$
Handheld Spray Applicator	$6.4x10^{-2}$	$7.4x10^{-1}$

Fosamine Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.2x10^{-2}$	$7.0x10^{-1}$
Ingestion of Drift Contaminated Berries (child)	$4.5x10^{-2}$	$2.7x10^{0}$
Ingestion of Drift Contaminated Lettuce	$3.8x10^{-2}$	$5.6x10^{0}$
Ingestion of Drift Contaminated Beans	$6.4x10^{-3}$	$8.5x10^{-1}$
Dermal Contact With Drift Contaminated Berries	$2.8x10^{-3}$	$1.4x10^{-1}$
Dermal Contact With Directly Sprayed Vegetation	$2.8x10^{-2}$	$1.3x10^{0}$

5.2.12 Glyphosate

5.2.12.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to glyphosate are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of glyphosate in the WSDOT vegetation management program.

Glyphosate Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$8.1x10^{-3}$	$4.5x10^{-1}$
Mixer/Loader	$2.4x10^{-2}$	$8.2x10^{-1}$
Applicator/Mixer/Loader	$2.4x10^{-2}$	$6.9x10^{-1}$
Handheld Spray Applicator	$1.2x10^{-2}$	$2.9x10^{-1}$

Glyphosate Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$6.1x10^{-4}$	$7.7x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$2.4x10^{-3}$	$3.0x10^{-1}$
Ingestion of Drift Contaminated Lettuce	$2.0x10^{-3}$	$6.2x10^{-1}$
Ingestion of Drift Contaminated Beans	$3.4x10^{-4}$	$9.4x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$5.2x10^{-4}$	$5.3x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$5.1x10^{-3}$	$4.8x10^{-1}$

5.2.13 Imazapyr

5.2.13.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to imazapyr are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of imazapyr in the WSDOT vegetation management program.

Imazapyr Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$2.4x10^{-3}$	$6.0x10^{-2}$
Mixer/Loader	$7.1x10^{-3}$	$1.1x10^{-1}$
Applicator/Mixer/Loader	$7.1x10^{-3}$	$9.3x10^{-2}$
Handheld Spray Applicator	$3.6x10^{-3}$	$3.9x10^{-2}$

Imazapyr Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$6.2x10^{-5}$	$3.6x10^{-3}$
Ingestion of Drift Contaminated Berries (child)	$2.4x10^{-4}$	$1.4x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$2.0x10^{-4}$	$2.9x10^{-2}$
Ingestion of Drift Contaminated Beans	$3.5x10^{-5}$	$4.4x10^{-3}$
Dermal Contact With Drift Contaminated Berries	$1.5x10^{-4}$	$7.1x10^{-3}$
Dermal Contact With Directly Sprayed Vegetation	$1.5x10^{-3}$	$6.5x10^{-2}$

5.2.14 Isoxaben

5.2.14.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to isoxaben are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of isoxaben in the WSDOT vegetation management program.

Isoxaben Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.0x10^{-2}$	$1.2x10^{-1}$
Mixer/Loader	$3.1x10^{-2}$	$2.2x10^{-1}$
Applicator/Mixer/Loader	$3.1x10^{-2}$	$1.9x10^{-1}$
Handheld Spray Applicator	$1.6x10^{-2}$	$7.7x10^{-2}$

Isoxaben Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$5.5x10^{-4}$	$1.5x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$2.1x10^{-3}$	$5.7x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$1.8x10^{-3}$	$1.2x10^{-1}$
Ingestion of Drift Contaminated Beans	$3.1x10^{-4}$	$1.8x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$6.6x10^{-4}$	$1.4x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$6.5x10^{-3}$	$1.3x10^{-1}$

5.2.15 Mecoprop

5.2.15.1 Systemic Effects - Average

Mecoprop is not expected to cause adverse health effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.15.2 Systemic Effects - Maximum

Mecoprop is also not expected to pose a hazard to the public by the maximum case exposure pathways. Contact with directly sprayed vegetation has the highest hazard quotient value of 0.79. Hazard quotient values for maximum case worker exposure scenarios range from 0.42 for handheld or backpack sprayers to 1.2 for mixer/loaders,

approaching a level of concern. Hazard quotients around one indicate that the expected acute human dose is equal to the RfD. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100. Mecoprop could present some low risk to workers, given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The average exposure risk estimates indicate that no systemic adverse health effects would be expected from use of mecoprop in the WSDOT vegetation management program. Under the conditions specified by the maximum case exposure estimates, mecoprop would have negligible to low potential for causing acute systemic effects in humans.

Mecoprop Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	7.8×10^{-2}	6.5×10^{-1}
Mixer/Loader	2.3×10^{-1}	1.2×10^{0}
Applicator/Mixer/Loader	2.3×10^{-1}	1.0×10^{0}
Handheld Spray Applicator	1.2×10^{-1}	4.2×10^{-1}

Mecoprop Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	2.1×10^{-3}	3.9×10^{-2}
Ingestion of Drift Contaminated Berries (child)	8.0×10^{-3}	1.5×10^{-1}
Ingestion of Drift Contaminated Lettuce	6.7×10^{-3}	3.1×10^{-1}
Ingestion of Drift Contaminated Beans	1.2×10^{-3}	4.8×10^{-2}
Dermal Contact With Drift Contaminated Berries	5.0×10^{-3}	7.7×10^{-2}
Dermal Contact With Directly Sprayed Vegetation	4.9×10^{-2}	7.0×10^{-1}

5.2.16 Mefluidide

5.2.16.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to mefluidide are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of mefluidide in the WSDOT vegetation management program.

Mefluidide Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$6.3x10^{-3}$	$8.0x10^{-2}$
Mixer/Loader	$1.9x10^{-2}$	$1.5x10^{-1}$
Applicator/Mixer/Loader	$1.9x10^{-2}$	$1.2x10^{-1}$
Handheld Spray Applicator	$9.6x10^{-3}$	$5.1x10^{-2}$

Mefluidide Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.7x10^{-3}$	$4.9x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$6.5x10^{-3}$	$1.9x10^{-1}$
Ingestion of Drift Contaminated Lettuce	$5.4x10^{-3}$	$3.9x10^{-1}$
Ingestion of Drift Contaminated Beans	$9.3x10^{-4}$	$5.9x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$4.0x10^{-4}$	$9.5x10^{-3}$
Dermal Contact With Directly Sprayed Vegetation	$4.0x10^{-3}$	$8.7x10^{-2}$

5.2.17 Metsulfuron Methyl

5.2.17.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to metsulfuron methyl are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of metsulfuron methyl in the WSDOT vegetation management program.

Metsulfuron Methyl Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$2.3x10^{-4}$	$7.2x10^{-3}$
Mixer/Loader	$7.0x10^{-4}$	$1.3x10^{-2}$
Applicator/Mixer/Loader	$7.0x10^{-4}$	$1.1x10^{-2}$
Handheld Spray Applicator	$3.6x10^{-4}$	$4.6x10^{-3}$

Metsulfuron Methyl Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$6.2x10^{-6}$	$4.4x10^{-4}$
Ingestion of Drift Contaminated Berries (child)	$2.4x10^{-5}$	$1.7x10^{-3}$
Ingestion of Drift Contaminated Lettuce	$2.0x10^{-5}$	$3.5x10^{-3}$
Ingestion of Drift Contaminated Beans	$3.5x10^{-6}$	$5.3x10^{-4}$
Dermal Contact With Drift Contaminated Berries	$1.5x10^{-5}$	$8.5x10^{-4}$
Dermal Contact With Directly Sprayed Vegetation	$1.5x10^{-4}$	$7.8x10^{-3}$

5.2.18 Napropamide

5.2.18.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to napropamide are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of napropamide in the WSDOT vegetation management program.

Napropamide Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$3.0x10^{-2}$	$2.4x10^{-1}$
Mixer/Loader	$9.0x10^{-2}$	$4.4x10^{-1}$
Applicator/Mixer/Loader	$9.0x10^{-2}$	$3.7x10^{-1}$
Handheld Spray Applicator	$4.6x10^{-2}$	$1.5x10^{-1}$

Napropamide Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.6x10^{-3}$	$2.9x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$6.2x10^{-3}$	$1.1x10^{-1}$
Ingestion of Drift Contaminated Lettuce	$5.2x10^{-3}$	$2.3x10^{-1}$
Ingestion of Drift Contaminated Beans	$8.9x10^{-4}$	$3.5x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$1.9x10^{-3}$	$2.8x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$1.9x10^{-2}$	$2.6x10^{-1}$

5.2.19 Oryzalin

5.2.19.1 Systemic Effects - Average

Oryzalin is not expected to cause adverse health effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.19.2 Systemic Effects - Maximum:

Oryzalin is also not expected to pose a hazard to the public by the maximum case exposure pathways. Contact with directly sprayed vegetation has the highest hazard quotient value of 0.78.

Hazard quotient values for maximum case worker exposure scenarios range from 0.46 for handheld spray applicators to 1.3 for mixer/loaders. Hazard quotients around one indicate that the expected acute human dose is equal to the RfD. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100. Oryzalin could present some risk given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

5.2.19.3 Cancer Effects - Average

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (one in one hundred thousand). The estimated cancer risk for both worker and public average case scenarios is less than this benchmark value. Values ranged from $2.8x10^{-9}$ (2.8 in one billion) for drift contaminated lettuce to $5.0x10^{-6}$ (5 in one million) calculated for mixer/loaders and applicator/mixer/loaders.

5.2.19.4 Cancer Effects - Maximum

Maximum cancer risk values for the public ingesting contaminated berries and dermal contact of directly sprayed vegetation exceed the $1x10^{-5}$ (1 in one hundred thousand) acceptable benchmark with values of $4.0x10^{-5}$ (4.0 in one hundred thousand) and $3.6x10^{-4}$ (3.6 in ten thousand), respectively. Oryzalin is sprayed in the spring. The chances of contaminating ripe berries are very remote. It is a preemergence herbicide and is not sprayed on established weeds. Therefore the likelihood of the public receiving a significant dermal exposure very doubtful.

Under the conditions specified for the maximum case exposure scenarios, all maximum worker cancer risk estimates exceed $1x10^{-5}$. Values range from $3.8x10^{-5}$ (3.8 in one hundred thousand) for handheld spray applicators to $1.1x10^{-4}$ (1.1 in ten thousand) for mixer/loaders. WSDOT workers can reduce exposure by wearing appropriate protective clothing when spraying Oryzalin and reducing application rates.

The risk assesment values for estimating the potential to cause acute systemic health effects indicate that the use of oryzalin in the WSDOT vegetation management program would be of negligile to low concern. The carcinogenic risks for average exposure

assumptions are below the level of concern and for maximum case exposure are negligible to moderate.

Oryzalin Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$4.7x10^{-2}$	$7.2x10^{-1}$
Mixer/Loader	$1.4x10^{-1}$	$1.3x10^{0}$
Applicator/Mixer/Loader	$1.4x10^{-1}$	$1.1x10^{0}$
Handheld Spray Applicator	$7.1x10^{-2}$	$4.6x10^{-1}$

Oryzalin Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$2.5x10^{-3}$	$8.7x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$9.6x10^{-3}$	$3.4x10^{-1}$
Ingestion of Drift Contaminated Lettuce	$8.0x10^{-3}$	$7.0x10^{-1}$
Ingestion of Drift Contaminated Beans	$1.4x10^{-3}$	$1.1x10^{-1}$
Dermal Contact With Drift Contaminated Berries	$3.0x10^{-3}$	$8.5x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$2.9x10^{-2}$	$7.8x10^{-1}$

Oryzalin Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$1.7x10^{-6}$	$5.9x10^{-5}$
Mixer/Loader	$5.0x10^{-6}$	$1.1x10^{-4}$
Applicator/Mixer/Loader	$5.0x10^{-6}$	$9.0x10^{-5}$
Handheld Spray Applicator	$2.5x10^{-6}$	$3.8x10^{-5}$

Oryzalin Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.7x10^{-7}$	$4.0x10^{-5}$
Ingestion of Drift Contaminated Lettuce	$2.8x10^{-9}$	$1.6x10^{-6}$
Ingestion of Drift Contaminated Beans	$1.2x10^{-8}$	$6.2x10^{-6}$
Dermal Contact With Drift Contaminated Berries	$4.1x10^{-8}$	$5.9x10^{-6}$
Dermal Contact With Directly Sprayed Vegetation	$1.1x10^{-6}$	$3.6x10^{-4}$

5.2.20 Oxadiazon

5.2.20.1 Systemic Effects - Average

Oxadiazon is not expected to cause adverse health effects to the public from any average case exposure pathway evaluated. All hazard quotient values are less than 1, which indicates that the expected human doses are less than the RfD. Hazard quotient values for average case worker exposure scenarios range from 0.6 for applicators to 1.8 for mixer/loaders and applicator/mixer/loaders. Hazard quotients around one indicate that the expected acute human dose is approximately equal to the RfD. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100.

5.2.20.2 Systemic Effects - Maximum

Oxadiazon might be expected to pose a hazard to the public given the maximum case exposure assumptions. Children ingesting drift contaminated berries, ingestion of drift contaminated lettuce, dermal contact with drift contaminated berries and contact with directly sprayed vegetation all have hazard quotient values over one, 2.3, 4.7, 1.1, and 10 respectively. However, oxadiazon is sprayed in the spring thus the chances of contaminating ripe berries or lettuce are very remote. Public exposure to directly sprayed vegetation is also greatly reduced since people tend to walk to school or work less in the spring time than estimated for the maximum exposure scenario. Shorts are less frequently worn in the cool spring months than in the summer as is assumed for the maximum exposure scenario.

Under the conditions specified for the maximum exposure scensrios, oxadiazon could present some risk to workers. Hazard quotient values range from 6.2 for handheld applicators to 18 for mixer/loaders. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

5.2.20.3 Cancer Effects - Average

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (one in one hundred thousand). The estimated cancer risk for both worker and public average case scenarios range from $1.9x10^{-9}$ (1.9 in one billion) for ingestion of contaminated lettuce to $6.9x10^{-6}$ (6.9 in one million) calculated for mixer/loaders and applicator/mixer/loaders.

5.2.20.4 Cancer Effects - Maximum

Maximum cancer risk values for the public ingesting contaminated berries and dermal contact of directly sprayed vegetation exceed the $1x10^{-5}$ acceptable benchmark with values of $2.9x10^{-5}$ (2.9 in one hundred thousand) and $5.1x10^{-4}$ (5.1 in ten thousand), respectively. Oxadiazon is sprayed in the spring. The chances of contaminating ripe berries are very remote. It is a preemergence herbicide and is usually not sprayed on established weeds. The reduced usage of the roadside discussed above and cool weather deterring people to wear shorts reduce the likelihood of the public receiving a significant dermal exposure.

Maximum worker cancer risk estimates exceed $1x10^{-5}$, values range from $5.4x10^{-5}$ (5.4 in one hundred thousand) for handheld applicators to $1.5x10^{-4}$ (1.5 in ten thousand) for mixer/loaders. WSDOT workers can reduce potential exposure by wearing appropriate protective clothing when spraying oxadiazon and application rates should not approach maximum recommended label rates.

The risk assesment values for estimating the potential to cause acute systemic health effects indicate that average use of oxadiazon in the WSDOT vegetation management program would be of negligible to low concern. Maximum exposure scenarios indicate it would have negligible to moderate potential for systemic effects. The carcinogenic risks for average exposure assumptions are below the level of concern and for maximum case exposure are negligible to moderate.

Oxadiazon Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$6.0x10^{-1}$	$9.6x10^{0}$
Mixer/Loader	$1.8x10^{0}$	$1.8x10^{1}$
Applicator/Mixer/Loader	$1.8x10^{0}$	$1.5x10^{1}$
Handheld Spray Applicator	$9.1x10^{-1}$	$6.2x10^{0}$

Oxadiazon Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.6x10^{-2}$	$5.8x10^{-1}$
Ingestion of Drift Contaminated Berries (child)	$6.2x10^{-2}$	$2.3x10^{0}$
Ingestion of Drift Contaminated Lettuce	$5.2x10^{-2}$	$4.6x10^{0}$
Ingestion of Drift Contaminated Beans	$8.9x10^{-3}$	$7.1x10^{-1}$
Dermal Contact With Drift Contaminated Berries	$3.8x10^{-2}$	$1.1x10^{0}$
Dermal Contact With Directly Sprayed Vegetation	$3.8x10^{-1}$	$1.0x10^{1}$

Oxadiazon Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$2.3x10^{-6}$	$8.4x10^{-5}$
Mixer/Loader	$6.9x10^{-6}$	$1.5x10^{-4}$
Applicator/Mixer/Loader	$6.9x10^{-6}$	$1.3x10^{-4}$
Handheld Spray Applicator	$3.5x10^{-6}$	$5.4x10^{-5}$

Oxadiazon Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.2x10^{-7}$	$2.9x10^{-5}$
Ingestion of Drift Contaminated Lettuce	$1.9x10^{-9}$	$1.2x10^{-6}$
Ingestion of Drift Contaminated Beans	$8.2x10^{-9}$	$4.4x10^{-6}$
Dermal Contact With Drift Contaminated Berries	$5.7x10^{-8}$	$8.4x10^{-6}$
Dermal Contact With Directly Sprayed Vegetation	$1.5x10^{-6}$	$5.1x10^{-4}$

5.2.21 Picloram

5.2.21.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to picloram are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that the potential human doses are less than the RfD .

5.2.21.2 Cancer Effects Summary

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (one in one hundred thousand). The estimated cancer risk for both worker and public average and maximum case scenarios is less than $8.6x10^{-7}$ (8.6 in ten million) calculated for public maximum dermal exposure to directly sprayed vegetation. Therefore, according to the lifetime cancer risk estimated from average and maximum exposure scenarios and the very low carcinogenic potency of picloram, 0.003 $(mg/kg/day)^{-1}$, the risks associated with use of picloram in the WSDOT vegetation management program are consider to be acceptable.

The risk assesment values for estimating the potential to cause acute systemic health effects indicate that the use of picloram in the WSDOT vegetation management program would be of negligible concern. Likewise, the carcinogenic risks for average and maximum exposure scenarios are below the level of concern.

Picloram Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$9.1x10^{-4}$	$1.8x10^{-2}$
Mixer/Loader	$2.7x10^{-3}$	$3.3x10^{-2}$
Applicator/Mixer/Loader	$2.7x10^{-3}$	$2.7x10^{-2}$
Handheld Spray Applicator	$1.4x10^{-3}$	$1.1x10^{-2}$

Picloram Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$5.0x10^{-4}$	$2.2x10^{-2}$
Ingestion of Drift Contaminated Berries (child)	$1.9x10^{-3}$	$8.7x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$1.6x10^{-3}$	$1.8x10^{-1}$
Ingestion of Drift Contaminated Beans	$2.8x10^{-4}$	$2.7x10^{-2}$
Dermal Contact With Drift Contaminated Berries	$5.8x10^{-5}$	$2.1x10^{-3}$
Dermal Contact With Directly Sprayed Vegetation	$5.7x10^{-4}$	$1.9x10^{-2}$

Picloram Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$3.1x10^{-9}$	$1.9x10^{-7}$
Mixer/Loader	$9.4x10^{-9}$	$3.4x10^{-7}$
Applicator/Mixer/Loader	$9.4x10^{-9}$	$2.9x10^{-7}$
Handheld Spray Applicator	$4.7x10^{-9}$	$1.2x10^{-7}$

Picloram Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.1x10^{-9}$	$3.3x10^{-7}$
Ingestion of Drift Contaminated Lettuce	$1.8x10^{-11}$	$1.3x10^{-8}$
Ingestion of Drift Contaminated Beans	$7.8x10^{-11}$	$5.1x10^{-8}$
Dermal Contact With Drift Contaminated Berries	$2.6x10^{-11}$	$4.7x10^{-9}$
Dermal Contact With Directly Sprayed Vegetation	$4.4x10^{-9}$	$8.6x10^{-7}$

5.2.22 Pronamide

5.2.22.1 Systenic Effects Summary

The risks to workers and the public associated with exposure to pronamide are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that the potential human doses are less than the RfD .

5.2.22.2 Cancer Effects Summary

The level of concern when estimating the potential lifetime cancer risk of a chemical is $1x10^{-5}$ (one in one hundred thousand). The estimated cancer risk for both worker and public average and maximum case scenarios (with the exception of public exposure to

directly sprayed vegetation) is less than 4.4×10^{-6} (calculated for worker mixer/loaders). Under the conditions specified for the maximum exposure scenario, the carcinogenic risk estimate for the public exposed dermally to directly sprayed vegetation is calculated to be 2×10^{-5} (2 in one hundred thousand). However, pronamide is sprayed only one month in the spring. Public exposure would be reduced since people tend to walk to school or work less in the spring time than estimated for the maximum exposure scenario. Shorts are less frequently worn in the cool spring months than in the summer, as is assumed for the maximum exposure scenario. Therefore, according to the lifetime cancer risk estimated from average and maximum exposure scenarios and the relatively low carcinogenic potency of pronamide, 0.016 $(mg/kg/day)^{-1}$, the risks associated with use of pronamide in the WSDOT vegetation management program are consider to be acceptable, with application rates below recommended maximum.

The risk assesment values for estimating the potential to cause acute systemic health effects indicate that the use of pronamide in the WSDOT vegetation management program would be of negligible concern. Likewise, the carcinogenic risks for average exposure scenarios are below the level of concern as are most maximum risk calculations. Given the maximum exposure assumptions, dermal exposure to the public from directly sprayed vegetation would be of low concern.

Pronamide Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	2.0×10^{-2}	3.2×10^{-1}
Mixer/Loader	6.0×10^{-2}	5.9×10^{-1}
Applicator/Mixer/Loader	6.0×10^{-2}	4.9×10^{-1}
Handheld Spray Applicator	3.0×10^{-2}	2.1×10^{-1}

Pronamide Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	1.1×10^{-3}	3.9×10^{-2}
Ingestion of Drift Contaminated Berries (child)	4.1×10^{-3}	1.5×10^{-1}
Ingestion of Drift Contaminated Lettuce	3.4×10^{-3}	3.1×10^{-1}
Ingestion of Drift Contaminated Beans	5.9×10^{-4}	4.7×10^{-2}
Dermal Contact With Drift Contaminated Berries	1.3×10^{-3}	3.8×10^{-2}
Dermal Contact With Directly Sprayed Vegetation	1.3×10^{-2}	3.5×10^{-1}

Pronamide Worker Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$6.6x10^{-8}$	$2.4x10^{-6}$
Mixer/Loader	$2.0x10^{-7}$	$4.4x10^{-6}$
Applicator/Mixer/Loader	$2.0x10^{-7}$	$3.7x10^{-6}$
Handheld Spray Applicator	$1.0x10^{-7}$	$1.5x10^{-6}$

Pronamide Public Risk Summary (Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.3x10^{-8}$	$3.3x10^{-6}$
Ingestion of Drift Contaminated Lettuce	$2.2x10^{-10}$	$1.3x10^{-7}$
Ingestion of Drift Contaminated Beans	$9.4x10^{-10}$	$5.1x10^{-7}$
Dermal Contact With Drift Contaminated Berries	$3.3x10^{-9}$	$4.8x10^{-7}$
Dermal Contact With Directly Sprayed Vegetation	$4.3x10^{-8}$	$2.0x10^{-5}$

5.2.23 Sethoxydim

5.2.23.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to sethoxydim are below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of sethoxydim in the WSDOT vegetation management program.

Sethoxydim Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$4.7x10^{-4}$	$6.1x10^{-3}$
Mixer/Loader	$1.4x10^{-3}$	$1.1x10^{-2}$
Applicator/Mixer/Loader	$1.4x10^{-3}$	$9.5x10^{-3}$
Handheld Spray Applicator	$7.1x10^{-4}$	$3.9x10^{-3}$

Sethoxydim Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$8.4x10^{-5}$	$3.7x10^{-3}$
Ingestion of Drift Contaminated Berries (child)	$4.8x10^{-4}$	$1.4x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$4.0x10^{-4}$	$3.0x10^{-2}$
Ingestion of Drift Contaminated Beans	$6.9x10^{-5}$	$4.5x10^{-3}$
Dermal Contact With Drift Contaminated Berries	$3.0x10^{-5}$	$7.3x10^{-4}$
Dermal Contact With Directly Sprayed Vegetation	$2.9x10^{-4}$	$6.6x10^{-3}$

5.2.24 Sulfometuron Methyl

5.2.24.1 Systemic Effects Summary

The risks to workers and the public associated with exposure to sulfometuron methyl is below levels of concern for all pathways evaluated. The hazard quotients from each worker and public exposure scenario, both average and maximum risk calculations, were less than one indicating that potential acute human doses are less than the RfD.

The estimated risks indicate that no adverse health effects would be expected from use of sulfometuron methyl in the WSDOT vegetation management program.

Sulfometuron Methyl Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	$5.7x10^{-4}$	$1.3x10^{-2}$
Mixer/Loader	$1.7x10^{-3}$	$2.5x10^{-2}$
Applicator/Mixer/Loader	$1.7x10^{-3}$	$2.1x10^{-2}$
Handheld Spray Applicator	$8.7x10^{-4}$	$8.6x10^{-3}$

Sulfometuron Methyl Public Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$1.5x10^{-4}$	$8.2x10^{-3}$
Ingestion of Drift Contaminated Berries (child)	$5.9x10^{-4}$	$3.2x10^{-2}$
Ingestion of Drift Contaminated Lettuce	$4.9x10^{-4}$	$6.5x10^{-2}$
Ingestion of Drift Contaminated Beans	$8.4x10^{-5}$	$9.9x10^{-3}$
Dermal Contact With Drift Contaminated Berries	$3.7x10^{-5}$	$1.6x10^{-3}$
Dermal Contact With Directly Sprayed Vegetation	$3.6x10^{-4}$	$1.5x10^{-2}$

5.2.25 Triclopyr

5.2.25.1 Systemic Effects - Average

Triclopyr is not expected to cause adverse health effects to workers or the public from any average case exposure pathway evaluated. All hazard quotient values are less than one, which indicates that the expected human doses are less than the RfD.

5.2.25.2 Systemic Effects - Maximum

Maximum risk estimates for the public are below the level of concern (≤ 1) with one exception. Triclopyr might be expected to pose a hazard to the public given the maximum case exposure assumptions for ingesting drift contaminated lettuce. This value was calculted to be 2.1. Triclopyr is sprayed all year round and could potentially contaminate lettuce in a home garden. Contamination of lettuce can be reduced by using caution when spraying during the summer months and reducing application rates.

Hazard quotient values for maximum case worker exposure scenarios range from 0.46 for handheld spray applicators to 1.3 for mixer/loaders. Hazard quotients around one indicate that the expected acute human dose is equal to the RfD. It should be noted that the RfD is estimated using the NOAEL from a subchronic or chronic study and applying an "uncertainty" or "safety" factor of 100. Triclopyr could present some risk given the maximum case assumptions. Exposure can be reduced by wearing protective clothing during all phases of applying herbicides and reducing application rates.

The risk assesment values for estimating the potential to cause acute systemic health effects indicate that the use of triclopyr in the WSDOT vegetation management program would be of negligible to low concern.

Triclopyr Worker Risk Summary (Noncancer Effects)

Exposure Pathway	Average	Maximum
Applicator	1.6×10^{-2}	7.1×10^{-1}
Mixer/Loader	4.9×10^{-2}	1.3×10^{0}
Applicator/Mixer/Loader	4.9×10^{-2}	1.1×10^{0}
Handheld Spray Applicator	2.5×10^{-2}	4.6×10^{-1}

Triclopyr Public Risk Summary (Non-Cancer Effects)

Exposure Pathway	Average	Maximum
Ingestion of Drift Contaminated Berries (adult)	$2.6x10^{-3}$	$2.6x10^{-1}$
Ingestion of Drift Contaminated Berries (child)	$1.0x10^{-2}$	$1.0x10^{0}$
Ingestion of Drift Contaminated Lettuce	$8.5x10^{-3}$	$2.1x10^{0}$
Ingestion of Drift Contaminated Beans	$1.5x10^{-3}$	$3.2x10^{-1}$
Dermal Contact With Drift Contaminated Berries	$1.1x10^{-3}$	$8.4x10^{-2}$
Dermal Contact With Directly Sprayed Vegetation	$1.0x10^{-2}$	$7.7x10^{-1}$

5.3 Synergistic, Additive and Antagonistic Effects

Synergistic effects of chemicals are those that occur from exposure to two chemicals either simultaneously or within a relatively short period of time. Synergism occurs when the combined effects of two chemicals is greater than the sum of the effects of each agent individually (simple additive effect). Instances of chemical combinations that cause synergistic effects are relatively rare. Kociba and Mullison (USDA, 1989) in describing toxicological interactions with agricultural chemicals state:

> "Our present scientific knowledge in toxicology indicates that an exposure to a mixture of pesticides is more likely to lead to additivity or antagonism rather than synergism when considering the toxicological effects of such a combination. To be conservative and for reasons of safety, an additive type of toxicological response is generally assumed rather than an antagonistic type of response.

In the case of registered pesticides, a great amount of toxicological information is developed during the research and development of each individual pesticide. In addition to this information on individual pesticides, short term toxicity studies are always done prior to selling of a pesticide mixture. Should synergism unexpectedly be present in a proposed commercial mixture of two pesticides, it would be identified in such cases and would be dealt with accordingly. In toxicological tests involving a combination of commercial pesticides, synergism has not generally been observed.

Synergistic toxic effects of herbicide combinations other than US EPA-registered commercial mixtures are not normally studied. Time and money normally limit toxicity testing to the effects of herbicides individually. Combinations that could be tested are too numerous to make that testing feasible. Based on the limited amount of data available on pesticide combinations, it is possible but quite unlikely that synergistic effects would occur as a result of exposure to two or more of the herbicides considered in this analysis.

5.4 Quantitative Risk From Multiple Pathways

5.4.1 Calculating Risk Associated With Exposure To A Single Chemical But Multiple Pathways

Several simultaneous exposures to subthreshold levels of the same herbicide could result in an adverse health effect due to additive effects. Therefore, the risk associated with multiple exposure pathways must also be estimated (i.e., dermal exposure picking berries and ingestion of the berries picked). Exposure to two different chemicals which have the same target organ may also cause an unexpected adverse effect. This risk assessment evaluates combined risks by calculating the hazard index (HI), which is the sum of hazard quotients.

$$HI = HQ1 + HQ2 + \ldots HQX$$

The maximum or "worst case" exposure repeatedly use conservative assumptions in their calculations. As a result the scenarios described are only remotely possible for a handful of persons. (Paustenbach, 1989) Adding hazard quotients calculated using maximum exposure estimates only magnifies these conservative estimates resulting in an out of porportion and unrealistic estimate of risk. Maximum label rates are rarely required to control vegetation and since the average single day dose is already a very conservative exposure estimate, average hazard quotients were used to calculate the risk associated with exposure from multiple pathways.

Hazard index values of one or less are typically considered to be of no concern. Values slightly larger than one are of limited concern, considering that hazard quotients are calculated by comparing a potential single acute exposure to a reference dose (RfD) value (which represents the NOAEL from a chronic exposure study plus "uncertainty" or "safety" factors).

Tables 4-25 and 4-26, presents the hazard index for each compound, multiple pathways of exposure. Multiple exposure scenarios for WSDOT workers have already been addressed in the pathways chosen for risk analysis (i.e., applicator/mixer/loader).

The combined public exposure pathways considered are:

1) Hiking through sprayed vegetation to pick blackberries which are consumed the same day.

2) Consuming drift contaminated vegetables and berries in the same day (lettuce, beans and berries).

5.4.2 Summary of results

Neither of the combined pathways evaluated had a hazard index over one, indicating little potential for additive effects of concern. Total risk values for hiking through sprayed vegetation to pick blackberries which are eaten the same day (without washing) ranged from 9.4×10^{-1} for diuron to 1.7×10^{-4} for metsulfuron methyl. Ingesting drift contaminated

lettuce, beans and berries produced hazard index values from 3.2×10^{-1} for diuron to 3.0×10^{-5} for metsulfuron methyl.

5.5 Quantitative Risk From Multiple Chemicals

5.5.1 Calculating Risk Associated With Exposure To Multiple Chemicals

In order to estimate the risk associated from exposure to multiple chemicals, one must first identify which herbicides are used in conjunction with each other. Active ingredients used most commonly together by the WSDOT are:

1) Glyphosate	Sulfometuron methyl
2) Glyphosate	Diuron
3) 2,4-D	Dicamba
4) 2,4-D	Chlorsulfuron
5) Glyphosate	Oryzalin
6) 2,4-D	Picloram
7) Diuron	Sulfometuron methyl
8) 2,4-D	Triclopyr
9) 2,4-D	Metsulfuron methyl
10) Bromacil	Glyphosate Sulfometuron methyl
11) Diuron	Bromacil Glyphosate
12) 2,4-D	Chlosulfuron Picloram
13) 2,4-D	Dicamba Chlorsulfuron
14) Diuron	Glyphosate Sulfometuron methyl
15) Bromacil	Diuron Sulfometuron methyl
16) Picloram	Chlorsulfuron Metsulfuron methyl

This risk assessment evaluates combined risks by calculating the hazard index (HI), which is the sum of hazard quotients.

$$HI = HQ1 + HQ2 + \ldots HQX$$

The maximum or "worst case" exposure repeatedly use conservative assumptions in their calculations. As a result the scenarios described are only remotely possible for a handful of persons. (Paustenbach, 1989) Adding hazard quotients calculated using maximum exposure estimates only magnifies these conservative estimates resulting in an out of porportion and unrealistic estimate of risk. Maximum label rates are rarely required to control vegetation and since the average single day dose is already a very conservative exposure estimate, average hazard quotients were used to calculate the risk associated with exposure to multiple chemicals.

Hazard index values of one or less are typically considered to be of no concern. Values slightly larger than one are of limited concern, considering that hazard quotients are calculated by comparing a potential single acute exposure to a reference dose (RfD) value (which represents the NOAEL from a chronic exposure study plus "uncertainty" or "safety" factors).

Tables 4-27 and 4-28 present the hazard index (or the sum of the hazard quotients) of herbicides frequently used together, for each exposure pathway.

5.5.2 Summary of results

No combination of herbicides in this evaluation would be expected to cause an adverse health effect in the public for any (average) exposure pathway. All hazard index values were less than one, indicating little potential for additive effects of concern.

Total risk values range from:

1) 5.8×10^{-4} to 7.0×10^{-2} for ingestion of drift contaminated berries, adult.

2) 2.2×10^{-3} to 2.8×10^{-1} for ingestion of drift contaminated berries, child.

3) 1.9×10^{-3} to 2.3×10^{-1} for ingestion of drift contaminated lettuce.

4) 3.3×10^{-4} to 3.9×10^{-2} for ingestion of drift contaminated beans.

5) 9.1×10^{-5} to 8.1×10^{-2} for dermal exposure to drift contaminated berries.

6) 9.1×10^{-4} to 8.0×10^{-1} for dermal exposure to directly sprayed vegetation.

All herbicide combinations with diuron as one of the active ingredients exceed one for all worker pathways. Values ranged from 1.3 to 3.8. A hazard index greater than one but less than 10 indicates a low potential for the additive effects from diuron containing combinations to be of concern. Workers can reduce risk by using precaution when spraying diuron combinations such as using protective clothing and lowering diuron application rates.

Total risk values for other (non-diuron containing) herbicide combinations were less than or equal to one for all worker exposure pathways, indicating little potential for the additive effects from these herbicide combinations to be of concern.

5.6 Qualitative Human Health Risk Analysis

5.6.1 Inert Ingredients

There are basically two groups of inerts of concern that are being addressed in this risk assessment. The first is the petroleum distillates that includes kerosene, diesel and naphthalenes. The second is xylene. Both of these are included on the EPA's list number 2 that identifies those inerts that have structures which suggests that they are of toxicological concern and thus are rated as a high priority for testing. All of the other inerts for the herbicides listed are on EPA's list 3 (unknown toxicity) or on EPA's list 4 (minimal hazard).

Information for the petroleum distillates indicates that acute toxicities (LD_{50}s) are comparatively high, indicating a low toxicity to mammals. There is, however, concern about the potential for petroleum distillates to cause cancer. Although kerosene and

diesel oil have not been shown to cause cancer, they are likely to to have a slight carcinogenic potency because they contain small amounts of chemicals known or suspected to cause cancer. However, even with these contaminants, the cancer potencies for these inerts is approximately 6,000 times lower than the potencies for some of the other herbicides being considered in this document.

Xylene is considered to be relatively non-toxic to mammals based on an acute LD_{50} for mammals of 4,300 mg/kg and it not listed as a carcinogen by the IARC, NTP, or OSHA.

Both of these inert ingredients are irritants. They can cause erythema, dryness and defatting of the skin. In addition, xylene can depress the central nervous system, cause gastrointestinal disturbances and possibly hematemesis (vomiting blood). The concentrations necessary to generate these symptoms are not likely to occur in well ventilated areas such as those encountered while making chemical applications.

The greatest human risk from these inert ingredients is for the mixer/applicators themselves. There is essential no risk to the general public as a result of the use of these inerts for roadside vegetation management. Because all herbicide applications occur outdoors, it is not likely the concentrations of these inerts will accumulate to levels that will cause symptoms. However, skin contact and irritation is a possibility and potective equipment should be worn to reduce this risk.

5.6.2 Adjuvants

Some uncertainty exists concerning the potential toxicity of the adjuvants used in applying herbicides. However, existing data indicate that toxicity of adjuvants is genenerally low. Also, the transport of these adjuvants from the target area is considered to be low, thereby reducing off target area contact to workers, the general public or ecological receptors. Surfactants and petroleum distillates bind to soil thereby reducing the amount available for surface runoff. The low volatility of these compounds indicate that volatolization to the air would be insignificant. Although data are not available to address the effect of these adjuvants on the potential toxicity of herbicides, their use is considered beneficial. This is due to the fact that the adjuvants enhance the biological effectiveness of herbicides by improving spray delivery, enhancing penetration, or by maximizing efficacy through enhanced phytotoxicity or selectivity. The net result is that, by using the adjuvants, smaller amounts of herbicide are required (Jones & Stokes, 1991)

5.6.3 Fertilizers

The greatest impact from the use of fertilizers, as it relates to human health, is the effect that fertilizers might have on water quality (surface and groundwater). This section is discussed further in Appendix A, Chapter 4. In general, the amount of fertilizer used by the WSDOT is insignificant compared to the amount of fertilizers used in agronomic practices throughout the state. Therefore, the impact on human health as a result of fertilizer use on roadsides by WSDOT is insignificant.

6.0 Risk Communication (Summary)

6.1 Understanding Risk

For the sake of putting the risk numbers into perspective for the layperson, the following scheme was developed (assuming an uncertainty factor of 100 is used to calculate the RfD value).

Hazard Quotient	Cancer Risk	Estimated Risk
≤ 1	$\leq 1x10^{-5}$	Negligible Risk
$> 1, \leq 10$	$> 1x10^{-5}$, $\leq 1x10^{-4}$	Low Risk Sensitive individuals may be at risk, margin of safety 10 to 100 times the NOEL. For small populations with a potential risk of $< 1x10^{-4}$, potential carcinogens are not regulated in any special way.
> 10, ≤ 100	$> 1x10^{-4}$, $\leq 4x10^{-3}$	Moderate Risk Humans may be at risk, margin of safety 1 to 10 times the NOEL. Some chemicals are regulated for small popu-lations with these individual cancer risks.
> 100	$> 4x10^{-3}$	High Risk The expected dose is larger than the NOEL, there is no margin of safety. Every potentially carcinogenic chem-ical with an individual lifetime risk above $4x10^{-3}$ is regulated with respect to its cancer potency.

Tables 4-29 through 4-32 summarize the risk data for each individual herbicide, assuming single routes of exposure. Compounds which are sprayed in the spring, fall or winter are not likely to contaminate ripe berries, lettuce or beans. It's also unlikely that the public will be dermally exposed to these chemicals when picking berries. Herbicides which fall into this category have not be include in the summary tables for these four public exposure routes. They are: 2,4-DP, bromacil and dichlobenil for winter or fall application and benefin, chlorthal dimethyl, diuron, fluazifop-butyl, imazapyr, isoxaben, mefluidide, napropamide, oryzalin, oxadiazon, pronamide, sethoxydim and sulfometuron methyl for spring application.

After examination of the risk summary tables it becomes obvious that most of the compounds can be used with negligible systemic or carcinogenic risk to the public and WSDOT workers under the average exposure conditions outlined in Section 3 of this chapter. The average worker exposure scenario using diuron and oxadiazon indicate that WSDOT employees would be at some low level of risk. However, workers in the 2,4-D field studies (which were used as models for estimating exposure) were reported as not wearing protective equipment such as respirators, impermeable gloves, coveralls, splash shields or rubber boots. Thus actual dose levels in WSDOT workers would be much lower than in the model used to estimate exposure. When applying these herbicides personal protection equipment should be used and application rates should not reach maximum allowable levels. (As a general rule when applying any pesticide product protective measures should always be taken).

2,4-D does not pose a significant risk in average case exposure scenarios. However, since it does pose a low to moderate risk in all maximum exposure scenarios the same precautions should be used when applying 2,4-D as suggested for diuron and oxadiazon.

Most of the compounds can also be used without risk given the maximum exposure scenario assumptions. Exposure to those compounds which pose a low, moderate or high risk can be reduced by spraying below maximum label rates and using protective equipment while mixing, loading and/or applying. The public should exercise common sense when using the roadsides and never eat berries picked by a right-of-way without washing them first. Vegetables should be washed before consumption irregardless of where they were grown or purchased.

Maximum (or worst case) scenarios are examined in an effort to ensure that the true risk to which anyone will be exposed will be less than that predicted. "...Although this might appear to be an admirable goal, such approaches to 'protect' everyone from potential harm have a number of shortcomings. One problem with the repeated use of exceedingly unlikely exposure scenarios is that it is extremely difficult to compare various assessments since different scientists can incorporate differing levels of conservatism in their assumptions. Many exposure scenarios have been called 'worst case,' as if to imply that they are feasible or plausible, but frequently, the scenarios described are remotely possible for only a handful of persons." (Paustenbach, 1989)

6.2 Uncertainties

The major uncertainties created in doing a risk assessment are:

1) The risk assessment process;

2) Toxicity values;

3) Exposure assumptions;

4) Chemical fate and transport;

and

5) Summing risks.

6.2.1 The Risk Assessment Process

The process of doing a risk assessment, as dictated by the scientific community, is constantly in the state of flux. The Food and Drug Administration (FDA) prefers the Margin of Safety (MOS) approach using NOEL's from acute, subchronic, chronic and reproduction/developmental studies to calculate a MOS for each systemic toxic effect category. The Environmental Protection Agency (EPA) recommends the use of a single Reference Dose (RfD) value for estimating the risk of systemic toxic effects, which calculated using the lowest NOEL, from the most sensitive toxicity study. An MOS over 100 is considered a negligible risk. An uncertainty factor of 100 is usually applied to calculate the RfD and values of one and less are considered to be a negligible risk. However, factors of 300 and 1000 also are frequently used to adjust for an inadequate data base or calculation of the RfD from a subchronic study. The EPA has tried to standardize the risk assessment process by publishing guideline documents and the exposure factors handbook. However, the risk assessment process is still subjective to some extent.

6.2.2 Toxicity Values

Data gaps or incomplete data packages exist for some of the chemicals. In the case of non-food use herbicides, these are the longer more expensive (reproduction/ developmental and/or chronic/oncogenicity) studies which generate the lowest NOEL's. The data can also be old and below the quality standards and guideline requirements of today. Studies accepted by the U.S. EPA may be found unacceptable by State Agencies. Who is right and does one Agencies objections render the study results as obsolete? This risk assessment went by the EPA's judgement of a studies quality and used their RfD and cancer potency calculations whenever possible.

In order to measure a chemicals potential to cause noncancer effects, an estimated single day dose is calculated and compared to the RfD in order arrive at a risk number. The RfD value is an estimated human dose (including sensitive populations) at which no adverse health effects would be expected over an entire lifetime of exposure. An acute dose will have to be many times greater (possibly orders of magnitude greater) than the RfD value before effect will occur.

Calculation of the extra lifetime risk due to chronic exposure to a potential carcinogen requires extrapolation of the effects seen in laboratory species to humans and extrapolation from high doses used in laboratory studies to low doses typically encountered by humans. The low dose extrapolation is done using a model which assumes that no level of exposure is safe. These models predict the risk to humans associated with doses which they might receive and are very conservative estimates. As stated by Paustenbach,(1989):

> "The upper bound risk estimates are based on data from the most sensitive sex, strain and species of test animal and for the cancer tumor type (often including benign tumors) and tissue that maximizes the estimated potency. Extrapolation of animal results to humans are calculated from the ratio of surface areas, an approach more conservative

than scaling by weight, and extrapolation from high- to low-dose response is based on a dose-response model that exhibits linearity at low doses." (Whipple, 1986)

The model selected for use in estimating the low dose risk adds uncertainty because different models can provide very different estimates of risk.

6.2.3 Exposure Assumptions

Numerous assumptions are made in the process of estimating exposure. Each assumption introduces a degree of uncertainty in the way of an over estimate or an under estimate. Realistic scientifically sound values are used and referenced. In order not to under estimate risk, conservative values are employed. This, in effect, causes risk assessments to become greatly exaggerated over estimates. The following represent sources of uncertainty when assessing exposure:

1) Subjective interpretation of published data.

2) Using data generated from an agricultural exposure field study verses a right-of-way field study.

3) Utilizing exposure data generated for another (surrogate) compound.

4) Estimating the amount of drift, distance of drift etc. generated by the application of a surrogate compound.

5) Estimating the number of berry pickers.

6) Estimating the amount and frequency of berries, lettuce, beans or other potentially exposed garden vegetable consumed in a day, month or year.

7) Estimating the number of people using the roadside going to and from work or school and frequency during each season.

8) Assuming the amount of clothing worn during outdoor activities per season.

9) Estimating the amount of time spent picking berries per month or spent jogging/hiking along the roadway.

10) Using a dermal penetration factor generated by a rodent study.

11) In the absence of animal data using a generic dermal penetration factor for herbicides (10%).

12) Estimating residue levels.

13) Assuming that berries are contaminated by spray drift. Given that it is WSDOT policy not to spray near bushes while blossoming or bearing fruit the likelihood that blackberries would become contaminated at all is very remote.

6.2.4 Chemical Fate and Transport

Environmental fate and transport of the chemical was not considered when doing the risk analysis calculations. 100% of the parent compound was considered available for consumption and dermal uptake in the public exposure scenarios when many of the compounds are rapidly hydrolyzed, photodegraded, biodegraded, volatilized, absorbed, metabolized or transported. The Figure 4-6 demonstrates some of the possible pathways a chemical can take in the environment.

6.2.5 Summing Risks

The risks from compounds which have the same target organ of toxicity could have an additive effect. Risks calculated for frequently used herbicides can be summed. In this risk assessment, however, target organ toxicity or mechanism of action was not considered when summing the risks associated with the use of multiple chemicals. This can generate misleading data and uncertainty. Since none of the 16 combinations evaluated generated an average risk value exceeding one, the uncertainty created by not considering target organs is of no consequence.

Several simultaneous exposures to subthreshold levels of a compound through different pathways could result in a health effect. Pathways were identified which might occur simultaneously or within a short period of time, for example picking berries and eating them later that night. To estimate this combined risk the hazard quotients for each pathway, for each individual chemical, are summed. Different routes of exposure to the same compound can have very different toxic effects and some chemicals are rapidly metabolized reducing the body burden before a second exposure occurs. Summing the risk estimates for multiple pathways without considering the mechanism of action or metabolic fate introduces another uncertainty.

The uncertainties involved with summed cancer risk estimates are similar to those generated by summing noncancer risk estimates. No consideration is given to mechanism of action or target organ. It assumes that all carcinogenic effects are additive without considering antagonistic or synergistic effects more common to cancer causing agents. Such as the combined effects of a promoter and a compound requiring promotion in order to cause tumor formation. Instead, all carcinogens are given equal weight. Compounds which are classified as possible human carcinogens (Group C) are treated the same as a known human carcinogen (Group A) which is extremely misleading. Lastly, the cancer potency factor (or slope factor) estimated in the mathematical model, represents the upper bound or 95th percent confidence limit. The summing of these 95% confidence limits leads to a very large over-estimate of risk if several chemicals are involved.

6.3 Differences Between Risk Assessments

In risk assessment documents of this magnitude there are many minor differences or location and use pattern specific parameters which vary. This subsection discusses only the major differences between the Environmental Impact Statements (Report) prepared for WSDOT, Caltrans (Jones & Stokes, 1991), BLM (USDI,1991), and the USFS (1988):

1) The USDI and USFS documents employ the margin of safety method to estimate risk while WSDOT and Jones & Stokes use the reference dose method recommended by the US EPA.

2) The amount of detail provided in the risk assessments (how the actual calculations were done) varies. In the USDI (1988) FEIS 4 routine-realistic and 4 routine-worse case scenarios were outlined. It is not clear how the worse case scenarios were used in the calculations described. Specifically backpack spraying of a 60 acre conifer release site by 14 applicators for 9 hours and hand treatment of 9 acres by 4 applicators for 9 hours. It is unclear how these values were used in the exposure assessment.

3) All four documents used urinary excretion rates reported in the 2,4-D field study by Nash, et al. (1982), to estimate worker doses. The data was interpreted differently in each assessment without resulting in significant differences. The most striking difference was in how the Jones & Stokes DEIR quantified the number of pounds of active ingredient Caltrans workers were exposed to while mixing, loading and/or applying herbicides. Urinary excretion rate was normalized to 1 lb active ingredient (ai) and reported in mg/kg/lb ai. To calculate dose, the application rate is multiplied by the number of acres sprayed (or to be sprayed) then multiplied by the appropriate urinary excretion rate. If the average application rate is 4 lb ai/acre and the average number of acres a ground crew can spray is 25 (average acreage sprayed by both USDI BLM and WSDOT crews), then workers are exposed to 100 pounds of active ingredient. Due to a lack of Caltrans specific data Jones & Stokes assumed 20 pounds of active ingredient for an average estimate and 40 lb ai for the maximum estimate. This would be equivalent to about 1 lb ai per acre. (The average application rate of 13 compounds in this WSDOT EIS exceed this level). The calculated number of pounds of active ingredient workers are exposed to in this WSDOT EIS range from 0.75 pounds for sethoxydim to 328 for chlorthal dimethyl, average case, and 3.5 for sethoxydim to 700 pounds for fosamine, maximum exposure scenario.

4) The WSDOT EIS basically follows the same methods used in the Jones & Stokes DEIR for calculating public exposures. USDI and the USFS did not go into enough detail in their exposure assessment and the calculations are difficult to reproduce. The models used to estimate public exposure were adequate in all the documents. The WSDOT EIS does not use the same method to calculate the exposure to people walking through sprayed vegetation as the other three. A 2,4,5-T field study by Lavy, et al. (1980) was used, in the other documents, in which a person walked through a field two hours after it was sprayed. No detectable amounts of 2,4,5-T were found on the patches worn by the pedestrian at a detection limit of 0.01 mg. They assumed a percentage of the detection limit was found on the patches and made their calculations. The WSDOT EIS takes a more conservative approach and calculates dermal exposure to the public walking through sprayed vegetation by using the dislodgeable foliar residue method utilized by all four assessments to estimate the dermal exposure to berry pickers. This made our calculations more conservative by approximately two orders of magnitude.

5) The Jones and Stokes' Caltrans DEIR used an incorrect value in calculating dietary exposure from drift contaminated lettuce. The fraction of the year lettuce is available for herbicide exposure is 0.019 not 0.0019 as they have reported, throwing their values off one order of magnitude.

6) The USDI BLM and USFS EIS's use 50 kg as the average body weight for an adult. Most of the scientific community recognizes 70 kg to be the average weight of an adult.

7) Risk associated with drinking potentially contaminated groundwater was not evaluated in this document. Pesticide contamination of groundwater is almost always from agricultural sources. A pilot groundwater monitoring study was performed by the Washington State Department of Ecology in three counties, Whatcom, Franklin and Yakima. A total of 81 wells were sampled in all. The only herbicide in WSDOT's program which was also detected in the WDOE program was bromacil, in one solitary well at a level of 12 to 15 μg/L. (The leaching potential of bromacil is well known and the label restricts its use in order to prevent groundwater contamination). This level of exposure would not be expected to significantly change the risk equations.

8) The risk connected with ingestion of contaminated surface water was not evaluated in this EIS. Roadways are designed to limit the movement of hydrocarbons, lead and particulate matter normally present in surface water runoff. Jones & Stokes assessed the risk potential associated with drinking runoff contaminated water. They found that all chemicals evaluated had a negligible noncancer and cancer risk. Inclusion of predicted contaminant levels in surface waters would not be expected to significantly alter the risk equations presented here.

9) The US Forestry Service EIS evaluates the carcinogenic potential of 2,4-DP. A positive study for oncogenicity could not be located in the literature, including the US EPA Science Chapter on 2,4-DP. The US EPA does not consider 2,4-DP to be a potential carcinogen. They are not even considering it for review. Therefore, 2,4-DP was not evaluated as a potential carcinogen in this assessment.

Figure 4-1. Components of a Risk Assessment.

HAZARD IDENTIFICATION	• List all relevant chemicals
	• Review available epidemiology data
	• Review all laboratory toxicology data for each active ingredient, formulation and inert ingredient
	  * Acute
	  * Sub-chronic/chronic
	  * Reproductive/developmental
	  * Carcinogenicity/mutagenicity
	• Review physical and chemical properties
	• Review environmental fate

EXPOSURE ASSESSMENT	• Identify potentially exposed populations • Identify major routes and pathways of exposure • Estimate exposure concentrations • Estimate frequency and duration of exposure • Discuss bioavailability information and assumptions • Calculate estimated chemical intake
DOSE-RESPONSE ASSESSMENT	• Identify the lowest NOEL's for chronic systemic effects and for reproductive/developmental effects • Identify Reference Doses (or acceptable daily intake) available from the EPA • Calculate Reference Doses, when not available, using the appropriate NOEL and an uncertainty factor • Identify cancer potency factors
RISK CHARACTERIZATION	• Quantify risk from individual chemicals • Quantify risk from multiple chemicals • Quantify risk from multiple pathways
RISK COMMUNICATION	• Summarize • Assess and present uncertainties • Present conflicting data from EIS's

Figure 4-2 Diagram of Quantal Dose-response Relationship

The abscissa is a long dosage of the chemical. In the top panel the ordinate is percent mortality; in the middle panel the ordinate is mortality frequency; and in the bottom panel the mortality is in probit units. (Klassen, et al., 1986)

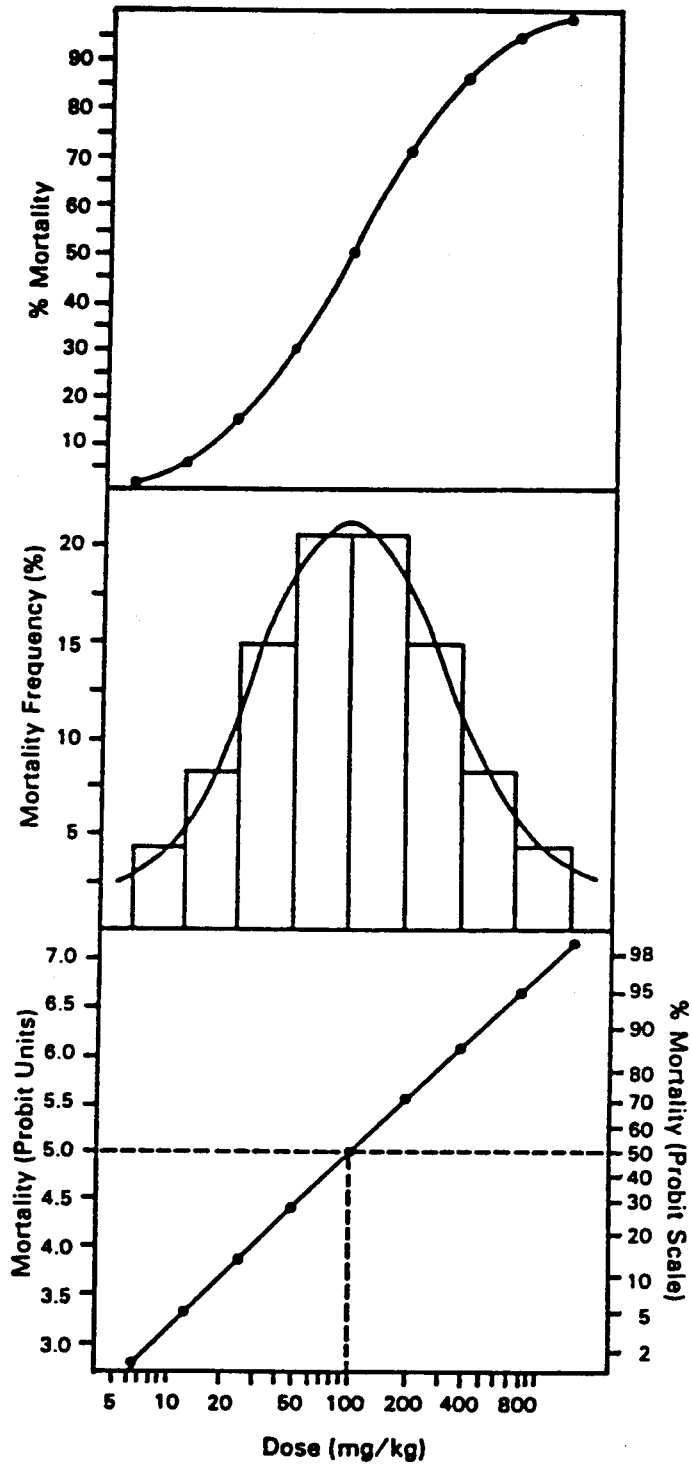


Figure 4-3 Diagram of Dose-response Relationships

Dose-response relationship is steeper from chemical B than chemical A. (Klassen, et al., 1986)

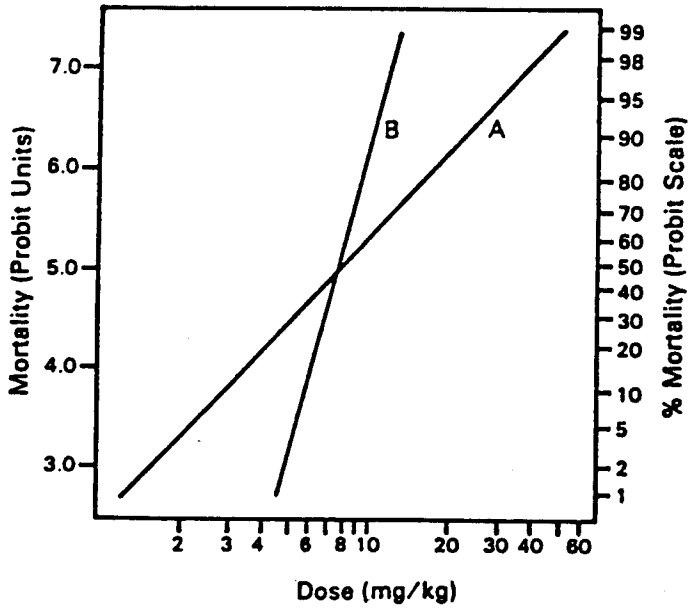


Figure 4-4 Dose Response Curve Resolved By Modeling

The rationale for using low-dose extrapolation models is that it is not possible to conduct animal studies at doses as low as those typically encountered in the environment. Using statistical principles, it is possible to estimate the likely range of results for those data collected at low doses and from this estimate the response at much lower doses. This figure illustrates how the models can describe the date in the high-dose region by at low doses the presumption of linearity and the need to account for the uncertainty introduced by studying only 100 animals per dose forces the models to predict risks that almost certainly are greater than those that exist. (Paustenbach, 1989)

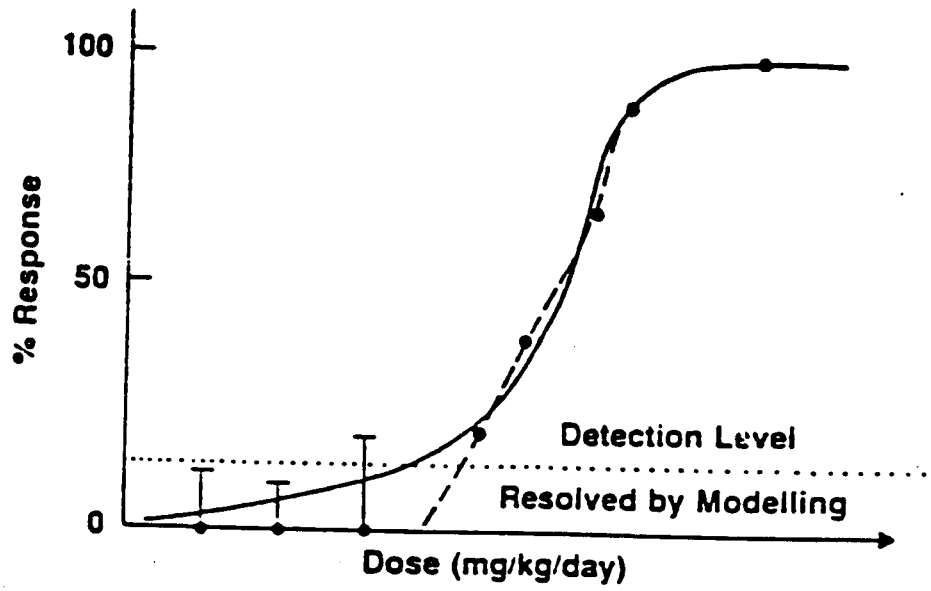


Figure 4-5 A Part Of Hazard Identification For Developmental Toxicants

A part of hazard identification for developmental toxicants is to evaluate the relationship between adult (A) and developmental (D) effective doses. In the plot where A/D is less than one, toxic effects are seen in the pregnant animal at doses lower than those which adversely affect the conceptus. In contrast where the A/D ration is greater than one, the conceptus in much more sensitive to the effects of the chemicals than the mother. (Paustenbach, 1989)

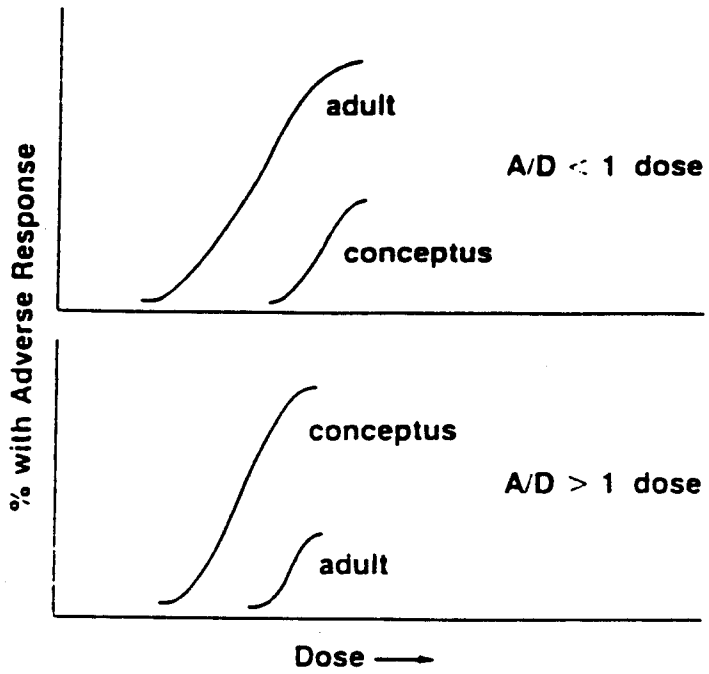


Figure 4-6 Schematic Illustrating the Various Avenues for Degradation and Movement of Xenobiotics in the Environment.

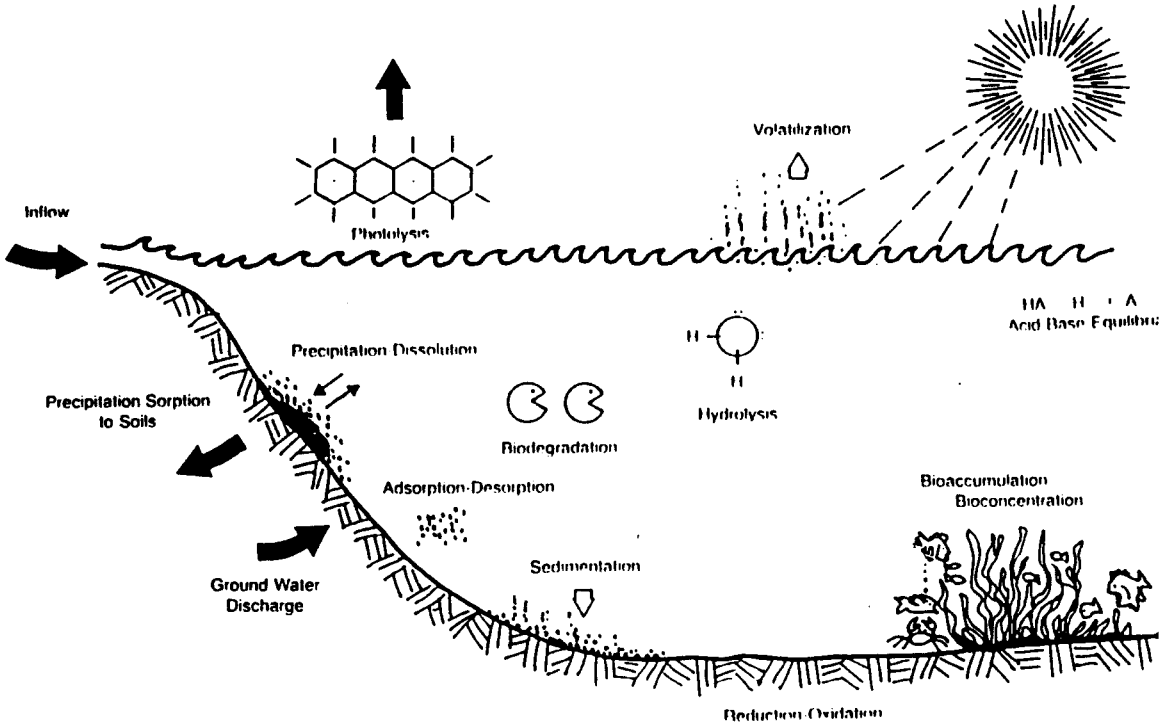


Table 4-1 Exposure Estimates for Workers Applying Herbicides with Truck Mounted Spray Equipment.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	3.3×10^{-3}	2.0×10^{-1}	5.4×10^{-5}	1.0×10^{-2}
2,4-DP	1.2×10^{-3}	1.3×10^{-1}		NA
Benefin	3.1×10^{-3}	3.6×10^{-2}		NA
Bromacil	3.1×10^{-4}	1.4×10^{-2}	1.7×10^{-6}	1.8×10^{-4}
Chlorsulfuron	1.4×10^{-5}	1.7×10^{-4}		NA
Chlorthal dimethyl	1.9×10^{-2}	1.8×10^{-1}		NA
Dicamba	8.4×10^{-4}	4.8×10^{-2}		NA
Dichlobenil	3.7×10^{-3}	1.2×10^{-1}		NA
Diuron	2.5×10^{-3}	2.4×10^{-1}		NA
Fluazifop-butyl	2.0×10^{-4}	2.3×10^{-3}		NA
Fosamine	1.1×10^{-3}	2.9×10^{-2}		NA
Glyphosate	8.1×10^{-4}	4.5×10^{-2}		NA
Imazapyr	7.1×10^{-4}	1.8×10^{-2}		NA
Isoxaben	5.2×10^{-4}	6.0×10^{-3}		NA
Mecoprop	7.8×10^{-4}	6.5×10^{-3}		NA
Mefluidide	9.5×10^{-5}	1.2×10^{-3}		NA
Metsulfuron methyl	5.9×10^{-5}	1.8×10^{-3}		NA
Napropamide	3.0×10^{-3}	2.4×10^{-2}		NA
Oryzalin	2.3×10^{-3}	3.6×10^{-2}	1.3×10^{-5}	4.5×10^{-4}
Oxadiazon	3.0×10^{-3}	4.8×10^{-2}	1.6×10^{-5}	6.0×10^{-4}
Picloram	6.3×10^{-5}	1.2×10^{-3}	1.0×10^{-6}	6.2×10^{-5}
Pronamide	1.5×10^{-3}	2.4×10^{-2}	4.1×10^{-6}	1.5×10^{-4}
Sethoxydim	4.2×10^{-5}	5.5×10^{-4}		NA
Sulfometuron methyl	2.9×10^{-5}	6.7×10^{-4}		NA
Triclopyr	4.1×10^{-4}	1.8×10^{-2}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-2 Exposure Estimates for Workers Mixing and Loading Herbicides into Truck Mounted Spray Equipment.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	9.9×10^{-3}	3.7×10^{-1}	1.6×10^{-4}	1.8×10^{-2}
2,4-DP	3.5×10^{-3}	2.3×10^{-1}		NA
Benefin	9.2×10^{-3}	6.6×10^{-2}		NA
Bromacil	9.2×10^{-4}	2.6×10^{-2}	5.0×10^{-8}	3.3×10^{-4}
Chlorsulfuron	4.3×10^{-5}	3.1×10^{-4}		NA
Chlorthal dimethyl	5.7×10^{-2}	3.3×10^{-1}		NA
Dicamba	2.5×10^{-3}	8.8×10^{-2}		NA
Dichlobenil	1.1×10^{-2}	2.2×10^{-1}		NA
Diuron	7.5×10^{-3}	4.4×10^{-1}		NA
Fluazifop-butyl	5.9×10^{-4}	4.2×10^{-3}		NA
Fosamine	3.3×10^{-3}	5.3×10^{-2}		NA
Glyphosate	2.4×10^{-3}	8.2×10^{-2}		NA
Imazapyr	2.1×10^{-3}	3.3×10^{-2}		NA
Isoxaben	1.6×10^{-3}	1.1×10^{-2}		NA
Mecoprop	2.3×10^{-3}	1.2×10^{-2}		NA
Mefluidide	2.8×10^{-4}	2.2×10^{-3}		NA
Metsulfuron methyl	1.8×10^{-4}	3.3×10^{-3}		NA
Napropamide	9.0×10^{-3}	4.4×10^{-2}		NA
Oryzalin	7.0×10^{-3}	6.6×10^{-2}	3.9×10^{-5}	8.3×10^{-4}
Oxadiazon	9.0×10^{-3}	8.8×10^{-2}	4.9×10^{-5}	1.1×10^{-3}
Picloram	1.9×10^{-4}	2.3×10^{-3}	3.1×10^{-6}	1.1×10^{-4}
Pronamide	4.5×10^{-3}	4.4×10^{-2}	1.2×10^{-5}	2.8×10^{-4}
Sethoxydim	1.3×10^{-4}	1.0×10^{-3}		NA
Sulfometuron methyl	8.6×10^{-5}	1.2×10^{-3}		NA
Triclopyr	1.2×10^{-3}	3.3×10^{-2}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-3 Exposure Estimates for Workers Applying, Mixing and Loading Herbicides Using Truck Mounted Spray Equipment.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	9.9×10^{-3}	3.1×10^{-1}	1.6×10^{-4}	1.5×10^{-2}
2,4-DP	3.5×10^{-3}	2.0×10^{-1}		NA
Benefin	9.2×10^{-3}	5.6×10^{-2}		NA
Bromacil	9.2×10^{-4}	2.2×10^{-2}	5.0×10^{-6}	2.8×10^{-4}
Chlorsulfuron	4.3×10^{-5}	2.6×10^{-4}		NA
Chlorthal dimethyl	5.7×10^{-2}	2.8×10^{-1}		NA
Dicamba	2.5×10^{-3}	7.4×10^{-2}		NA
Dichlobenil	1.1×10^{-2}	1.9×10^{-1}		NA
Diuron	7.5×10^{-3}	3.7×10^{-1}		NA
Fluazifop-butyl	5.9×10^{-4}	3.5×10^{-3}		NA
Fosamine	3.3×10^{-3}	4.4×10^{-2}		NA
Glyphosate	2.4×10^{-3}	6.9×10^{-2}		NA
Imazapyr	2.1×10^{-3}	2.8×10^{-2}		NA
Isoxaben	1.6×10^{-3}	9.3×10^{-3}		NA
Mecoprop	2.3×10^{-3}	1.0×10^{-2}		NA
Mefluidide	2.8×10^{-4}	1.9×10^{-3}		NA
Metsulfuron methyl	1.8×10^{-4}	2.8×10^{-3}		NA
Napropamide	9.0×10^{-3}	3.7×10^{-2}		NA
Oryzalin	7.0×10^{-3}	5.6×10^{-2}	3.9×10^{-5}	7.0×10^{-4}
Oxadiazon	9.0×10^{-3}	7.4×10^{-2}	4.9×10^{-5}	9.3×10^{-4}
Picloram	1.9×10^{-4}	1.9×10^{-3}	3.1×10^{-6}	9.6×10^{-5}
Pronamide	4.5×10^{-3}	3.7×10^{-2}	1.2×10^{-5}	2.3×10^{-4}
Sethoxydim	1.3×10^{-4}	8.5×10^{-4}		NA
Sulfometuron methyl	8.6×10^{-5}	1.0×10^{-3}		NA
Triclopyr	1.2×10^{-3}	2.8×10^{-2}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-4 Exposure Estimates for Workers Using Backpack or Handheld Spray Equipment.*

COMPOUND	Lifetime Single Day Dose (mg/kg/day) Average	Lifetime Single Day Dose (mg/kg/day) Maximum**	Lifetime Average Daily Dose (mg/kg/day) Average	Lifetime Average Daily Dose (mg/kg/day) Maximum**
2,4-D	5.0×10^{-3}	1.3×10^{-1}	8.3×10^{-5}	6.4×10^{-3}
2,4-DP	1.8×10^{-3}	8.2×10^{-2}		NA
Benefin	4.7×10^{-3}	2.3×10^{-2}		NA
Bromacil	4.7×10^{-4}	9.2×10^{-3}	2.6×10^{-6}	1.2×10^{-4}
Chlorsulfuron	2.2×10^{-5}	1.1×10^{-4}		NA
Chlorthal dimethyl	2.9×10^{-2}	1.2×10^{-1}		NA
Dicamba	1.3×10^{-3}	3.1×10^{-2}		NA
Dichlobenil	5.6×10^{-3}	7.7×10^{-2}		NA
Diuron	3.8×10^{-3}	1.5×10^{-1}		NA
Fluazifop-butyl	3.0×10^{-4}	1.5×10^{-3}		NA
Fosamine	1.7×10^{-3}	1.9×10^{-2}		NA
Glyphosate	1.2×10^{-3}	2.9×10^{-2}		NA
Imazapyr	1.1×10^{-3}	1.2×10^{-2}		NA
Isoxaben	7.9×10^{-4}	3.9×10^{-3}		NA
Mecoprop	1.2×10^{-3}	4.2×10^{-3}		NA
Mefluidide	1.4×10^{-4}	7.7×10^{-4}		NA
Metsulfuron methyl	8.9×10^{-5}	1.2×10^{-3}		NA
Napropamide	4.6×10^{-3}	1.5×10^{-2}		NA
Oryzalin	3.6×10^{-3}	2.3×10^{-2}	2.0×10^{-5}	2.9×10^{-4}
Oxadiazon	4.6×10^{-3}	3.1×10^{-2}	2.5×10^{-5}	3.9×10^{-4}
Picloram	9.6×10^{-5}	8.0×10^{-4}	1.6×10^{-6}	4.0×10^{-5}
Pronamide	2.3×10^{-3}	1.5×10^{-2}	6.3×10^{-6}	9.6×10^{-5}
Sethoxydim	6.4×10^{-5}	3.5×10^{-4}		NA
Sulfometuron methyl	4.3×10^{-5}	4.3×10^{-4}		NA
Triclopyr	6.2×10^{-4}	1.1×10^{-2}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-5 Exposure Estimates for the Public-Ingestion of Drift Contaminated Berries, Adults.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	8.7×10^{-5}	1.2×10^{-2}	9.2×10^{-7}	8.6×10^{-4}
2,4-DP	4.8×10^{-5}	1.2×10^{-2}		NA
Benefin	8.1×10^{-5}	2.2×10^{-3}		NA
Bromacil	8.1×10^{-5}	8.7×10^{-3}	8.6×10^{-7}	6.2×10^{-4}
Chlorsulfuron	3.8×10^{-6}	1.0×10^{-4}		NA
Chlorthal dimethyl	5.1×10^{-4}	1.1×10^{-2}		NA
Dicamba	4.4×10^{-5}	5.8×10^{-3}		NA
Dichlobenil	2.0×10^{-4}	1.5×10^{-2}		NA
Diuron	1.3×10^{-4}	2.9×10^{-2}		NA
Fluazifop-butyl	1.0×10^{-5}	2.7×10^{-4}		NA
Fosamine	2.9×10^{-4}	1.8×10^{-2}		NA
Glyphosate	6.1×10^{-5}	7.7×10^{-3}		NA
Imazapyr	1.9×10^{-5}	1.1×10^{-3}		NA
Isoxaben	2.7×10^{-5}	7.3×10^{-4}		NA
Mecoprop	2.1×10^{-5}	3.9×10^{-4}		NA
Mefluidide	2.5×10^{-5}	7.3×10^{-4}		NA
Metsulfuron methyl	1.6×10^{-6}	1.1×10^{-4}		NA
Napropamide	1.6×10^{-4}	2.9×10^{-3}		NA
Oryzalin	1.2×10^{-4}	4.4×10^{-3}	1.3×10^{-6}	3.1×10^{-4}
Oxadiazon	7.9×10^{-5}	2.9×10^{-3}	8.4×10^{-7}	2.1×10^{-4}
Picloram	3.5×10^{-5}	1.6×10^{-3}	3.7×10^{-7}	1.1×10^{-4}
Pronamide	7.9×10^{-5}	2.9×10^{-3}	8.4×10^{-7}	2.1×10^{-4}
Sethoxydim	7.5×10^{-6}	3.4×10^{-4}		NA
Sulfometuron methyl	7.5×10^{-6}	4.1×10^{-4}		NA
Triclopyr	6.5×10^{-5}	6.6×10^{-3}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-6 Exposure Estimates for the Public-Ingestion of Drift Contaminated Berries, Child.*

COMPOUND	Single Day Dose (mg/kg/day)		Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	3.4×10^{-4}	4.7×10^{-2}		
2,4-DP	1.9×10^{-4}	4.7×10^{-2}		
Benefin	3.2×10^{-4}	8.5×10^{-3}		
Bromacil	3.2×10^{-4}	3.4×10^{-2}		
Chlorsulfuron	1.5×10^{-5}	4.0×10^{-4}		
Chlorthal dimethyl	2.0×10^{-3}	4.3×10^{-2}		
Dicamba	1.7×10^{-4}	2.3×10^{-2}		
Dichlobenil	7.6×10^{-4}	5.7×10^{-2}		
Diuron	5.1×10^{-4}	1.1×10^{-1}		
Fluazifop-butyl	4.0×10^{-5}	1.1×10^{-3}		
Fosamine	1.1×10^{-3}	6.8×10^{-2}		
Glyphosate	2.4×10^{-4}	3.0×10^{-2}		
Imazapyr	7.3×10^{-5}	4.3×10^{-3}		
Isoxaben	1.1×10^{-4}	2.8×10^{-3}		
Mecoprop	8.0×10^{-5}	1.5×10^{-3}		
Mefluidide	9.7×10^{-5}	2.8×10^{-3}		
Metsulfuron methyl	6.0×10^{-6}	4.3×10^{-4}		
Napropamide	6.2×10^{-4}	1.1×10^{-2}		
Oryzalin	4.8×10^{-4}	1.7×10^{-2}		
Oxadiazon	3.1×10^{-4}	1.1×10^{-2}		
Picloram	1.4×10^{-4}	6.1×10^{-3}		
Pronamide	3.1×10^{-4}	1.1×10^{-2}		
Sethoxydim	4.3×10^{-5}	1.3×10^{-3}		
Sulfometuron methyl	2.9×10^{-5}	1.6×10^{-3}		
Triclopyr	2.5×10^{-4}	2.6×10^{-2}		

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-7 Exposure Estimates for the Public-Ingestion of Drift Contaminated Vegetables, Lettuce.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	2.8×10^{-4}	9.7×10^{-2}	1.5×10^{-8}	3.5×10^{-5}
2,4-DP	1.6×10^{-4}	9.7×10^{-2}		NA
Benefin	2.6×10^{-4}	1.7×10^{-2}		NA
Bromacil	2.6×10^{-4}	7.0×10^{-2}	1.4×10^{-8}	2.5×10^{-5}
Chlorsulfuron	1.2×10^{-5}	8.1×10^{-4}		NA
Chlorthal dimethyl	1.6×10^{-3}	8.7×10^{-2}		NA
Dicamba	1.4×10^{-4}	4.7×10^{-2}		NA
Dichlobenil	6.4×10^{-4}	1.2×10^{-1}		NA
Diuron	4.3×10^{-4}	2.3×10^{-1}		NA
Fluazifop-butyl	3.4×10^{-5}	2.2×10^{-3}		NA
Fosamine	9.4×10^{-4}	1.4×10^{-1}		NA
Glyphosate	2.0×10^{-4}	6.2×10^{-2}		NA
Imazapyr	6.1×10^{-5}	8.7×10^{-3}		NA
Isoxaben	8.9×10^{-5}	5.8×10^{-3}		NA
Mecoprop	6.7×10^{-5}	3.1×10^{-3}		NA
Mefluidide	8.1×10^{-5}	5.8×10^{-3}		NA
Metsulfuron methyl	5.0×10^{-6}	8.7×10^{-4}		NA
Napropamide	5.2×10^{-4}	2.3×10^{-2}		NA
Oryzalin	4.0×10^{-4}	3.5×10^{-2}	2.2×10^{-8}	1.2×10^{-5}
Oxadiazon	2.6×10^{-4}	2.3×10^{-2}	1.4×10^{-8}	8.3×10^{-6}
Picloram	1.1×10^{-4}	1.3×10^{-2}	6.1×10^{-9}	4.5×10^{-6}
Pronamide	2.6×10^{-4}	2.3×10^{-2}	1.4×10^{-8}	8.3×10^{-6}
Sethoxydim	3.6×10^{-5}	2.7×10^{-3}		NA
Sulfometuron methyl	2.5×10^{-5}	3.3×10^{-3}		NA
Triclopyr	2.1×10^{-4}	5.2×10^{-2}		NA

*Numbers rounded off to one decimal point.

**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-8 Exposure Estimates for the Public-Ingestion of Drift Contaminated Vegetables, Beans.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	4.9×10^{-5}	1.5×10^{-2}	6.5×10^{-8}	1.3×10^{-4}
2,4-DP	2.7×10^{-5}	1.5×10^{-2}		NA
Benefin	4.6×10^{-5}	2.7×10^{-3}		NA
Bromacil	4.5×10^{-5}	1.1×10^{-2}	6.0×10^{-8}	9.5×10^{-5}
Chlorsulfuron	2.1×10^{-8}	1.3×10^{-4}		NA
Chlorthal dimethyl	2.8×10^{-4}	1.3×10^{-2}		NA
Dicamba	2.5×10^{-5}	7.1×10^{-3}		NA
Dichlobenil	1.1×10^{-4}	1.8×10^{-2}		NA
Diuron	7.4×10^{-5}	3.5×10^{-2}		NA
Fluazifop-butyl	5.8×10^{-8}	3.4×10^{-4}		NA
Fosamine	1.6×10^{-4}	2.1×10^{-2}		NA
Glyphosate	3.4×10^{-5}	9.4×10^{-3}		NA
Imazapyr	1.0×10^{-5}	1.3×10^{-3}		NA
Isoxaben	1.5×10^{-5}	8.9×10^{-4}		NA
Mecoprop	1.2×10^{-5}	4.8×10^{-4}		NA
Mefluidide	1.4×10^{-5}	8.9×10^{-4}		NA
Metsulfuron methyl	8.7×10^{-7}	1.3×10^{-4}		NA
Napropamide	8.9×10^{-5}	3.5×10^{-3}		NA
Oryzalin	6.9×10^{-5}	5.3×10^{-3}	9.2×10^{-8}	4.8×10^{-5}
Oxadiazon	4.4×10^{-5}	3.5×10^{-3}	5.9×10^{-8}	3.2×10^{-5}
Picloram	2.0×10^{-5}	1.9×10^{-3}	2.6×10^{-8}	1.7×10^{-5}
Pronamide	4.4×10^{-5}	3.5×10^{-3}	5.9×10^{-8}	3.2×10^{-5}
Sethoxydim	6.2×10^{-8}	4.1×10^{-4}		NA
Sulfometuron methyl	4.2×10^{-8}	5.0×10^{-4}		NA
Triclopyr	3.7×10^{-5}	8.0×10^{-3}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-9 Exposure Estimates for the Public-Dermal Exposure from Drift Contaminated Berries.*

COMPOUND	Lifetime Single Day Dose (mg/kg/day) Average	Lifetime Single Day Dose (mg/kg/day) Maximum**	Lifetime Average Daily Dose (mg/kg/day) Average	Lifetime Average Daily Dose (mg/kg/day) Maximum**
2,4-D	2.1×10^{-4}	2.4×10^{-2}	4.5×10^{-7}	2.5×10^{-4}
2,4-DP	7.4×10^{-5}	1.5×10^{-2}		NA
Benefin	2.0×10^{-4}	4.3×10^{-3}		NA
Bromacil	2.0×10^{-5}	1.7×10^{-3}	4.1×10^{-8}	1.8×10^{-5}
Chlorsulfuron	9.1×10^{-7}	2.0×10^{-5}		NA
Chlorthal dimethyl	1.2×10^{-3}	2.1×10^{-2}		NA
Dicamba	5.4×10^{-5}	5.7×10^{-3}		NA
Dichlobenil	2.4×10^{-4}	1.4×10^{-2}		NA
Diuron	1.6×10^{-4}	2.8×10^{-2}		NA
Fluazifop-butyl	1.3×10^{-5}	2.7×10^{-4}		NA
Fosamine	7.0×10^{-5}	3.4×10^{-3}		NA
Glyphosate	5.2×10^{-5}	5.3×10^{-3}		NA
Imazapyr	4.5×10^{-5}	2.1×10^{-3}		NA
Isoxaben	3.3×10^{-5}	7.1×10^{-4}		NA
Mecoprop	5.0×10^{-5}	7.7×10^{-4}		NA
Mefluidide	6.1×10^{-6}	1.4×10^{-4}		NA
Metsulfuron methyl	3.8×10^{-6}	2.1×10^{-4}		NA
Napropamide	1.9×10^{-4}	2.8×10^{-3}		NA
Oryzalin	1.5×10^{-4}	4.3×10^{-3}	3.2×10^{-7}	4.5×10^{-5}
Oxadiazon	1.9×10^{-4}	5.7×10^{-3}	4.1×10^{-7}	6.0×10^{-5}
Picloram	4.1×10^{-6}	1.5×10^{-4}	1.0×10^{-8}	1.6×10^{-6}
Pronamide	9.6×10^{-5}	2.8×10^{-3}	2.0×10^{-7}	3.0×10^{-5}
Sethoxydim	2.7×10^{-6}	6.5×10^{-5}		NA
Sulfometuron methyl	1.8×10^{-6}	8.0×10^{-5}		NA
Triclopyr	2.6×10^{-5}	2.1×10^{-3}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-10 Exposure Estimates for the Public-Dermal Exposure from Contaminated Vegetation.*

COMPOUND	Single Day Dose (mg/kg/day)		Lifetime Average Daily Dose (mg/kg/day)	
	Average	Maximum**	Average	Maximum**
2,4-D	2.1×10^{-3}	2.2×10^{-1}	7.6×10^{-5}	4.6×10^{-2}
2,4-DP	7.3×10^{-4}	1.4×10^{-1}		NA
Benefin	1.9×10^{-3}	3.9×10^{-2}		NA
Bromacil	1.9×10^{-4}	1.6×10^{-2}	1.1×10^{-6}	1.1×10^{-3}
Chlorsulfuron	9.0×10^{-8}	1.8×10^{-4}		NA
Chlorthal dimethyl	1.2×10^{-2}	2.0×10^{-1}		NA
Dicamba	5.3×10^{-4}	5.2×10^{-2}		NA
Dichlobenil	2.3×10^{-3}	1.3×10^{-1}		NA
Diuron	1.6×10^{-3}	2.6×10^{-1}		NA
Fluazifop-butyl	1.2×10^{-4}	2.5×10^{-3}		NA
Fosamine	6.9×10^{-4}	3.1×10^{-2}		NA
Glyphosate	5.1×10^{-4}	4.8×10^{-2}		NA
Imazapyr	4.4×10^{-4}	2.0×10^{-2}		NA
Isoxaben	3.3×10^{-4}	6.5×10^{-3}		NA
Mecoprop	4.9×10^{-4}	7.0×10^{-3}		NA
Mefluidide	6.0×10^{-5}	1.3×10^{-3}		NA
Metsulfuron methyl	3.7×10^{-5}	2.0×10^{-3}		NA
Napropamide	1.9×10^{-3}	2.6×10^{-2}		NA
Oryzalin	1.5×10^{-3}	3.9×10^{-2}	8.3×10^{-6}	2.7×10^{-3}
Oxadiazon	1.9×10^{-3}	5.2×10^{-2}	1.1×10^{-5}	3.7×10^{-3}
Picloram	4.0×10^{-5}	1.4×10^{-3}	1.5×10^{-6}	2.9×10^{-4}
Pronamide	9.5×10^{-4}	2.6×10^{-2}	2.7×10^{-6}	1.2×10^{-3}
Sethoxydim	2.7×10^{-5}	6.0×10^{-4}		NA
Sulfometuron methyl	1.8×10^{-5}	7.3×10^{-4}		NA
Triclopyr	2.6×10^{-4}	1.9×10^{-2}		NA

*Numbers rounded off to one decimal point.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-11 Critical Exposure Estimate Criteria.

	Application Rate (lbs. A.I./Acre) Average	Maximum*	Season Applied (# months)		Dermal Bioavailibility Factor (DBF)	Correction Factor (DBCF)
2,4-D	2.2	16.65	All	(12)	.1	1.0
2,4-DP	1.2	16.65	W	(3)	.064	0.64
Benefin	2.05	3	Sp	(2)	.1	1.0
Bromacil	2.04	12	F or W	(2)	.01	0.1
Chlorsulfuron	0.095	0.14	All	(12)	.01	0.1
Chlorthal dimethyl	12.75	15	Sp	(2)	.1	1.0
Dicamba	1.12	8	All	(12)	.05	0.5
Dichlobenil	4.93	20	W	(1)	.05	0.5
Diuron	3.33	40	Sp	(2)	.05	0.5
Fluazifop-butyl	0.26	0.38	Sp	(2)	.05	0.5
Fosamine	7.28	24	S	(2)	.01	0.1
Glyphosate	1.55	10.6	All	(12)	.035	0.35
Imazapyr	0.47	1.5	Sp	(2)	.1	1.0
Isoxaben	0.69	1	Sp	(2)	.05	.05
Mecoprop	0.52	0.54	Sp,S	(4)	.1	1.0
Mefluidide	0.63	1	Sp	(2)	.01	0.1
Metsulfuron methyl	0.039	0.15	Sp,S	(4)	.1	1.0
Napropamide	4	4	Sp	(2)	.05	0.5
Oryzalin	3.12	6	Sp	(2)	.05	0.5
Oxadiazon	2	4	Sp	(2)	.1	1.0
Picloram	0.88	2.16	All	(12)	.0048	048
Pronamide	2	4	Sp	(1)	.05	0.5
Sethoxydim	0.05	0.46	Sp	(2)	.01	0.1
Sulfometuron methyl	0.19	0.56	Sp	(2)	.01	0.1
Triclopyr	1.65	9	All	(12)	.0165	0.165

*WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-12 List Of Reference Doses.

COMPOUND	RfD[a] (mg/kg/d)	UF	NOAEL	Confidence
2,4-D	1×10^{-2}	100	•rat, 91 day •1 mg/kg/day diet •Hematologic, hepatic & renal toxicity.	NA
2,4-DP	4×10^{-2}[b]	100	•rat, 2 year •4 mg/kg/day •Kidney & liver toxicity.	med.
Benefin	3×10^{-1}	100	•dog, chronic •25 mg/kg/day •Dep. erythrocyte counts.	med.
Bromacil	2.5×10^{-2}[b]	100	•rat, 2 year •2.5 mg/kg/day •Decreased body weight.	med.
Chlorsulfuron	5×10^{-2}	100	•rat, 2 year •5 mg/kg/day •Decreased body weight.	high
Chlorthal dimethyl	5×10^{-1}	100	•rat, 2 year •50 mg/kg/day •Inc. kidney & adrenal weights.	med.
Dicamba	3×10^{-2}	100	•rabbit,teratology •3 mg/kg/day •Maternal & fetal toxicity.	high
Dichlobenil	2.5×10^{-2}[b]	100	•rat, 2 year •2.5 mg/kg/day •Dec. body weight & inc. organ weights	med.
Diuron	2×10^{-3}	300	•dog, 2 year •0.625 mg/kg/day •Abnormal pigments in blood.	low

[a] US EPA derived unless stated otherwise.
[b] CSI derived reference dose, see compound in dose-response section for more detail.

Table 4-12 List Of Reference Doses (cont'd).

COMPOUND	RfD[a] (mg/kg/day)	UF	NOAEL	Confidence
Fluazifop-butyl	1×10^{-2}[c]	100	•rat, 2 year •1.0 mg/kg/day •Dec. survival	NA
Fosamine	2.5×10^{-2}[b]	1000	•dog, 6 months •25 mg/kg/day	low
Glyphosate	1×10^{-1}	100	•rat, reproduction •10 mg/kg/day •Renal tubular dilation in F3b offspring.	high
Imazapyr	3×10^{-1}[b]	1000	•rat, teratology •300 mg/kg/day •Maternal toxicity.	low
Isoxaben	5×10^{-2}[c]	100	•rat, 2 year •5 mg/kg/day •Inc. organ/body weight ratio, dec. food consumption & changes in blood parameters.	high
Mecoprop	1×10^{-2}[b]	100	•rat, 2 year •1 mg/kg/day, 20ppm •Changes in blood parameters & inc. kidney weights. (90 day study)	low
Mefluidide	1.5×10^{-2}[b]	100	•dog, 1 year •1.5 mg/kg/day, •Decreased body weight & kidney toxicity.	low
Metsulfuron methyl	2.5×10^{-1}	100	•rat, 2 year •25 mg/kg/day •Decreased body weight.	high
Napropamide	1×10^{-1}	300	•rat, reproduction •30 mg/kg/day •Decreased body weight gain.	medium

[a] US EPA derived unless stated otherwise.
[b] CSI derived reference dose, see compound in dose-response section for more detail.
[c] Reported in Jones & Stokes, (1991).

Table 4-12 List Of Reference Doses (cont'd).

COMPOUND	RfD[a] (mg/kg/day)	UF	NOAEL	Confidence
Oryzalin	5×10^{-2}	100	•dog, 1 year •5 mg/kg/day •Changes in blood parameters, inc. liver & kidney weights & dec. adrenal weights.	high
Oxadiazon	5×10^{-3}	100	•rat, 2 year •0.5 mg/kg/day •Inc. serum proteins & inc. liver weight.	medium
Picloram	7×10^{-2}	100	•dog, 6 month •7 mg/kg/day •Increased liver weights.	medium
Pronamide	7.5×10^{-2}	100	•dog, 2 year •7.5 mg/kg/day •No adverse effect.	medium
Sethoxydim	9×10^{-2}	100	•dog 1 year •8.9/9.4 mg/kg/d •Mild anemia.	high
Sulfometuron methyl	5×10^{-2}[b]	100	•dog 1 year •5 mg/kg/day,200ppm •Changes in blood parameters & inc. liver weights.	low
Triclopyr	2.5×10^{-2}[b]	100	•dog, 1 year •2.5 mg/kg/day •Pigment deposits in the kidney & changes in serum parameters.	high

[a] US EPA derived unless stated otherwise.
[b] CSI derived reference dose, see compound in dose-response section for more detail.

Table 4-13 Potential Carcinogens

Compound	CPF $(mg/kg/d)^{-1}$	Weight of Evidence Classification	Tumor Type
2,4-D	1.9×10^{-2} [e]	C [e]	Increased rate of tumor formation in rats
Bromacil	3.8×10^{-3} [e]	C [b]	Hepatocellular adenomas & carcinomas in mice
Dichlobenil	----	C [b]	Male and female rat liver tumors.
Glyphosate	----	D [d]	----
Isoxaben	----	pending [b] C [e]	----
Oryzalin	3.4×10^{-2} [e]	pending [c] C [e]	Tumors at multiple sites in rats
Oxadiazon	1.3×10^{-1} [e]	B2 [e] pending [c]	Hepatocellular adenomas & carcinomas in rats & mice
Picloram	3.0×10^{-3} [a]	pending [d] D [e]	Neoplastic nodules in the liver of female rats
Pronamide	1.6×10^{-2} [e]	pending [c] C [e]	Liver neoplasms in male mice

(a) USDI, 1989.
(b) Jones & Stokes, 1991.
(c) US EPA, 1991 (a).
(d) US EPA, 1990
(e) US EPA, 1991 (b)

Weight of Evidence Classification:
Group A, human carcinogens- Sufficient evidence from human studies to support a causal association between exposure and cancer.
Group B, probable human carcinogens-Sufficient evidence of carcinogenicity from animal studies but limited evidence in human studies (B1) or inadequate evidence in human studies (B2).
Group C, possible human carcinogens-Limited evidence of carcinogenicity in animal studies and no human data.
Group D, not classifiable as to human carcinogenicity- Inadequate evidence of human or animal carcinogenicity or for which no data are available.

Tumor Type:
Species and tumor(s) produced in the oncogenicity study used to calculate the Cancer Potency Factor.

Table 4-14 Reference Doses and Cancer Potency Factors Used in Risk Assessment

COMPOUND	RfD/EPA [g] (mg/kg/day)	RfD/Other (mg/kg/day)	CPF $(mg/kg/day)^{-1}$
2,4-D	1×10^{-2}	----	1.9×10^{-2} [d]
2,4-DP	----	1.24×10^{-2} [a]	----
Benefin	3×10^{-1}	----	----
Bromacil	----	2.5×10^{-2} [a]	3.8×10^{-3} [d]
Chlorsulfuron	5×10^{-2}	----	----
Chlorthal dimethyl	5×10^{-1}	----	----
Dicamba	3×10^{-2}	----	----
Dichlobenil	----	2.5×10^{-2} [a]	----
Diuron	2×10^{-3}	----	----
Fluazifop-butyl	1×10^{-2} [b]	----	----
Fosamine	----	2.5×10^{-2} [a]	----
Glyphosate	1×10^{-1}	----	----
Imazapyr	----	3×10^{-1} [a]	----
Isoxaben	5×10^{-2} [b]	----	----
Mecoprop	----	1×10^{-2} [a]	----
Mefluidide	----	1.5×10^{-2} [a,c]	----
Metsulfuron methyl	2.5×10^{-1}	----	----
Napropamide	1×10^{-1}	----	----
Oryzalin	5×10^{-2}	----	3.4×10^{-2} [d]
Oxadiazon	5×10^{-3}	----	1.3×10^{-1} [d]
Picloram	7×10^{-2}	----	3×10^{-3} [e]
Pronamide	7.5×10^{-2}	----	1.63×10^{-2} [f]
Sethoxydim	9×10^{-2}	----	----
Sulfometuron methyl	----	5×10^{-2} [a,c]	----
Triclopyr	----	2.5×10^{-2} [a]	----

(a) CSI,
(b) Jones & Stokes, 1991- citing US EPA 1990,
(c) Jones & Stokes, 1991- citing Clement,
(d) US EPA, 1991 (b)
(e) USDI, 1991 - citing US EPA 1988,
(f) Jones & Stokes, 1991- citing EPA 1986.
(g) US EPA, 1990; US EPA, 1991 (a)

Table 4-15 Estimates of Non-cancer and Cancer Risks for Workers Applying Herbicides Using Truck-mounted Spray Equipment*

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	3.3×10^{-1}	2.0×10^{1}	1.0×10^{-6}	1.9×10^{-4}
2,4-DP	2.9×10^{-2}	3.2×10^{0}		
Benefin	1.0×10^{-2}	1.2×10^{-1}		
Bromacil	1.2×10^{-2}	5.8×10^{-1}	6.4×10^{-9}	6.8×10^{-7}
Chlorsulfuron	2.9×10^{-4}	3.4×10^{-3}		
Chlorthal dimethyl	3.8×10^{-2}	3.6×10^{-1}		
Dicamba	2.8×10^{-2}	1.6×10^{0}		
Dichlobenil	3.0×10^{-1}	9.6×10^{0}		
Diuron	1.3×10^{0}	1.2×10^{2}		
Fluazifop-butyl	2.0×10^{-2}	2.3×10^{-1}		
Fosamine	4.4×10^{-2}	1.2×10^{0}		
Glyphosate	8.1×10^{-3}	4.5×10^{-1}		
Imazapyr	2.4×10^{-3}	6.0×10^{-2}		
Isoxaben	1.0×10^{-2}	1.2×10^{-1}		
Mecoprop	7.8×10^{-2}	6.5×10^{-1}		
Mefluidide	6.3×10^{-3}	8.0×10^{-2}		
Metsulfuron methyl	2.3×10^{-4}	7.2×10^{-3}		
Napropamide	3.0×10^{-2}	2.4×10^{-1}		
Oryzalin	4.7×10^{-2}	7.2×10^{-1}	1.7×10^{-8}	5.9×10^{-5}
Oxadiazon	6.0×10^{-1}	9.6×10^{0}	2.3×10^{-8}	8.4×10^{-5}
Picloram	9.1×10^{-4}	1.8×10^{-2}	3.1×10^{-9}	1.9×10^{-7}
Pronamide	2.0×10^{-2}	3.2×10^{-1}	6.6×10^{-8}	2.4×10^{-6}
Sethoxydim	4.7×10^{-4}	6.1×10^{-3}		
Sulfometuron methyl	5.7×10^{-4}	1.3×10^{-2}		
Triclopyr	1.6×10^{-2}	7.1×10^{-1}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-16 Estimates of Non-cancer and Cancer Risk for Workers Mixing and Loading Herbicides into Truck-mounted Spray Equipment

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	9.9×10^{-1}	3.7×10^{1}	3.1×10^{-6}	3.5×10^{-4}
2,4-DP	8.7×10^{-2}	5.9×10^{0}		
Benefin	3.1×10^{-2}	2.2×10^{-1}		
Bromacil	3.7×10^{-2}	1.1×10^{0}	1.9×10^{-8}	1.3×10^{-6}
Chlorsulfuron	8.6×10^{-4}	6.2×10^{-3}		
Chlorthal dimethyl	1.2×10^{-1}	6.6×10^{-1}		
Dicamba	8.4×10^{-2}	2.9×10^{0}		
Dichlobenil	8.9×10^{-1}	1.8×10^{1}		
Diuron	3.8×10^{0}	2.2×10^{2}		
Fluazifop-butyl	5.9×10^{-2}	4.2×10^{-1}		
Fosamine	1.3×10^{-1}	2.1×10^{0}		
Glyphosate	2.4×10^{-2}	8.2×10^{-1}		
Imazapyr	7.1×10^{-3}	1.1×10^{-1}		
Isoxaben	3.1×10^{-2}	2.2×10^{-1}		
Mecoprop	2.3×10^{-1}	1.2×10^{0}		
Mefluidide	1.9×10^{-2}	1.5×10^{-1}		
Metsulfuron methyl	7.0×10^{-4}	1.3×10^{-2}		
Napropamide	9.0×10^{-2}	4.4×10^{-1}		
Oryzalin	1.4×10^{-1}	1.3×10^{0}	5.0×10^{-6}	1.1×10^{-4}
Oxadiazon	1.8×10^{0}	1.8×10^{1}	6.9×10^{-6}	1.5×10^{-4}
Picloram	2.7×10^{-3}	3.3×10^{-2}	9.4×10^{-9}	3.4×10^{-7}
Pronamide	6.0×10^{-2}	5.9×10^{-1}	2.0×10^{-7}	4.4×10^{-6}
Sethoxydim	1.4×10^{-3}	1.1×10^{-2}		
Sulfometuron methyl	1.7×10^{-3}	2.5×10^{-2}		
Triclopyr	4.9×10^{-2}	1.3×10^{0}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-17 Estimates of Non-cancer and Cancer Risks for Workers Applying, Mixing and Loading Using Truck-mounted Spray Equipment

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	9.9×10^{-1}	3.1×10^{1}	3.1×10^{-6}	2.9×10^{-4}
2,4-DP	8.7×10^{-2}	4.9×10^{0}		
Benefin	3.1×10^{-2}	1.9×10^{-1}		
Bromacil	3.7×10^{-2}	8.9×10^{-1}	1.9×10^{-8}	1.1×10^{-6}
Chlorsulfuron	8.6×10^{-4}	5.2×10^{-3}		
Chlorthal dimethyl	1.2×10^{-1}	5.6×10^{-1}		
Dicamba	8.4×10^{-2}	2.5×10^{0}		
Dichlobenil	8.9×10^{-1}	1.5×10^{1}		
Diuron	3.8×10^{0}	1.9×10^{2}		
Fluazifop-butyl	5.9×10^{-2}	3.5×10^{-1}		
Fosamine	1.3×10^{-1}	1.8×10^{0}		
Glyphosate	2.4×10^{-2}	6.9×10^{-1}		
Imazapyr	7.1×10^{-3}	9.3×10^{-2}		
Isoxaben	3.1×10^{-2}	1.9×10^{-1}		
Mecoprop	2.3×10^{-1}	1.0×10^{0}		
Mefluidide	1.9×10^{-2}	1.2×10^{-1}		
Metsulfuron methyl	7.0×10^{-4}	1.1×10^{-2}		
Napropamide	9.0×10^{-2}	3.7×10^{-1}		
Oryzalin	1.4×10^{-1}	1.1×10^{0}	5.0×10^{-6}	9.0×10^{-5}
Oxadiazon	1.8×10^{0}	1.5×10^{1}	6.9×10^{-8}	1.3×10^{-4}
Picloram	2.7×10^{-3}	2.7×10^{-2}	9.4×10^{-9}	2.9×10^{-7}
Pronamide	6.0×10^{-2}	4.9×10^{-1}	2.0×10^{-7}	3.7×10^{-6}
Sethoxydim	1.4×10^{-3}	9.5×10^{-3}		
Sulfometuron methyl	1.7×10^{-3}	2.1×10^{-2}		
Triclopyr	4.9×10^{-2}	1.1×10^{0}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-18 Estimates of Non-cancer and Cancer Risk for Workers Applying Herbicides Using Backpack or Hand-held Spray Equipment

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	5.0×10^{-1}	1.3×10^{1}	1.6×10^{-6}	1.2×10^{-4}
2,4-DP	4.4×10^{-2}	2.1×10^{0}		
Benefin	1.6×10^{-2}	7.7×10^{-2}		
Bromacil	1.9×10^{-2}	3.7×10^{-1}	9.7×10^{-9}	4.4×10^{-7}
Chlorsulfuron	4.3×10^{-4}	2.2×10^{-3}		
Chlorthal dimethyl	5.8×10^{-2}	2.3×10^{-1}		
Dicamba	4.3×10^{-2}	1.0×10^{0}		
Dichlobenil	4.5×10^{-1}	6.2×10^{0}		
Diuron	1.9×10^{0}	7.7×10^{1}		
Fluazifop-butyl	3.0×10^{-2}	1.5×10^{-1}		
Fosamine	6.6×10^{-2}	7.4×10^{-1}		
Glyphosate	1.2×10^{-2}	2.9×10^{-1}		
Imazapyr	3.6×10^{-3}	3.9×10^{-2}		
Isoxaben	1.6×10^{-2}	7.7×10^{-2}		
Mecoprop	1.2×10^{-1}	4.2×10^{-1}		
Mefluidide	9.6×10^{-3}	5.1×10^{-2}		
Metsulfuron methyl	3.6×10^{-4}	4.6×10^{-3}		
Napropamide	4.6×10^{-2}	1.5×10^{-1}		
Oryzalin	7.1×10^{-2}	4.6×10^{-1}	2.5×10^{-6}	3.8×10^{-5}
Oxadiazon	9.1×10^{-1}	6.2×10^{0}	3.5×10^{-6}	5.4×10^{-5}
Picloram	1.4×10^{-3}	1.1×10^{-2}	4.7×10^{-9}	1.2×10^{-7}
Pronamide	3.0×10^{-2}	2.1×10^{-1}	1.0×10^{-7}	1.5×10^{-6}
Sethoxydim	7.1×10^{-4}	3.9×10^{-3}		
Sulfometuron methyl	8.7×10^{-4}	8.6×10^{-3}		
Triclopyr	2.5×10^{-2}	4.6×10^{-1}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-19 Estimates of Non-cancer and Cancer Risk from Ingestion of Drift Contaminated Berries, Adults

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	8.7×10^{-3}	1.2×10^{0}	1.8×10^{-8}	1.6×10^{-5}
2,4-DP	1.2×10^{-3}	3.0×10^{-1}		
Benefin	2.7×10^{-4}	7.3×10^{-3}		
Bromacil	3.2×10^{-3}	3.5×10^{-1}	3.3×10^{-9}	2.3×10^{-6}
Chlorsulfuron	7.5×10^{-5}	2.0×10^{-3}		
Chlorthal dimethyl	1.0×10^{-3}	2.2×10^{-2}		
Dicamba	1.5×10^{-3}	1.9×10^{-1}		
Dichlobenil	1.6×10^{-2}	1.2×10^{0}		
Diuron	6.6×10^{-2}	1.5×10^{1}		
Fluazifop-butyl	1.0×10^{-3}	2.8×10^{-2}		
Fosamine	1.2×10^{-2}	7.0×10^{-1}		
Glyphosate	6.1×10^{-4}	7.7×10^{-2}		
Imazapyr	6.2×10^{-5}	3.6×10^{-3}		
Isoxaben	5.5×10^{-4}	1.5×10^{-2}		
Mecoprop	2.1×10^{-3}	3.9×10^{-2}		
Mefluidide	1.7×10^{-3}	4.9×10^{-2}		
Metsulfuron methyl	6.2×10^{-6}	4.4×10^{-4}		
Napropamide	1.6×10^{-3}	2.9×10^{-2}		
Oryzalin	2.5×10^{-3}	8.7×10^{-2}	1.7×10^{-7}	4.0×10^{-5}
Oxadiazon	1.6×10^{-2}	5.8×10^{-1}	1.2×10^{-7}	2.9×10^{-5}
Picloram	5.0×10^{-4}	2.2×10^{-2}	1.1×10^{-9}	3.3×10^{-7}
Pronamide	1.1×10^{-3}	3.9×10^{-2}	1.3×10^{-8}	3.3×10^{-6}
Sethoxydim	8.4×10^{-5}	3.7×10^{-3}		
Sulfometuron methyl	1.5×10^{-4}	8.2×10^{-3}		
Triclopyr	2.6×10^{-3}	2.6×10^{-1}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-20 Estimates of Non-cancer and Cancer Risk from Ingestion of Drift Contaminated Berries, Child

COMPOUND	Non-cancer Hazard Quotient Average	Maximum**	Cancer Risk Average	Maximum**
2,4-D	3.4×10^{-2}	4.7×10^{0}		
2,4-DP	4.6×10^{-3}	1.2×10^{0}		
Benefin	1.1×10^{-3}	2.8×10^{-2}		
Bromacil	1.3×10^{-2}	1.4×10^{0}		
Chlorsulfuron	2.9×10^{-4}	7.9×10^{-3}		
Chlorthal dimethyl	3.9×10^{-3}	8.5×10^{-2}		
Dicamba	5.8×10^{-3}	7.6×10^{-1}		
Dichlobenil	6.1×10^{-2}	4.5×10^{0}		
Diuron	2.6×10^{-1}	5.7×10^{1}		
Fluazifop-butyl	4.0×10^{-3}	1.1×10^{-1}		
Fosamine	4.5×10^{-2}	2.7×10^{0}		
Glyphosate	2.4×10^{-3}	3.0×10^{-1}		
Imazapyr	2.4×10^{-4}	1.4×10^{-2}		
Isoxaben	2.1×10^{-3}	5.7×10^{-2}		
Mecoprop	8.0×10^{-3}	1.5×10^{-1}		
Mefluidide	6.5×10^{-3}	1.9×10^{-1}		
Metsulfuron methyl	2.4×10^{-5}	1.7×10^{-3}		
Napropamide	6.2×10^{-3}	1.1×10^{-1}		
Oryzalin	9.6×10^{-3}	3.4×10^{-1}		
Oxadiazon	6.2×10^{-2}	2.3×10^{0}		
Picloram	1.9×10^{-3}	8.7×10^{-2}		
Pronamide	4.1×10^{-3}	1.5×10^{-1}		
Sethoxydim	4.8×10^{-4}	1.4×10^{-2}		
Sulfometuron methyl	5.9×10^{-4}	3.2×10^{-2}		
Triclopyr	1.0×10^{-2}	1.0×10^{0}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-21 Estimates of Non-cancer and Cancer Risk from Ingestion of Drift Contaminated Vegetables, Lettuce

COMPOUND	Non-cancer Hazard Quotient		Cancer Risk	
	Average	Maximum**	Average	Maximum**
2,4-D	2.8×10^{-2}	9.7×10^{0}	2.9×10^{-10}	6.6×10^{-7}
2,4-DP	3.9×10^{-3}	2.4×10^{0}		
Benefin	8.8×10^{-4}	5.8×10^{-2}		
Bromacil	1.1×10^{-2}	2.8×10^{0}	5.4×10^{-11}	9.5×10^{-8}
Chlorsulfuron	2.4×10^{-4}	1.6×10^{-2}		
Chlorthal dimethyl	3.3×10^{-3}	1.7×10^{-1}		
Dicamba	4.8×10^{-3}	1.6×10^{0}		
Dichlobenil	5.1×10^{-2}	9.3×10^{0}		
Diuron	2.2×10^{-1}	1.2×10^{2}		
Fluazifop-butyl	3.4×10^{-3}	2.2×10^{-1}		
Fosamine	3.8×10^{-2}	5.6×10^{0}		
Glyphosate	2.0×10^{-3}	6.2×10^{-1}		
Imazapyr	2.0×10^{-4}	2.9×10^{-2}		
Isoxaben	1.8×10^{-3}	1.2×10^{-1}		
Mecoprop	6.7×10^{-3}	3.1×10^{-1}		
Mefluidide	5.4×10^{-3}	3.9×10^{-1}		
Metsulfuron methyl	2.0×10^{-5}	3.5×10^{-3}		
Napropamide	5.2×10^{-3}	2.3×10^{-1}		
Oryzalin	8.0×10^{-3}	7.0×10^{-1}	2.8×10^{-9}	1.6×10^{-6}
Oxadiazon	5.2×10^{-2}	4.6×10^{0}	1.9×10^{-9}	1.2×10^{-6}
Picloram	1.6×10^{-3}	1.8×10^{-1}	1.8×10^{-11}	1.3×10^{-8}
Pronamide	3.4×10^{-3}	3.1×10^{-1}	2.2×10^{-10}	1.3×10^{-7}
Sethoxydim	4.0×10^{-4}	3.0×10^{-2}		
Sulfometuron methyl	4.9×10^{-4}	6.5×10^{-2}		
Triclopyr	8.5×10^{-3}	2.1×10^{0}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-22 Estimates of Non-cancer and Cancer Risk from Ingestion of Drift Contaminated Vegetables, Beans

COMPOUND	Non-cancer Hazard Quotient		Cancer Risk	
	Average	Maximum**	Average	Maximum**
2,4-D	4.9×10^{-3}	1.5×10^{0}	1.2×10^{-9}	2.5×10^{-6}
2,4-DP	6.7×10^{-4}	3.7×10^{-1}		
Benefin	1.5×10^{-4}	8.8×10^{-3}		
Bromacil	1.8×10^{-3}	4.2×10^{-1}	2.3×10^{-10}	3.6×10^{-7}
Chlorsulfuron	4.2×10^{-5}	2.5×10^{-3}		
Chlorthal dimethyl	5.7×10^{-4}	2.7×10^{-2}		
Dicamba	8.3×10^{-4}	2.4×10^{-1}		
Dichlobenil	8.7×10^{-3}	1.4×10^{0}		
Diuron	3.7×10^{-2}	1.8×10^{1}		
Fluazifop-butyl	5.8×10^{-4}	3.4×10^{-2}		
Fosamine	6.4×10^{-3}	8.5×10^{-1}		
Glyphosate	3.4×10^{-4}	9.4×10^{-2}		
Imazapyr	3.5×10^{-5}	4.4×10^{-3}		
Isoxaben	3.1×10^{-4}	1.8×10^{-2}		
Mecoprop	1.2×10^{-3}	4.8×10^{-2}		
Mefluidide	9.3×10^{-4}	5.9×10^{-2}		
Metsulfuron methyl	3.5×10^{-6}	5.3×10^{-4}		
Napropamide	8.9×10^{-4}	3.5×10^{-2}		
Oryzalin	1.4×10^{-3}	1.1×10^{-1}	1.2×10^{-8}	6.2×10^{-6}
Oxadiazon	8.9×10^{-3}	7.1×10^{-1}	8.2×10^{-9}	4.4×10^{-6}
Picloram	2.8×10^{-4}	2.7×10^{-2}	7.8×10^{-11}	5.1×10^{-8}
Pronamide	5.9×10^{-4}	4.7×10^{-2}	9.4×10^{-10}	5.1×10^{-7}
Sethoxydim	6.9×10^{-5}	4.5×10^{-3}		
Sulfometuron methyl	8.4×10^{-5}	9.9×10^{-3}		
Triclopyr	1.5×10^{-3}	3.2×10^{-1}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-23 Estimates of Non-cancer and Cancer Risk from Dermal Contact with Drift Contaminated Berries

COMPOUND	Non-cancer Hazard Quotient		Cancer Risk	
	Average	Maximum**	Average	Maximum**
2,4-D	2.1×10^{-2}	2.4×10^{0}	8.5×10^{-9}	4.7×10^{-8}
2,4-DP	1.9×10^{-3}	3.8×10^{-1}		
Benefin	6.6×10^{-4}	1.4×10^{-2}		
Bromacil	7.8×10^{-4}	6.8×10^{-2}	1.6×10^{-10}	6.8×10^{-8}
Chlorsulfuron	1.8×10^{-5}	4.0×10^{-4}		
Chlorthal dimethyl	2.5×10^{-3}	4.3×10^{-2}		
Dicamba	1.8×10^{-3}	1.9×10^{-1}		
Dichlobenil	1.9×10^{-2}	1.1×10^{0}		
Diuron	8.0×10^{-2}	1.4×10^{1}		
Fluazifop-butyl	1.3×10^{-3}	2.7×10^{-2}		
Fosamine	2.8×10^{-3}	1.4×10^{-1}		
Glyphosate	5.2×10^{-4}	5.3×10^{-2}		
Imazapyr	1.5×10^{-4}	7.1×10^{-3}		
Isoxaben	6.6×10^{-4}	1.4×10^{-2}		
Mecoprop	5.0×10^{-3}	7.7×10^{-2}		
Mefluidide	4.0×10^{-4}	9.5×10^{-3}		
Metsulfuron methyl	1.5×10^{-5}	8.5×10^{-4}		
Napropamide	1.9×10^{-3}	2.8×10^{-2}		
Oryzalin	3.0×10^{-3}	8.5×10^{-2}	4.1×10^{-8}	5.9×10^{-6}
Oxadiazon	3.8×10^{-2}	1.1×10^{0}	5.7×10^{-8}	8.4×10^{-6}
Picloram	5.8×10^{-5}	2.1×10^{-3}	2.6×10^{-11}	4.7×10^{-9}
Pronamide	1.3×10^{-3}	3.8×10^{-2}	3.3×10^{-9}	4.8×10^{-7}
Sethoxydim	3.0×10^{-5}	7.3×10^{-4}		
Sulfometuron methyl	3.7×10^{-5}	1.6×10^{-3}		
Triclopyr	1.1×10^{-3}	8.4×10^{-2}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-24 Estimates of Non-cancer and Cancer Risk from Dermal Contact with Sprayed Vegetation

COMPOUND	Non-cancer Hazard Quotient		Cancer Risk	
	Average	Maximum**	Average	Maximum**
2,4-D	2.1×10^{-1}	2.1×10^{1}	1.5×10^{-6}	8.8×10^{-4}
2,4-DP	1.8×10^{-2}	3.5×10^{0}		
Benefin	6.5×10^{-3}	1.3×10^{-1}		
Bromacil	7.7×10^{-3}	6.2×10^{-1}	4.1×10^{-9}	4.2×10^{-6}
Chlorsulfuron	1.8×10^{-4}	3.6×10^{-3}		
Chlorthal dimethyl	2.4×10^{-2}	3.9×10^{-1}		
Dicamba	1.8×10^{-2}	1.7×10^{0}		
Dichlobenil	1.9×10^{-1}	1.0×10^{1}		
Diuron	7.9×10^{-1}	1.3×10^{2}		
Fluazifop-butyl	1.2×10^{-2}	2.5×10^{-1}		
Fosamine	2.8×10^{-2}	1.3×10^{0}		
Glyphosate	5.1×10^{-3}	4.8×10^{-1}		
Imazapyr	1.5×10^{-3}	6.5×10^{-2}		
Isoxaben	6.5×10^{-3}	1.3×10^{-1}		
Mecoprop	4.9×10^{-2}	7.0×10^{-1}		
Mefluidide	4.0×10^{-3}	8.7×10^{-2}		
Metsulfuron methyl	1.5×10^{-4}	7.8×10^{-3}		
Napropamide	1.9×10^{-2}	2.6×10^{-1}		
Oryzalin	2.9×10^{-2}	7.8×10^{-1}	1.1×10^{-6}	3.6×10^{-4}
Oxadiazon	3.8×10^{-1}	1.0×10^{1}	1.5×10^{-6}	5.1×10^{-4}
Picloram	5.7×10^{-4}	1.9×10^{-2}	4.4×10^{-9}	8.6×10^{-7}
Pronamide	1.3×10^{-2}	3.5×10^{-1}	4.3×10^{-8}	2.0×10^{-5}
Sethoxydim	2.9×10^{-4}	6.6×10^{-3}		
Sulfometuron methyl	3.6×10^{-4}	1.5×10^{-2}		
Triclopyr	1.0×10^{-2}	7.7×10^{-1}		

*Numbers rounded off to one decimal.
**WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-25 Hiking Through Sprayed Vegetation to Pick Contaminated Blackberries Which Are Consumed the Same Day

	Dermal Vegetation Risk	Dermal Berry Risk	Oral Berry Risk	Total Risk*
2,4-D	$2.1x10^{-1}$	$2.1x10^{-2}$	$8.7x10^{-3}$	$2.4x10^{-1}$
2,4-DP	$1.8x10^{-2}$	$1.9x10^{-3}$	$1.2x10^{-3}$	$2.1x10^{-2}$
Benefin	$6.5x10^{-2}$	$6.6x10^{-3}$	$2.7x10^{-4}$	$7.4x10^{-3}$
Bromacil	$7.7x10^{-3}$	$7.8x10^{-4}$	$3.2x10^{-3}$	$1.2x10^{-2}$
Chlorsulfuron	$1.8x10^{-4}$	$1.8x10^{-5}$	$7.5x10^{-5}$	$2.7x10^{-4}$
Chlorthal dimethyl	$2.4x10^{-2}$	$2.5x10^{-3}$	$1.0x10^{-3}$	$2.8x10^{-2}$
Dicamba	$1.8x10^{-2}$	$1.8x10^{-3}$	$1.5x10^{-3}$	$2.1x10^{-2}$
Dichlobenil	$1.9x10^{-1}$	$1.9x10^{-2}$	$1.6x10^{-2}$	$2.3x10^{-1}$
Diuron	$7.9x10^{-1}$	$8.0x10^{-2}$	$6.6x10^{-2}$	$9.4x10^{-1}$
Fluazifop-butyl	$1.2x10^{-2}$	$1.3x10^{-3}$	$1.0x10^{-3}$	$1.4x10^{-2}$
Fosamine	$2.8x10^{-2}$	$2.8x10^{-3}$	$1.2x10^{-2}$	$4.3x10^{-2}$
Glyphosate	$5.1x10^{-3}$	$5.2x10^{-4}$	$6.1x10^{-4}$	$6.2x10^{-3}$
Imazapyr	$1.5x10^{-3}$	$1.5x10^{-4}$	$6.2x10^{-5}$	$1.7x10^{-3}$
Isoxaben	$6.5x10^{-3}$	$6.6x10^{-4}$	$5.5x10^{-4}$	$7.7x10^{-3}$
Mecoprop	$4.9x10^{-2}$	$5.0x10^{-3}$	$2.1x10^{-3}$	$5.6x10^{-2}$
Mefluidide	$4.0x10^{-3}$	$4.0x10^{-4}$	$1.7x10^{-3}$	$6.1x10^{-3}$
Metsulfuron methyl	$1.5x10^{-4}$	$1.5x10^{-5}$	$6.2x10^{-6}$	$1.7x10^{-4}$
Napropamide	$1.9x10^{-2}$	$1.9x10^{-3}$	$1.6x10^{-3}$	$2.3x10^{-2}$
Oryzalin	$2.9x10^{-2}$	$3.0x10^{-3}$	$2.5x10^{-3}$	$3.5x10^{-2}$
Oxadiazon	$3.8x10^{-1}$	$3.8x10^{-2}$	$1.6x10^{-2}$	$4.3x10^{-1}$
Picloram	$5.7x10^{-4}$	$5.8x10^{-5}$	$5.0x10^{-4}$	$1.1x10^{-3}$
Pronamide	$1.3x10^{-2}$	$1.3x10^{-3}$	$1.1x10^{-3}$	$1.5x10^{-2}$
Sethoxydim	$2.9x10^{-4}$	$3.0x10^{-5}$	$8.4x10^{-5}$	$4.0x10^{-4}$
Sulfometuron methyl	$3.6x10^{-4}$	$3.7x10^{-5}$	$1.5x10^{-4}$	$5.5x10^{-4}$
Triclopyr	$1.0x10^{-2}$	$1.1x10^{-3}$	$2.6x10^{-3}$	$1.4x10^{-2}$

*Numbers rounded off to one decimal.

Table 4-26 Ingestion of Contaminated Lettuce, Beans and Berries in the Same Day

	Oral Lettuce Risk	Oral Bean Risk	Oral Berry Risk	Total Risk*
2,4-D	$2.8x10^{-2}$	$4.9x10^{-3}$	$8.7x10^{-3}$	$4.2x10^{-2}$
2,4-DP	$3.9x10^{-3}$	$6.7x10^{-4}$	$1.2x10^{-3}$	$5.8x10^{-3}$
Benefin	$8.8x10^{-4}$	$1.5x10^{-4}$	$2.7x10^{-4}$	$1.3x10^{-3}$
Bromacil	$1.1x10^{-2}$	$1.8x10^{-3}$	$3.2x10^{-3}$	$1.6x10^{-2}$
Chlorsulfuron	$2.4x10^{-4}$	$4.2x10^{-5}$	$7.5x10^{-5}$	$3.6x10^{-4}$
Chlorthal dimethyl	$3.3x10^{-3}$	$5.7x10^{-4}$	$1.0x10^{-3}$	$4.9x10^{-3}$
Dicamba	$4.8x10^{-3}$	$8.3x10^{-4}$	$1.5x10^{-3}$	$7.1x10^{-3}$
Dichlobenil	$5.1x10^{-2}$	$8.8x10^{-3}$	$1.6x10^{-2}$	$7.6x10^{-2}$
Diuron	$2.2x10^{-1}$	$3.7x10^{-2}$	$6.6x10^{-2}$	$3.2x10^{-1}$
Fluazifop-butyl	$3.4x10^{-3}$	$5.8x10^{-4}$	$1.0x10^{-3}$	$5.0x10^{-3}$
Fosamine	$3.8x10^{-2}$	$6.4x10^{-3}$	$1.2x10^{-2}$	$5.6x10^{-2}$
Glyphosate	$2.0x10^{-3}$	$3.4x10^{-4}$	$6.1x10^{-4}$	$3.0x10^{-3}$
Imazapyr	$2.0x10^{-4}$	$3.5x10^{-5}$	$6.2x10^{-5}$	$3.0x10^{-4}$
Isoxaben	$1.8x10^{-3}$	$3.1x10^{-4}$	$5.5x10^{-4}$	$2.7x10^{-3}$
Mecoprop	$6.7x10^{-3}$	$1.2x10^{-3}$	$2.1x10^{-3}$	$1.0x10^{-2}$
Mefluidide	$5.4x10^{-3}$	$9.3x10^{-4}$	$1.7x10^{-3}$	$8.0x10^{-3}$
Metsulfuron methyl	$2.0x10^{-5}$	$3.5x10^{-6}$	$6.2x10^{-6}$	$3.0x10^{-5}$
Napropamide	$5.2x10^{-3}$	$8.9x10^{-4}$	$1.6x10^{-3}$	$7.7x10^{-3}$
Oryzalin	$8.0x10^{-3}$	$1.4x10^{-3}$	$2.5x10^{-3}$	$1.2x10^{-2}$
Oxadiazon	$5.2x10^{-2}$	$8.9x10^{-3}$	$1.6x10^{-2}$	$7.7x10^{-2}$
Picloram	$1.6x10^{-3}$	$2.8x10^{-4}$	$5.0x10^{-4}$	$2.4x10^{-3}$
Pronamide	$3.4x10^{-3}$	$5.9x10^{-4}$	$1.1x10^{-3}$	$5.1x10^{-3}$
Sethoxydim	$4.0x10^{-4}$	$6.9x10^{-5}$	$8.4x10^{-5}$	$5.5x10^{-4}$
Sulfometuron methyl	$4.9x10^{-4}$	$8.4x10^{-5}$	$1.5x10^{-4}$	$7.2x10^{-4}$
Triclopyr	$8.5x10^{-3}$	$1.5x10^{-3}$	$2.6x10^{-3}$	$1.3x10^{-2}$

*Numbers rounded off to one decimal.

Table 4-27 Hazard Indices For WSDOT Worker Risk From Multiple Chemicals*

Compound Mixture	Applicator	Mixer Loader	Applicator Mixer Loader	Hand-held Spray Equipment
Glyphosate Sulfometuron methyl	$8.7x10^{-3}$	$2.6x10^{-2}$	$2.6x10^{-2}$	$1.3x10^{-2}$
Glyphosate Diuron	$1.3x10^{0}$	$3.8x10^{0}$	$3.8x10^{0}$	$1.9x10^{0}$
2,4-D Dicamba	$3.6x10^{-1}$	$1.1x10^{0}$	$1.1x10^{0}$	$5.4x10^{-1}$
2,4-D Chlorsulfuron	$3.3x10^{-1}$	$9.9x10^{-1}$	$9.9x10^{-1}$	$5.0x10^{-1}$
Glyphosate Oryzalin	$5.5x10^{-2}$	$1.6x10^{-1}$	$1.6x10^{-1}$	$8.3x10^{-2}$
2,4-D Picloram	$3.3x10^{-1}$ Cancer: $1.0x10^{-6}$	$9.9x10^{-1}$ Cancer: $3.1x10^{-6}$	$9.9x10^{-1}$ Cancer: $3.1x10^{-6}$	$5.0x10^{-1}$ Cancer: $1.6x10^{-6}$
Diuron Sulfometuron methyl	$1.3x10^{0}$	$3.8x10^{0}$	$3.8x10^{0}$	$1.9x10^{0}$
2,4-D Triclopyr	$3.5x10^{-1}$	$1.0x10^{0}$	$1.0x10^{0}$	$5.3x10^{-1}$
2,4-D Metsulfuron methyl	$3.3x10^{-1}$	$9.9x10^{-1}$	$9.9x10^{-1}$	$5.0x10^{-1}$
Glyphosate Sulfometuron methyl Bromacil	$2.1x10^{-2}$	$6.3x10^{-2}$	$6.3x10^{-2}$	$3.2x10^{-2}$
Glyphosate Diuron Bromacil	$1.3x10^{0}$	$3.9x10^{0}$	$3.9x10^{0}$	$1.9x10^{0}$
2,4-D Chlorsulfuron Picloram	$3.3x10^{-1}$	$9.9x10^{-1}$	$9.9x10^{-1}$	$5.0x10^{-1}$
2,4-D Chlorsulfuron Dicamba	$3.6x10^{-1}$	$1.1x10^{0}$	$1.1x10^{0}$	$5.4x10^{-1}$
Glyphosate Sulfometuron methyl Diuron	$1.3x10^{0}$	$3.8x10^{0}$	$3.8x10^{0}$	$1.9x10^{0}$
Diuron Sulfometuron methyl Bromacil	$1.3x10^{0}$	$3.8x10^{0}$	$3.8x10^{0}$	$1.9x10^{0}$
Chlorsulfuron Metsulfuron methyl Picloram	$1.4x10^{-3}$	$4.3x10^{-3}$	$4.3x10^{-3}$	$2.2x10^{-3}$

*Numbers rounded off to one decimal.

Table 4-28 Hazard Indices For Public Risk From Exposure to Multiple Chemicals*

COMPOUND MIXTURE	Oral Berry Adult	Oral Berry Child	Oral Lettuce	Oral Beans	Dermal Berry	Dermal Vegetation
Glyphosate Sulfometuron methyl	$7.6x10^{-4}$	$3.0x10^{-3}$	$2.5x10^{-3}$	$4.2x10^{-4}$	$5.6x10^{-4}$	$5.5x10^{-3}$
Glyphosate Diuron	$6.7x10^{-2}$	$2.6x10^{-1}$	$2.2x10^{-1}$	$3.7x10^{-2}$	$8.1x10^{-2}$	$8.0x10^{-1}$
2,4-D Dicamba	$1.0x10^{-2}$	$4.0x10^{-2}$	$3.3x10^{-2}$	$5.7x10^{-3}$	$2.3x10^{-2}$	$2.3x10^{-1}$
2,4-D Chlorsulfuron	$8.8x10^{-3}$	$3.4x10^{-2}$	$2.8x10^{-2}$	$4.9x10^{-3}$	$2.1x10^{-2}$	$2.1x10^{-1}$
Glyphosate Oryzalin	$3.1x10^{-3}$	$1.2x10^{-2}$	$1.0x10^{-2}$	$1.7x10^{-3}$	$3.5x10^{-3}$	$3.5x10^{-2}$
2,4-D Picloram	9.2x10-3 Cancer: $1.9x10^{-8}$	$3.6x10^{-2}$	$3.0x10^{-2}$ Cancer: $3.1x10^{-10}$	$5.2x10^{-3}$ Cancer: $1.3x10^{-9}$	$2.1x10^{-2}$ Cancer: $8.5x10^{-9}$	$2.1x10^{-1}$ Cancer: $1.5x10^{-6}$
Diuron Sulfometuron methyl	$6.6x10^{-2}$	$2.6x10^{-1}$	$2.2x10^{-1}$	$3.7x10^{-2}$	$8.0x10^{-2}$	$7.9x10^{-1}$
2,4-D Triclopyr	$1.1x10^{-2}$	$4.4x10^{-2}$	$3.7x10^{-2}$	$6.4x10^{-3}$	$2.2x10^{-2}$	$2.2x10^{-1}$
2,4-D Metsulfuron methyl	$8.7x10^{-3}$	$3.4x10^{-2}$	$2.8x10^{-2}$	$4.9x10^{-3}$	$2.1x10^{-2}$	$2.1x10^{-1}$
Glyphosate Sulfometuron methyl Bromacil	$4.0x10^{-3}$	$1.6x10^{-2}$	$1.3x10^{-2}$	$2.2x10^{-3}$	$1.3x10^{-3}$	$1.3x10^{-2}$
Glyphosate Diuron Bromacil	$7.0x10^{-2}$	$2.8x10^{-1}$	$2.3x10^{-1}$	$3.9x10^{-2}$	$8.1x10^{-2}$	$8.0x10^{-1}$
2,4-D Chlorsulfuron Picloram	$9.3x10^{-3}$	$3.6x10^{-2}$	$3.0x10^{-2}$	$5.2x10^{-3}$	$2.1x10^{-2}$	$2.2x10^{-1}$
2,4-D Chlorsulfuron Dicamba	$1.0x10^{-2}$	$4.0x10^{-2}$	$3.3x10^{-2}$	$5.8x10^{-3}$	$2.3x10^{-2}$	$2.3x10^{-1}$
Glyphosate Sulfometuron methyl Diuron	$6.7x10^{-2}$	$2.6x10^{-1}$	$2.2x10^{-1}$	$3.7x10^{-2}$	$8.1x10^{-2}$	$8.0x10^{-1}$
Diuron Sulfometuron methyl Bromacil	$6.9x10^{-2}$	$2.7x10^{-1}$	$2.3x10^{-1}$	$3.9x10^{-2}$	$8.1x10^{-2}$	$8.0x10^{-1}$
Chlorsulfuron Metsulfuron methyl Picloram	$5.8x10^{-4}$	$2.2x10^{-3}$	$1.9x10^{-3}$	$3.3x10^{-4}$	$9.1x10^{-5}$	$9.0x10^{-4}$

*Numbers rounded off to one decimal.

Table 4-29 Compounds of Negligible Risk

	SYSTEMIC EFFECTS		CANCER EFFECTS	
	Average	Maximum*	Average	Maximum*
Applicator	4D,DP,BE,BR,CS,CD, DC,DB,FB,FO,GL,IM, IS,MC,MF,MM,NA,OR, OX,PI,PR,SE,SM,TR	BE,BR,CS,CD,FB,GL, IM,IS,MC,MF,MM, NA,OR,PI,PR,SE,SM, TR	4D,BR,OR,OX,PI,PR	BR,PI,PR
Mixer/Loader	4D,DP,BE,BR,CS,CD, DC,DB,FB,FO,GL,IM, IS,MC,MF,MM,NA,OR, PI,PR,SE,SM,TR	BE,CS,CD,FB,GL,IM, IS,MF,MM,NA,PI,PR, SE,SM	4D,BR,OR,OX,PI,PR	BR,PI,PR
Applicator/Mixer/Loader	4D,DP,BE,BR,CS,CD, DC,DB,FB,FO,GL,IM, IS,MC,MF,MM,NA,OR, PI,PR,SE,SM,TR	BE,BR,CS,CD,FB,GL, IM,IS,MC,MF,MM, NA,PI,PR,SE,SM	4D,BR,OR,OX,PI,PR	BR,PI,PR
Hand-held Spray Applicator	4D,DP,BE,BR,CS,CD, DC,DB,FB,FO,GL,IM, IS,MC,MF,MM,NA,OR, OX,PI,PR,SE,SM,TR	DP,BE,BR,CS,CD,DC, FB,FO,GL,IM,IS,MC, MF,MM,NA,OR,PI, PR,SE,SM,TR	4D,BR,OR,OX,PI,PR	BR,PI,PR
Ingestion of Drift Contaminated Berries (adult)	4D,CS,DC,FO,GL,MC, MM,PI TR (all)	CS,DC,FO,GL,MC, MM,PI,TR	4D,PI	PI
Ingestion of Drift Contaminated Berries (child)	4D,CS,DC,FO,GL,MC, MM,PI, TR (all)	CS,DC,GL,MC,MM,PI TR	Not applicable	Not applicable
Ingestion of Drift Contaminated Lettuce	4D,CS,DC,FO,GL,MC, MM,PI, TR (all)	CS,GL,MC,MM,PI	4D,PI	4D,PI
Ingestion of Drift Contaminated Beans	4D,CS,DC,FO,GL,MC, MM,PI, TR (all)	CS,DC,FO,GL,MC, MM,PI,TR	4D,PI	4D,PI
Dermal Contact with Drift Contaminated Berries	4D,CS,DC,FO,GL,MC, MM,PI TR (all)	CS,DC,FO,GL,MC, MM,PI,TR	4D,PI	4D,PI
Dermal Contact with Directly Sprayed Vegetation	4D,DP,BE,BR,CS,CD, DC,DB,DU,FB,FO,GL, IM,IS,MC,MF,MM,NA, OR,OX,PI,PR,SE,SM, TR (all)	BE,BR,CS,CD,FB,GL, IM,IS,MC,MF,MM, NA,OR,PI,PR,SE,SM, TR	4D,BR,OR,OX,PI,PR	BR,PI

Legend:

4D=2,4-D
DP=2,4-DP
BE=Benefin
BR=Bromacil
CS=Chlorsulfuron
CD=Chlorthal dimethyl
DC=Dicamba
DB=Dichlobenil
DU=Diuron
FB=Fluazifop-butyl
FO=Fosamine
GL=Glyphosate
IM=Imazapyr
IS=Isoxaben
MC=Mecoprop
MF=Mefluidide
MM=Metsulfuron methyl
NA=Napropamide
OR=Oryzalin
OX=Oxadiazon
PI=Picloram
PR=Pronamide
SE=Sethoxydim
SM=Sulfometuron methyl
TR=Triclopyr

*WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-30 Compounds of Low Risk

	SYSTEMIC EFFECTS		CANCER EFFECTS	
	Average	Maximum*	Average	Maximum*
Applicator	DU	DP,DC,DB,FO,OX	---	OR,OX
Mixer/Loader	DU,OX	DP,BR,DC,DB,FO MC,OR,TR	---	---
Applicator/Mixer/Loader	DU,OX	DP,DC,DB,FO,OR,TR	---	OR
Hand-held Spray Applicator	DU	DB,OX	---	OR,OX
Ingestion of Drift Contaminated Berries (adult)	---	4D	---	4D,OR,OX
Ingestion of Drift Contaminated Berries (child)	---	4D,FO	not applicable	not applicable
Ingestion of Drift Contaminated Lettuce	---	4D,DC,FO,TR	---	---
Ingestion of Drift Contaminated Beans	---	4D	---	---
Dermal Contact with Drift Contaminated Berries	---	4D	---	---
Dermal Contact with Directly Sprayed Vegetation	---	DP,DC,DB,FO,OX	---	PR

Legend:

4D-2,4-D
DP-2,4-DP
BE-Benefin
BR-Bromacil
CS-Chlorsulfuron
CD-Chlorthal dimethyl
DC-Dicamba
DB-Dichlobenil
DU-Diuron
FB-Fluazifop-butyl
FO-Fosamine
GL-Glyphosate
IM-Imazapyr
IS-Isoxaben
MC-Mecoprop
MF-Mefluidide
MM-Metsulfuron methyl
NA-Napropamide
OR-Oryzalin
OX-Oxadiazon
PI-Picloram
PR-Pronamide
SE-Sethoxydim
SM-Sulfometuron methyl
TR-Triclopyr

*WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-31 Compounds of Moderate Risk

	SYSTEMIC EFFECTS		CANCER EFFECTS	
	Average	Maximum*	Average	Maximum*
Applicator	---	4D	---	4D
Mixer/Loader	---	4D,OX	---	4D,OR,OX
Applicator/Mixer/Loader	---	4D,OX	---	4D,OX
Hand-held Spray Applicator	---	4D,DU	---	4D
Ingestion of Drift Contaminated Berries (adult)	---	---	---	---
Ingestion of Drift Contaminated Berries (child)	---	---	not applicable	not applicable
Ingestion of Drift Contaminated Lettuce	---	---	---	---
Ingestion of Drift Contaminated Beans	---	---	---	---
Dermal Contact with Drift Contaminated Berries	---	---	---	---
Dermal Contact with Directly Sprayed Vegetation	---	4D	---	4D,OR,OX

Legend:

4D-2,4-D
DP-2,4-DP
BE-Benefin
BR-Bromacil
CS-Chlorsulfuron
CD-Chlorthal dimethyl
DC-Dicamba
DB-Dichlobenil
DU-Diuron
FB-Fluazifop-butyl
FO-Fosamine
GL-Glyphosate
IM-Imazapyr
IS-Isoxaben
MC-Mecoprop
MF-Mefluidide
MM-Metsulfuron methyl
NA-Napropamide
OR-Oryzalin
OX-Oxadiazon
PI-Picloram
PR-Pronamide
SE-Sethoxydim
SM-Sulfometuron methyl
TR-Triclopyr

*WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-32 Compounds of High Risk

SYSTEMIC EFFECTS			CANCER EFFECTS	
	Average	Maximum*	Average	Maximum*
Applicator	---	DU	---	---
Mixer/Loader	---	DU	---	---
Applicator/Mixer/Loader	---	DU	---	---
Hand-held Spray Applicator	---	---	---	---
Ingestion of Drift Contaminated Berries (adult)	---	---	---	---
Ingestion of Drift Contaminated Berries (child)	---	---	not applicable	not applicable
Ingestion of Drift Contaminated Lettuce	---	---	---	---
Ingestion of Drift Contaminated Beans	---	---	---	---
Dermal Contact with Drift Contaminated Berries	---	---	---	---
Dermal Contact with Directly Sprayed Vegetation	---	DU	---	---

Legend:
4D-2,4-D
DP-2,4-DP
BE-Benefin
BR-Bromacil
CS-Chlorsulfuron
CD-Chlorthal dimethyl
DC-Dicamba
DB-Dichlobenil
DU-Diuron
FB-Fluazifop-butyl
FO-Fosamine
GL-Glyphosate
IM-Imazapyr
IS-Isoxaben
MC-Mecoprop
MF-Mefluidide
MM-Metsulfuron methyl
NA-Napropamide
OR-Oryzalin
OX-Oxadiazon
PI-Picloram
PR-Pronamide
SE-Sethoxydim
SM-Sulfometuron methyl
TR-Triclopyr

*WSDOT rarely uses maximum label rate see Table 4-36 for maximum WSDOT rates.

Table 4-33 Toxicity Categories

Hazard Indicator	I	II	III	IV
SIGNAL WORD	Severe	Moderate	Slight	Very Slight
Oral LD50.....	Up to and including 50 mg/kg	From 50 thru 500 mg/kg	From 500 thru 5000 mg/kg	Greater than 5000 mg/kg
Inhalation LC50.	Up to and including 0.2 mg/liter	From 0.2 thru 2 mg/liter	From 2 thru 20 mg/liter	Greater than 20 mg/liter
Dermal LD50.....	Up to and including 200 mg/kg	From 200 thru 2000 mg/kg	From 2000 thru 20000 mg/kg	Greater than 20000 mg/kg
Eye Effects.....	Corrosive; corneal opacity not reversible within 7 days	Corneal opacity reversible within 7 days; irritation persisting for 7 days	No corneal opacity; irritation reversible within 7 days	No irritation
Skin Effects....	Corrosive	Severe irritation at 72 hours	Moderate irritation at 72 hours	Mild or Slight irritation at 72 hours

Table 4-34 Precautionary Statements

TOXICITY CATEGORY	Oral, Inhalation or Dermal Toxicity	Skin Eye and Local Effects
I.....	Fatal (poisonous) if swallowed [inhaled or absorbed through skin]. Do not breathe vapor [dust or spray mist]. Do not get in eyes, on skin, or on clothing. [Front panel statement of practical treatment required].	Corrosive, causes eye and skin damage [or skin irritation]. Do not get in eyes, on skin, or on clothing. Wear goggles or face shield and rubber gloves when handling. Harmful or fatal if swallowed. [Appropriate first aid statement required].
II.....	May be fatal if swallowed [inhaled or absorbed through skin]. Do not breathe vapors [dust or spray mist]. Do not get in eyes, on skin, or on clothing. [Appropriate first aid statement required].	Causes eye [and skin] irritation. Do not get in eyes, on skin, or on clothing. Harmful if swallowed. [Appropriate first aid statement required].
III.....	Harmful if swallowed [inhaled or absorbed through skin]. Avoid breathing vapors [dust or spray mist]. Avoid contact with skin [eyes or clothing]. [Appropriate first aid statement required].	Avoid contact with skin, eyes or clothing. In case of contact immediately flush eyes or skin with plenty of water. Get medical attention if irritation persists.
IV.....	[No precautionary statements required].	[No precautionary statements required].

Table 4-35 List of Classification Set By EPA

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogeniologic	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer.
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies.
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans.
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals.
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals.
Group E	No Evidence of Carcinogenicity	No evidence for carcinogenicity in at least two adequate animal tests or in humans in epidemiologic and animal studies.

Table 4-36 WSDOT's average and maximum application rates for the 25 herbicides from July 1989 through June 1990. These rates (average and maximum) are compared with the maximum label rate for each of the herbicides.

CHEMICAL	max. label rate lbs/acre	WSDOT's avg. appl. rate lbs/acre	% of max. label rate	WSDOT's max. appl. rate lbs/acre	% of max. label rate
2,4-D	16.65	2.16	13%	5.82	35%
2,4-DP	16.65	1.21	7%	1.28	8%
Benefin	3.00	2.05	68%	2.50	83%
Chlorsulfuron Methyl	0.14	0.10	71%	0.13	93%
Bromacil	12.00	2.04	17%	2.52	21%
Chlorthal Dimethyl	15.00	12.75	85%	12.75	85%
Dicamba	8.00	1.12	14%	3.98	50%
Dichlobenil	20.00	4.93	25%	6.50	33%
Diuron	40.00	3.33	8%	10.30	26%
Fosamine Ammonium	24.00	7.28	30%	9.30	39%
Glysphosate	10.60	1.55	15%	5.45	51%
Imazapyr	1.50	0.47	31%	0.60	40%
Isoxaben	1.00	0.69	69%	0.75	75%
Mecoprop	0.54	0.52	96%	0.60	111%
Mefluidide	1.00	0.63	63%	0.63	63%
Metsulfuron Methyl	0.15	0.04	27%	0.07	47%
Napropamide	4.00	4.00	100%	4.00	100%
Oryzalin	6.00	3.12	52%	3.90	65%
Picloram	4.00	0.88	22%	1.63	41%
Pronamide	4.00	2.00	50%	2.00	50%
Sulfometuron	0.56	0.19	34%	0.27	48%
Triclopyr	9.00	1.65	18%	5.00	56%

Appendix B - Chapter 5

Draft EIS
Roadside Vegetation Management

Introduction

The tables in this chapter are intended to summarize the information that is critical for conducting a wildlife and human health risk assessment. There are eight separate tables in this chapter. The first table (Table 5-1) identifies the active ingredients for the herbicides that have been used by WSDOT and which they plan to use in the future. The table also contains the product names that contain the active ingredient. It also includes the "signal word" that is used to indicate the hazards associated with using the product. If the signal word is either "danger" or "warning," then a reason is given that explains why the product is dangerous. All of the herbicides that are considered to be dangerous are classified as such because of their likelihood of causing eye damage. None of the herbicides are considered dangerous based on their acute toxicities.

Table 5-2 is set up very similar to Table 5-1. It includes the active ingredients and their products. It also presents the "formulation" or the amount of active ingredient in each product and the maximum label rate of each product. In addition, this table lists the inert ingredients in the products that are considered by the EPA to be of toxicological concern.

Tables 5-3 through 5-6 list each of the active ingredients and presents the physical data, toxicity data, environmental data and wildlife data for each of these active ingredients, respectively.

Tables 5-7 and 5-8 present the same data as above (Tables 5-3 through 5-6) for the inert ingredients of concern and the adjuvants that have been used by WSDOT in the past, respectively.

The units for these tables have been standardized as much as possible. However, there are cases in which the results for a study from one source is presented in different units than that from another source. All units presented in these tables are as they were reported in the data source.

A minimum amount of text has been included in these tables in order to make them short and concise. However, all of the values in these tables that were needed for an analysis of risk, have been discussed in their appropriate chapters of this appendix.

Tables 5-9 through 5-33 are presented here as a summary of the historical use of herbicides by WSDOT from July of 1989 to June of 1990. These numbers were generated by analyzing the application records for this time period and determining products used that contain each active ingredient, application methods and mode of action for each active ingredient, average application rates, minimum application rates, maximum application rates, total amount of each active ingredient used and the target vegetation for each active ingredient. These numbers were then broken down by district so differences in use patterns could be observed.

The application rates that were used in the risk assessments were based on these figures. The total amount of each active ingredient used is included in these tables in order to provide a means by which the amount of each active ingredient used in each of the districts can be compared. These numbers are not intended to be used as absolute amounts of active ingredient used, because they are based on actual and estimated usage.

The target vegetation is listed in these tables as they were listed on the application records. Therefore, some categories are very general, such as grass and broadleaf weeds. These categories are not more specific because the information was not available.

Occasionally, categories for the active ingredients are listed as "Unknown." This means that there was an application record for the use of this active ingredient, but there was not sufficient data on the application record to extract the data. This occurs for dichlobenil, oryzalin and sethoxydim. In two of the active ingredients (fluazifop-butyl and oxadiazon) they are listed as not having been used by WSDOT. This means that WSDOT plans to use these in the future and they may have used them in the past, but they were not used in the time period from July, 1989 to June of 1990; therefore, there was no basis for estimating application rates or total amounts used.

Table 5-1 Active ingredients and their formulated products that will be used by WSDOT for roadside vegetation managment. Included are the signal words and the toxicity catagories assigned to each formulation and a reason for assignment of signal words (Danger or Warning).

COMPOUND	PRODUCT	SIGNAL WORD	USEPA TOXICITY CATEGORY	REASON
2,4-D	Amine#4 - (46.8% AI,38.9% AE), Dimethylamine salt	Danger	I	Eye irritant
	Envy - (58.1% AI, 42.8% AE), Triethylamine salt	Danger	I	Eye irritant
	Formula 40 - (56.5 % AI, 36.6% AE), Alkanoamine salt	Danger	I	Eye irritant
	Trimec Classic - (25.9% AI, 21.5% AE), Dimethylamine salt	Danger	I	Eye irritant
	Visko-Rhap A-3D - (61.1% AI, 33.9% AE), N-alkylamine salt	Caution	III	*
	Visko-Rhap LV2D - (35.6% AI, 23.6% AE), 2-ethylhexyl ester	Caution	III	*
	Weedar 64 - (46.8% AI, 38.9% AE), Dimethylamine salt	Danger	I	Eye irritant
	Weedone LV4 - (60.8% AI, 41.9% AE), Butoxyethyl ester	Caution	III	*
	Weedone 170 - (29.9% AI, 20.6% AE), Butoxyethanol ester	Caution	III	*
	Weedmaster - (35.7% AI, 29.6% AE), Dimethylamine salt	Caution	III	*
	Crossbow - (34.4% AI,23.7% AE), Butoxyethyl ester	Caution	III	*
	Tordon RTU - (20.9% AI, 11.2% AE), Triisopropylamine salt	Warning	II	Eye irritant
	Tordon 101 Mixture (39.6% AI, 21.2% AE), Triisopropylamine salt	Warning	II	Eye irritant
2,4-DP	Weedone 170 - (29.3% AI, 20.6% AE), Butoxyethanol ester	Caution	III	*
Benefin	XL-2G - (1.0% AI)	Caution	III	*
Bromacil	Krovar 1DF - (40% AI)	Caution	III	*
Chlorsulfuron	Telar (dispersible granule) - (75% AI)	Caution	III	*
Chlorthal Dimethyl	Dacthal 75W - (75% AI)	Caution	III	*

* - NOTE: Reasons were only given for "Danger" and "Warning" signal words since chemicals with "Caution" as a signal word are less serious irritants.

COMPOUND	PRODUCT	SIGNAL WORD	USEPA TOXICITY CATEGORY	REASON
Dicamba	Banvel 4WS - (48.2% AI, 40% AE)	Warning	II	Eye irritant
	Weedmaster - (15.5% AI, 10.3% AE)	Caution	III	*
	Trimec - (2.8% AI, 2.3% AE), Dimethylamine salt	Danger	I	Eye irritant
Dichlobenil	Casoron 4G - (4% AI)	Caution	III	*
	Casoron 50W - (50 % AI)	Caution	III	*
	Norosac 4G - (4% AI)	Caution	III	*
Diuron	Karmex DF - (80% AI)	Warning	II	Eye irritant
	Direx 4L - (40% AI)	Caution	III	*
	Krovar 1DF - (40% AI)	Caution	III	*
Fluazifop-Butyl	Fusilade 2000 - (13% AI)	Caution	III	*
Fosamine	Krenite S - (41.5% AI), Ammonium salt	Warning	II	Eye irritant
Glyphosate	Roundup - (41% AI), Isopropylamine salt	Warning	II	Eye irritant
	Rodeo - (53.8% AI), Isopropylamine salt	Caution	III	*
Imagzapyr	Arsenal - (27.6% AI, 22.6% AE), Isopropylamine salt	Caution	III	*
Isoxaben	Gallery - (75% AI)	Caution	III	*
Mecoprop	Trimec - (13.9 AI, 11.5% AE), Dimethylamine salt	Danger	I	Eye irritant
Mefluidide	Embark 2-S (28.0% AI, 21% AE), Diethanolamine salt	Caution	III	*
Metsulfuron Methyl	Escort - (60% AI)	Caution	III	*
Napropamide	Devrinol - (50% AI)	Caution	III	*
Oryzalin	XL-2G - (1.0% AI)	Caution	III	*
	Surflan - (40.4% AI)	Caution	III	*
Oxadiazon	Ronstar G - (2.0% AI)	Warning	II	Eye irritant
	Ronstar 50WP - (50% AI)	Warning	II	Eye irritant

* - NOTE: Reasons were only given for "Danger" and "Warning" signal words since chemicals with "Caution" as a signal word are less serious irritants.

COMPOUND	PRODUCT	SIGNAL WORD	USEPA TOXICITY CATEGORY	REASON
Picloram	Tordon 101R - (10.2% AI, 5.7% AE), Triisopropylamine salt.	Warning	II	Eye irritant
	Tordon 22K - (24.4% AI, 21.1% AE), Potassium salt	Warning	II	*
	Tordon RTU - (5.4% AI, 3.0% AE), Triisopropylamine salt	Warning	II	Eye irritant
	Tordon K - (24.4% AI, 21.1% AE), Potassium salt	Warning	II	Eye irritant
Pronamide	Kerb 50W - (50% AI)	Caution	III	*
Sethoxydim	Poast - (18% AI)	Warning	II	Eye irritant
Sulfometuron methyl	Oust - (75% AI)	Caution	III	*
Triclopyr	Garlon 3A - (44.4% AI, 31.8% AE), Triethylamine salt	Danger	I	Eye irritant
	Garlon 4 - (61.6% AI, 44.3% AE), Butoxyethyl ester	Caution	III	*
	Crossbow - (16.5% AI, 11.9% AE), Butoxyethyl ester	Caution	III	*

* - NOTE: Reasons were only given for "Danger" and "Warning" signal words since chemicals with "Caution" as a signal word are less serious irritants.

Table 5-2 Active ingredients and their formulated products that will be used by WSDOT for roadside vegetation managment. Included are the amounts of active ingredient in each formulation and the maximum label rate for each of the products. Also listed are inerts for each product that are on either EPA's list 1 or 2.

COMPOUND	PRODUCT	FORMULATION	MAXIMUM RATE	INERTS OF CONCERN
2,4-D	Amine#4 - (46.8% AI,38.9% AE), Dimethylamine salt	3.8 lbs/gal	2.0 gal/acre	None
	Envy - (58.1% AI, 42.8% AE), Triethylamine salt	3.8 lbs/gal	1.0 gal/acre	None
	Formula 40 - (56.5 % AI, 36.6% AE), Alkanoamine salt	3.8 lbs/gal	0.75 gal/acre	None
	Trimec Classic - (25.9% AI, 21.5% AE), Dimethylamine salt	2.03 lbs/gal	0.5 gal/acre	None
	Visko-Rhap A-3D - (61.1% AI, 33.9% AE), N-alkylamine salt	2.87 lbs/gal	1.0 gal/acre	Xylene
	Visko-Rhap LV2D - (35.6% AI, 23.6% AE), 2-ethylhexyl ester	1.92 lbs/gal	3.0 gal/acre	Xylene
	Weedar 64 - (46.8% AI, 38.9% AE), Dimethylamine salt	3.8 lbs/gal	1.0 gal/acre	None
	Weedone LV4 - (60.8% AI, 41.9% AE), Butoxyethyl ester	3.8 lbs/gal	2.5 gal/acre	Petroleum Distillates
	Weedone 170 - (29.9% AI, 20.6% AE), Butoxyethanol ester	1.85 lbs/gal	9.0 gal/acre	Petroleum Distillates
	Weedmaster - (35.7% AI, 29.6% AE), Dimethylamine salt	2.87 lbs/gal	0.5 gal/acre	None
	Crossbow - (34.4% AI,23.7% AE), Butoxyethyl ester	2.0 lbs/gal	4.0 gal/acre	Kerosene
	Tordon RTU - (20.9% AI, 11.2% AE), Triisopropylamine salt	N/A	Use undilluted	None
	Tordon 101 Mixture (39.6% AI, 21.2% AE), Triisopropylamine salt	2.0 lbs/gal	4.0 gal/acre	None
2,4-DP	Weedone 170 - (29.3% AI, 20.6% AE), Butoxyethanol ester	1.85 lbs/gal	9.0 gal/acre	None
Benefin	XL-2G - (1.0% AI)	0.5 lbs/50 lb bag	300.0 lbs/acre	None
Bromacil	Krovar 1DF - (40% AI)	20.0 lbs/50 lb bag	30.0 lbs/acre	None
Chlorsulfuron	Telar (dispersible granule) - (75% AI)	0.75 lbs/1 lb	3.0 oz/acre	None
Chlorthal Dimethyl	Dacthal 75W - (75% AI)	0.75 lbs/1 lb	20.0 lbs/acre	None

COMPOUND	PRODUCT	FORMULATION	MAXIMUM RATE	INERTS OF CONCERN
Dicamba	Banvel 4WS - (48.2% AI, 40% AE)	4.0 lbs/gal	2.0 gal/acre	None
	Weedmaster - (15.5% AI, 10.3% AE)	1.0 lb/gal	0.5 gal/acre	None
	Trimec - (2.8% AI, 2.3% AE), Dimethylamine salt	0.21 lbs/gal	0.5 gal/acre	None
Dichlobenil	Casoron 4G - (4% AI)	0.04 lbs/lb	200.0 lbs/acre	None
	Casoron 50W - (50 % AI)	0.5 lbs/lb	12.0 lbs/acre	None
	Norosac 4G - (4% AI)	0.04 lbs/lb	500.0 lbs/acre	None
Diuron	Karmex DF - (80% AI)	40.0 lbs/50 lb bag	15.0 lbs/acre	None
	Direx 4L - (40% AI)	4.0 lbs/gal	10.0 gal/acre	None
	Krovar 1DF - (40% AI)	20.0 lbs/50 lb bag	30.0 lbs/acre	None
Fluazifop-butyl	Fusilade 2000 - (13% AI)	1.0 lb/gal	48.0 oz/acre	None
Fosamine	Krenite S - (41.5% AI), Ammonium salt	4.0 lbs/gal	6.0 gal/acre	None
Glyphosate	Roundup - (41% AI), Isopropylamine salt	4.0 lbs/gal	2.65 gal/acre	None
	Rodeo - (53.8% AI), Isopropylamine salt	5.4 lbs/gal	7.5 pts/acre	None
Imazapyr	Arsenal - (27.6% AI, 22.6% AE), Isopropylamine salt	2.0 lbs/gal	6.0 pts/acre	None
Isoxaben	Gallery - (75% AI)	0.75 lbs/lb	1.33 lbs/acre	None
Mecoprop	Trimec - (13.9 AI, 11.5% AE), Dimethylamine salt	1.08 lbs/gal	0.5 gal/acre	None
Mefluidide	Embark 2-S (28.0% AI, 21% AE), Diethanolamine salt	2.0 lbs/gal	0.5 gal/acre	None
Metsulfuron Methyl	Escort - (60% AI)	0.6 lbs/lb	4.0 oz/acre	None
Napropamide	Devrinol - (50% AI)	0.5 lbs/lb	8.0 lbs/acre	None
Oryzalin	XL-2G - (1.0% AI)	0.5 lb/50 lb bag	300.0 lbs/acre	None
	Surflan - (40.4% AI)	4.0 lbs/gal	1.5 gal/acre	None
Oxadiazon	Ronstar G - (2.0% AI)	0.02 lbs/lb	200.0 lbs/acre	None
	Ronstar 50WP - (50% AI)	0.5 lbs/lb	8.0 lbs/acre	None

COMPOUND	PRODUCT	FORMULATION	MAXIMUM RATE	INERTS OF CONCERN
Picloram	Tordon 101R - (10.2% AI, 5.7% AE), Triisopropylamine salt.	0.54 lbs/gal	4.0 gal/acre	None
	Tordon 22K - (24.4% AI, 21.1% AE), Potassium salt	2.0 lbs/gal	0.5 gal/acre	None
	Tordon RTU - (5.4% AI, 3.0% AE), Triisopropylamine salt	N/A	Use undilluted	None
	Tordon K - (24.4% AI, 21.1% AE), Potassium salt	2.0 lbs/gal	1.0 gal/acre	None
Pronamide	Kerb 50W - (50% AI)	0.5 lbs/lb	8.0 lb/acre	None
Sethoxydim	Poast - (18% AI)	1.5 lbs/gal	2.5 pts/acre	Petroleum Distillates
Sulfometuron methyl	Oust - (75% AI)	0.75 lbs/lb	12 oz/acre	None
Triclopyr	Garlon 3A - (44.4% AI, 31.8% AE), Triethylamine salt	3.0 lbs/gal	3.0 gal/acre	
	Garlon 4 - (61.6% AI, 44.3% AE), Butoxyethyl ester	4.0 lbs/gal	2.0 gal/acre	Kerosene
	Crossbow - (16.5% AI, 11.9% AE), Butoxyethyl ester	1.0 lbs/gal	4.0 gal/acre	Kerosene

Table 5-3 Formulated products, manufacturers for the active ingredients and physical data that is relevant to conducting an environmetal risk assessment.

	2,4-D	2,4-DP
FORMULATED PRODUCTS	Amine #4; Envy; Formula 40; Trimec; Visko-Rhap; Weedar; Weedone; Weedmaster; Crossbow.	Weedone (170)
MANUFACTUER	Wilbur Ellis; Lilly Miller; Rhone-Poulenc PBI Gordon; Agrolinz; Sandoz; Dow/Elanco	Rhone-Poulenc
CHEMICAL DATA		
CHEMICAL NAME	CA and Manufacturers: (2,4-Dichlorophenoxy) acetic acid	CA and Rhone-Poulenc: 2-(2,4-Dichlorophenoxy) propanoic acid
SIGNAL WORD/ TOXICITY CLASS	Danger/I (Dimethylamine salt) (106) Caution/III,IV (EC) (106)	Caution/III (106)
MOLECULAR FORMULA	$C_8H_6Cl_2O_3$ (274)	$C_9H_8Cl_2O_3$ (189)
MOLECULAR WEIGHT	221.04 (274)	235.05 (189)
CAS NUMBER	94-75-7 (106)	120-36-5 (106)
SOLUBILITY (25°C)	Water: 900 ppm (162) Water: 620 mg/l (297) Water: Amine salts are soluble. (189) Oils: esters are soluble. (106) Alcohols: soluble (106)	Water: 825 ppm (room temp) (106) 350 ppm @ 20°C (189,87) Organic solvents: Readily soluble (189,87)
MELTING POINT	135-142°C (98)	116-118°C (98)
VAPOR PRESSURE	$< 10^{-7}$ mbar (106) 8×10^{-6} Torr (101) 0.4 mmHg @ 106°C (98)	0.45 mPa @ 20°C (117) Negligible @ 20°C (98)
DISSOCIATION CONSTANT	pKa 2.64 (297)	DNF
HENRY'S LAW CONSTANT	2.59×10^{-10} (101)	DNF
Log K_{ow}	1.6 (162) 0.27 (10)	-1.59 @ pH 7 and 1.77 as undissociated acid (155)
PH	DNF	5.1 (10% suspension in isopropanol (155) 4.6 (16 ppm solution) (155)
STABILITY	Stable up to 3 years (286) Stable up to melting point (98)	Acid is very stable (117) Stable @ pH 5, 7 and 9 for up to 8 days @ 70°C (155)
CORROSIVENESS	Non-corrosive (286)	Corrosive (189)

	BENEFIN	BROMACIL
FORMULATED PRODUCTS	XL-2G	Krovar
MANUFACTUER	Dow/Elanco	Dupont
CHEMICAL DATA		
CHEMICAL NAME	CA: N-Butyl-N-ethyl-2,6-dinitro-4-(trifluoromethyl)benzenamine Dow/Elanco: N-Butyl-N-ethyl-a,a,a-trifluroro-2,6-dinitro-p-toluidine	CA and DuPont: 5-Bromo-3-sec-butyl-6-methyluracil
SIGNAL WORD/ TOXICITY CLASS	Caution/IV (106)	Caution/III (106)
MOLECULAR FORMULA	$C_{13}H_{16}F_3N_3O_4$ (189)	$C_6H_{13}BrN_2O_2$ (189)
MOLECULAR WEIGHT	335.29 (189)	261.11 (189)
CAS NUMBER	1861-40-1 (106)	314-40-9 (106)
SOLUBILITY (25°C)	Water: 0.1 ppm @ 25°C (187) Acetone: 650 g/l @ 25°C (117) Ethanol: 24 g/l @ 25°C (297)	Water: 8.5 ppm (162) Water: 815 ppm @ 20°C (189) Acetone: moderately soluble, 201 g/kg (189,297) Ethanol: 155 g/kg (297) Xylene 33 g/kg (297)
MELTING POINT	65-66.5°C (297,95)	158-159°C (297)
VAPOR PRESSURE	6.6×10^{-5} mmHg @ 25°C (187) 3.7 mPa @ 25°C (117)	8×10^{-4} mmHg @ 100°C (189) 3.1×10^{-3} mmHg @ 25°C (287)
DISSOCIATION CONSTANT	Does not dissociate (187)	pKa = 9.1 (287)
HENRY'S LAW CONSTANT	3.44×10^{-10} (101)	3.37×10^{-10} (101)
Log K_{ow}	1.95×10^5 (297) 5.3 @ 20°C (297,162)	Mean valve = 75.6 (287) 2.02 (101)
PH	Does not disperse in water (187)	DNF
STABILITY	Relatively stable (187)	Stable up to the melting point (287)
CORROSIVENESS	Non-corrosive (187,117)	Non-corrosive (287,117)

	CHLORSULFURON	CHLORTHAL DIMETHYL
FORMULATED PRODUCTS	Telar	Dacthal
MANUFACTUER	DuPont	Fermenta
CHEMICAL DATA		
CHEMICAL NAME	CA and DuPont: 2-Chloro-N[(4-methoxy-6-methyl-1,3,5 triazin-2-yl) amino carbonyl] benzenesulfonamide	CA and Fermenta: Dimethyl tetrachloroterephthalate
SIGNAL WORD/ TOXICITY CLASS	Caution/IV (106)	Caution/IV (106)
MOLECULAR FORMULA	$C_{12}H_{12}ClN_5O_4S$ (189)	$C_{10}H_6ClO_4$ (189)
MOLECULAR WEIGHT	357.78 (189)	331.99 (189)
CAS NUMBER	64902-72-3 (106)	1861-32-1 (106)
SOLUBILITY (25°C)	Water: 27.9 g/l (pH7 @ 25°C) (297) Water: 587 ppm (pH5 @ 25°C) (70) Hydrocarbon solvents: low solubility (106) Acetone, Acetonitrile: moderate (106)	Water: <0.5 ppm (106) Acetone: 100 g/kg (297) Benzene: 250 g/kg (297)
MELTING POINT	174-178°C (297)	156°C (297)
VAPOR PRESSURE	2.3×10^{-11} mmHg (70)	<67 Pa @ 40°C (117) 2.5×10^{-6} (101)
DISSOCIATION CONSTANT	pKa = 3.6 (70,297)	DNF
HENRY'S LAW CONSTANT	1.73×10^{-8} (101)	2.18×10^{-6} (101)
Log K_{ow}	pH5 @ 25°C = 2.13 (70)	3.83 (101)
PH	3.5 (70,297)	7-8 (107)
STABILITY	Stable (70,117)	Stable to heat and CV light (117)
CORROSIVENESS	Non-corrosive (70,117)	DNF

	DICAMBA	DICHLOBENIL
FORMULATED PRODUCTS	Banvel; Weedmaster; Trimec	Casoron; Norosac
MANUFACTUER	Sandoz; PBI Gordon	Uniroyal; PBI Gordon
CHEMICAL DATA		
CHEMICAL NAME	Sandoz: 2-methoxy-3,6-dichlorobenzoic acid IUPAC: 3,6-dichloro-o-ansinic acid CA: 3,6-dichloro-2-methoxybenzoic acid	CA and Manufacturers: 2,6-dichlorobenzonitrile IUPAC: 2,6-dichlorobenzonitrile
SIGNAL WORD/ TOXICITY CLASS	Warning/III((106)	Caution/III (106)
MOLECULAR FORMULA	$C_8H_6Cl_2O_3$ (257)	$C_7H_3Cl_2N$ (189)
MOLECULAR WEIGHT	221.0 (257)	172.02 (189)
CAS NUMBER	1918-00-9 (189)	1194-65-6 (106)
SOLUBILITY (25°C)	Water: 4.5-6.5 g/l (257) Ethanol: 922 g/l (257) Acetone: 810 g/l (257)	Water: 25 ppm @ 20°C (270,89) Xylene: 53 g/l @ 25°C (55) Ethanol: 15 g/l @ 25°C (55) Cycloheyane: 3.7 g/l @ 25°C (55)
MELTING POINT	114-116°C (297)	139-145°C (297)
VAPOR PRESSURE	9.24×10^{-6} mmHg (115) 3.4×10^{-5} mmHg (257)	0.135 Pa @ 25°C (152) 5.5×10^{-4} @ 25°C (270) 1.6×10^{-2} @ 50°C (270)
DISSOCIATION CONSTANT	1.16×10^{-2} (257)	Data Not Applicable (81)
HENRY'S LAW CONSTANT	2.3×10^{-6} (101)	2.3×10^{-6} (101)
Log K_{ow}	3.5 (257) 2.46 (101)	2.7 (117,162,269) 3.6 (89) 1.64 (101)
PH	2.5 - 3.0 (257)	6.2 (55)
STABILITY	Stable in acids & alkalis; Decomposses at about 200°C. Acidic in reaction, forming water-soluble salts. (115)	No appreciable deterioration if stored in a tight container in a dry place. (270) Stable in acids (81)
CORROSIVENESS	Slight (257)	Non-corrosive (269,81)

	DIURON	FLUAZIFOP-BUTYL
FORMULATED PRODUCTS	Karmex; Krovar; Direx	Fusilade 2000
MANUFACTUER	DuPont; Griffin	ICI
CHEMICAL DATA		
CHEMICAL NAME	CA: N-(3,4-dichlorophenyl)-N,N-dimethyl urea Manufacturers: [3-(3,4-dichlorophenyl)-1,1-dimethylurea	CA: Butyl (R)2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy]propanoate
SIGNAL WORD/ TOXICITY CLASS	Caution/III (106)	Caution/IV (106)
MOLECULAR FORMULA	$C_9H_{10}Cl_2N_2O$ (189)	$C_{18}H_{20}F_3NO_4$ (189)
MOLECULAR WEIGHT	233.10 (189)	383.38 (189)
CAS NUMBER	330-54-1 (106)	69806-50-4 (106)
SOLUBILITY (25°C)	Water: 42 mg/l @ 25°C (297,162) Water: 0.035 g/l @ 20°C (106) Hydrocarbon solvents: Very low (189) Acetone: 53 g/kg @ 27°C (297)	Soluble in most organic solvents (106) water: 1 mg/l @ 20°C (297)
MELTING POINT	158-159°C (297)	13°C (297)
VAPOR PRESSURE	2.3×10^{-9} mmHg @ 20°C (106) 3.1×10^{-6} mmHg @ 50°C (189)	2.5×10^{-7} mmHg @ 20°C (165) 4.12×10^{-6} mmHg @ 40°C (165)
DISSOCIATION CONSTANT	Does not ionize (287)	DNF
HENRY'S LAW CONSTANT	2.26×10^{-8} (101)	1.39×10^{-5} (101)
Log K_{ow}	0.747 (287) 1.97 (162) 2.81 (101)	4.5 (162,165)
PH	DNF	DNF
STABILITY	Stable at normal temperatures and conditions. (79)	Rapidly hydrolyzed in alkaline Stable under normal conditions. (165)
CORROSIVENESS	Non-corrosive (287)	Non-corrosive (117)

	FOSAMINE	GLYPHOSATE
FORMULATED PRODUCTS	Krenite S	Rodeo; Roundup
MANUFACTUER	DuPont	Monsanto
CHEMICAL DATA		
CHEMICAL NAME	CA: Ammonium ethyl carbamoylphosphonate DuPont: [ethyl hydrogen (aminocarbonyl) phosphonate]	Monsanto, IUPAC and CA: N-(phosphonomethyl)glycine
SIGNAL WORD/ TOXICITY CLASS	Warning/IV (106)	Warning/II (Roundup) (106) Caution/III (Rodeo) (106)
MOLECULAR FORMULA	$C_3H_{11}N_2O_3P$ (189)	$C_3H_8NO_5P$ (106)
MOLECULAR WEIGHT	154.07 (189)	169.08 (189)
CAS NUMBER	25954-13-6 (106)	1071-83-6 (106)
SOLUBILITY (25°C)	Water: 179 g/100 g (106) Methanol: 15.8 g/100 g (106)	Water: 12 g/l (189,162) Insoluble in common organic solvents (189)
MELTING POINT	175°C (297)	200°C (297,95)
VAPOR PRESSURE	Negligible, 4 X 10^{-8} @ 25°C (109)	Negligible (155)
DISSOCIATION CONSTANT	pKa = 9.25 (109)	pKa 1 = 2.6 (8) pKa 2 = 5.6 (8) pKa 3 = 10.6 (8)
HENRY'S LAW CONSTANT	4.51 X 10^{-13} (101)	2.00 X 10^{-11} (101)
Log K_{ow}	-2.9 @ 25°C (109)	-3.8 @ 20 ppm (162) -4.6 (297)
PH	5.8 @ 25°C (1% soln. by wt.) (109)	Glyphosate (1% solution in water) = 2.5 (8) Roundup Herbicide = 4.4 - 4.9 (8)
STABILITY	Stable in neutral or alkaline solutions. (117)	Indefinite (8)
CORROSIVENESS	Corrosive to copper and brass (117) Not corrosive (109)	Corrosive to iron and galvanized metal (95)

	IMAZAPYR	ISOXABEN
FORMULATED PRODUCTS	Arsenal	Gallery
MANUFACTUER	American Cyanamid	Dow/Elanco
CHEMICAL DATA		
CHEMICAL NAME	IUPAC: 2-(4-isopropyl-4-methyl-5-oxo-2-imidazolin-2-yl)nicotinic acid. American Cyanamid and CA: 2-(4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl)-3-pyridinecarboxylic acid.	CA and Dow/Elanco: N-[3-(1-ethyl-1-methylpropyl)-5-isoxazolyl]-2,6-dimethoxybenzamide IUPAC: N-[3-(1-ethyl-1-Methylpropyl) isoxazol-5-yl]-2,6-dimethoxybenzamide
SIGNAL WORD/ TOXICITY CLASS	Caution/III (106)	Caution/III (106)
MOLECULAR FORMULA	$C_{13}H_{15}N_3O_3$ (3)	$C_{18}H_{24}N_2O_4$ (106)
MOLECULAR WEIGHT	261.3 (3)	332.40 (189)
CAS NUMBER	81334-34-1 (106)	82558-53-7 (156)
SOLUBILITY (25°C)	Water: 1.0 - 1.5% @ 25°C (3) Acetone: 11 g/l (297) Acetone: 6 g/l (297)	Water: .001-.002 mg/ml (189) Water: 1-2 mg/l (297) Methanol: 50-100 mg/ml (189) Methanol: 70-80 mg/l (297) Hexane: 0.07-0.08 mg/ml (189)
MELTING POINT	128-130°C (297)	176-179°C (297)
VAPOR PRESSURE	<0.03 mPa @ 45°C (177) (2.0×10^{-7} mmHg) (177)	< 3.9×10^{-7} mmHg @ 30°C (189)
DISSOCIATION CONSTANT	1.9 and 3.6 (177)	434 (pH 7, 25°C) (297)
HENRY'S LAW CONSTANT	6.8×10^{-15} (101)	6×10^{-7} to 9×10^{-8} (155)
Log K_{ow}	0.11 @ 22°C (3,101)	Low (75) 2.64 (136,297)
PH	3.0-3.5 as 1% water solution @ 25°C (3)	(Aqueous 50/50) 6.5 to 7.5 (156)
STABILITY	Stable at least 18 months at 25°C (3)	Stable to hydrolysis at pH 5-9 (117)
CORROSIVENESS	Non-corrosive to packaging material. (177)	Non-corrosive (156)

	MECOPROP	MEFLUIDIDE
FORMULATED PRODUCTS	Trimec	Embark
MANUFACTUER	PBI Gordon	PBI Gordon
CHEMICAL DATA		
CHEMICAL NAME	CA: 2-(4-chloro-2-methylphenoxy)-propionic acid	CA: N-[2,4-Dimethyl-5-[[(trifluoromethyl)-sulfonyl]amino]phenyl]acetamide
SIGNAL WORD/ TOXICITY CLASS	Caution/III (106)	Caution/III (106)
MOLECULAR FORMULA	$C_{10}H_{11}ClO_3$ (189)	$C_{11}H_{13}F_3N_2O_3S$ (189)
MOLECULAR WEIGHT	214.65 (189)	310.29 (189)
CAS NUMBER	7085-19-0 (106)	53780-34-0 (106)
SOLUBILITY (25°C)	Water: 620 ppm @ 20°C (106) Organic solvents: soluble (106)	Water: 0.18 g/l @ 25°C (189) Acetone: 350 g/l @ 25°C (189) Methanol: 310 g/l @ 25°C (189)
MELTING POINT	94-95°C (297)	183-185°C (297)
VAPOR PRESSURE	1.4×10^{-4} mmHg @ 20°C (94) 0 - 30 ± 0.05 pa @ 25°C (176)	< 10^{-4} mmHg @ 25°C (189)
DISSOCIATION CONSTANT	pKa = 3.78 (20-25°C) (297) 3.07 (176)	pka = 4.6 (255)
HENRY'S LAW CONSTANT	2.28×10^{-9} (101)	< 2.3×10^{-8} (155)
Log K_{ow}	1.26 (pH 7) (297) -7 (101)	1.98 (255)
PH	>8.6 (117)	7.5 - 8.5 (Diethanolamine salt)(236,235)
STABILITY	Good at ambient temperature. (176)	Not hydrolyzed at pH 5,7 and 9 at ambient temperatures. (255)
CORROSIVENESS	Non-corrosive (176) Corrosive to metals (117)	Mildly corrosive (189,255)

	METSULFURON METHYL	NAPROPAMIDE
FORMULATED PRODUCTS	Escort	Devrinol
MANUFACTUER	DuPont	ICI
CHEMICAL DATA		
CHEMICAL NAME	CA and DuPont: 2-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)-amino]carbonyl]amino]sulfonyl]benzoate	CA: 2-(a-Naphoxy)-N,N-diethylpropionamide ICI: N,N-Diethyl-2-(1-naphthalenyloxy)-propionamide
SIGNAL WORD/ TOXICITY CLASS	Caution/IV (106)	Caution/III (106)
MOLECULAR FORMULA	$C_{14}H_{15}N_5O_6S$ (189)	$C_{17}H_{21}NO_2$ (189)
MOLECULAR WEIGHT	381.36 (189)	271.37 (189)
CAS NUMBER	74223-64-6 (106)	15299-99-7 (106)
SOLUBILITY (25°C)	Water: 0.27 g/l @ pH 4.6 (189,86) Water: 9.5 g/l @ pH 6.1 (189,86) Ethanol: 2.3 g/l (297) Xylene: 580 g/l (297)	Water: 70 ppm @ 20°C (189,165) Xylene: 500 g/l (297)
MELTING POINT	150°C (297)	68-70°C (297)
VAPOR PRESSURE	5.8×10^{-5} mmHg @ 25°C (189,86) 2.5×10^{-12} mmHg @ 25°C (70)	0.53 m Pa @ 25°C (117) 4.0×10^{-6} mmHg (155)
DISSOCIATION CONSTANT	pKa 3.3 (70,297)	DNF
HENRY'S LAW CONSTANT	3.06×10^{-9} (101)	1.96×10^{-8} (101)
Log K_{ow}	-1.74 (101) -1.85 (297)	3.4 @ 25°C (117) 3.3 (165)
PH	acidic (297) 5.0 (1% slurry in water) (70)	8.2 - 8.9 (1%) solution (165)
STABILITY	Stable in air up to 140°C, in neutral and alkaline soils @ 25°C (70,117)	Stable under normal conditions (165)
CORROSIVENESS	Non-corrosive (70)	Non-corrosive (117,165)

	ORYZALIN	OXADIAZON
FORMULATED PRODUCTS	XL-2G; Surflan	Ronstar
MANUFACTUER	Dow/Elanco	Rhone-Poulenc
CHEMICAL DATA		
CHEMICAL NAME	CA and Dow/Elanco: 3,5-Dinitro-N_4,N_4-dipropylsulfanilamide	CA: 2-Tert-butyl-4-(2,4-dichloro-5-isopropoxyphenyl)-d^2-1,3,4-oxadiazolin-5-one
SIGNAL WORD/ TOXICITY CLASS	Caution/IV (106)	Danger (EC)/IV (106) Warning (G, WP)/IV (106)
MOLECULAR FORMULA	$C_{12}H_{18}N_4O_6S$ (189)	$C_{15}H_{18}Cl_2N_2O_3$ (189)
MOLECULAR WEIGHT	346.36 (189)	345.22 (189)
CAS NUMBER	19044-88-3 (106)	19666-30-9 (106)
SOLUBILITY (25°C)	Water: 2.5 ppm @ 25°C (74,106) Polar organic solvents: readily soluble (74,106) Hexane: insoluble (74,106)	Water: 0.7 mg/l @ 20°C (189) Ethanol: 100 g/l @ 20°C (189) Acetone: 600 g/l @ 20°C (189)
MELTING POINT	141-142°C (297)	90°C (297)
VAPOR PRESSURE	$< 1 \times 10^{-7}$ mmHg @ 30°C (189)	$< 1 \times 10^{-8}$ mmHg @ 20°C (189)
DISSOCIATION CONSTANT	pKa 9.4 (57, 297)	DNF
HENRY'S LAW CONSTANT	1.82×10^{-8} (101)	4.54×10^{-7} (101)
Log K_{ow}	3.7 (57,297)	4.7 @ 25°C (191) 4.8 (297) 8.13 (101)
PH	6.3 - 6.6 (50%) (57)	DNF
STABILITY	Stable for 36 months @ 25°C (57)	Stable under normal storage conditions (117) Stable (191)
CORROSIVENESS	Non-corrosive (57,117)	Non-corrosive (117)

	PICLORAM	PRONAMIDE
FORMULATED PRODUCTS	Tordon	Kerb
MANUFACTUER	Dow/Elanco	Rohm and Haas
CHEMICAL DATA		
CHEMICAL NAME	CA and Dow/Elanco: 4-amino-3,5,6-trichloropicolinic acid	CA and Rohm and Haas: 3,5-Dichloro-N-(1,1-dimethyl-2-proponyl) benzamide
SIGNAL WORD/ TOXICITY CLASS	Caution-Warning/IV (106)	Caution/IV (106)
MOLECULAR FORMULA	$C_6H_3Cl_3N_2O_2$ (189)	$C_{12}H_{11}Cl_2NO$ (189)
MOLECULAR WEIGHT	241.48 (189)	256.13 (189)
CAS NUMBER	1918-02-1 (106)	23950-58-5 (106)
SOLUBILITY (25°C)	Water: 430 mg/l @ 25°C (189) Acetone: 19.8 g/l @ 25°C (189) Ethanol: 10.5 g/l (91)	Water: 15 ppm @ 25°C (189,162) (low water solubility) Aliphatic and aromatic solvents: soluble (189)
MELTING POINT	Decomposes @ 215°C w/o melting (297)	155-156°C (297)
VAPOR PRESSURE	6.16×10^{-7} mmHg @ 35°C (189)	8.5×10^{-5} mmHg @ 25°C (189,87)
DISSOCIATION CONSTANT	2.3 (159)	DNF
HENRY'S LAW CONSTANT	4.58×10^{-10} (101)	1.91×10^{-8} (101)
Log K_{ow}	5.90 (+ or - 0.06) (159) 0.25 (101)	3.1-3.3 (297)
PH	DNF	DNF
STABILITY	Protect from freezing and store <50°C (159)	Hydrolyzed by strong acids and alkalis (117) Relatively stable (87)
CORROSIVENESS	TIPA and salt not corrosive to stainless steel (159)	Non-corrosive (117)

	SETHOXYDIM	SULFOMETURON METHYL
FORMULATED PRODUCTS	Poast	Oust
MANUFACTUER	BASF	DuPont
CHEMICAL DATA		
CHEMICAL NAME	CA and BASF: 2[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one	IUPAC: methyl 2-(3(-4,6-dimethylpyrimidin-2-yl)ureidosulphonyl)benzoate Dupont and CA: Methyl 2-(((((4,6-dimethyl-2-pyrimidinyl)amino)carbonyl)amion)sulfonyl) benzoate.
SIGNAL WORD/ TOXICITY CLASS	Warning/III (106)	Caution/III (106)
MOLECULAR FORMULA	$C_{17}H_{29}NO_3S$ (189)	$C_{15}H_{16}N_4O_5S$ (189)
MOLECULAR WEIGHT	327.86 (189)	364.4 (189)
CAS NUMBER	74051-80-2 (106)	74222-97-2 (106)
SOLUBILITY (25°C)	Water: 4,700 ppm @ pH 7 (106) Organic solvents: readily soluble (106) Methanol/Hexane/Xylene: soluble (106)	Water: 8 mg/l @ pH5 (297) Water: 70 mg/kg @ pH7 (297) Ethanol: 137 mg/kg (297) 70 ppm @ 20°C (101)
MELTING POINT	>90°C (boiling point) (297)	203-205°C (297)
VAPOR PRESSURE	<0.1 mPa @ 20°C (25) (117) <7.5 X 10^{-6} mmHg (155)	DNF
DISSOCIATION CONSTANT	pKa = 4.16 @ 25°C (25)	pka = 5.2 (297)
HENRY'S LAW CONSTANT	1.44 X 10^{-9} (101)	3.84 X 10^{-7} (101)
Log K_{ow}	45.1 @ pH7 (25) 4.51 @ pH5 (297)	7.0 (77) 0.84 (101)
PH	3.95 @ 20°C (24)	DNF
STABILITY	Stable to metal @ 24°C (24) Unstable to sunlight and at elevated temperatures. (24) Stable under normal storage conditions (117)	Stable at normal temperatures and storage conditions (78)
CORROSIVENESS	Non-corrosive (117, 24)	DNF

	TRICLOPYR
FORMULATED PRODUCTS	Garlon
MANUFACTUER	Dow/Elanco
CHEMICAL DATA	
CHEMICAL NAME	Dow, IUPAC: 3,5,6-trichloro-2-pyridyloxyacetic acid CA: ((3,5,6-trichloro-2-pyridinyl)oxy)acetic acid
SIGNAL WORD/ TOXICITY CLASS	Danger/I (Garlon 3A) (106) Caution/III (Garlon 4) (106)
MOLECULAR FORMULA	$C_7H_4Cl_3NO_3$ (71)
MOLECULAR WEIGHT	256.5 (71)
CAS NUMBER	55335-06-3 (106)
SOLUBILITY (25°C)	Water: 430-440 ppm @ 24.5°C (71) Ethanol: very soluble (71) Benzene: slightly soluble (71) Acetone: 989 g/kg (297)
MELTING POINT	148-150°C (297)
VAPOR PRESSURE	1.26×10^{-6} mmHg @ 25°C (71) 5.30×10^{-6} mmHg @ 40°C (71)
DISSOCIATION CONSTANT	pKa 2.68 (297)
HENRY'S LAW CONSTANT	9.66×10^{-10} (101)
Log K_{ow}	0.48 (162) -0.69 (101)
PH	acidic (297)
STABILITY	Stable to hydrolysis but subject to photo decomposition in < 24 hours (117)
CORROSIVENESS	DNF

Table 5-4 Toxicity data for the active ingredients to be used by WSDOT for roadside vegetation management.

	2,4-D	2,4-DP
TOXICITY		
ACUTE ORAL LD_{50}	375 mg/kg (rat) (275) 370 mg/kg (rat) (274) 639-764 mg/kg (rat) (92)	500-800 mg/kg (rat) (277,297,117,93) 400-650 mg/kg (mouse) (277,297,117,93)
ACUTE DERMAL LD_{50}	3,980 mg/kg (rabbit) (275) 1,400 mg/kg (rabbit) (274) 1,500 mg/kg (rat) (274)	1,400 mg/kg (rat) (274) 1,400 mg/kg (mouse) (274)
ACUTE INHALATION LC_{50}	1.79 mg/l (92)	70.65 mg/l (rats, 4hr) (117)
EYE IRRITATION	Severe eye irritant (275)	Slight irritation (rabbit) (277)
SKIN IRRITATION	DNF	Slight irritation (rabbit) (277)
SKIN SENSITIZATION	Not a sensitizer (92)	DNF
SUBCHRONIC (RAT)	NOEL (90 day): 1 mg/kg/day (275)	LOEL (90 day): 5 mg/kg/day (93)
SUBCHRONIC (NONRODENT)	DNF	DNF
CHRONIC (RAT)	NOEL (2 year): 1 mg/kg/day (275, 100)	LOEL (2 year): 12 mg/kg/day (93,95) NOEL (2 year): 4 mg/kg/day (93,95)
CHRONIC (OTHER)	NOEL (2 year): 500 ppm (dog) (286)	DNF
ONCOGENICITY (MOUSE)	Not oncogenic (62)	LOEL (2 year): 25 mg/kg (93) (No NOEL; study supplementory)
ONCOGENICITY (OTHER)	Controversial evidence that 2,4-D caused tumors in rats. EPA believes there is not enough evidence to make a decision. No EPA cancer potency value established. Cancer potency of 0.019 mg/kg/day used for risk assessment as reported by the OPP. 0.019 per (mg/kg/day) (275)	Study was conducted but was found to be invalid by the EPA. (93)
TERATOGENICITY (RAT)	No teratogenic effects (275) NOEL (maternal and dev): 25 mg/kg (62)	No effect at 100 mg/kg (rat) (277) (Study found invalid) (93)
TERATOGENICITY (OTHER)	DNF	Teratogenic at 25 mg/kg (rabbit) (277)
REPRODUCTIVE EFFECTS	NOEL (maternal): 5 mg/kg/day (rat) (275) NOEL (parental and rep): 20 mg/kg (rat) (62)	LOEL: 100 mg/kg (offspring mortality) (93)
GENE MUTATION	Non-mutagenic (275)	Positive in Ames and yeast assay. (93) There is a positive trend towards mutagenic effects. (19)

	BENEFIN	BROMACIL
TOXICITY		
ACUTE ORAL LD_{50}	> 2,000 mg/kg (dogs and rabbits) (117) > 10,000 mg/kg (female rat) (274) > 5,000 mg/kg (mouse) (117)	3,998 mg/kg (rat) (275)
ACUTE DERMAL LC_{50}	> 5,000 mg/kg (rabbit) (187)	2,000 mg/kg (rabbit) (275)
ACUTE INHALATION LC_{50}	> 2.16 mg/l (rat, 4hr.) (187)	> 57.6 mg/l (rat) (275)
EYE IRRITATION	Irritant, (rabbit) (187)	Mild irritant (275)
SKIN IRRITATION	Slight irritant (rabbit) (187)	Very slightly irritating (rabbit) (275)
SKIN SENSITIZATION	DNF	Not a sensitizer (287)
SUBCHRONIC (RAT)	NOEL: 5,000 ppm/day (187)	NOEL (90 day): 25 mg/kg/day (275)
SUBCHRONIC (NONRODENT)	NOEL: 12.5 mg/kg/day (187)	DNF
CHRONIC (RAT)	NOEL: 1,000 ppm/day (187)	NOEL (2 year): 12.5 mg/kg/day (275) NOEL (2 year): 2.5 mg/kg/day (62)
CHRONIC (OTHER)	NOEL (2year): 5 mg/kg/day (187) NOEL: 25 mg/kg/day (dog) (100)	NOEL (2 year): 6.25 mg/kg/day (275)
ONCOGENICITY (MOUSE)	Not oncogenic (187)	EPA class., Group C, "possible human carcinogen" A 2 year study at 5,000 ppm (750mg/kg/day) demonstrated increased hepatocellular adenomas and carcinomas (275). EPA Cancer Potency - 0.0038 mg/kg/day (275)
ONCOGENICITY (OTHER)	Not oncogenic (187)	Not oncogenic at levels up to 2,500 ppm (rat) (287)
TERATOGENICITY (RAT)	Not teratogenic (187)	Not teratogenic (275) NOEL (maternal): 20 mg/kg (62) NOEL (developmental): 75 mg/kg (62)
TERATOGENICITY (OTHER)	Not teratogenic (rabbit) (187)	Not teratogenic (rabbit) (275) NOEL (maternal): 100 mg/kg (62) NOEL (developmental): 100 mg/kg (62)
REPRODUCTIVE EFFECTS	No reproductive impairment (187)	No reproductive effects at 12.5 mg/kg/day (rat) (275)
GENE MUTATION	Not mutagenic (187)	Very weak mutagen (275)

	CHLORSULFURON	CHLORTHAL DIMETHYL
TOXICITY		
ACUTE ORAL LD_{50}	3,053 mg/kg (rat,male) (80,70) 2,341 mg/kg (rat,female) (80,70)	> 3,000 mg/kg (rat) (117) > 10,000 mg/kg (rat) (107)
ACUTE DERMAL LD_{50}	> 2,000 mg/kg (rabbit) (80,70)	10,000 mg/kg (rabbit) (117)
ACUTE INHALATION LC_{50}	> 5.9 mg/l/4 hours (rat) (275)	> 5.7 mg/l (rat) (107)
EYE IRRITATION	Mild irritation at 10 mg (rabbit) (275,80)	Mild irritant (rabbit) (117)
SKIN IRRITATION	Not a primary irritant (275,80)	Non-irritating (rabbit) (117)
SKIN SENSITIZATION	Nonsensitizing (80)	DNF
SUBCHRONIC (RAT)	NOEL (6 month): 2500 ppm (80) NOEL (90 day): 5 mg/kg/day (275,100) NOEL (90 day): 365 mg/kg/day (mouse) (275)	NOEL: 500 mg/kg/day (100)
SUBCHRONIC (NONRODENT)	NOEL (180 day): 62.5 mg/kg/day (dog)(275,100)	DNF
CHRONIC (RAT)	NOEL (2 year): 5 mg/kg/day (275,100) NOEL (2 year): 100 ppm (80,100)	LOEL (2 year): 10,000 ppm (117) NOEL (2 year): 50 mg/kg/day (100)
CHRONIC (OTHER)	NOEL (2 year): 75 mg/kg/day (mice) (275,100)	NOEL (2 year): 10,000 ppm (dog) (117,100)
ONCOGENICITY (MOUSE)	Not oncogenic (275,80)	DNF
ONCOGENICITY (OTHER)	Not oncogenic (275,80)	DNF
TERATOGENICITY (RAT)	Not teratogenic (275) NOEL (maternal): 25 mg/kg/day (62) NOEL (developmental): 125 mg/kg/day (62)	NOEL (maternal and developmental): 2,000 mg/kg/day (rat) (100)
TERATOGENICITY (OTHER)	Not teratogenic (275) NOEL (maternal and developmental): 25 mg/kg/day (rabbit) (62)	DNF
REPRODUCTIVE EFFECTS	NOEL: 75 mg/kg/day (rabbit) (80) NOEL (reproductive and maternal): 25 mg/kg/day (rat) (275)	NOEL: 500 mg/kg/day (rat) (100)
GENE MUTATION	Not mutagenic (275)	DNF

	DICAMBA	DICHLOBENIL
TOXICITY		
ACUTE ORAL LD_{50}	757-1,701 mg/kg (rat) (257) 1,707 mg/kg (rat) (256) 1,581-2,740 mg/kg (144,143)	>3,160 mg/kg (rat) (270) 2,710 mg/kg (rat) (274) 4,250 mg/kg (rat) (89) 4,460 mg/kg (rat) (7) 2,056 mg/kg (mouse) (274,270) 681 mg/kg (guinea pig) (274)
ACUTE DERMAL LD_{50}	>2,000 mg/kg (rabbit) (257,256,144)	1,350 mg/kg (rabbit) (274,270)
ACUTE INHALATION LC_{50}	>9.6 mg/l (rat) (257,256,144)	>5 mg/l (rat) (270,54)
EYE IRRITATION	Severe eye irritant (115,275,256,295)	Nonirritating (rabbit) (270,170)
SKIN IRRITATION	Slight (275,256,145)	Nonirritating (rabbit) (270,171)
SKIN SENSITIZATION	Moderate (guinea pig) (275,256,56)	Nonsensitizing (66,269)
SUBCHRONIC (RAT)	NOEL:5000 ppm in diet (250 mg/kg/day) (257,256,147)	NOEL (90 day): 1.25 mg/kg/day (mouse) (269) NOEL: 2.5 mg/kg/day (rat) (103)
SUBCHRONIC (NONRODENT)	NOEL (21 day, dermal): <100 mg/kg/day (rabbit) (256)	NOEL (90 day): 1.25 mg/kg/day (dog) (269,263)
CHRONIC (RAT)	NOEL (2 year): >2500 ppm (0.125 mg/kg/day) (256,88,148)	NOEL (2 year): 2.5 mg/kg/day (269,142)
CHRONIC (OTHER)	NOEL: 1000 ppm in diet (115 mg/kg/day) (mouse) (256,99) NOEL (1 year): > 2500 ppm (50-60 mg/kg/day) (dog) (256,90,149)	NOEL (2 year): 1.25 mg/kg/day (dog) (269) NOEL: 50 ppm in diet (dog) (264,62)
ONCOGENICITY (MOUSE)	Not oncogenic (256,99,279)	Not oncogenic (hamster) (136)
ONCOGENICITY (OTHER)	Not oncogenic (rat) (256,88,148)	Not oncogenic (rat) (270,142)
TERATOGENICITY (RAT)	Not teratogenic (275,257,266) NOEL (maternal): 160 mg/kg/day (100,85) NOEL (dev): 400 mg/kg/day (100,85)	NOEL: 20 mg/kg/day (269) Not teratogenic (169)
TERATOGENICITY (OTHER)	Not teratogenic (rabbit) NOEL: 3.0 mg/kg/day (257,100,256,146)	NOEL: 60 mg/kg (mouse) (270) Not teratogenic (rabbit) (12,13)
REPRODUCTIVE EFFECTS	NOEL: 500 ppm in diet (25 mg/kg/day) (rat) (256)	NOEL: 50 mg/kg/day (rat) (269) NOEL: 60 ppm in diet (rat) (175)
GENE MUTATION	Not mutagenic (275,257)	Not mutagenic (1,2,153,182,203,254,259,269)

	DIURON	FLUAZIFOP-BUTYL
TOXICITY		
ACUTE ORAL LD_{50}	3,750 mg/kg (rat) (275) 2,900 mg/kg (rat) (79) 3,400 mg/kg (rat) (84)	621 mg/kg (rabbits) (117) 3,328 mg/kg (rat) (165,117,141) 2,659 mg/kg (guinea pig) (117) 1,630 mg/kg (mouse) (117)
ACUTE DERMAL LD_{50}	>10,000 mg/kg (rat) (275) >2,000 mg/kg (rabbit) (79) >20,000 mg/kg (rabbit) (84)	>6,050 mg/kg (rat) (117) >2,420 mg/kg (rabbit) (165,117,141)
ACUTE INHALATION LC_{50}	DNF	> 5.2 mg/l (rat) (165,117,141)
EYE IRRITATION	Very slightly toxic (rabbit) (275) Mild irritant (84)	Practically non-irritating (117) Slight irritant (165)
SKIN IRRITATION	Slight irritant (rabbit) (275) Mild conjuctival irritant (84)	Mild (165,117,141)
SKIN SENSITIZATION	Nonsensitizing (79)	Nonsensitizing (117,141)
SUBCHRONIC (RAT)	DNF	NOEL: 0.5 mg/kg/day (165) NOEL (90 day): 10 mg/kg/day (117)
SUBCHRONIC (NONRODENT)	DNF	NOEL (90 day): 25 mg/kg/day (dog) (117,141)
CHRONIC (RAT)	NOEL (2 year): 1.25 mg/kg/day (rat) (275) NOEL (2 year): 25 ppm (rat) (84)	Systemic Tox. NOEL: 1.0 mg/kg/day (165)
CHRONIC (OTHER)	NOEL (2 year): 0.625 mg/kg/day (dog) (275,100) NOEL (2 year): 25 ppm (dog) (84)	Systemic Tox. NOEL: 1.0 mg/kg/day (mouse) (165) NOEL (1 year): 5 mg/kg/day (dog) (155)
ONCOGENICITY (MOUSE)	Not oncogenic (275) Additional studies in progress (287)	Not oncogenic (165)
ONCOGENICITY (OTHER)	Not oncogenic (rat,dog) (275) Additional studies in progress (287)	Not oncogenic (rat) (165)
TERATOGENICITY (RAT)	Not teratogenic (275) NOEL (maternal): 16 mg/kg/day (62) NOEL (developmental): 80 mg/kg/day (62)	NOEL (teratogenic and mat): 10 mg/kg/day (165) NOEL (fetotoxic): 1.0 mg/kg/day (165)
TERATOGENICITY (OTHER)	Not teratogenic (dog) (275) NOEL (maternal): 10 mg/kg/day (rabbit) (62) NOEL (dev): 50 mg/kg/day (rabbit) (62)	Not teratogenic (rabbit) (165) NOEL (fetotoxic): 10 mg/kg/day (165)
REPRODUCTIVE EFFECTS	NOEL: 6.25 mg/kg/day (rat) (275)	NOEL: 80 ppm (rat) (165)
GENE MUTATION	Not mutagenic (275,79)	Not mutagenic (165)

	FOSAMINE	GLYPHOSATE
TOXICITY		
ACUTE ORAL LD_{50}	24,400 mg/kg (rat) (276) 7,380 mg/kg (guinea pig) (276)	4,300 mg/kg (rat) (95)
ACUTE DERMAL LD_{50}	1,683 mg/kg (rabbit) (276)	> 5,010 mg/kg (mouse,rabbit) (95) >794 mg/kg (rabbit) (95)
ACUTE INHALATION LC_{50}	3 mg/l (rat) (109)	Roundup: > 3.18 mg/l (rat) (196) Rodeo: > 1.3 mg/l (rat) (196)
EYE IRRITATION	Not irritating (rabbit) (277)	Slightly irritating (95,195)
SKIN IRRITATION	Transient, mild to moderate irritation (277)	Non-irritating (rabbit) (95,195)
SKIN SENSITIZATION	Non-sensitizing (276)	Non-irritating (8)
SUBCHRONIC (RAT)	NOEL (90 day): 250/500 mg/kg/day (276)	NOEL (90 day): 20,000 ppm (rat) (194) NOEL (90 day): 10,000 ppm (mouse) (194)
SUBCHRONIC (NONRODENT)	NOEL (6 month): 25 mg/kg/day (dog) (276)	DNF
CHRONIC (RAT)	Data not required (109)	NOEL (2 year): 8000 ppm (194) NOEL: 31 mg/kg/day (95)
CHRONIC (OTHER)	Data not required (109)	NOEL (2 year): 500 mg/kg/day (dog) (194) NOEL (1 year): 20 mg/kg/day (dog) (100,95)
ONCOGENICITY (MOUSE)	Data not required (109)	NOEL (systemic): 710 mg/kg/day (100)
ONCOGENICITY (OTHER)	Data not required (109)	Class D oncogen (100)
TERATOGENICITY (RAT)	NOEL $\geq$ 10,000 ppm (109) Not Teratogenic (109)	NOEL (dev): $\geq$ 3500 mg/kg/day (95) NOEL (fetotoxicity): 1000 mg/kg/day (95) NOEL (mat & dev): 1,000 mg/kg/day (100,95)
TERATOGENICITY (OTHER)	Teratogenic when sprayed into eggs of quail and chicken (277) Teratogenic (study rejected by CDFA) (276)	NOEL (maternal): 175 mg/kg/day (100,95) NOEL (developmental): >350 mg/kg/day (100,95)
REPRODUCTIVE EFFECTS	No reproductive effects (Study rejected by CDFA) (276)	NOEL: 30 mg/kg/day (3 generation, rat) (194) Not a reproductive toxin (100,95)
GENE MUTATION	Not mutagenic (276)	Not mutagenic (194,95)

	IMAZAPYR	ISOXABEN
TOXICITY		
ACUTE ORAL LD_{50}	> 5,000 mg/kg (rat) (3) > 2,000 mg/kg (female mouse) (3) 4,800 mg/kg (rabbit) (3)	> 10,000 mg/kg (rat) (156) (117) >10,000 mg/kg (mouse) (156) > 5,000 mg/kg (dog) (156) (117)
ACUTE DERMAL LD_{50}	>2,000 mg/kg (rabbit) (3) > 2,000 mg/kg (rat) (3)	> 200 mg/kg (rabbit) (156) (117)
ACUTE INHALATION LC_{50}	>5.1 mg/l (rat) (3)	> 1.99 mg/l (rat) (156) (117)
EYE IRRITATION	Irritating (rabbit) (3)	Slight eye irritant. (156)
SKIN IRRITATION	Mildly irritating (rabbit) (3)	Slight irritant (rabbit) (156)
SKIN SENSITIZATION	Non-sensitizing (guinea pig) (3)	Non-sensitizing (guinea pig) (156)
SUBCHRONIC (RAT)	NOEL: ≥ 10,000 ppm in diet 500 mg/kg/day (275)	50,000 mg/kg maximum dose. No serious effects. (156)
SUBCHRONIC (NONRODENT)	Data not required by EPA. (177)	1,000 mg/kg maximum dose. No serious effects (dog). (156)
CHRONIC (RAT)	Data not required by EPA. (177)	NOEL (2 year.): 5.0 mg/kg/day (117,155) NOEL: 6.6 mg/kg/day (156)
CHRONIC (OTHER)	Data currently under EPA review. (177)	NOEL: 10 mg/kg/day (dog) (156)
ONCOGENICITY (MOUSE)	Data currently under EPA review. (177)	NOEL: 12 mg/kg/day (Class C oncogenic) (156) Not oncogenic (75)
ONCOGENICITY (OTHER)	Data currently under EPA review. (177) Non-oncogenic (rat) (4)	DNF
TERATOGENICITY (RAT)	NOEL: ≥ 1,000 mg/kg/day (rat) (3) Not teratogenic (277) NOEL (maternal and dev): 300 mg/kg/day (275)	NOEL: 1,000 mg/kg/day (maximum dose) NOEL (developmental): 320 mg/kg/day (155) Not teratogenic (156)
TERATOGENICITY (OTHER)	NOEL: ≥ 400 mg/kg/day (rabbit) (3) Not teratogenic	NOEL (maternal): 320 mg/kg/day (rabbit) (156) NOEL (prenatal): >1,000 mg/kg/day (rabbit) (156) Not teratogenic
REPRODUCTIVE EFFECTS	NOEL: ≥ 1000 mg/kg/day (rat) NOEL: ≥ 400 mg/kg/day (rabbit) Not a reproductive toxin (3)	No reproductive effects. (75) NOEL (developmental): 200 mg/kg/day (rat) (156) NOEL (parental): 40 mg/kg/day (rat) (156) Not a reproductive toxin
GENE MUTATION	Not mutagenic (3)	Not mutagenic (156)

	MECOPROP	MEFLUIDIDE
TOXICITY		
ACUTE ORAL LD_{50}	650 mg/kg (rat) (274) 650 mg/kg (mouse) (274) 558 mg/kg (rat) (96)	4,000 mg/kg (rat) (237) 1,920 mg/kg (mouse) (215)
ACUTE DERMAL LD_{50}	900 mg/kg (rabbit) (274) > 4,000 mg/kg (rat) (297) > 2,000 mg/kg (rabbit) (96)	>4,000 mg/kg (rabbit) (275,221)
ACUTE INHALATION LC_{50}	> 12.5 mg/l (rat) (176) (297)	>8.5 mg/l/4hr (rat) (275) >5.4 mg/l in air (rat) (222)
EYE IRRITATION	Not corrosive (176) Severe irritant (96)	Minimal irritation (rabbit) (275,223)
SKIN IRRITATION	Moderate skin irritant (176) Not an irritant (96)	Non-irritating (rabbit) (275,224)
SKIN SENSITIZATION	Not a sensitizer (176)	Not a sensitizer (209)
SUBCHRONIC (RAT)	NOEL (90 days): 3 mg/kg (176,96) NOEL (7 month): 100 mg/kg in diet (117) NOEL (3 week): 65 mg/kg/day (297)	NOEL (90 day): 300 mg/kg/day (rat) (275) NOEL: 6,000 ppm (211,220)
SUBCHRONIC (NONRODENT)	NOEL: 114 ppm (dog) (176)	NOEL (90 day): 25 mg/kg/day (dog) (275) NOEL: 1,000 ppm (dog) (219)
CHRONIC (RAT)	NOEL (2 year): 1.1 mg/kg (297) NOEL: 20 ppm (176)	NOEL (2 year): 30 mg/kg/day (rat) (275) NOEL: 2,000 ppm (213)
CHRONIC (OTHER)	DNF	NOEL (18 mon): 90 mg/kg/day (mouse) (275) NOEL (1 year): 1.5 mg/kg/day (dog) (275) NOEL: 60 ppm (dog) (212)
ONCOGENICITY (MOUSE)	DNF	Non-carcinogenic (275) Non-tumorigenic at 600, 1,800, and 6,000 ppm (210)
ONCOGENICITY (OTHER)	DNF	Data not required by EPA (6)
TERATOGENICITY (RAT)	Not teratogenic or embroyotoxic at doses up to and including 0.25 mg/kg (176) NOEL (maternal): 125 mg/kg (96) NOEL (developmental): 50 mg/kg (96) NOEL: 125 mg/kg (96)	NOEL: >60 mg/kg/day (rat) (275) Diethanolamine salt: NOEL (maternal and developmental): 200 mg/kg/day (231) Additional studies in progress (6)
TERATOGENICITY (OTHER)	Rabbit: Not teratogenic or embryotoxic at doses up to and including 7.5 mg/kg (176) NOEL (maternal): 30 mg/kg (96) NOEL (developmental): 75 mg/kg (96)	NOEL: >60 mg/kg/day (rabbit) (155)
REPRODUCTIVE EFFECTS	DNF	NOEL: 90 mg/kg/day (rat) (275) No effect on reproductive capacity or health of rats dosed with 600, 1,800, and 6,000 ppm. (214)
GENE MUTATION	Not mutagenic (176,96)	Not mutagenic (275,218,216,217)

	METSULFURON METHYL	NAPROPAMIDE
TOXICITY		
ACUTE ORAL LD_{50}	> 5,000 mg/kg (rat) (275)	>5,000 mg/kg (rat) (274)
ACUTE DERMAL LD_{50}	> 2,000 mg/kg (rabbit) (275)	> 4,640 mg/kg (rabbit) (117,165)
ACUTE INHALATION LC_{50}	> 5.0 mg/l (rat, 4 hrs) (275)	> 5.1 ml/l (165)
EYE IRRITATION	Slight corneal clouding, moderate iritis and severe to moderate conjunctivitis in unwashed eyes. (rabbit) (275)	Not irritating (117) Mildly irritating (165)
SKIN IRRITATION	Moderately irritating (rabbit) (275)	Mildly irritating (165) Not irritating (117)
SKIN SENSITIZATION	Not a sensitizer (117)	Not a sensitizer (165)
SUBCHRONIC (RAT)	NOEL (90 day): 50 mg/kg/day (rat) (275)	NOEL (13 week): 50 mg/kg/day (117) NOEL (13 week): 25 mg/kg/day (100)
SUBCHRONIC (NONRODENT)	NOEL (90 day): >125 mg/kg/day (dog) (275)	NOEL (90 day): 40 mg/kg/day (dog) (297,100)
CHRONIC (RAT)	NOEL (2 year): 25 mg/kg/day (rat) (275,100) NOEL (2 year): >750 mg/kg/day (mouse)(275)	NOEL (2 year): 30 mg/kg/day (165,297)
CHRONIC (OTHER)	NOEL (1 year): 1.25 mg/kg/day (dog) (275,86) NOEL (1 year): > 125 mg/kg/day (dog) (100)	NOEL: 10 mg/kg/day (dog) (165)
ONCOGENICITY (MOUSE)	Not oncogenic (275,86)	NOEL: 450 ppm (165) Not oncogenic (165)
ONCOGENICITY (OTHER)	Not oncogenic (275,86)	NOEL: 30 mg/kg/day (165) Not oncogenic (rat) (165)
TERATOGENICITY (RAT)	Fetotoxic and teratogenic NOEL (maternal): < 40 mg/kg/day (100,86) NOEL: >1,000 mg/kg/day (275,100,86)	Not fetotoxic or embryotoxic at 100 mg/kg/day (165)
TERATOGENICITY (OTHER)	Fetotoxic and teratogenic NOEL: 700 mg/kg/day (rabbit) (275,100) NOEL (maternal): 25 mg/kg/day (275,100)	Not fetotoxic or embryotoxic at 100 mg/kg/day (165)
REPRODUCTIVE EFFECTS	NOEL (maternal): 25 mg/kg/day (rat) (275,100) NOEL (reproductive and fetotoxic): >250 mg/kg/day (rat) (275,100)	NOEL (parental and rep): 30 mg/kg/day (165,100)
GENE MUTATION	Not mutagenic (275) Caused chromosome aberrations (86)	Not mutagenic (165)

	ORYZALIN	OXADIAZON
TOXICITY		
ACUTE ORAL LD_{50}	>10,000 mg/kg (rat) (274,74) >1,000 mg/kg (dog, chicken) (74)	3,500 mg/kg (rat) (274)
ACUTE DERMAL LD_{50}	> 5,000 mg/kg (57) > 2,000 mg/kg (rabbit) (117,91)	>2,000 mg/kg (rabbit) (191) > 8,000 mg/kg (rat) (117)
ACUTE INHALATION LC_{50}	>3.56 mg/l (91)	> 200 mg/l (rats) (117) > 1.94 mg/l (4 hr, rat) (191)
EYE IRRITATION	Slightly irritating (57,91) Non-irritating (rabbit) (117)	Slightly (117)
SKIN IRRITATION	Slight (rabbit) (74,57)	Slightly (117)
SKIN SENSITIZATION	Positive (57)	No sensitization (191)
SUBCHRONIC (RAT)	NOEL: > 0.275 - < 56.25 mg/kg/day (57) NOEL (1 year): 15 mg/kg/day (100)	NOEL: <25 mg/kg/day (155)
SUBCHRONIC (NONRODENT)	NOEL: > 18.75 - < 56.25 mg/kg/day (57) NOEL (3 month): 18.8 mg/kg/day (dog) (100)	NOEL: 100 mg/kg/day (155)
CHRONIC (RAT)	NOEL (2 year): 300 mg/kg/day diet (117) NOEL (1 year): 15 mg/kg/day (100)	NOEL (2 year): 100 mg/kg/day (rat) (117,62) 10 mg/kg/day (297,100) NOEL: 0.5 mg/kg/day (100)
CHRONIC (OTHER)	NOEL (1year): 5 mg/kg (dog) (100)	NOEL (2 year): 100 mg/kg/day (dog) (117,100) NOEL: 2.5 mg/kg/day (100)
ONCOGENICITY (MOUSE)	Class C oncogen (57) Negative for oncogenicity (91)	Class C oncogen (83) Cancer potency factor: 0.14 (83)
ONCOGENICITY (OTHER)	Class C oncogen (57) Increases in thyroid gland, skin and mammary gland tumors (91) Cancer Potency Factor: 0.13	Minimum tox level: 1,000 ppm (191) Max no-effect-level: 100 ppm (191)
TERATOGENICITY (RAT)	Not teratogenic (57) NOEL (maternal and dev): 225 mg/kg/day (100)	NOEL (developmental): 12 mg/kg/day (62) NOEL (maternal): > 40 mg/kg/day (62)
TERATOGENICITY (OTHER)	Not teratogenic (57) NOEL (fetotoxic and mat): 25 mg/kg/day (100) NOEL: 125 mg/kg/day (teratogenic) (100)	NOEL (maternal and fetal): 20 mg/kg/day (62)
REPRODUCTIVE EFFECTS	Not a reproductive toxin (57) NOEL: 12.5 mg/kg/day (fetotoxic) (100)	NOEL: 200 ppm (62) NOEL: 5 mg/kg/day (100)
GENE MUTATION	Not mutagenic (57)	Not mutagenic (62)

	PICLORAM	PRONAMIDE
TOXICITY		
ACUTE ORAL LD_{50}	8,200 mg/kg (rat) (277) 6,000 mg/kg (chicken) (277) 2,000-4,000 mg/kg (mouse) (73) 4,012-5,000 mg/kg (rat) (97)	5,620 mg/kg (rat) (274) >10,000 mg/kg (dog) (117) 16,000 mg/kg (rat) (87)
ACUTE DERMAL LD_{50}	>3,980 mg/kg (rabbit) (73)	>3,160 mg/kg (rabbit) (117) >10,000 mg/kg (rabbit) (87)
ACUTE INHALATION LC_{50}	minimal/slight: No effect in saturated atmosphere for 2 weeks. (159) K salt >1.5 mg/l (97) Acid >0.035 mg/l (97)	> 5 mg/l (rat) (297) >3.2 mg/l (rat) (87)
EYE IRRITATION	Mild (rabbit) (277) Moderate (97)	Slightly (117,87)
SKIN IRRITATION	Mild (rabbit) (277) Non-irritating (73,97)	Slightly (117) Non-irritating (87)
SKIN SENSITIZATION	Not a sensitizer (73) K salt is a sensitizer (97)	Nonsensitizing (87)
SUBCHRONIC (RAT)	NOEL: 50 mg/kg/day (rat) (73,100)	NOEL (90 day): 2.5 mg/kg/day (100)
SUBCHRONIC (NONRODENT)	NOEL: 7 mg/kg/day (male dog) (73) NOEL: 35 mg/kg/day (female dog) (73) NOEL (6 mos): 7 mg/kg/day (dog) (100)	NOEL (90 day): 33.7 mg/kg/day (100) NOEL: 1,350 ppm, 30 mg/kg/day (87)
CHRONIC (RAT)	NOEL (2 year): 150 mg/kg/day (159) NOEL (2 year): 20 mg/kg/day (100)	NOEL (2 year): 300 mg/kg/day (297,100) NOEL (2 year): 15 mg/kg/day (100)
CHRONIC (OTHER)	NOEL (90 day): 150 mg/kg/day (159)	NOEL (2 year): 300 mg/kg/day (dog) (297,100) NOEL (2 year): 7.5 mg/kg/day (100)
ONCOGENICITY (MOUSE)	Not oncogenic (159,202) Class D oncogen	Class C oncogen (83) Cancer potency factor: 0.016 (83) Positive (87)
ONCOGENICITY (OTHER)	Not oncogenic (rat) (202) Not oncogenic (dog) (159) Cancer potency factor: 0.003 (275)	DNF
TERATOGENICITY (RAT)	Not teratogenic at 1,000 mg/kg (277,100)	DNF
TERATOGENICITY (OTHER)	Not teratogenic at 400 mg/kg (rabbit) (277)	NOEL (maternal): 5 mg/kg/day (rabbit) (100) NOEL (developmental): 20 mg/kg/day (87)
REPRODUCTIVE EFFECTS	NOEL: 50 mg/kg/day (100)	NOEL: 15 mg/kg/day (100)
GENE MUTATION	Not mutagenic (73)	DNF

	SETHOXYDIM	SULFOMETURON METHYL
TOXICITY		
ACUTE ORAL LD_{50}	2,676 mg/kg (rat) (female) (23) 3,125 mg/kg (male rat) (117)	> 5,000 mg/kg (male rat) (78)
ACUTE DERMAL LD_{50}	5,000 mg/kg (rabbits) (31) > 5,000 mg/kg (rat) (117)	> 8,000 mg/kg (male rabbit) (78) > 2,000 mg/kg (female rabbit) (78)
ACUTE INHALATION LC_{50}	> 6.03 mg/l (rat, males) (35) > 6.28 mg/l (rat, females) (35)	5 mg/l (rat) (78)
EYE IRRITATION	Non-irritating (39, 117)	Slightly irritating (rabbit) (277,78)
SKIN IRRITATION	Non-irritating (46, 117)	Not a primary skin irritant. (78)
SKIN SENSITIZATION	Not a sensitizer (117, 40)	Non-sensitizing (guinea pig) (78)
SUBCHRONIC (RAT)	NOEL (14 week): 300 ppm (44) NOEL (14 week): 15 mg/kg/day (155) NOEL (90 day): 900 mg/kg//day (mice) (151)	NOEL (90 day): 50 mg/kg/day (155)
SUBCHRONIC (NONRODENT)	NOEL (14 week): 45 mg/kg/day (mouse) (155) Data not required (258) NOEL (6 months): 20 mg/kg/day (dog) (100)	NOEL (21 day, dermal): >2,000 mg/kg (rabbit) (155)
CHRONIC (RAT)	NOEL (2 year): 17.2 mg/l (297) NOEL (2 year): 360 ppm (53) NOEL (2 year): 18 mg/kg/day (100)	DNF
CHRONIC (OTHER)	NOEL (2 year): 120 ppm (mouse) (51) NOEL (1 year): 300 ppm in diet (dog) (45) NOEL (1 year): 8.6-9.4 mg/kg/day (dog) (100)	NOEL (1 year): 5 mg/kg/day (155)
ONCOGENICITY (MOUSE)	Not oncogenic (51)	Not oncogenic (78)
ONCOGENICITY (OTHER)	Not oncogenic (rat) (53)	Not oncogenic (78)
TERATOGENICITY (RAT)	Not teratogenic (rat) (14) NOEL (maternal): 40 mg/kg/day (100) NOEL (developmental): 250 mg/kg/day (100)	Not teratogenic (277) NOEL (maternal and dev): 50 mg/kg/day (155)
TERATOGENICITY (OTHER)	Not teratogenic (rabbit) (18) NOEL (maternal): 160 mg/kg/day (100) NOEL (developmental): 480 mg/kg/day (100)	Not teratogenic (rabbit) (277) NOEL: 300 mg/kg/day (155)
REPRODUCTIVE EFFECTS	NOEL: 150 ppm (15) NOEL: 18 mg/kg/day (100)	NOEL: 500 ppm (78) NOEL: 250 mg/kg/day (155)
GENE MUTATION	Not mutagenic (42)	Not mutagenic (78)

	TRICLOPYR
TOXICITY	
ACUTE ORAL LD_{50}	630-2560 mg/kg (male, female rat) (71)
ACUTE DERMAL LD_{50}	> 2,000 mg/kg (rabbit) (71)
ACUTE INHALATION LC_{50}	DNF
EYE IRRITATION	Slightly irritating (71)
SKIN IRRITATION	Non-irritating (71)
SKIN SENSITIZATION	DNF
SUBCHRONIC (RAT)	NOEL: 100 mg/kg/day (female) (72) 30 mg/kg/day (male) (72)
SUBCHRONIC (NONRODENT)	LOEL (7.5 month): 2.5 mg/kg/day (dog) (72) NOEL: >30 mg/kg/day(monkey) (72,62)
CHRONIC (RAT)	NOEL (2 year): 3 mg/kg/day (71,62)
CHRONIC (OTHER)	NOEL (2 year): 240 ppm (5mg/kg/day) (mouse) (71) NOEL (1 year): 5mg/kg/day (dog) (71)
ONCOGENICITY (MOUSE)	Not oncogenic (71) NOEL (1 year): 2.5 mg/kg/day (dog) (62)
ONCOGENICITY (OTHER)	Not oncogenic (71)
TERATOGENICITY (RAT)	NOEL: 100 mg/kg/day (71) NOEL (maternal): 50 mg/kg/day (62) NOEL (developmental): 100 mg/kg/day (62)
TERATOGENICITY (OTHER)	NOEL: 75 mg/kg/day (rabbit) (71) NOEL (maternal): 25 mg/kg/day (62) NOEL (developmental): >75 mg/kg/day (62)
REPRODUCTIVE EFFECTS	NOEL: $\geq$ 30 mg/kg/day (3 generations)(rat) (72) Not a reproductive toxin (72)
GENE MUTATION	Not mutagenic (71)

Table 5-5 Environmental fate of active ingredients to be used by WSDOT for roadside vegetation management.

	2,4-D	2,4-DP
ENVIRONMENTAL FATE		
MODE OF ACTION	Post-emergent herbicide. Uptake is primarily through foliage and stems (276). 2,4-D interferes with cell wall development by acting as plant growth hormone (166)	Systemic, hormone-type herbicide. Absorbed by leaves with translocation to the roots. Acts as a growth inhibitor. (117)
HYDROLYSIS	No hydrolyzable groups (2,4-D). (137)	DNF
PHOTODEGRE-DATION	Aquatic photolysis half-life: 2-4 days (137) Photooxidation half-life (air): 1.8-18 hours (137) In water above 300 ppm 2,4-D, 2- and 4-CPA were the major photoproducts. At dilute concentrations the major photoproducts were 2,4-dichlorophenol and products with hydroxy groups in place of chlorines. (188)	DNF
SOIL HALF-LIFE	<28 days in sandy loam soil. (277) <7 days in prairie soils. (188) 10-50 days (137)	10 days in sandy loam soil. (277)
AQUATIC HALF-LIFE	aerobic - 10-5 days (137); anaerobic - 4 weeks-6 months (137)	DNF
SOIL LEACHING/ MOBILITY	K_{oc} = 20 (162)	DNF
SOIL METABOLISM	2,4-D dimethylamine salt and octyl ester were metabolized similarly and both were degraded to CO_2. (188)	In Saskatchewan soils, i-octyl 2,4-DP ester was rapidly hydrolyzed to unknown products. (188) Degradation at side chain to 2,4-dichlorophenol, ring hydroxylation and ring opening. (117)
AQUATIC METABOLISM	2,4-DBEE degradation in ground water with watermilfoil is greatest under high temperatures and high light levels. 50% of 2,4-DBEE is degraded in 12 hours under ultraviolet light. (188)	DNF
ANIMAL METABOLISM	Rapidly eliminated (277). Primarily excreted in the urine in unchanged form. Rats converted 2,4-D to glycine and taurine conjugates. (188)	DNF
PLANT METABOLISM	In wheat, ring hydroxylation and conjugation with sugars was major pathway for 2,4-D metabolism after 6-8 days. 60% metabolism in 20 days for milkweed but no detectable metabolism of 2,4-D in dogbane roots after 20 days. Soybean roots metabolized 2,4-D to 2,4-D-Glu and 2,4-D-asp acid conjugates. (188)	Susceptible and resistant plants converted 2,4-D and 2,4-DP to 2,4-Dichlorophenol; kidney bean, soybean, pea, brome grass, wild oat, yellow foxtail, barley, timothy and orchard grass. (188)
ACCUMULATION IN NON-TARGET SPECIES	Does not bioaccumulate. (276) BCF = 0 (162)	Slight accumulation. (277)

	BENEFIN	BROMACIL
ENVIRONMENTAL FATE		
MODE OF ACTION	Inhibits root development and interferes with tubulin synthesis (187,117)	Absorbed by the plant roots. Moisture is needed to activate bromacil. It inhibits photosynthesis within the chloroplasts. (276)
HYDROLYSIS	No degradation @ pH 5,7,9 @ 25°C (187)	Stable to hydrolysis regardless of pH. (287)
PHOTODEGRA-DATION	Half-life 9.9 hrs. (187)	Half-life at pH 5: 102 days pH 9: 7 days (287)
SOIL HALF-LIFE	Lab: Aerobic sandy loam soil: 1.7 months Anaerobic sandy loam soil: 15 days (187) Field: Bare soil; 117-131 Turf; 70 days (187)	165 days - East Coast (DE) 137 days - West Coast (CA) (287)
AQUATIC HALF-LIFE	Data not required (187)	Stable in absence of radiation. (287)
SOIL LEACHING/ MOBILITY	Adsorption coefficients from 27 (sand) to 117 (clay loam) (187) K_{oc} = 10,700 (162) Considered immobile in soil. (187)	Mobile in low organic matter soils. (287) K_{oc} = 72 (162) K_{oc} = 18 ml/gm (155)
SOIL METABOLISM	Degrades primarily by dealkylation, nitro group reduction, oxidation of alkylamine group and ring formation to form benzimidazoles. In aerobic soil, 9 metabolites were identified. (187)	Products: Carbon dioxide. 5 other metabolites. (<3% each) (287)
AQUATIC METABOLISM	Data not required (187)	Main metabolite: debrominated analog of bromacil. (287)
ANIMAL METABOLISM	Poorly absorbed in dairy cows. No residue in milk or edible tissues suggesting, that which is absorbed is rapidly metabolized or excreted. (187)	Main metabolite: 5-bromo-3-secbutyl-6-hydroxymethyluracil. (287)
PLANT METABOLISM	Very little uptake and translocation occurs. Extensively metabolized. Very little metabolized benefin is detected in tissues. (187)	Same as above. (287)
ACCUMULATION IN NON-TARGET SPECIES	Estimated BCF = 620-850 (161)	Does not accumulate. (287) Estimated BCF = 15 (155)

	CHLORSULFURON	CHLORTHAL DIMETHYL
ENVIRONMENTAL FATE		
MODE OF ACTION	Systemic. Absorbed by roots and foliage. Inhibits division and growth. Inhibits valine and isoleucine biosynthesis. (70)	Non-systemic. Kills germinating seeds. (117)
HYDROLYSIS	Half-life of 23 days at pH 5. No hydrolytic activity observed at pH 7 and pH 9. (68)	DNF
PHOTODEGRA-DATION	Photodegradation plays a minor role in aqueous solutions (69). Half-life of 65 days in silty loam soils with artificial irradiation; equal to 158 days of natural summer sunlight. (128)	The half-life of 6.1 - 61.1 days. (photooxidation in air) (137)
SOIL HALF-LIFE	1 - 2 weeks in silt loam soil at 25°C. (242)	Half-life of 18 - 92 days (137)
AQUATIC HALF-LIFE	Anaerobic: 20 - 22 weeks. Flooded soils: 7 - 8 weeks. (64)	Half-life of 18 - 92 days (137)
SOIL LEACHING/ MOBILITY	Chlorsulfuron is weakly adsorbed to loam, sandy loam and silt loam soils. It's metabolites, however, adsorb more readily. The relative mobility of chlorsulfuron and its metabolites are: chlorsulfuron > sulfonamide >> triazine amine. (241) K_{oc} = 89 ml/gm (155)	DNF
SOIL METABOLISM	Degradation in soils increases with lower pH values and decreases with increased soil clay content. (157) Microbial degradation is temperature and moisture sensitive. (242)	Microbial degradation to monomethyl tetrachloroterephthalate acid (chlorthal) (117)
AQUATIC METABOLISM	Degradates include: 2-chlorobenzene sulfonamide 2-chlorophenyl sulfonyl urea 2-amino-4-methoxy-6-methyl-1,3,5-triazine Rate increases with pH and amount of oxygen. (64)	DNF
ANIMAL METABOLISM	After 24 hrs. of invitro incubation, 785% of the Chlorsulfuron remained intact. In male rats, 95% of the Chlorsulfuron was eliminated in the urine, 4% in feces and 1% was retained in the body after 72 hrs. Major urine components were parent material (86%),2-Chlorobenzene-sulfonamide (5%) and other minor metabolites (4%). (70)	Following oral administration, metabolized to mono-methyl tetrachloroterephthalate and 2,3,5,6-tetrachloroterephthalatic acid, and eliminated in the urine. (117)
PLANT METABOLISM	Main plant (wheat and barley) metabolite is 5-hydroxyphenyl analog of chlorsulfuron and its glucose conjugate. From this, further degradation produces 2-chloro-5-hydroxybenzenesulfonamide and its glucose conjugate. Main broadleaf metabolite is 6-hydroxymethyl triazine. (70)	Carrots that were analyzed for residues at harvest contained unchanged chlorthal dimethyl and the monoacid. (188) Not metabolized by plants. (117)
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate. (70) Estimated BCF = 4 (155)	Accumulation appears to occur in some pine seedlings (white pine, monterey pine). (188)

	DICAMBA	DICHLOBENIL
ENVIRONMENTAL FATE		
MODE OF ACTION	Selective systemic herbicide. Absorbed by the leaves and roots and readily translocated throughout the plant. Acts as an auxin-like growth regulator (115)	Inhibits the incorporation of glucose into cellulose which is the main constituent of the cell wall. (269) Also inhibits germination and actively dividing meristems. (135,160,190,201)
HYDROLYSIS	Resistant to hydrolysis under normal environmental conditions. (257)	Resistent to hydrolysis between pH 5-9.(59) Hydrolyzed under strong alkaline conditions into 2,6-dichlorobenzamide. (269)
PHOTODEGRA-DATION	Subject to photodegradation (257)	Half-life of 10.5 days in natural light @ 40 degrees North Latitude. (58) Stable in sunlight. (269)
SOIL HALF-LIFE	< 25 days (257) 31 days in aerobic soils. 58 days in anaerobic soils (173).	2-5 months depending on moisture, temperature and soil type. (270) Average: 70 days. (208,282)
AQUATIC HALF-LIFE	DNF	15-69 days. (150,151)
SOIL LEACHING/ MOBILITY	K_{oc} = 0.4 (162) K_{oc} = 4 ml/gm (155) K_d:0 to 0.9 (115) Dicamba readily leaches in moist soils except those with high organic content and kaolinite clays. The degradation product 3,6-dichloro-salicylic acid adsorbs more readily and is less mobile (115)	K_{oc}: 235 (162) K_{oc}: 890 ml/gm (155) K_{oc}: 125 (281) Low mobility in various soils. The primary soil metabolite 2.6-dichlorobenzamide has a greater mobility. (281)
SOIL METABOLISM	Undergoes microbial breakdown to 3,6-dichlorosalicylic acid and thereafter largely to CO_2. (257)	Metabolized rapidly. (269) Microbial transformation to 2.6 dichlorobenzamide. This metabolism is very slowly converted to CO_2 through 2,6 dichlorobenzoic acid. (208,282)
AQUATIC METABOLISM	DNF	Similar to soil metabolism. (81)
ANIMAL METABOLISM	Readily absorbed in the GI tract and rapidly (80-90% in 24 hours) eliminated in urine, mainly as dicamba and partly as 3,6-dichloro-salicylic acid (257,115)	Primary hydroxylation followed by conjugation. Within 168 hrs. over 90% of the dose is excreted of which 95% is excreted within the first 24 hrs. (172,158,174,280) Metabolized in liver to 3-hydroxy-2,6 dichlorobenzonitrile and 4-hydroxy-2,6-dichlorobenzonitrile. Detoxified to glucuronides, sulphates and mercapturic acid conjugates. (269)
PLANT METABOLISM	Several metabolites are formed, including: major: 5-hydroxy-3,6-dichloro-o-anisic acid minor: 3-6 dichlorosalicylic acid minor: 5-hydroxy-3,6-dichloro-o-ansinic acid (115)	After application to water cress, 3-OH and 4-OH was found in the roots and 2,6-dichlorobenzamide was found in the stem. (188) Major residue in edible plant tissue is 2,6-dichlorobenzamide. (138,139)
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate in animals (275,257) BCF = 0 (162)	BCF of 57, although only 0.30 days are required to accomplish 50% clearance after transfer to clean water. (108)

	DIURON	FLUAZIFOP-BUTYL
ENVIRONMENTAL FATE		
MODE OF ACTION	Rapidly absorbed by the roots and translocated throughout the plant. It inhibits photosynthesis. (276)	Interferes with ATP production. Systemic. Absorbed by leaves. Hydrolyzed to fluazifop, translocated and accumulates in meristematic tissue. (117)
HYDROLYSIS	> 500 days at all pH's (287)	Stable at pH 5 (165) Half-life: 78 days @ pH 7 (165) Half-life: 29 hours @ pH 9 (165)
PHOTODEGRA-DATION	Half-life 43 days in water. Half-life 173 days in soil. (287)	Soil Half-life: 195 days (165)
SOIL HALF-LIFE	Relatively persistent in soils. (276) 130 days in Hilo, Hawaii and 160 days in Molokai, Hawaii. (188)	1 - 3 weeks (117) 1 - 13 days (165)
AQUATIC HALF-LIFE	>500 days in absence of radiation (287)	DNF
SOIL LEACHING/ MOBILITY	Will not leach more than 6 inches into the soil in typical texture and moisture conditions. (276,275) K_{oc}: 400 (162) K_{oc}: 560 ml/gm (155)	K_d: 8.2 - 13.9 (165) K_{oc}: 12,000 ml/gm (155)
SOIL METABOLISM	Microbial decomposition is the main mechanism of decomposition. (276) Persistent, especially in high clay and organic matter content. (275) The main degradation product is monomethyl diuron. Conjugated residues were also formed. Residue levels in soil are phytotoxic to oats for three years following last application. (188)	Moist soils, rapid degradation occurs, half-life < 1 week. Major degradation product is fluazifop, which has a half-life < 3 weeks in moist soils. (117)
AQUATIC METABOLISM	Stable - No metabolite formed. (287)	DNF
ANIMAL METABOLISM	DNF	DNF
PLANT METABOLISM	Sugarcane converted diuron to methyl diuron, 3,4-dichlorophenol urea and glucose conjugates of N-hydroxy-methyl-diuron and desmethyl diuron. (188)	Rapidly hydrolyzed to fluazifop. (117) Forms glutathione conjugates. (165)
ACCUMULATION IN NON-TARGET SPECIES	There is evidence of contamination in carp and some aquatic invertebrates. (276,275) Low tendency for bioaccumulation, estimated BCF = 276 (155)	Moderate potential to bioaccumulate, estimated BCF = 160 (155)

	FOSAMINE	GLYPHOSATE
ENVIRONMENTAL FATE		
MODE OF ACTION	Absorbed by the buds, stem and foliage. It inhibits or prevents bud formation. (277)	Broad spectrum herbicide. Absorbed by the foliage and rapidly translocated throughout the plant. Acts on various enzyme systems, interfering with amino acid and other biosynthesis reactions. (189)
HYDROLYSIS	Does not hydrolyze at pH 5.7 and 9 @ 25°C. (167)	Stable in soils. (188)
PHOTODEGRA-DATION	Negligible: Photolysis half-life in water and soil is > 30 days. (164,163)	Negligible in soil. (188)
SOIL HALF-LIFE	< 10 days in sandy loam soil. (277) 2 - 5 days. (82)	Typically less than 60 days. (296) Persistent in sandy loam soils. (275)
AQUATIC HALF-LIFE	Data not required. (109)	DNF
SOIL LEACHING/ MOBILITY	Fosamine is weakly to highly adsorbed onto different soil types where it is converted to carbamyl phosphonic acid (CPA) that does bind strongly to soils. (240)	Very little potential mobility. Rapidly and strongly adsorbed to soil, particularly to clay particles and at lower pH values. (194) K_{oc}: 2,640 (162) K_{oc}: 25.4 ml/gm (155)
SOIL METABOLISM	Rapid microbial degradation to carbamyl phosphonic acid. (116)	Soil microbial attack is the major and possibly only mechanism of breakdown in the soil. The primary metabolite is aminomethylphosphonic acid which is also biodegradable; three other phosphonic acids are also usually produced. Further microbial action results in conversion to CO_2. (188)
AQUATIC METABOLISM	Data not required. (109)	No significant chemical degradation/hydrolysis occurs. (188)
ANIMAL METABOLISM	Total elimination from the body by rats in 72 hours. Carbamoylphosphonate and fosamine were identified as products. (277)	Eliminated largely in feces, some in urine in rabbits. (194)
PLANT METABOLISM	Rapidly metabolized in pasture flora, half-life 7 days. Metabolized to CPA. (109)	Not metabolized in thistle after one week. (188)
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate (109)	Does not accumulate in fish or other animals. BCF = 1 (155)

	IMAZAPYR	ISOXABEN
ENVIRONMENTAL FATE		
MODE OF ACTION	Non-selective systemic herbicide. Absorbed by roots and foliage and rapidly translocated to the meristematic regions where it accumulates. Inhibits synthesis of branched chain amino acids leading to interference of cell growth. (3,177)	Selective preemergence herbicide for control of certain broadleaf and grass weeds. Absorbed by roots and translocated to the stem and leaf tissues. It disrupts leaf and shoot development. It must be activated by rainfall or irrigation. (75,156)
HYDROLYSIS	In aqueous solution, is stable in the dark, but breaks down very rapidly in the presence of light. (177)	Does not hydrolyze. Stable in the presence of soil moisture. (75) Negligible (156)
PHOTODEGRA-DATION	Stable in light in the absence of water. (177)	Susceptible to photodegradation in water. (156)
SOIL HALF-LIFE	27 days in sandy loam soil. (277) High soil moisture and warm temperatures increase rate of degradation. (177)	34 days for spring turf application and 146 days for fall bare soil applications. (75,156) 5 - 6 months (117)
AQUATIC HALF-LIFE	Stable under dark conditions. Currently under EPA review. (177)	3.9 - 65.3 days: Photodegradation occurs in water. (75,156)
SOIL LEACHING/ MOBILITY	K_d: 0 (clay), 0.07 (clay loam), 0.17 (loamy sand), 0.19 (sandy clay loam). (177) Fairly mobile. Relationship between soil pH and mobility is poorly understood. (177)	K_d = 0.81-6.63 (156) K_{oc}: 190-570 (156) K_{oc}: 2,980-4,370 ml/gm (155) High affinity for soil particles and organic matter. Low water solubility prevents leaching. (75,156)
SOIL METABOLISM	Degrades slowly in soil. Main route is by photodegradation with some contribution from microbes. (177)	Degraded primarily by oxidation and dealkylation of the alkyl side chain with degradation of the isoxazole ring also occurring. (156)
AQUATIC METABOLISM	Stable under dark conditions. Currently under EPA review. (177)	Relatively stable in anaerobic environments. (156)
ANIMAL METABOLISM	Studies in rats indicate approximately 87% of the dose is excreted in urine and feces within 24 hours as the unmetabolized acid. (3)	Rapidly metabolized and excreted. (156)
PLANT METABOLISM	Few plant species metabolize imazapyr. Excreted through roots largely as unmetabolized acid. A half-life of 12-40 days has been reported for trees and shrubs. (177)	Metabolized primarily through hydroxylation of the alkyl side chain. Very low levels of plant uptake. (117,156)
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate in animals, including fish. (177)	Low bioaccumulation potential. (75) Moderate potential, estimated BCF = 420-620 (155) Does not accumulate in animal tissue. (156)

	MECOPROP	MEFLUIDIDE
ENVIRONMENTAL FATE		
MODE OF ACTION	Selective systemic, hormone type herbicide. Absorbed by leaves, translocated to roots. (117)	Inhibits growth and development of meristematic regions of responsive species. (6)
HYDROLYSIS	Stable (155)	Stable @ pH 5.7 and 9 @ 25°C. (230)
PHOTODEGRE-DATION	Half-life: 83 days @ pH 5 88 days @ pH 7 64 days @ pH 9 (94,155)	Study in progress. (6)
SOIL HALF-LIFE	Finnish soils: north Finland - 20 days south Finland - 60 days (188)	Study in progress. (6)
AQUATIC HALF-LIFE	DNF	Data not required by EPA. (6)
SOIL LEACHING/ MOBILITY	K_d: 0.19 (sand) 0.29 (sandy loam) 0.43 (silt loam) (155)	Study in progress. (6) K_{oc}: 250 ml/gm (155)
SOIL METABOLISM	Aerobic half-life: 13.3 days (176). Degradation of side chain to 4-chloro-2-methylphenol. Ring hydroxylation and ring opening. (117)	Study in progress. (6)
AQUATIC METABOLISM	DNF	Data not required by EPA. (6)
ANIMAL METABOLISM	80% excreted in urine within 8 hrs. (176)	Study in progress. (6)
PLANT METABOLISM	85% hydroxylated methyl group. Minor ring metabolism. (176) Similar to that of soil. (225)	Mefluidide applied to soybeans was metabolized to form 4,6-diamino-m-xylene, 6-acetamido-4-amino-m-xylene and conjugates. (188)
ACCUMULATION IN NON-TARGET SPECIES	Slight bioaccumulation in bluegill sunfish (1.2X in edible tissue, 5.5X in non-edible tissue and 3.0X in the whole fish). (155)	No bioaccumulation observed in catfish from soil applied mefluidide-C^{14}. (233) Low potential for bioaccumulation, calculated BCF = 33 (155)

	METSULFURON METHYL	NAPROPAMIDE
ENVIRONMENTAL FATE		
MODE OF ACTION	Systemic, absorbed through roots and foliage. Inhibits cellular division in the meristems. (117)	Systemic herbicide. Absorbed by the roots and translocated. Inhibits root development and growth (117).
HYDROLYSIS	Half-life @ 25°C: 15 hours (pH 2) 33 day (pH 5) >41 days (pH 7-9) (117)	Stable between pH 4 and pH 10 (117)
PHOTODEGRA-DATION	Stable (86)	Soil half-life: 28 days (165)
SOIL HALF-LIFE	Approximately 8 months in silt loam soils @ 25°C and 70 - 75% moisture retention capacity. (246) 120-180 days (silt loam soil) (86)	8 - 12 weeks (117) 9 - 10 days (165)
AQUATIC HALF-LIFE	36 days (161) 139 days in sterile water. (161)	DNF
SOIL LEACHING/ MOBILITY	Poorly adsorbed on neutral sandy loam and slightly alkaline loam soils (K_d = 0.05 and 0.15 ml/g). Weakly adsorbed on slightly alkaline sandy clay loam and clay loam soils (K_d = 0.3 and 0.6 ml/g). Weakly adsorbed on acidic clay loam and sandy loam soils (K_d = 0.3 and 4.9 ml/g). (298)	K_d = 3.4 - 14.8 (165) K_{oc}: 680 (162) K_{oc}: 568 ml/gm (155)
SOIL METABOLISM	Main degradates include: dihydroxy methyl triazine and CO_2. (246)	Photodegradation to 2-(x naphthoxy)-N-ethylpropionanide 2-(naphthowy) propionamide, 1-naphthol and 1-4 naphthoquinone (117)
AQUATIC METABOLISM	Anaerobic metabolism: Metabolites include: 1) Free acid and O-demethyl analogs. 2) O-demethyl triazine amine. 3) bis-O-demethyl analog. 4) dihydroxymethyl triazine. Final products are CO_2 and methane. (161,261)	DNF
ANIMAL METABOLISM	Rapidly eliminated, 91% in 96 hours (86)	Oral 98.6% excreted within 96 hrs. (117)
PLANT METABOLISM	In cereals rapid hydrolysis to non-toxic metabolites. (117)	Rapidly metabolized to water soluble metabolites (117)
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate in fish (86)	Moderate potential to bioaccumulate, estimated BCF = 188 (155)

	ORYZALIN	OXADIAZON
ENVIRONMENTAL FATE		
MODE OF ACTION	Effects seed germination and prevents weed growth by inhibition of root development. (74)	Preemergent herbicide. (191) Selective contact herbicide (117)
HYDROLYSIS	None @ pH 5,7,9 (57)	pH 5.0 = half-life of 124 days. pH 7.0 = half-life of 114 days. pH 9.0 = half-life of 14 days. (191)
PHOTODEGRA-DATION	Subject to decomposition from UV light. (74) Half-life is 2-4 hours in aqueous solutions. (57)	Rapid photodegradation. Half-life: 30 minutes. (191)
SOIL HALF-LIFE	2.1 months (57)	180-365 days. (191) 3-6 months (117)
AQUATIC HALF-LIFE	Data not required (57)	DNF
SOIL LEACHING/ MOBILITY	Strongly adsorbed to the soil and resists movement by water. (74) K_{oc}: 848 ml/gm (155) No leaching below 6 inches. (57) Soil adsorption constant: 10.2 (loam soil) (297)	Immobile. (191) K_{oc}: 3,241 (162) K_{oc} 5,310 ml/gm (155) Strongly absorbed by soil colloids and humus with very little migration or leaching. (117)
SOIL METABOLISM	Rapid degradation (188)	Slowly degraded. Some CO_2 was formed. (188)
AQUATIC METABOLISM	Data not required (57)	DNF
ANIMAL METABOLISM	Rapidly metabolized and excreted in urine and feces. Rat, 97% is excreted in 72 hours. (74) No residues in milk or edible tissues. (57)	Oral admin. 93% eliminated within 72 hrs. After stopping the consumption of oxadiazon in their diet, cows and quail eliminated all residues within 12 days. (188) (117)
PLANT METABOLISM	Uptake and metabolism is minimal. Unmetabolized oryzalin is rarely detected. (57)	DNF
ACCUMULATION IN NON-TARGET SPECIES	Data no required (57) Potential to bioaccumulate, estimated BCF=368 (155)	Moderate potential to bioaccumulate, estimated BCF = 800 (155)

	PICLORAM	PRONAMIDE
ENVIRONMENTAL FATE		
MODE OF ACTION	Absorbed by plants roots and foliage and then translocated to the rest of the plant. Acts as a plant growth regulator. (276)	Systemic absorbs by roots with translocation. Inhibits photosynthesis. (117)
HYDROLYSIS	Half-life of 18 years (1 ppm @ pH 5,7,9 and 25°C). (202)	Stable @ pH 4.7-8.8 (87)
PHOTODEGRA-DATION	Rapid. (276) Half-life of 7 days. (202)	DNF
SOIL HALF-LIFE	Moderately to high persistent in soils. (276) > 4 years in arid regions and 1 month in favorable conditions. (276) 63 days in sandy loam soils. (277)	1 to 3 months depending on temperature and moisture content in the soil. High pH decreases half-life. 30 days @ 25°C (117)
AQUATIC HALF-LIFE	2.5 days (159)	DNF
SOIL LEACHING/ MOBILITY	Relatively mobile. K-salts and acids easily leach. (276) K_{oc}: 17 (162)	K_d = 3.15(clay) - 10.1(loam) (206) Low mobility and not easily leached (206,251) K_{oc}: 200 (162) K_{oc}: 980 ml/gm (155)
SOIL METABOLISM	Slow microbial degradation, especially in cold climates. (276) Microbial decomposition ceases in anaerobic conditions, but there is no adverse effect on soil microbes. (202)	Rate increases with temperature and moisture content. Primarily degraded by microbes. (207) Final degradate is CO_2 (207)
AQUATIC METABOLISM	Metabolites are non-halogenated. (159) Primarily degraded by photodegradation, especially in clear, shallow water. (202)	DNF
ANIMAL METABOLISM	Rapidly excreted unmetabolized in the urine. (276,277) 82-98% excreted as picloram (97) 97.7% eliminated in 5 days (cows). (202) Primarily excreted in urine by rats and dogs. (73)	DNF
PLANT METABOLISM	Accumulates in plants. (276) Degrades to CO_2, oxalic acid, and the metabolites 4-amino-2,3,5-trichloropyridine and 4-amino-3,5-dichloro-6-hydroxypicolinic acid (97)	DNF
ACCUMULATION IN NON-TARGET SPECIES	Does not accumulate. (276) BCF = 0.02 (162)	Has the potential to bioaccumulated, estimated BCF = 150 (155)

	SETHOXYDIM	SULFOMETURON METHYL
ENVIRONMENTAL FATE		
MODE OF ACTION	Systemic, selective broad spectrum postemergence herbicide. Absorbed by foliage and translocated to root system. (117)	Broad-spectrum herbicide. Absorbed rapidly by the roots and foliage then translocated throughout the plant. Stops cell division in the growing tips of roots and plants by mitotic inhibition. (117)
HYDROLYSIS	Unstable at pH5 but stable at pH 7 and 9. Half-lives of 8.7, 155 and 284 respectively (49)	In the dark, the acid is stable at pH 7 and 9 (approximately 10% loss after 30 days). At pH 5, the acid breaks down to methyl-2-(aminosulfonyl)-benzoate and saccharine. The dark half-life at pH 7 is approximately 14 days. For light condition see photodegradation. (118,119)
PHOTODEGRA-DATION	Soil: Half-life is 3.8 hrs. @ 25°C degrades to sulfoxide metabolite. Water: Half-life is 5.5 days @ pH 6.7 and 25°C. (27, 28)	Degradation in soil was 1.5 times faster in sunlight irradiated soil than in soil shaded from light (255). Photolysis plays a minor role in degradation of sulfometuron methyl in aqueous solutions. (204)
SOIL HALF-LIFE	Field half-life: 3 - 181 days. Low soil moisture increases half-life. (36) Aerobic half-life: 0.9 days in sandy loam. 0.7 days in sandy clay loam. Anaerobic half-life: 60 days in sandy loam. 30 days in sandy clay loam. (41,43)	23 - 34 days in soil. (255) 10 days in sandy loam soil. (277)
AQUATIC HALF-LIFE	Data under development (258)	While no data were found, photodegradation and hydrolysis data suggest that the half-life in neutral and alkaline waters for the acid would be a couple days. See hydrolysis.
SOIL LEACHING/ MOBILITY	No evidence of leaching by sethoxydim or its metabolites. No residues found greater than 8 inches. (36) K_{oc}: 48 ml/gm (155)	Inverse correlation between movement of sulfometuron methyl and adsorption on the soil. (69). Sulfometuron leaches through soils in the following order: Keyport silt loam > Fallsington sandy loam > Myakka sand > Flanagan silt loam. (76) K_{oc}: 89 ml/gm (155) K_{oc}: 171 ml/gm (101)
SOIL METABOLISM	DNF	Degraded mainly by hydrolysis, also by soil microbial action. In acid soils, hydrolysis is the most important factor, while microbial action is more important in neutral and alkaline soils. In addition to CO_2 and saccharin, at least three major aromatic sulfonyl breakdown products have been identified in soil. (5)
AQUATIC METABOLISM	Data under development (258)	DNF
ANIMAL METABOLISM	Rat: 98% excreted as sulfoxide and sulfone metabolites. 2% remained in body in liver and kidney (37,38,48)	93% excreted in the urine and feces after 7 days. Primary metabolite (hydroxymethyl) pyrimidine sufometuron methyl. Minor metabolites formed from cleavage are sulfonamide and (hydroxymethyl) pyrimidin-amine. Accumulation in the fat milk and meat was minimal. (168)
PLANT METABOLISM	Rapidly oxidizes to sulfoxides and sulfones. (30,32,50,47,26)	DNF
ACCUMULATION IN NON-TARGET SPECIES	Little tendency to bioaccumulate estimated BCF = 4 (155)	Minimal accumulation in goats (168) estimated BCF = 6 (155)

	TRICLOPYR
ENVIRONMENTAL FATE	
MODE OF ACTION	Selective systemic herbicide, absorbed by foliage and roots and translocated throughout plant and concentrated in meristematic tissue. Induces auxin-type responses in growing plants. (71)
HYDROLYSIS	In aqueous solution, the acid is stable in the dark, but breaks down very rapidly in the presence of light. The triethylamine (TEA) salt (Garlon 3A) and butoxyethyl ester (BEE) Garlon 4) break down rapidly in water to the acid. (71)
PHOTODEGRADATION	Rapid photodegradation, 8 - 10 hours. (71)
SOIL HALF-LIFE	Varies over a large range, depending primarily on soil moisture and temperature. It averages 30 days. (71)
AQUATIC HALF-LIFE	Rapid hydrolysis indicates that a short half-life would be expected in surface waters exposed to sunlight, but a longer half-life in deeper waters. (72)
SOIL LEACHING/ MOBILITY	The acid has low leaching potential. (71) K_{oc}: 27 (162) K_{oc}: 154 ml/gm (155)
SOIL METABOLISM	Undergoes rapid microbial breakdown in soil. Degradation is dependent on soil moisture and temperature. (71)
AQUATIC METABOLISM	Exists as a salt. Rapid degradation due to sunlight. (71)
ANIMAL METABOLISM	Is rapidly excreted primarily via urine as unaltered triclopyr. (72)
PLANT METABOLISM	DNF
ACCUMULATION IN NON-TARGET SPECIES	BCF = 0.02 (162)

Table 5-6 Wildlife toxicity and efficacy of active ingredients to be used by WSDOT for roadside vegetation management.

	2,4-D	2,4-DP
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	472 mg/kg (pheasant) (275) > 2,000 mg/kg (mallard) (275,277)	DNF
AVIAN (DIETARY) LC50	> 5,000 ppm (275) (Japanese quail, bobwhite, pheasant, mallard)	> 20,000 ppm. (191) > 10,000 ppm (mallard,quail) (98)
AVIAN REPRODUCTION	Not teratogenic (chicken, pheasant) (275) LC50: 215 kg/ha (mallard eggs) (275) (equivalent to field application rate)	Reduced chicken hatching success at 10 ppm and complete inhibition at 200 ppm. (277)
FISH LC50 (96 HR)	Amine > 100 ppm (rainbow trout) (277) 1.0 mg/l (rainbow trout) (273) > 100 ppm (chinook salmon) (277) 168 ppm (bluegill) (277) Butoxyethanol ester 10 ppm (rainbow trout) (277) 1.49 mg/l (rainbow trout) (273) 1.2 ppm (bluegill) (277)	Slightly toxic to fish. (276) 1.1 ppm (bluegill, 48 hrs) (277) Butoxyethyl ester: Highly toxic (273)
INVERTEBRATE LC50	Amine > 100 ppm (crayfish, 48 hrs) (277) 8 ppm (seed shrimp, 48 hrs) (277) No effect @ 2 ppm (eastern oyster, 96 hrs) (277) Butoxyethanol ester > 100 ppm (crayfish, 48 hrs) (277) EC50: 2.2 ppm (seed shrimp, 48 hrs) (277) EC50: 3.75 PPM (eastern oyster, 48 hrs) (277) 5.6 mg/l (daphnia, 96 hrs) (273)	Butoxyethyl ester: Highly toxic (273)
MARINE EC50	DNF	DNF
HONEY BEE EFFECTS	LD50: 11.5 - 105 ug/bee (276) Bees experienced a shortened lifespan from 1,000 ppm of purified 2,4-D, but not from 1,000 ppm of butoxyethanol ester, isoostyl ester or the dimethylamine salt. (276)	Not toxic to bees (117)

	BENEFIN	BROMACIL
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	> 2,000 mg/kg (187) (chickens, mallard, quail) (117)	2,250 mg/kg (bobwhite) (287)
AVIAN (DIETARY) LC50	> 5,000 ppm (bobwhite) (187) > 5,000 ppm (mallard) (187)	10,000 ppm (mallard, quail) (275,117)
AVIAN REPRODUCTION	Studies in progress. (187)	Data not required. (287)
FISH LC50 (96 HR)	65 ppb (bluegill) (187) 0.8 ppb (rainbow trout) (187) > 1.0 mg/l (flathead minnow) (154) 0.8 mg/l (goldfish) (154)	127 mg/l (bluegill) (287) 38 mg/l (rainbow trout) (287) 164 mg/l (carp) (117)
INVERTEBRATE LC50	>100 ppb (187) 1.1 mg/l (scud, 96 hrs) (154)	EC50: 119 mg/l (daphnia, 48 hrs) (287) 40 mg/l (crafish, 72 hrs) (273)
MARINE EC50	LC50 (96 hr): 43 ppm (mysid shrimp) (187) 1.7 ppm (sheepshead minnow, 7 days) (187) EC50 (96 hr for shell deposition): 100 ppb (oysters) (187)	EC50: 130 mg/l (48 hrs) (287)
HONEY BEE EFFECTS	LD50: > 105 ug a.i./bee (187)	No effects (117,287)

	CHLORSULFURON	CHLORTHAL DIMETHYL
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	> 5,000 mg/kg (bobwhite, mallard) (133,134,275)	DNF
AVIAN (DIETARY) LC50	> 5,000 ppm/day (bobwhite, mallard) (275,131,132)	DNF
AVIAN REPRODUCTION	Studies currently underway. (187)	DNF
FISH LC50 (96 HR)	>250 ppm (rainbow trout) (80) >300 ppm (flathead minnow) (80) >300 ppm (bluegill) (122)	Not toxic to fish. (117) 700 mg/l (bluegill, 48 hrs) (140)
INVERTEBRATE LC50	370.9 ppm (daphnia, 48 hrs) (121)	EC50: 0.25 mg/l (oyster, 96 hrs) (61)
MARINE EC50	Data not required. (187)	DNF
HONEY BEE EFFECTS	> 25 ug/bee (184)	Slightly toxic to bees. (117)

	DICAMBA	DICHLOBENIL
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	2000-2009 mg/kg (mallard) (275,256) 637 (female pheasant) (275)	1,000 ppm (quail) (270) >50 mg/kg (248)
AVIAN (DIETARY) LC50	>10,000 ppm (mallard, quail, 8 day) (275,256)	>5,200 mg/kg (mallard, bobwhite) (248,249,250) 1,500 mg/kg (pheasant) (269) > 5,000 mg/kg (Japanese quail) (269)
AVIAN REPRODUCTION	Decreased hatching at 400 ppm but not at 300 ppm. (275)	Data not required. (81)
FISH LC50 (96 HR)	28 mg/l (rainbow trout) (275) 50-135 mg/l (bluegill) (273,256) 135 mg/l (rainbow trout) (256) >180 mg/l (sheepshead minnow) (256)	5-25 mg/l (depending on species) (114) 18 mg/l (guppies) (117) 15-30 mg/l (rainbow, bluegill, bass) (268) 8.1 mg/l (rainbow trout) (265) 11.0 mg/l (bluegill) (273)
INVERTEBRATE LC50	> 100 mg/l @ 15-21°C (6 spp freshwater crustaceans, 48 hrs) (61) 111 mg/l (daphnia) (256) >100 mg/l (*Grammarus fasciatus*) (273) EC50: 11 mg/l (daphnia, 48 hrs) (275)	6.2 mg/l (48 hrs) (60) EC50: 4-10 mg/L (daphnia, 48 hrs) (269,297) EC50: 0.3 ppm (D. pulex) (262)
MARINE EC50	>100 mg/l (grass shrimp) (256) >180 mg/l (fiddlers crab) (256)	Data not required. (81)
HONEY BEE EFFECTS	LD50 contact: > 100 ug/bee (260) oral: >10 ug/bee (260) Essentially non-toxic (260)	LD50 contact > 100 ug/bee (9) oral > 10 ug/bee (9) Essentially non-toxic (9)

	DIURON	FLUAZIFOP-BUTYL
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	> 2,000 mg/kg (mallard) (275,84)	>17,000 mg/kg (mallard) (117)
AVIAN (DIETARY) LC50	> 5,000 ppm (mallard, pheasant) (275) 1,730 ppm (bobwhite) (275,84)	>25,000 ppm (mallard) (117) >18,500 ppm (pheasant) (117)
AVIAN REPRODUCTION	Data not required. (287)	DNF
FISH LC50 (96 HR)	3.5 ppm (rainbow trout) (79) 5.6 mg/l (trout) (117) 5.9 mg/l (bluegill) (117) 0.25 mg/l (guppies) (117) 3-60 ppm (84)	1.37 mg/l (trout) (117) 1.31 mg/l (carp) (117) 0.53 mg/l (bluegill) (117)
INVERTEBRATE LC50	Toxic to lower aquatic organisms. (276,275) 0.16 mg/l (scud) (275) 1.8 mg/l (oyster) (61)	Low toxicity to aquatic invertebrates (117)
MARINE EC50	EC50: 3.2 mg/l (oyster, 96 hrs) (287)	DNF
HONEY BEE EFFECTS	No effects (287)	Very low toxicity both orally and by contact. (117) >0.2 mg/bee (297)

	FOSAMINE	GLYPHOSATE
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	> 5,000 mg/kg (bobwhite) (244) > 5,000 mg/kg (mallard) (243,276)	> 2,000 mg/kg (quail) (275,95)
AVIAN (DIETARY) LC50	> 10,000 ppm (mallard, bobwhite) (245,239,277)	> 4,640 ppm (mallard, quail) (194)
AVIAN REPRODUCTION	Low embryotoxicity (277)	No effect in long term feeding study with bobwhites up to 2,150 ppm in feed. No adverse effect on mallards. (8) NOEL: >1,000 ppm (mallard,quail) (95)
FISH LC50 (96 HR)	8.9 ppm cause avoidance in salmon.(276) > 300 mg/l (rainbow trout) (10) 590 mg/l (bluegill) (11) 670 ppm (bluegill) (277)	86-170 mg/l (rainbow trout) (194,95) 10-197 mg/l (rainbow trout) (257) 120 mg/l (bluegill) (194,95) 24-140 mg/l (bluegill) (98) 115 mg/l (carp) (194) 97 mg/l (fathead minnow) (194)
INVERTEBRATE LC50	1,524 ppm (daphnia, 48hr) (120) 3,547 ppm (crayfish) (277) > 100 ppm (midge, 48 hrs) (277)	43-780 mg/l (daphnia, 48 hrs) (194) 281 mg/l (grass shrimp) (194)
MARINE EC50	Data not required (109)	281 mg/l (grass shrimp) (194) 934 mg/l (fiddler crab) (194) > 10 mg/l (oyster) (194)
HONEY BEE EFFECTS	> 10,000 ppm when sprayed. (277) > 200 ug/bee (277,183)	> 100 ug/bee (194)

	IMAZAPYR	ISOXABEN
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	>2,150 mg/kg (mallard, quail) (3)	> 2,000 mg/kg (bobwhite) (75,156)
AVIAN (DIETARY) LC50	>5,000 ppm diet (mallard, quail) (3)	> 5,000 ppm (bobwhite, mallard) (75,156)
AVIAN REPRODUCTION	Data not required by EPA. (177)	NOEL: 98.6 mg/kg (bobwhite) (156) NOEL: 60.0 mg/kg (mallard) (156)
FISH LC50 (96 HR)	>100 mg/l (rainbow trout, bluegill, channel catfish). (3)	> 1.1 mg/l (rainbow trout, bluegill) (75,156) Solubility limits effective dose. (156)
INVERTEBRATE LC50	> 100 mg/l (water flea, 48 hrs) (3,277)	> 100 ppm (earthworm) (75) EC50: > 1.3 mg/l (daphnia) (75,156)
MARINE EC50	Currently under EPA review. (177)	DNF
HONEY BEE EFFECTS	Contact LD50: >100 ug/bee (3)	LD50 > 100 ug/bee (75) Non-toxic at 101.7 ug/bee (156)

	MECOPROP	MEFLUIDIDE
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	500 mg/kg (quail) (297) 700 mg/kg (quail) (253) >486 mg/kg (mallard) (176)	>4,640 mg/kg (mallard) (275) NOEL: 2,000 mg/kg (bobwhite) (234)
AVIAN (DIETARY) LC50	>5,620 ppm (mallard) (176) 5,000 ppm (quail) (155)	> 10,000 ppm (mallard,bobwhite) (275) Technical: NOEL: >5,000 ppm (bobwhite,mallard) (226,228) Diethanolamine salt: >5,000 ppm (bobwhite, mallard) (227,232)
AVIAN REPRODUCTION	DNA	Pending EPA requirement of phase IV review. (6)
FISH LC50 (96 HR)	150-220 mg/l (rainbow trout) (176,297) low toxicity to fish (117) 124 ppm (rainbow trout) (155)	Technical: Study in progress. (6) Diethanolamine salt: > 94.4 mg/l (bluegill) (238) >91.3 mg/l (rainbow trout) (238)
INVERTEBRATE LC50	DNF	Technical: Study in progress. (6) Diethanolamine salt: NOEL: 103 mg/l (daphnia) (229)
MARINE EC50	DNF	Study in progress. (6)
HONEY BEE EFFECTS	Not toxic to bees (117)	Study in progress. (6)

	METSULFURON METHYL	NAPROPAMIDE
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	> 2,510 mg/kg (mallard) (275,293)	>486 mg/kg (mallard) (165)
AVIAN (DIETARY) LC50	> 5,620 ppm (mallard, quail) (275,291,294) 75,620 ppm (quail) (86)	>5,600 ppm (quail) (297) >5,600 ppm (mallard) (165)
AVIAN REPRODUCTION	DNF	DNF
FISH LC50 (96 HR)	> 150 ppm (bluegill, trout) (125,86)	1.3 mg/l (bluegill) (165) 6.6 mg/l (trout) (165)
INVERTEBRATE LC50	> 150 ppm (daphnia, 48 hrs) (126,86)	14.3-28 mg/l (daphnia, 48 hrs) (165,155) 18 mg/l (pink shrimp, 96 hrs) (155)
MARINE EC50	DNF	LC50: 42 mg/l (mysid shrimp) (165)
HONEY BEE EFFECTS	>25 ug/bee (185,86)	Not toxic to bees (117) 0.121 mg/bee (165,297)

	ORYZALIN	OXADIAZON
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	>500 mg/kg (quail, mallard) (117) 507 ppm (57)	>1,000 mg/kg (mallard) >6,000 mg/kg (quail) (191,117)
AVIAN (DIETARY) LC50	>5,000 ppm (57)	NOEL: 5,000 ppm (bobwhite) 5,000 ppm (8 day) (191)
AVIAN REPRODUCTION	NOEL: >1,000 ppm (57)	No information. (191)
FISH LC50 (96 HR)	>1.4 (goldfish) (297) 2.88 mg/l (bluegill) (57) 3.26 mg/l (trout) (57,117)	1.76 (carp) (297) >15.4 (catfish) (297) Toxic to fish (117)
INVERTEBRATE LC50	71.7 ppm (57) 0.19 mg/l (scud) (275)	0.5 - 8.0 mg/l (daphnia) (297)
MARINE EC50	Data not required	DNF
HONEY BEE EFFECTS	Not dangerous to bees if used as directed. (117) 0.011 mg/bee (oral) (297)	No information. (191) Toxic to bees (117)

	PICLORAM	PRONAMIDE
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	>2,000 mg/kg (mallard, pheasant) (277) 2,250 mg/kg (quail) (97)	>14,000 mg/kg (mallard) (178) 8,770 mg/kg (Japanese quail) (178) 20,000 mg/kg (mallard) (87)
AVIAN (DIETARY) LC50	>5,000 ppm (bobwhite, Japanese quail, pheasant and mallard) (277,97)	>10,000 ppm (bobwhite) (178,87) >10,000 ppm (mallard) (178,87)
AVIAN REPRODUCTION	No effect (277)	DNF
FISH LC50 (96 HR)	4.3-19.3 (rainbow trout) (97) 24-34 ppm (rainbow trout) (277) 3.1 mg/l (rainbow trout) (273) 21-26.5 ppm (bluegill) (277) 13.5 mg/l (bluegill) (273) 14.5-23 ppm (bluegill) (97) 27 mg/l (scud) (273)	72 mg/l (rainbow trout) (179,87) 54 mg/l (rainbow trout) (180) 150 mg/l (guppy) (87,179) 350 mg/l (goldfish) (87,179) >7,200 < 500 mg/l (catfish) (87,179) 150-375 mg/l (catfish) (180) >100 mg/l (bluegill) (87)
INVERTEBRATE LC50	10-68.3 ppm (daphnids, gammaras, pterorarcella, pteronarcys) (97) NOEL: 1 ppm (brown shrimp, 48 hrs) (277) NOEL: 380 ppm (water flea, 48 hrs) (277) 50.7 mg/l (daphnia, 48 hrs) (273)	>5.6 mg/l (daphnia, 48 hr) (180,87)
MARINE EC50	>1,000 ppm (11.6% K salt) (oyster, pink shrimp, fiddler crab) (159)	DNF
HONEY BEE EFFECTS	LD50: 14 ug/bee (277,97)	Not dangerous to bees. (117)

	SETHOXYDIM	SULFOMETURON METHYL
WILDLIFE AND AQUATIC ORGANISMS		
AVIAN LD50	5,620 mg/kg (mallard) (16)	>5,000 mg/kg (mallard) (130,277)
AVIAN (DIETARY) LC50	5,620 mg/kg (mallard) (21) >5,620 mg/kg (quail) (21)	>5,000 ppm (mallard) (78,129,277) >5,620 ppm (quail) (78,292,277)
AVIAN REPRODUCTION	Data not required (258)	DNF
FISH LC50 (96 HR)	8.24 ppm (bluegill) (19) 6.22 ppm (trout) (17)	>12.5 ppm (rainbow trout) (78,124) >12.5 ppm (bluegill) (123,277) 45 mg/l (sheepshead minnow) (285)
INVERTEBRATE LC50	13.47 ppm (daphnia) (22)	>1,000 ppm (earthworm) (186) >12.5 ppm (water flea, 48 hrs) (277) >5,000 ppm (crayfish, 96 hrs) (127,277) 38.2 mg/l (oyster embryos and larvae) (284)
MARINE EC50	4.4 pp.m (shrimp) (52) 5.0 ppm (oyster) (34) 19.4 ppm (sheepshead minnow) (29)	DNF
HONEY BEE EFFECTS	>10.0 mg/bee (33) Not toxic to bees (117)	> 12.5 ug/bee (277)

	TRICLOPYR
WILDLIFE AND AQUATIC ORGANISMS	
AVIAN LD50	1,698 mg/kg (mallard) (71)
AVIAN (DIETARY) LD50	> 5,600 ppm (mallard) (71) 2,935 ppm (quail) (71)
AVIAN REPRODUCTION	NOEL: > 500 ppm (mallard, quail) (71)
FISH LC50 (96 HR)	117 mg/l (rainbow trout) (71) BE ester: 0.74 mg/l (rainbow trout) (155) TEA salt: 552 mg/l (rainbow trout) (155) 148 mg/l (bluegill) (71)
INVERTEBRATE LC50	133 mg/l (daphnia, 48 hrs) (71) 56-87 mg/l (oyster, 48 hrs) (275) TEA salt: 5,687 mg/l (oyster, 48 hrs) (155)
MARINE EC50	TEA salt: >1,000 mg/l (crab) (155) TEA salt: 895 mg/l (shrimp) (155)
HONEY BEE EFFECTS	DNF

Table 5-7 Physical data, toxicity information, environmental fate and wildlife toxicity of the inert ingredients found in the herbicides to be used by WSDOT for roadside vegetation management. Diesel is an adjuvant but is considered here because it is a petroleum distillate.

	Petroleum Distillates (diesel, kerosene, naphtha)	Xylene
Manufacturer	Numerous	Numerous (228)
Physical Data		
Ingredients	Hydrocarbons	o-xylene m-xylene p-xylene (228)
Signal Word/ Toxicity Class	DNF	DNF
Mode of Action	Wetting and dispersing agents.	Unknown
Solubility	Insoluble in water (113,110,271,205)	Insoluble in water (112)
Stability	Stable in closed containers at room temperature. Extremely flammable! (113,110,271,205)	Stable in closed containers (112)
pH	DNF	DNF
Boiling Point	170 to 400°C (113,110,205)	275-293°F (112)
Melting Point	Variable (205)	-13°F (112)
Vapor Pressure	Variable (205)	7-9 torrs @ 68°F (112)
% Volatile	About 100% (110,111)	100% (112)
Toxicity Data		
LD50	2,800mg/kg (kerosene) to 9,000 mg/kg (diesel) (rat) (113,110)	4,300 mg/kg (rat) (112)
Acute Dermal	DNF	DNF
Acute Inhalation	DNF	> 10,000 ppm/6hr (human) (112)
Eye Irritation	Irritant (113,110,271)	Irritant (112)
Skin Irritation	Irritant (113,110,271)	Irritant (112)
Environmental Fate	Half-lives: Soil: 16.6 - 48 days (137) Water: 20 days (aerobic) (137) Water: 25-258 days (anaerobic) (137) Does not hydrolyze (137)	Half-lives: Soil: 1 to 4 weeks (137) Water: 1 to 4 weeks (aerobic) (137) Water: 6 to 12 months (anaerobic) (137) Does not hydrolyze. (137)
Wildlife and Aquatic Toxicity	LC50: >0.19 mg/l (freshwater fish, 96 hrs) (67,273) (Diesel) LC50: 0.21 mg/l (pink shrimp) (No. 2 fuel) (273)	TLm: 22 ppm (bluegill, 96 hrs) (272) TLm: >100 (D. magna, 96 hrs) (272) LC50: 20.87 mg/l (bluegill, 96 hrs) (65) LC50: 26.7 mg/l (fathead minnow, 96 hrs) (65) LC50: 7.4 mg/l (grass shrimp, 96 hrs) (65)

Table 5-8 Physical data, toxicity information, environmental fate and wildlife toxicity information for the adjuvants used for roadside vegetation management by WSDOT.

	Blazon	Get-Down
Manufacturer	Milliken Chemical Company	Exacto Chemical Company
Physical Data		
Ingredients	Polymer bound aromatic organic colorant. (193)	Polyacrylamide (105)
Signal Word/ Toxicity Class	DNA	Caution/III (105)
Mode of Action	Colors the spray to identifiy areas that have been sprayed. (193)	Increases the deposition and coverage of a spray while reducing drift by increasing the surface tension of the spray droplets. (105)
Solubility	Soluble in water. (193)	Soluble in water. (105)
Stability	Stable. (193)	Stable. (105)
pH	7.0 (193)	NA
Boiling Point	215°F (193)	NA
Melting Point	DNF	DNA (105)
Vapor Pressure	18 mmHg (193)	NA
% Volatile	60% @ 100°C	NA
Toxicity Data		
LD50	>5,000 mg/kg (rat) (193)	> 2,500 mg/kg (rat) (105)
Acute Dermal	> 2,000 mg/kg (rabbits) (193)	> 10,000 mg/kg (rabbit) (105)
Acute Inhalation	DNF	DNF
Eye Irritation	Not an irritant. (193)	Non-irritating (105)
Skin Irritation	Not a skin irritant. (193)	Non-irritating (105)
Environmental Fate	Decomposition may produce fumes, smoke, oxides of carbon and /or hydrocarbons. (193)	Thermal decomposition may produce carbon monoxide, carbon dioxide and/or oxides of N.
Wildlife and Aquatic Toxicity	> 10,000 ppm (bluegill, daphnia) (193)	DNF

	LI-700	Mor-Act
Manufacturer	Loveland Industries	Wilbur-Ellis Company
Physical Data		
Ingredients	Mixture of phosphatidylcholine and methylacetic acid. Hazardous ingredient: Propionic acid. (181)	Non-phytotoxic paraffin based petroleum oil Polyol fatty acid esters and polythoxylated derivatives. (290)
Signal Word/ Toxicity Class	DNF	Caution/III (290)
Mode of Action	DNF	Combines the penetrating activity of phytobland oil with the wetting and dispersing characteristics of a surfactant. (290)
Solubility	Miscible-forms emulsion. (181)	Emulsifiable. (290)
Stability	Stable. (181)	Stable. (290)
pH	DNF	DNF
Boiling Point	Not established. (181)	DNF
Melting Point	DNF	DNF
Vapor Pressure	Not established. (181)	< 0.1 @ 20°C (290)
% Volatile	Not established. (181)	DNA (290)
Toxicity Data		
LD50	DNF	DNF
Acute Dermal	Irritant. (181)	DNF
Acute Inhalation	DNF	DNF
Eye Irritation	Irritant. (181)	Mildly irritating. (290)
Skin Irritation	Irritant. (181)	Mildly irritating. (290)
Environmental Fate	DNF	DNF
Wildlife and Aquatic Toxicity	DNF	DNF

	More	Nalcotrol
Manufacturer	Exacto Chemical Company	Monterey Chemical Company
Physical Data		
Ingredients	Polyvinly polymer Unidentified inert ingredients (104)	Ethoxylated octylphenol Hydrotreated light distillate (200)
Signal Word/ Toxicity Class	Caution/III	Caution/III (200)
Mode of Action	Deposition improvement and drift retardation. (104)	Mist and Drift control and deposition improvement. (200)
Solubility	Appreciably soluble in water. (104)	Dispersible in water. (200)
Stability	Stable. (104)	DNF
pH	4-6 (diluted with water) (104)	DNF
Boiling Point	>212F (>100°C) (104)	DNF
Melting Point	0F (-18°C) (104)	DNF
Vapor Pressure	DNA (104)	DNF
% Volatile	70% (104)	DNF
Toxicity Data		
LD50	> 10 ml/kg (rat) (104)	> 5,000 mg/kg (albino rat) (200)
Acute Dermal	> 10 ml/kg (rabbit) (104)	DNF
Acute Inhalation	DNF	DNF
Eye Irritation	Irritant (104)	Moderately irritating. (rabbit) (200)
Skin Irritation	Irritant (104)	Slightly irritating. (rabbit) (200)
Environmental Fate	Thermal decomposition or combustion may produce carbon monoxide, carbon dioxide and/or oxides of N. (104)	Thermal decomposition products may include carbon monoxide, carbon dioxide and/or oxides of nitrogen. (200)
Wildlife and Aquatic Toxicity	DNF	Essentially non-toxic to rainbow trout and sheepshead minnows. Slightly toxic to mysid shrimp and daphnia. (200)

	No Foam	Odor Mask
Manufacturer	Monterey Chemical Company	Monterey Chemical Company
Physical Data		
Ingredients	POE Nonylphenol Isopropyl alcohol (198)	Methyl salicylate hydroxybenzoic acid ester. (199)
Signal Word/ Toxicity Class	Caution/III (198)	DNF
Mode of Action	Insures quick wetting and uniform coverage of agricultural chemicals. (198)	Masks undesirable odors. (199)
Solubility	Miscible. (198)	Negligible. (199)
Stability	Stable. (198)	Stable. (199
pH	6.8 (198)	DNF
Boiling Point	210°C (198)	222.8°C (199)
Melting Point	DNF	DNF
Vapor Pressure	0.5 psi (198)	<1 mmHG @ 20°C (199)
% Volatile	NA (198)	DNF
Toxicity Data		
LD50	>5,000 mg/kg (rat) (198)	887 mg/kg (rat) (199)
Acute Dermal	DNF	DNF
Acute Inhalation	May be an irritant to nasal membranes.	May irritate nasal membranes. (199)
Eye Irritation	Mild irritant. (198)	Irritant. (199)
Skin Irritation	Mild irritant. (198)	Mild irritant. (199)
Environmental Fate	Carbon oxides may result from burning. (198)	Burning will generate carbon monoxide, phenols and other toxic fumes. (199)
Wildlife and Aquatic Toxicity	DNF	DNF

	R-11	Sta-Put
Manufacturer	Wilbur Ellis Company	Monterey Chemical Company
Physical Data		
Ingredients	Isopropyl alcohol Non-ionic surfactants (289)	Isopropanol (1-10%) (197)
Signal Word/ Toxicity Class	Caution/III (289)	Caution/III (197)
Mode of Action	Increases absorption and translocation. (289)	Improve spray deposition and retard spray drift. (197)
Solubility	Soluble. (289)	Completely soluble. (197)
Stability	Stable. (289)	DNF
pH	DNF	DNF
Boiling Point	DNF	212°F @ 760 mmHg. (197)
Melting Point	DNA (289)	DNF
Vapor Pressure	33 @ 20°C. (289)	DNF
% Volatile	DNA (289)	DNF
Toxicity Data		
LD50	5,840 mg/kg (rat) (289)	No toxicity studies available. (197)
Acute Dermal	13 mg/kg (rabbit) (289)	No toxicity studies available. (197)
Acute Inhalation	DNF	No toxicity studies available. (197)
Eye Irritation	Irritant. (289)	Irritant. (197)
Skin Irritation	Irritnat. (289)	Irritant. (197)
Environmental Fate	Carbon monoxide and carbon dioxide are thermal decomposition products. (289)	Thermal decomposition may produce carbon monoxide, carbon dioxide and oxides of nitrogen. (197)
Wildlife and Aquatic Toxicity	DNF	DNF

	Super Spred 200	X-77
Manufacturer	Wilbur Ellis Company	Valent
Physical Data		
Ingredients	Isopropyl alcohol Triton X-114 (288)	Alkylarylpolyoxyethylene glycols Fatty acids Isopropyl alcohol (278)
Signal Word/ Toxicity Class	Caution/III (288)	Danger/I (278)
Mode of Action	Improves leaf coverage and chemical deposition. (288)	Increases coverage and retention of sprays by reducing surface tension of the spray droplets. (278)
Solubility	Soluble. (288)	Soluble in water. (278)
Stability	Stable. (288)	Stable (278)
pH	DNF	DNF
Boiling Point	DNF	175-176°F (278)
Melting Point	DNA (288)	DNA (278)
Vapor Pressure	Nil @ 20°C (288)	DNA (278)
% Volatile	10% (288)	20% (278)
Toxicity Data		
LD50	Isopropanol: 5,840 mg/kg (rat) (288) Triton X-114: 7,500 mg/kg (rat) (288)	> 5,000 mg/kg (rat) (278)
Acute Dermal	Isopropanol: 1,300 mg/kg (rabbit) (288) Triton X-114: > 3,000 mg/kg (rabbit) (288)	DNA (278)
Acute Inhalation	DNF	DNA (278)
Eye Irritation	Severe irritant. (288)	Corrosive (278) May cause irreversible eye damage.
Skin Irritation	Mild irritant. (288)	Moderate irritant (278)
Environmental Fate	Thermal decomposition may produce carbon monoxide and carbon dioxide. (288)	Combustion products are carbon dioxide and water. (278)
Wildlife and Aquatic Toxicity	DNF	Toxic to aquatic organisms. (278)

Active Ingredient: **2,4-D**

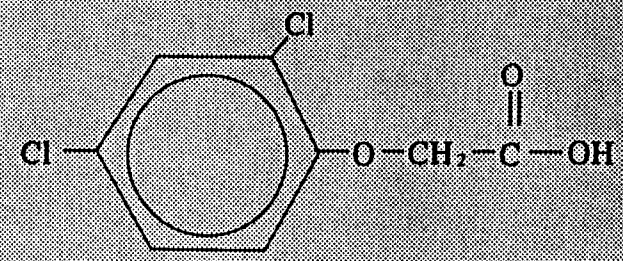
Product(s): Amine #4; Envy; Formula 40; Trimec Classic; Visko-Rhap (A-3D, LV2D); Weedar 64; Weedone LV4; Weedone 170; Weedmaster; Crossbow; Tordon (RTU, Mixture 101)

Maximum Label Rate: (Weedone 170) 16.65 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray

Mode of Action: Post-emergence control of broadleaf weeds

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	2.4	0.6	5	850	pigweed, curly dock, clover, japanese knotweed, willow, salsify, toadflax,mullien, maple, cedar, cottonwood, fir/evergreens, salmonberry, thistle, alder, tansy, pine, blackberry, knapweed, scotchbroom, ragweed, nightshade, chickory
2	2.6	0.29	6	22160	knapweed, toadflax, thistles, mullien, sumac, puncturevine, bindweedgoatshead, bitterbrush, poison oak/ivy, whitetop, clover, mustard, sagebrush, alder,tarweed, morning glory, cattail, loosestrife, prickly, lettuce, willow, fireweed
3	3.1	1	4	490	blackberry, willow, thistles, tansy, alder, horsetail, scotchbroom, elderberry,maple, elderberry, fir/evergreens, seeds and grasses
4	1.9	0.4	5.5	4930	thistles, blackberry, mullien, alder, tansy, knapweed, maple, cedar,cottonwood, toadflax, scotchbroom, willow, morning glory, strife, rose, ash, huckleberry,cascara, daisy, St. Johnswort, Queen Annes lace, salmonberry, carrot, nightshade, vetchmore
5	1.9	0.9	4	16480	thistles, mullien, knapweed, cedar, fir/evergreens, toadflax, scotchbroom, chickory, morning glory, pigweed, kochia, sumac, prickly lettuce, whitetop, rose, mustard, pepperwood, tumbleweed
6	1.08	0.23	4.3	3020	thistle, mullien, knapweed, toadflax, scotchbroom, kochia, morning glory,clover, mustard, St. Johnswort, hoary cress, rye

Table 5-9 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for 2,4-D.

* Note - Total pounds of active ingredient used for each district are combinationsof actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **2,4-DP**

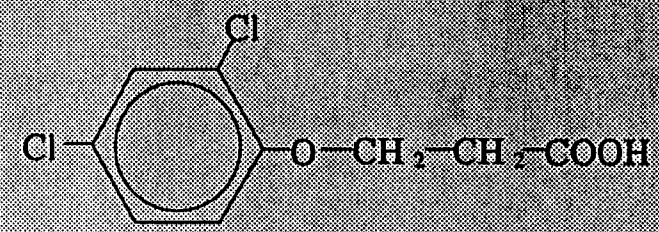
Product(s): Weedone 170

Maximum Label Rate: 16.65 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray.

Mode of Action: Post-emergence control of woody plants.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	1.62	0.49	1.85	80	thistles, alder, blackberry, knapweed, maple, toadflax, tansy, cottonwood, pigweed, fir/evergreens, scotchbroom, curly dock
2	Not used	Not used	Not used	Not used	
3	0.98	0.98	0.98	40	alder, thistles, blackberry, willow, tansy
4	1.02	1.02	1.02	220	fir/evergreens, thistles, tansy, scotchbroom, blackberry, alder, pine, rose, maple
5	Not used	Not used	Not used	Not used	
6	↓	↓	↓	↓	

Table 5-10 WSDOT Roadside Vegetation Management DEIS. Herbicide uses rates and information for 2,4-DP.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Benefin**

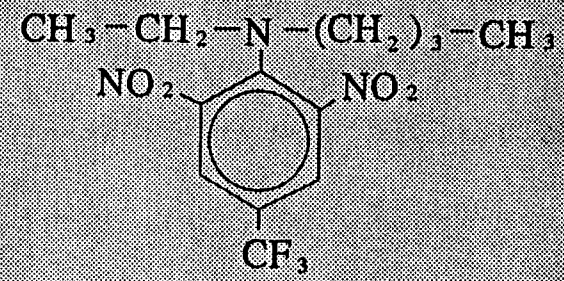
Product(s): XL-2G

Maximum Label Rate: 3 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray.

Mode of Action: Selective pre-emergence control of annual grasses and certain broadleaf weeds.

Chemical Structure



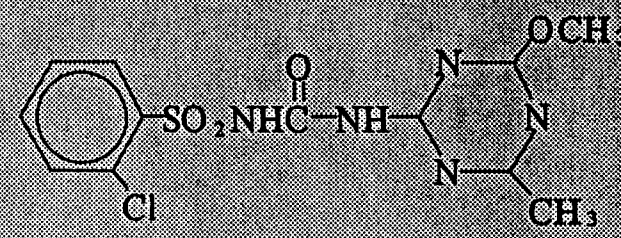
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	1.5	1	2	10	weeds, clover, grass, horsetail
2	Not used	Not used	Not used	Not used	
3	2.6	3	3	210	grass, broadleaf weeds, seeds
4	Not used	Not used	Not used	Not used	
5	↓	↓	↓	↓	
6	↓	↓	↓	↓	

Table 5-11 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for benefin.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Chlorsulfuron methyl**
Product(s):	Telar
Maximum Label Rate:	0.14 lbs active ingredient (ai)/acre
Application Method(s):	Foliar an soil-applied spray
Mode of Action:	Pre and postemergence control of annual and perennial broadleaf weeds and grasses.

Chemical Structure



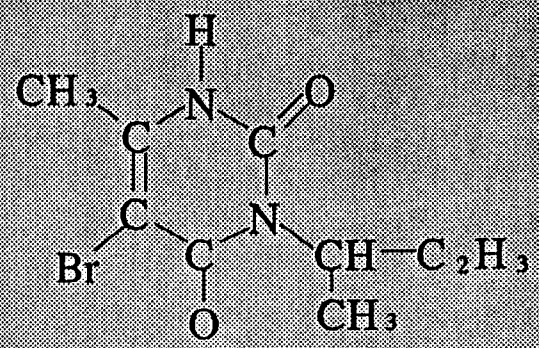
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	0.11	0.07	0.13	40	horsetail, thistle, knapweed, blackberry, tansy
2	0.12	0.06	0.13	270	sumac, cattail, thistles, knapweed, toadflax, mullien
3	0.09	0.08	0.09	3	horsetail, thistle, grass
4	0.09	0.05	0.14	30	horsetail, blackberry, mullien, clover, toadflax
5	0.06	0.03	0.14	110	seeds, rose, thistles, kochia, knapweed, mullien, tumbleweed, morning glory
6	0.10	0.09	0.13	220	kochia, toadflax, mullien, thistle, knapweed, St. Johnswort, horsetail

Table 5-12 | WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for chlorsulfuron methyl.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Bromacil**
Product(s):	Krovar 1DF
Maximum Label Rate:	12 lbs active ingredient (ai)/acre
Application Method(s):	Foliar and soil-applied spray.
Mode of Action:	Pre and early post-emergence control of annual and perennial broadleaves and grasses.

Chemical Structure



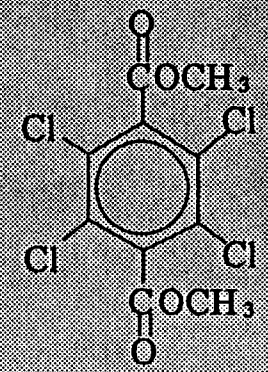
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990).				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	2	1.5	2.5	1350	weeds, grasses.
2	Not used	Not used	Not used	Not used	
3	2.1	0.9	2.5	830	all vegetation
4	2	2	2	980	weeds, grasses, broadleaf weeds, clover
5	1.8	1.2	3.2	980	weeds, seeds
6	2.3	1.2	2.4	1360	weeds, grasses, thistles, toadflax, mullien

Table 5-13 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for bromacil.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Chlorthal dimethyl**
Product(s):	Dacthal 75W
Maximum Label Rate:	15 lbs active ingredient (ai)/acre
Application Method(s):	Soil-applied spray
Mode of Action:	Preemergence control of annual grasses and broadleaf weeds.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	Not used	Not used	Not used	Not used	
2	↓	↓	↓	↓	
3	↓	↓	↓	↓	
4	15	15	15	400	broadleaves and grasses
5	10.5	10.5	10.5	170	all vegetation
6	Not used	Not used	Not used	Not used	

Table 5-14 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for chlorthal dimethyl.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Dicamba**

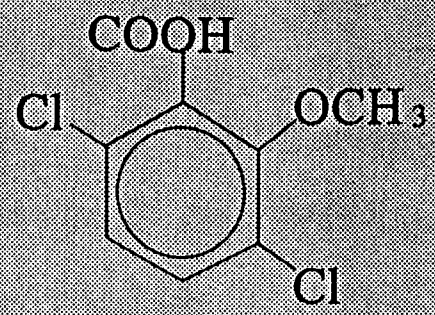
Product(s): Banvel; Weedmaster; Trimec Classic

Maximum Label Rate: 8 lbs active ingredient (ai)/acre

Application Method(s): Foliar applied spray.

Mode of Action: Postemergent control of broadleaf vegetation.

Chemical Structure



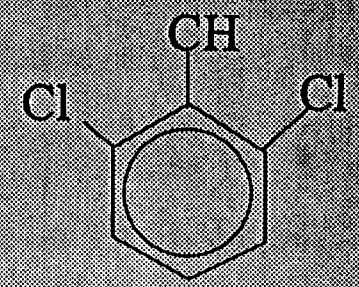
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	0.46	0.04	4	400	thistles, alder, tansy, blackberry, maple, cedar, knapweed, toadflax, cottonwood pine fir/evergreens, scotchbroom, ragweed, horsetail, salsify, chickory, nightshade,clover willow, japanese knotweed, pigweed, curly dock, mullien
2	1.41	0.003	4	4770	thistles, mullien, alder, knapweed, toadflax, clover, kochia, sumac, puncturevine, bindweed, cattail, mustard, horsetail, bitterbrush, willow, morning glory prickly lettuce, sagebrush, maple, cottonwood, milkweed, goatshead, firweed
3	2.8	0.5	4	530	thistles, blackberry, alder, tansy, knapweed, maple, fir/evergreens, toadflax, willow, scotchbroom, broadleaves, seeds, elderberry, gorst
4	1.04	0.14	4	1130	thistles, blackberry, mullien, alder, tansy, knapweed, maple, cottonwood, pine, St Johnswort, fir/evergreens, toadflax, scotchbroom, morning glory, willow, carrot, nightshade, elderberry, ash, rose, huckleberry, cascara, daisy, groundsel,
5	0.57	0.07	5	4790	thistles, mullien, knapweed, cedar, fir/evergreens, toadfalx, scotchbroom, rose, morning glory, chickory, pigweed, kochia, sumac, prickly lettuce, pepperwood, tumbleweed
6	0.45	0.0001	2.9	1040	thistles, mullien, knapweed, toadflax, scotchbroom, morning glory, clover, kochia, mustard, St Johnswort, hoarycress, rye

Table 5-15 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for dicamba.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Dichlobenil**
Product(s):	Casoron 4G; Casoron 50W; Norosac 4G
Maximum Label Rate:	20 lbs active ingredient (ai)/acre
Application Method(s):	Broadcast and soil-applied spray.
Mode of Action:	Preemergence control of annual and perennial and grasses.

Chemical Structure



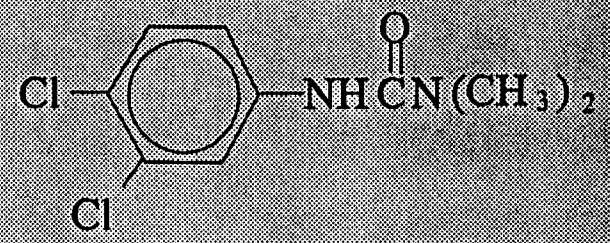
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990).				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	4.4	2	6	1810	grasses, horsetail, seeds
2	Not used	Not used	Not used	Not used	
3	8	4	12	40	grasses, seeds, broadleaves, horsetail
4	2	2	2	Unknown	horsetail
5	5.3	4	6	60	grasses and broadleaves
6	Not used	Not used	Not used	Not used	

Table 5-16 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for dichlobenil.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Diuron**
Product(s):	Karmex DF; Direx 4L; Krovar 1DF
Maximum Label Rate:	40 lbs active ingredient (ai)/acre
Application Method(s):	Foliar and soil-applied spray
Mode of Action:	Pre and early postemergence for control of annual and perennial broadleaves and grasses.

Chemical Structure



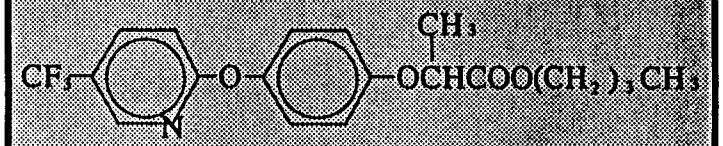
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990).				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	3.5	1.5	16	7120	grasses, blackberry, tansy, horsetail
2	6	1.7	10	12410	grass, sumac
3	3.3	0.8	5	2900	grass, all vegetation
4	2.7	1	6	7870	cottonwood, grass, broadleaves, clover, horsetail, thistle,
5	1.8	1	21	5460	seeds, broadleaves
6	2.7	1.2	4	2970	grass, toadflax, thistles, mullien, kochia

Table 5-17 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for diuron.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Fluazifop-butyl**
Product(s):	Fusilade 2000
Maximum Label Rate:	0.38 lbs active ingredient (ai)/acre
Application Method(s):	Foliar-applied spray
Mode of Action:	Postemergence control of annual and perennial grasses.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990).				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total Used (lbs ai)	
1	Not used	Not used	Not used	Not used	
2					
3					
4					
5					
6					

Table5-18 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for fluazifop-butyl.

Active Ingredient: **Fosamine ammonium**

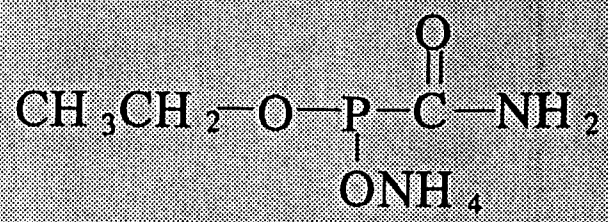
Product(s): Krenite S

Maximum Label Rate: 24 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray

Mode of Action: Control of woody species

Chemical Structure



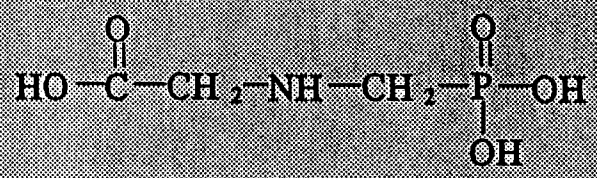
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	7.8	2.4	12	3480	willow, salmonberry, thimbleberry, pine, blackberry, alder, maple, cedar, fir/evergreens, scotchbroom
2	15	8	16	1240	maple, cottonwood, willow, elderberry, bitterbrush, sumac, elm, hardwoods, poison oak/ivy, wild cherry, apple trees
3	4.8	4	6.5	1090	alder, blackberry, maple, broadleaves, wilow, salmonberry, cattail
4	4.8	3	8	2870	grass, blackberry, mullien, alder, maple, cedar, cottonwood, scotchbroom, fir/evergreens, broadleaves, salmonberry, poison oak/ivy, milkweed, hardwoods
5	4	4	4	80	rose, woody plants
6	Not used	Not used	Not used	Not used	

Table 5-19 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for fosamine ammonium.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Glyphosate**
Product(s):	Roundup; Rodeo
Maximum Label Rate:	10.6 lbs active ingredient (ai)/acre
Application Method(s):	Foliar-applied spray
Mode of Action:	Non-selective postemergent control

Chemical Structure



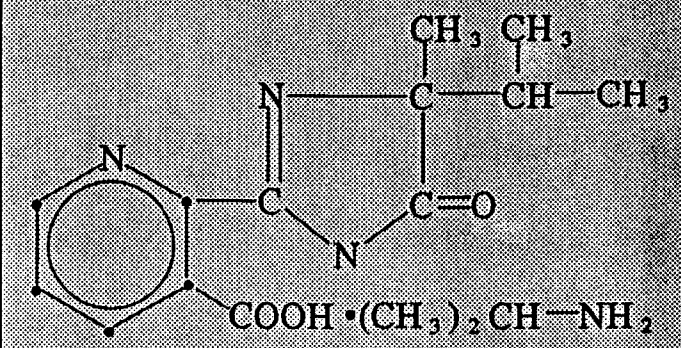
		Rates used by WSDOT from July (1989) to June (1990).				
		Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	**Target Vegetation**
WSDOT Districts	1	1.11	0.1	6	2170	knapweed, mullien, salmonberry, broadleaves, morning glory, sedge, clover, scothbroom, sweat pea, grass, seeds, horsetail, blackberry, tansy, thistles
	2	2.7	0.68	4	1770	horsetail, grass, thistles, mullien, knapweed, toadflax, willow, clover, pine, dropseed, sumac, puncturevine, foxtail, cattail, loosestrife, watercress, sandbur, dogbane
	3	1.2	0.3	8	1440	grasses, alder, knapweed, maple, broadleaves, seeds, japanese knotweed, cattail, bamboo, knotweed, blackberry
	4	2.1	0.61	8	640	grass, horsetail, thistles, blackberry, tansy, knapweed, cottonwood, clover, fir/evergreens, broadleaves, seeds, salmonberry, vetch
	5	1.2	0.1	4	680	grass, thistles, mullien, scotchbroom, broadleaves, kochia, prickly lettuce, cattail, rye, alfalfa
	6	1	0.16	2.7	9	cattail, rye

Table 5-20 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for glyphosate.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Imazapyr**
Product(s):	Arsenal
Maximum Label Rate:	1.5 lbs active ingredient (ai)/acre
Application Method(s):	Foliar-applied spray
Mode of Action:	Pre and postemergence control of annual and perennial grasses and broadleaf weeds.

Chemical Structure



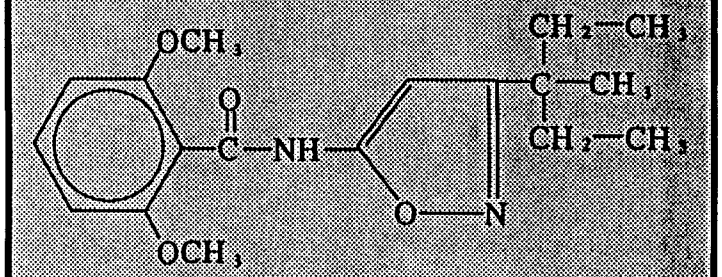
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	Not used	Not used	Not used	Not used	
2	↓	↓	↓	↓	
3	0.47	0.2	0.6	58	grass, horsetail, tansy, knapweed, toadflax
4	Not used	Not used	Not used	Not used	
5	↓	↓	↓	↓	
6	↓	↓	↓	↓	

Table 5-21 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for imazapyr.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Isoxaben**
Product(s):	Gallery
Maximum Label Rate:	1 lbs active ingredient (ai)/acre
Application Method(s):	Soil-applied spray
Mode of Action:	Preemergence control of annual and perennial and suppression of grasses.

Chemical Structure



		Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
		Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
WSDOT Districts	1	Not used	Not used	Not used	Not used	
	2					
	3					
	4					
	5	0.69	0.58	0.75	7	thistles, kochia, prickly lettuce
	6	Not used	Not used	Not used	Not used	

Table5-22 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for isoxaben.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Mecoprop**

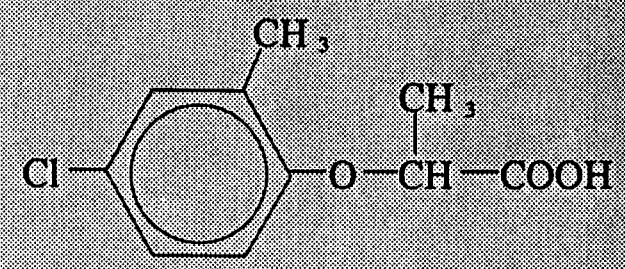
Product(s): Trimec classic

Maximum Label Rate: (Trimec Classic) 0.54 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray

Mode of Action: Postemergence control of broadleaf weeds.

Chemical Structure



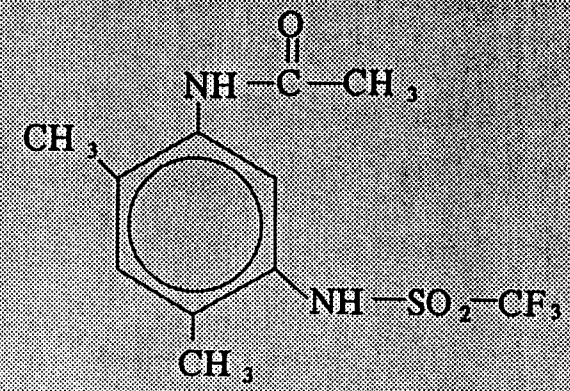
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	0.54	0.5	0.83	100	thistles, blackberry, mullien, alder, tansy, maple, scotchbroom, broadleaves, fir/evergreens, knapweed, salmonberry, nightshade, pine, ragweed
2	0.54	0.54	0.54	40	thistles, knapweed, sumac
3	Not used	Not used	Not used	Not used	
4	0.54	0.54	0.54	16	broadleaves
5	Not used	Not used	Not used	Not used	
6	0.47	0.47	0.47	2	thistles, clover

Table 5-23 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for mecoprop.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Mefluidide**
Product(s):	Embark 2S
Maximum Label Rate:	1 lbs active ingredient (ai)/acre
Application Method(s):	Foliar-applied spray
Mode of Action:	Slows growth. Suppresses seed head formation on broadleaf weeds and grasses.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990). Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	Target Vegetation
1	Not used	Not used	Not used	Not used	
2	↓	↓	↓	↓	
3	↓	↓	↓	↓	
4	0.5	0.5	0.5	10	ivy
5	Not used	Not used	Not used	Not used	
6	0.75	0.75	0.75	41	none listed

Table 5-24 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for mefluidide.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Metsulfuron methyl**

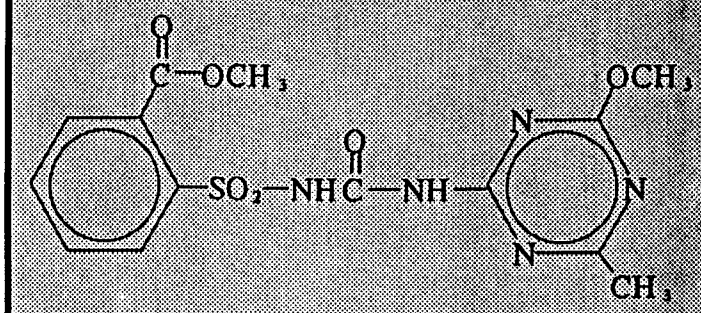
Product(s): Escort

Maximum Label Rate: 0.15 lbs active ingredient (ai)/acre

Application Method(s): Foliar and soil-applied spray

Mode of Action: Pre and postemergence control of annual and perennial broadleaves and grasses and woody species.

Chemical Structure



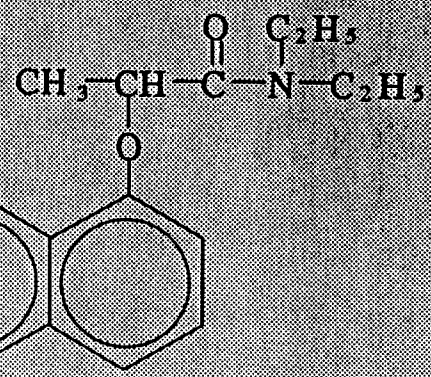
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	0.056	0.019	0.125	490	grasses, thistles,blackberry, mullien, alder, tansy, knapweed, maple, cottonwood, toadflax, scotchbroom, salsify
2	Not used	Not used	Not used	Not used	
3	0.026	0.017	0.038	1	thistles, blackberry, alder, knapweed, maple, fir/evergreens, pine
4	0.021	0.019	0.038	5	mullien, thistles, knapweed, strife, cactus, blackberry
5	0.075	0.05	0.1	0.5	thistles
6	0.019	0.01	0.031	300	grasses, thistles, mullien, knapweed, toadfalax, kochia

Table 5-25 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for metsulfuron methyl.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Napropamide**
Product(s):	Devrinol
Maximum Label Rate:	4 lbs active ingredient (ai)/acre
Application Method(s):	Soil-applied spray
Mode of Action:	Preemergence control of annual and perennial grasses and broadleaves.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	Not used	Not used	Not used	Not used	
2	↓	↓	↓	↓	
3	↓	↓	↓	↓	
4	4	4	4	108	weeds
5	Not used	Not used	Not used	Not used	
6	↓	↓	↓	↓	

Table5-26 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for napropamide.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Oryzalin**

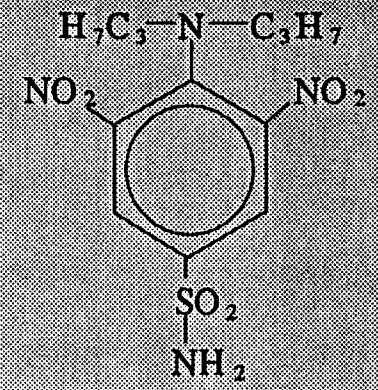
Product(s): XL-2G; Surflan

Maximum Label Rate: 6 lbs active ingredient (ai)/acre

Application Method(s): Soil-applied spray

Mode of Action: Preemergence control of annual and perennial grasses and broadleaf weeds.

Chemical Structure



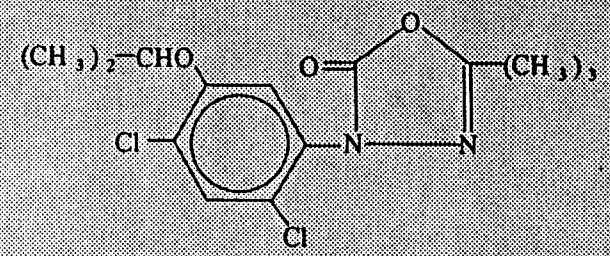
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	3.7	1	4	370	braodleaf weeds, grass, morning glory, seeds, sedge
2	Unknown	Unknown	Unknown	Unknown	broadleaf weeds, grass, thistles, sumac, clover
3	4	4	4	1230	broadleaf weeds, grass, seeds
4	3.25	1	6	130	broadleaf weeds and grasses
5	2.16	1.1	3	30	broadleaf weeds, thistles, kochia, prickly lettuce
6	2.5	2.5	2.5	5	grasses, broadleaf weeds

Table 5-27 WSDOT Roadside Vegetation Mangement DEIS. Herbicide use rates and information for oryzalin.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Oxadiazon**
Product(s):	Ronstar G; Ronstar 50WP
Maximum Label Rate:	4 lbs active ingredient (ai)/acre
Application Method(s):	Broadcast on soil or soil-applied spray.
Mode of Action:	Preemergence control of annual broadleaves and grasses.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total Used (lbs ai)	
1	Not used	Not used	Not used	Not used	
2					
3					
4					
5					
6					

Table 5-28 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for oxadiazon.

Active Ingredient: **Picloram**

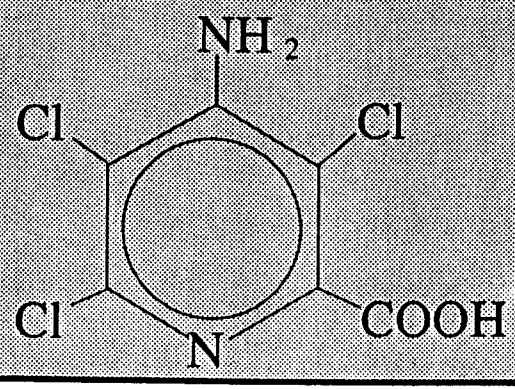
Product(s): Tordon (101R, 22K, RTU, K)

Maximum Label Rate: 2.16 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray

Mode of Action: Control of annual and perennial broadleaves, woody plants and vines.

Chemical Structure



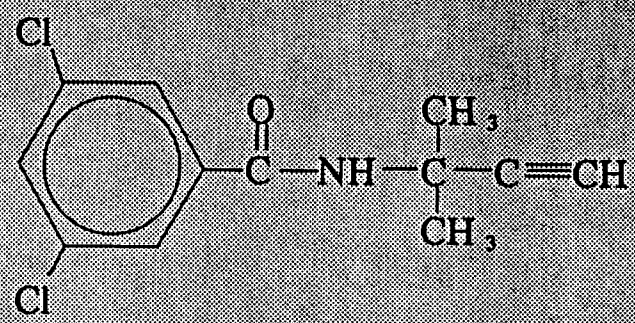
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	0.5	0.5	0.5	4	thistles, blackberry, knapweed, maple, alder, tansy, cedar
2	0.47	0.2	2	380	grass, horsetail, thistles, mullien, knapweed, toadflax, willow, clover, sumac, prickly lettuce, fireweed, tarweed
3	Not used	Not used	Not used	Not used	
4	↓	↓	↓	↓	
5	1.16	0.5	2	320	thistles, morning glory, broadleaves
6	1.4	0.25	2	190	broadleaves, whitetop, grasses, thistles, mullien, knapweed, toadflax, kochia

Table5-29 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for picloram.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Pronamide**
Product(s):	Kerb 50W
Maximum Label Rate:	4 lbs active ingredient (ai)/acre
Application Method(s):	Soil-applied spray
Mode of Action:	Pre and early postemergence control of annual and perennial broadleaves and grasses.

Chemical Structure



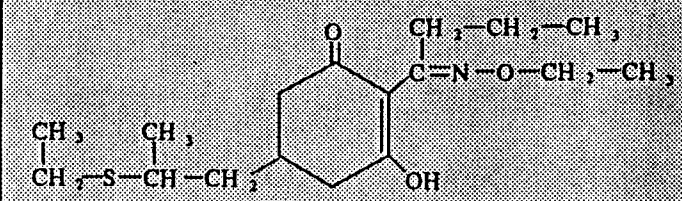
WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	2	2	2	102	weeds, grasses, seeds
2	Not used	Not used	Not used	Not used	
3	↓	↓	↓	↓	
4	2	2	2	30	weeds, grasses
5	Not used	Not used	Not used	Not used	
6	↓	↓	↓	↓	

Table 5-30 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates adn information for pronamide.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient:	**Sethoxydim**
Product(s):	Poast
Maximum Label Rate:	0.46 lbs active ingredient (ai)/acre
Application Method(s):	Foliar-applied spray
Mode of Action:	Postemergence control of annual and perennial broadleaves and grasses.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	Not used	Not used	Not used	Not used	
2	↓	↓	↓	↓	
3	↓	↓	↓	↓	
4	Unknown	Unknown	Unknown	Unknown	broadleaf weeds and grasses.
5	Not used	Not used	Not used	Not used	
6	↓	↓	↓	↓	

Table5-31 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for sethoxydim.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Sulfometuron methyl**

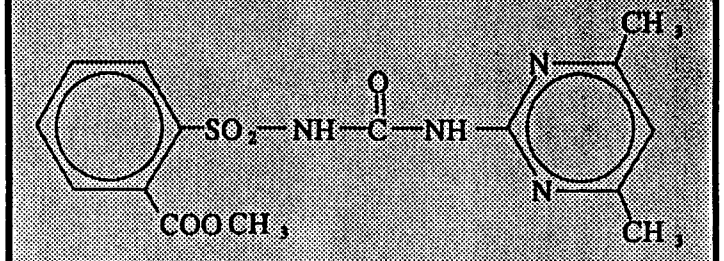
Product(s): Oust

Maximum Label Rate: 0.56 lbs active ingredient (ai)/acre

Application Method(s): Foliar or soil-applied spray

Mode of Action: Pre and early postemergence control of annual and perennial broadleaves and grasses.

Chemical Structure



		Rates used by WSDOT from July (1989) to June (1990)				
		Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	**Target Vegetation**
WSDOT Districts	1	0.23	0.19	0.33	130	broadleaf weeds, grass
	2	0.21	0.19	0.31	100	horsetail, willow, maple, sumac, grass, kochia, mullien, bitterbrush, dropseed
	3	0.18	0.11	0.25	200	broadleaf weeds, grasses
	4	0.15	0.09	0.25	70	broadleaf weeds
	5	0.19	0.15	0.28	530	broadleaf weeds, seeds, thistles
	6	0.19	0.19	0.19	820	broadleaf weeds, grasses

Table5-32 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for sulfometuron methyl.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

Active Ingredient: **Triclopyr**

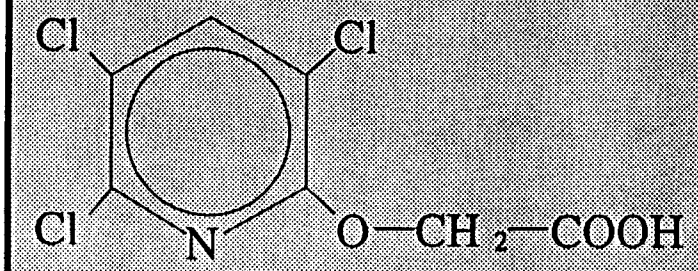
Product(s): Garlon (3A, 4); Crossbow

Maximum Label Rate: 9 lbs active ingredient (ai)/acre

Application Method(s): Foliar-applied spray

Mode of Action: Postemergence control of brush and annual and perennial broadleaves and grasses.

Chemical Structure



WSDOT Districts	Rates used by WSDOT from July (1989) to June (1990)				Target Vegetation
	Average Application Rate (lbs ai/acre)	Minimum Application Rate (lbs ai/acre)	Maximum Application Rate (lbs ai/acre)	Total* Used (lbs ai)	
1	1.82	0.25	6	1880	thistles, blackberry, mullien, alder, tansy, knapweed, maple, cottonwood, pine, fir/evergreen, toadflax, scotchbroom, willow, salmonberry, thimbleberry, vetch, wild carrot, nightshade, elderberry, ragweed, japanese knotweed
2	2.13	0.5	4	1030	horsetail, thistles, mullien, alder, knapweed, cottonwood, toadflax, willow, sumac, elm, apple tree, olive, prickly lettuce, bindweed, fireweed
3	1.5	0.063	6	270	thistles, blackberry, alder, tansy, knapweed, maple, cottonwood, willow, seeds, toadflax, scotchbroom, salmonberry, nightshade, pine, gorst
4	1.13	0.5	4	1080	horsetail, thistles, blackberry, mullien, alder, tansy, knapweed, maple, salal, cottonwood, fir/evergreen, toadflax, scotchbroom, willow, pepperwood, vetch, salmonberry, wild carrot, poison oak/ivy, daisy, teasle, Queen Anne's lace
5	Not used	Not used	Not used	Not used	
6	↓	↓	↓	↓	

Table 5-33 WSDOT Roadside Vegetation Management DEIS. Herbicide use rates and information for triclopyr.

* Note - Total pounds of active ingredient used for each district are combinations of actual and estimated figures derived from WSDOT spray records.

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Appendix B - Chapter 6

Draft EIS
Roadside Vegetation Management

1.0 Federal Laws and Regulations

1.1 The Federal Insecticide, Fungicide, and Rodenticide Act

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) is the federal law governing pesticides. FIFRA regulates:

- pesticide registration and labeling;
- protection of trade secrets and confidential data;
- pesticide suspension and cancellation;
- designation and use of restricted-use pesticide;
- standards for pesticide applicator certification;
- pesticide storage, disposal, and transportation; and
- penalties for pesticide misuse.

The U.S. Environmental Protection Agency (EPA) is responsible for administering and enforcing FIFRA, and is the federal agency responsible for regulating pesticides. Regulations for implementing FIFRA are contained in 40 CFR 150-189.

1.2 The Resource Conservation and Recovery Act

The Resource Conservation and Recovery Act (RCRA) regulates the generation, treatment, storage, transportation, and disposal of solid wastes. RCRA defines which solid wastes are hazardous and specifies handling procedures for hazardous wastes.

Discarding pesticides, unrinsed pesticide containers, pesticide rinsewater, and soil contaminated by pesticide is regulated by RCRA's hazardous waste provisions. Pesticide waste can be stored for a maximum of 270 days (9 months), and then must be shipped offsite by a permitted hauler to an approved disposal facility. All shipping and transportation procedures must conform with federal and state transportation department regulations for hazardous waste. (40 CFR 261)

Pesticide users are exempt from RCRA requirements if pesticides, containers, and rinsewater are handled in accordance with FIFRA regulations.

1.3 Other Federal Laws and Regulations

Other federal agencies, such as the U.S. Fish and Wildlife Service, monitor the environment for the presence of pesticides and other toxic substances. These agencies may request EPA to restrict or modify certain uses of pesticides, but do not regulate pesticides independently (Cheney pers. Comm.).

2.0 Washington State Laws and Regulations

2.1 Washington Department of Agriculture Regulations

While users in all states must comply with FIFRA, each state may pass additional laws regulating pesticide use. Because Washington's requirements are both more comprehensive and more stringent than the FIFRA provisions listed above, the remainder of this appendix will focus on Washington laws and regulations. Washington laws regulating pesticide use are included in the Washington Department of Agriculture (WDOA) Code.

The director of the Department of Agriculture may adopt rules to allow the Department of Agriculture to take possession and dispose of canceled, suspended, or otherwise unusable pesticides held by persons licensed under Chapter 15.58 RCW Washington Pesticide Control Act or regulated under Chapter 17.21 RCW Washington Pesticide Application Act.

1) Upon review and determination that a pesticide is no longer useable, WDOA may declare a pesticide to be a "waste pesticide."

2) WDOA may take possession of a waste pesticide with the owner's written consent for the purpose of disposal.

3) For the purpose of waste pesticide disposal, WDOA may:
   a. Become identified as a hazardous waste generator;
   b. Enter into contracts or cooperative agreements to carry out portions of or all of the waste pesticide disposal program. WDOA may also enter into cooperative agreements to carry out portions of or all of the development of education programs relating to waste pesticide disposal and programs for dissemination of information concerning the WDOA's disposal program.

4) WDOA may accept pesticides whose active ingredients are not clearly identifiable for disposal. The pesticides may be analyzed by either WDOA or a private laboratory. If upon an anlysis the material is not a pesticide, not identified or not acceptable for disposal, it shall be returned to the owner and/or not accepted for disposal. (Notice WSR 89-24-029, Filed November 30, 1989, Effective 12/31/89)

2.2 Other Laws and Regulations Applicable to WDOT Program

2.2.1 The Federal Endangered Species Act

The Federal Endangered Species Act (ESA) (16 USC 1361 et seq.) is the federal law governing the protection of fish, wildlife, and plant species in danger of or threatened with extinction. Section 9 of the ESA, which applies to all persons (including any officer, employee, agent, or department of a state government), prohibits the taking of any fish or wildlife species listed under Section 4 of the ESA as endangered (16 USC 1538). The ESA also provides for regulations which have been enacted to prohibit the

taking of any fish or wildlife species listed under Section 4 of the ESA as threatened (16 USC 1538, 50 CFR 17.31).

The ESA broadly defines the term "take" to include harassment (act or lack of action which creates the likelihood of injury to wildlife by significantly disrupting normal behavior patterns), harm (act which kills or injures wildlife, including significant habitat modification or degradation), pursuing, hunting, shooting, wounding, killing, trapping, capturing, or collection, or any attempt to engage in such conduct (16 USC 1532, 50 CFR 17.3). The use of herbicides which harm endangered species or destroy valuable habitat would be included in the definition of "take".

Under Section 10 of the ESA, the U.S. Fish and Wildlife Service or the National Marine Fisheries Service may issue an "incidental take permit" for any taking which is incidental to, and not for the purpose of, carrying out an otherwise lawful activity that would otherwise violate Section 9 of the ESA (16 USC 1539, 50 CFR 17.22, 17.32). With the application for an incidental take permit, the applicant must submit a conservation plan that specifies impacts likely to result from the taking, mitigation measures to minimize those impacts, funding for the mitigation, and description of project alternatives analyzed (16 USC 1539). If vegetation management activities might disrupt endangered species or their habitat, WDOT would need to apply for a permit for incidental taking of federally listed threatened and endangered species.

2.2.2 Endangered & Threatened Wildlife and Plants

The Washington Department of Wildlife Nongame Program was established in the 1970s to manage, preserve and protect the state's nongame wildlife species. The department is responsible for reviewing species for endangered or threatened status, monitoring their status and restoring populations.

The Endangered Species Act (ESA) of 1973 provides the criteria for determining endangered plant and animal species. Section 7 requires each federal agency to ensure its actions that authorize, permit, or fund a project, do not jeopardize the continued existence of any threatened or endangered species. The habitat for these species is not to be adversely modified.

Procedures

1. When the project prospectus indicates that there is the potential to affect a listed species (see below), the district must write to the U.S. Fish and Wildlife Service (USFWS) (rather than to the Washington Department of Wildlife) and request a listing of threatened and endangered species that may occur in the project vicinity. USFWS is to respond within 30 days. The listing is valid for 90 days. If a biological assessment is not begun within 90 days, an updated listing must be obtained by calling or writing the USFWS. WSDOT, acting on behalf of Federal Highway Adminstration (FHWA), must obtain a listing, if there is any question of the project affecting endangered species. Listings should be obtained early in project development, due to the time it takes to do appropriate studies and prepare reports. Normally, Protected and Endangered Species (P&ES) cannot be approved until the process is complete.

The following kinds of projects have the potential for affecting threatened or endangered species:

- Projects requiring an EIS, EA, or SEPA checklist;
- Projects requiring new right-of-way;
- Projects with earth moving, grading, pile driving, and other noisy activities;
- Bridge replacements;
- Projects with activity near shorelines, near major rivers, or other large water bodies; and
- Projects that affect natural vegetation or native habitats such as prairies, shrub-steppe, old growth forests or wetlands.

2. A biological assessment (BA) is an evaluation of how the project may affect species listed in the ESA. It is required under Section 7(c) of the ESA if threatened or endangered species or critical habitat may be present in the area affected by the project. The BA must be completed within 180 days of the listing; otherwise, a new listing must be obtained

   The BA is written by staff biologists at the headquarters Design Office. This is initiated by a written request from the district which must include a copy of the listing from USFWS, vicinity map, project description, contact person, and charge number.

   The BA usually contains conservation measures to protect the listed species.

   The BA is reviewed by the district for accuracy of project description and design details. District comments are incorporated into the report before it is sent to USFWS.

3. USFWS is to respond to the BA within 30 days; they normally concur. If not, USFWS will respond with a list of additional conservation measures needed for compliance with the ESA.

4. The district reviews the listed conservation measures to determine whether they can be included in the project. If not, WSDOT can enter a lengthy formal consolation process with USFWS through FHWA.

5. The district includes the conservation measures in the environmental commitment file, environmental document, design report, and PS&E documents.

2.2.3 Air Quality

Washington Clean Air Act RCW 70.94: Secure and maintain air quality levels that will protect human health and safety.

Motor Vehicle Emission Control RCW 70.120: Allow Department of Ecology (DOE) to conduct programs that will enhance the successful implementation of the air pollution

control system established for motor vehicles and give authority to establish motor vehicle emission standards.

Motor Vehicle Emission Inspections WAC 173-422: Implements both RCW 70.94 and 70.120 by establishing emission standards, testing procedures, and associated activities necessary for an effective air pollution prevention program.

Implementation of Regulations for Air Contaminant Sources WAC 173-403: Provides for the systematic control of air pollution from air contaminant sources and progressive reduction where needed.

Open Burning WAC 173-425: Implements the provisions of the Clean Air Act by establishing rules for open burning.

Ambient Air Quality Standards for Carbon Monoxide, Ozone, and Nitrogen Dioxide WAC 173-475: Establishes statewide air quality standards for the three pollutants identified.

Solid/Hazardous Waste

The Hazardous Waste Management Act RCW 70.105: Establishes a comprehensive statewide framework for planning, regulation, control, and management of hazardous waste.

The Model Toxics Control Act RCW 70.105A: Provides DOE with a funding source to implement hazardous waste regulations control and removal program.

Dangerous Waste Regulations WAC 173-303: Rules implementing the provisions of RCW 70.105. Provides lists of materials that are controlled hazardous waste.

Solid Waste Management (Recovery and Recycling Act) RCW 70.95: Directs local agencies or jurisdictions to develop regulations or ordinances governing solid waste management.

Minimum Functional Standards for Solid Waste Handling WAC 173-304: Establishes minimum standards for solid waste management as required by RCW 70.95.

3.0 Hazardous Waste Recycling

From the practice of pesticide application in vegetation management, WSDOT becomes a generator of the hazardous waste who causes the pesticide waste (outdated or damaged product) to become subject to state regulation. If unused or leftover pesticide materials cannot be legally applied in a manner consistent with the pesticide label use instructions, then the material would be a waste.

WSDOT is financially and legally responsible for proper management and disposal of its waste. If disposal practices lead to environmental contamination, site cleanup can be very costly. Therefore, waste reduction practices can help reduce the chance of your property becoming a hazardous waste site, and also reduce the effect of the hazardous

waste regulations on your operation. In essence, less waste results in less regulation and lower cost.

Some ideas for reducing and managing some common pesticide wastes are described below:

3.1 Pesticide Formulation (including unwanted, unusable, suspended, or cancelled pesticides).

- Pesticides should be used for their intended purpose and disposed of only as a last resort. If you have excess product that can still be used according to the pesticide registration, try to find a legitimate user for the product.

- Check with the WDOA for a definitive answer about authorized usage of the product. Their answer will help you to determine if the pesticide is a "waste."

- Select the least hazardous option for each pest management situation, and avoid using formulations that could be designated as "hazardous waste."

- Before buying quantities of pesticide, accurately calculate your volume needs and buy only the amount needed.

- Routinely check inventory and rotate your stock so that the oldest material is used first.

- Provide adequate storage conditions such as temperature and moisture controls in order to avoid damage to the product. Store dry formulations such as wetable powders or dusts above liquids and store containers off of the floor and on pallets.

Storage areas should also be: curbed, totally sealed (concrete pad) containment areas sloped to a sealed sump to assist in recovery of any spilled material; secured against unauthorized entry and identified with appropriate warning signs.

Residue spray solution is the leftover tank contents at completion of job and residue from cleaning the interior of tank and plumbing.

3.2 Waste Volume

Agricultural surveys point out that this waste stream represents approximately 70 percent of the total agrichemical waste volume. This waste can be almost eliminated using the following practices:

- Sequence applications to minimize change-overs from one spray solution to another.

- Dedicate equipment to compatible spray activities in order to reduce cleaning needs.

- Mix only what is needed, and calibrate your equipment to achieve precise application of the entire spray mixture.

- If spray solution is left over and cannot be applied to the target field, save it for a future application or for make-up solution for the next batch. It is very important to record the kind and amount of residual material and the date it is stored for future use. Careful record keeping can help make sure the material is used in a time frame and for a purpose where the material would be most effective. Materials should not be stored indefinitely as an alternative to disposal, because they could be designed as regulated hazardous waste.

3.3 Use equipment modifications and source reduction practices to reduce waste volumes. Some options include:

- Use an injection pump system for metering pesticide into the sprayer boom or lines. With the formulation and water held separately, the normal mixing operation and volume of spray solution mixture are eliminated. Injection systems offer improved accuracy in application and help reduce the amount of rinsate that is generated.

- Ultra-low-volume (ULV) applications (undiluted formulation) also eliminate mixing and spray solution volumes. Be sure to carefully review use instructions. Very few label directions allow for ULV application.

- If possible, the application equipment interior (tank, pump, spray lines) should be cleaned in the field where the rinsate can be applied to the target field, per label instructions. Include a small onboard saddle tank or nurse tank for carrying clean washwater to the application site.

3.4 Washwater from rinsing and cleaning spray equipment exterior

- Use high-pressure, low-volume cleaning equipment. Cleaning of the equipment exterior should ideally be done at a mixing-loading/cleaning station designed to collect washwater for reuse. Washwater containment makes sense economically. Proper containment can reduce costs of product loss, and reduce liability claims from environmental contamination.

- The washwater management facility should include a curbed concrete pad that is sloped to a sealed sump. Sump contents can be pumped into temporary holding tanks and the washwater reused as make-up for the next spray solution of the same pesticide. The more washwater that can be reused, the less waste volume there will be to deal with. If necessary, segregate washwater resulting from different spray mixtures in order to avoid accidental crop damage or other problems which could result from cross-contamination of the collected washwater.

3.5 Empty Pesticide Containers

- Never abandon empty uncleaned pesticide containers or accumulate them where unauthorized people have access to them. These containers can be dangerous to people, pets, livestock, wildlife, and our environment.

- Liquid containers should be triple-rinsed after use. Many fiber containers have plastic linings and these can also be rinsed. All rinsate should be added to the sprayer during the mixing procedure and applied to the target area in a manner consistent with the pesticide label directions.

- Paper containers should be thoroughly emptied by shaking and striking the package over the spray tank opening. Burning of pesticide containers can produce highly toxic fumes and such burning is prohibited by state air quality regulations. Be sure that containers are thoroughly emptied before attempting to burn them. Burning should never be done when it affects people, animals, occupied buildings, or public roads.

- The economic benefits of triple rinsing and cleaning of pesticide containers can be significant. For example, laboratory tests show that a drum that has been drained, but not rinsed, will generally still contain measurable amounts of pesticide. A five gallon container will usually still hold about six ounces and a 55-gallon drum will usually still hold about 32 ounces of pesticide product.

- When containers are triple-rinsed, they are not considered to be hazardous waste. They may be disposed in most landfills. Call your local landfill before taking containers there for disposal. Disposal requirements can vary depending on the landfill.

- Consult with your dealer-distributor or salvage company to determine if recycling of containers is feasible. These professionals sometimes offer container collection and recycling programs.

3.6 Spill Cleanup Residues

3.6.1 Spill Response

Before responding to a pesticide spill or accident, be sure you are wearing adequate protective clothing.

1) Stop the leak or flow.

2) Contain the spillage. Powder spills can be covered with plastic and/or dampened, if necessary, to prevent drift.

3) Pick up spills with absorbent material such as wood shavings, kitty litter, etc. Put the contaminated debris in secure nonleaking containers for disposal. Alternatively, the debris could possibly be used for its original intended purpose. (For example, if the pesticide that was spilled is intended for application to soil.) Check with WDOA for a

definitive answer about authorized usage of the pesticide. Otherwise, check with DOE for a determination of disposal requirements. If the debris is a hazardous waste, the Dangerous Waste Regulations would apply when the volume of waste exceeds 220 pounds.

3.6.2 Waste Oil Handling and Recycling

Waste oil reclaimed resulting from normal operation of the equipment and transportation practices of vegetation should be handled and recycled according to CFR40. 261.6. The preferred method is to use up the oils completely or recycle within WSDOT facilities. By doing so, the waste oil is exempt from hazardous waste regulations as well as cost savings on disposal.

In order to reduce the chance of spillage to the ground surface, it is recommended to drain and transfer the waste oil directly from the maintenance equipment to a oil service tank truck and then transport it to the designated high volume storage tank for recyling later on. Also, WSDOT has no underground storage tank on the maintenance area which can reduce the hazards of any underground soil/water contamination in the future.

The waste oils collected can be recycled or disposed by the method of incineration at the state approved facility.

3.6.3 Used Oil Filter Recycling

Recently issued directives from the EPA, Oregon DEQ, and Washington DOE require new handling and disposal practices for used oil filters. The new restrictions stem from concerns about whether used oil filters will pass the EPA's new Toxicity Characteristic (TC) rule; if they do not, they are a hazardous waste under RCRA.

In March 1991, WDOE issued a Regulatory Alert which stated that "For the next six months DOE will not require generators to test, designate, or manage used oil filters as hazardous waste. Over the next six months, DOE will test used oil filters to determine how they should be regulated."

For the present, Washington DOE and Oregon DEQ have recommended that generators do four things:

1) Drain (24+ hrs.), crush, or fillet/open the filters;

2) Recycle the oil by sending it to a re-refiner or burner;

3) Segregate and store the oil filters in a suitable container or drum; and

4) Send the filters to:
    a) Scrap metal recycler
    b) Hazardous Waste TSD
    c) Municipal/solid waste landfill (with prior approval).

Optionally, generators may choose to handle their used oil filters as hazardous waste.

The preferred method is to drain and/or crush the used oil filters and send the oil and filters to legitimate recyclers. By so doing, both the oil and filters are exempt from hazardous waste regulations.

Additionally, there are other significant environmental concerns. Virtually all of the 10,000,000 used oil filters generated per year in Washington are going to landfills. Add another 6,000,000 in Oregon.

Landfilling used oil filters in Washington results in the waste of 5,000,000 pounds of high grade scrap metal. It results in nearly 1,000,000 gallons of used lubricating oil being spilled and potentially contaminating the state's groundwater. And it results in our importing many times that amount of crude oil to replace what we have wasted.

4.0 Hazardous Materials and Waste

4.1 Criteria for identifying the characteristics of hazardous waste:

4.1.1 The Administrator shall identify and define a characteristic of hazardous waste only upon determining that:

a) A solid waste that exhibits the characteristic may: cause, or significantly contribute to an increase in mortality, or an increase in serious irreversible or incapacitating reversible illness; or pose a substantial present or potential hazard to human health or the environment when it is improperly treated, stored, transported, disposed of or otherwise managed; and

b) The characteristic can be measured by an available standardized test method which is reasonably within the capability of generators of solid waste or private sector laboratories that are available to serve generators of solid waste; or reasonably detected by generators of solid waste through their knowledge of their waste.

4.1.2 The Administrator shall list a solid waste as a hazardous waste only upon determinining that the solid waste meets one of the following criteria:

1) It exhibits any of the characteristics of hazardous waste identified in 40CFR Section 260.20, Subpart C.

2) It has been found to be fatal to humans in low doses or, in the absence of data on human toxicity, it has been shown in studies to have an oral LD_{50} toxicity (rat) of less than 50 milligrams per kilogram; an inhalation LC_{50} toxicity (rat) or less than 2 milligrams per kilogram or is otherwise capable of causing or significantly contributing to an increase in serious irreversible, or incapacitating reversible, illness. (Waste listed in accordance with these criteria will be designated Acute Hazardous Waste.)

3) It contains any of the toxic constituents listed in CFR40, Appendix VIII and, after considering the following factors, the Administrator concludes that the waste is capable

Solid Waste Management, Recovery and Recycling Act. RCW 70.95.

Applies to disposal of waste products, excluding hazardous waste, generated by department operations or encountered on department projects.

Dangerous Waste Regulations, WAC 173-303.

Implements state and state-enforced portions of federal statutes pertaining to hazardous waste. The basic rules concerning hazardous waste under which WSDOT must operate.

Common Law.

Traditional legal principles generally recognized by courts give persons, organizations, or agencies the right to bring legal action seeking to halt, or to recover damages for, environmental harm. These common law remedies encompass complaints based on nuisance, negligence, assault, trespass, strict liability, or various water rights. In the hazardous waste context, anyone who is adversely affected by the existence or disposal of hazardous waste could pursue common law remedies, in addition to CERCLA/RCRA--driven actions and settlements.

Indemnification, hold harmless, or contingency agreements cannot shield a person from liability under CERCLA. However, outside of CERCLA liability determinations, such agreements remain effective between the parties who enter into them (42 U.S.C. Section 9607 (e) (1982) for recovery of costs or settlement of claims and suits. Use of such provisions and agreements are recommended, if appropriate and useful, in particular hazardous waste site purchase or cleanup situations. (From FHWA's "Interim Guidance - Hazardous Waste Sites Affecting Highway Project Development," August 1988.)

4.2.1 Responsibility

Washington State Department of Ecology is responsible for implementing state statutes and programs, and federal programs contemplated for state participation or administration under the act. The FHWA and WSDOT have no direct responsibilities unless:

There is release of a hazardous substance as a result of a project;
or
WSDOT becomes the owner of property containing hazardous waste.

When WSDOT acquires a direct responsibility, which should only be after a thorough investigation of other alternatives, WSDOT will coordinate with DOE to arrive at a mutually agreeable solution. DOE will coordinate with EPA as required.

4.2.2 Procedures

Early Planning Phase

When a project is in the programming or early development phase, the district consults with their regional office of the DOE and reviews the lists of known hazardous waste sites that may be in the project vicinity and affect possible alternatives. Sites listed on

the EPA's National Priorities List (NPL) should obviously be avoided. Other known sites should also be avoided unless other environmental or engineering considerations indicate the risks and costs of involvement with such sites may be warranted.

Environmental/Design Phase

During development of environmental impact analysis and design information, the proposed right-of-way for all alternatives is studied in sufficient detail to locate any hazardous waste or general dumping sites, and to determine the nature of any hazardous material. The following are evidence of the potential presence of hazardous waste:

- Lack of vegetation
- Unusual odors, such as petroleum
- Present or previous use for storage of waste materials
- Stained soil or unnaturally colored water
- Proximity to known hazardous waste sites or sources
- Containers with unknown contents
- Stories, articles, historic records, verbal communications, etc.

Site Characterization

When potential right-of-way is suspected of containing hazardous waste, the district should further characterize the potential of the site by investigating past ownerships, talking with local officials and residents, reviewing old site photos, and conducting any other necessary investigations. When necessary, consult with DOE to see what additional information they may have. If the right-of-way is critical or other alternatives cause more severe problems, a more complete site characterization by the department's hazardous waste consultant is required.

Preliminary Assessment

Based on the information gained from the above site characterization, the district determines whether known or suspected hazardous waste contamination warrants more detailed assessment and sampling/testing of the soil and groundwater. The district then develops an appropriate scope of work for a preliminary assessment of the site by the hazardous waste consultant. The consultant is first requested to provide an estimate of costs. The estimate and scope of work are sent to the Planning Branch of DOT so that a new or supplemental budget can be issued, authorizing and funding the consultant services.

Site Remediation

When a right-of-way that contains hazardous waste must be acquired, the district prepares a hazardous waste management plan (HWMP) during project design. The HWMP is prepared with assistance from the HW consultant and in coordination with DOE.

Hazardous Waste Potential on Properties with Underground Storage Tanks (USTs)

When department projects require property from lands that contain USTs, then first determine the approximate location of the proposed right-of-way limits. Second, determine the location and approximate date of installation. This includes gasoline tanks, diesel tanks, waste oil tanks, chemical storage tanks, etc. These tanks are normally covered with a minimum of 3 feet of soil. Larger tanks may be 8 feet to 12 feet in diameter and 20 feet to 30 feet long. Tanks can be located by finding fill tubes, vent pipes, location of concrete slabs, or by metal detectors.

Recordkeeping and Reporting

When the Department becomes involved in site remediation (cleanup), it also becomes a hazardous waste generator. Generators are required by WAC 173-303 to maintain documentation on disposal actions. The requirements are listed in the Washington State Code WAC 173-303-210, and WAC 173-303-220.

Groundwater Monitoring

Groundwater monitoring may be required by DOE, DSHS, or the local health department. Monitoring well locations, monitoring frequency, and duration are negotiated with the controlling agency. Monitoring may be by consultants under contract. The department may negotiate with DOE, DSHS, or the local agency to assume monitoring and testing.

Discovery during Construction

If a previously undiscovered waste site is unearthed during construction, excavation, or removal, activities in the area are immediately suspended. If any of the materials appear dangerous or their health threat is uncertain, remove all workers and cordon off the area. A thorough record should be kept of all circumstances and actions taken including coordination with authorities, worker/public safety plan actions, and step-by-step remedial measures to deal with the problem. Contractual and contract administration problems, such as extra costs and time extensions for delays caused by the changed condition, should be handled like any other changed condition problem.

Right-of-way Procedural Guidance

When the department finds it necessary to acquire property contaminated with hazardous substances/wastes, it may be necessary to control, treat and/or dispose of existing wastes, contaminated soil, or other material in accordance with EPA or other state or local government regulations. Generally, the property owner is responsible for this effort and must bear the costs.

4.2.3 Worker Protection Standards for Agriculture Pesticides (CFR40, Part 170)

Application: No owner or lessee shall permit the application of a pesticide in such a manner as to directly or through drift exposed workers or other persons except those

knowingly involved in the application. The area being treated must be vacated by unprotected persons.

Reentry times: No owner or lessee shall permit any worker not wearing protective clothing (CFR40, 170.2d) to enter a field treated with pesticides, until sprays have dried or dusts have settled, unless exempted from such requirements, or a longer reentry time has been assigned to that pesticide.

Pesticides containing the following active ingredients have a reentry time of at least the interval indicated:

Ethyl Parathion	----------	48 hrs
Methyl Parathion	----------	48 hrs
Guthion	----------	24 hrs
Demeton	----------	48 hrs
Azodrin	----------	48 hrs
Phosalone	----------	24 hrs
Carbophenothion	----------	48 hrs
Metasystox-R	----------	48 hrs
EPN	----------	24 hrs
Bidrin	----------	48 hrs
Endrin	----------	48 hrs
Ethion	----------	24 hrs

The workers should not be permitted to enter treated fields if special circumstances exist, which would lead a reasonable man to conclude that such entry would be unsafe.

Warnings: When workers are expected to be working in a field treated or to be treated with a pesticide, appropriate and timely warning to such workers shall be given. The warning may be given orally and/or by posting warning signs at the usual points of entrance to the field, and/or on bulletin boards at points where the workers usually assemble for instructions. Where any person has reason to believe that a farm worker is unable to read, he shall give the farm worker oral warning and make reasonable effort to ensure understanding of such warning. When required, warnings shall be given in appropriate languages other than the English language. Oral warnings should be give in such a manner as to inform workers of areas or fields which should not be entered without protective clothing; the period of time the area or field should be vacated, and actions to take in case of accidental exposure.

of posing a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported or disposed.

4.2 Identifying and Handling Hazardous Waste

It is WSDOT practice to avoid known hazardous waste locations. When avoidance is not possible, or alternatives create greater adverse environmental impacts, early evaluation of hazardous waste locations allows time for site management and environmental protection. The chances of discovering unknown hazardous waste sites are remote; however, the potential added cost and adverse environmental impact support careful consideration early in the planning process. Federal law places the responsibility and cost of cleaning up hazardous waste on the current property owner.

Disposal of hazardous waste in landfills, surface impoundments, lagoons, pit sites or other locations was once common. Federal laws now give the Environmental Protection Agency (EPA) responsibility for controlling hazardous waste. EPA has delegated much of that authority within Washington State to the Department of Ecology. EPA is inventorying uncontrolled sites and has published the Hazardous Waste Sites National Priorities List (NPL).

Legal requirements pertaining to hazardous waste are contained in the following federal and state laws and regulations:

Resource Conservaton and Recovery Act (RCRA), Public Law 94-580.

The purpose is to regulate treatment, storage, transportation and disposal of hazardous waste. EPA regulations in 40 CFR and state regulations in WAC 173-303 implement RCRA.

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) or "Superfund."

Amended by SARA. Subject to certain defenses, CERCLA provides that all "persons" that own a hazardous waste site (i.e., a site contaminated with hazardous substances/wastes), own or operate a hazardous waste facility, arrange for the disposal or treatment of such waste, or cause a release or threat of release of hazardous substance are liable under CERCLA for costs of removal or remedial action incurred by others, and damages to natural resources (42 U.S.C.A. Section 9607 (1983 and West Supp. 1987)). Liability is strict (not dependent on fault), joint and several (any or all current and previous owners may be held totally liable), and retroactive. Federal and state governments are included in CERCLA's definition of "persons" (42 U.S.C.A. Section 9601 (21) (West Supp. 1987)). The department can be liable as an owner of a hazardous waste or releaser of hazardous substance/waste if it purchases property which has been or is being contaminated with such substance, or discovers/releases such substances during construction. WSDOT can also be held liable for the actions of lessees on WSDOT property resulting in contamination or release. (From FHWA's "Interim Guidance - Hazardous Waste Sites Affecting Highway Project Development," August 1988).

Superfund Amendments and Reauthorization Act (SARA) or "Superfund."

The purpose of CERCLA/SARA/Superfund is "to provide for liability, compensation, cleanup, and emergency response for hazardous substances released into the environment and cleanup of inactive hazardous waste disposal sites." Implemented by WAC 173-303. SARA expanded the defenses to liability under CERCLA to provide a defense for both private and public "innocent landowners" of land upon which hazardous waste attributed to a previous owner (third party) is discovered. One such defense provides that a government entity is not liable under CERCLA if property on which hazardous waste is located was acquired by the government entity "through the exercise of eminent domain authority by purchase or condemnation" after the hazardous waste was placed on the property by a previous owner, and the governmental body "exercised due care with respect to the hazardous substance concerned, taking into consideration the characteristics of such hazardous substance in light of all relevant facts and circumstances, and ...took precautions against foreseeable acts or omissions of any...third party and the consequences that could foreseeably result from such acts or omissions."

A second defense applies to "innocent landowners" who acquired property without knowing any hazardous substance/waste was present on or in the property, provided the purchaser took due care with respect to the potential for hazardous waste contamination and took precautions against foreseeable acts of third parties (i.e., possible contamination by a previous owner). This defense would shield the department, if the record shows that adequate measures were taken to identify hazardous substances/wastes at the site prior to purchase.

SARA also requires the Occupational Safety and Health Administration (OSHA) to issue regulations to protect the health and safety of workers engaged in hazardous waste operations, including those involved in early site assessments and those who may be inadvertently exposed to such substances (e.g., highway drill crews/materials test teams). (From FHWA's "Interim Guidance - Hazardous waste Sites Affecting Highway Project Development," August 1988).

National Oil and Hazardous Substance Contingency Plan (NCP) 40CFR300.

Implements CERCLA/SARA and provides detailed regulations on the cleanup of hazardous substances and oil discharges.

Hazardous Waste Management, RCW 70.105.

Established framework for statewide management of hazardous waste. Empowers DOE "to participate fully in... administer...RCRA."

Hazardous Waste Fees, RCW 70.105A.

State "superfund" act. Provides collection of fees to provide monies for the control and removal of hazardous waste. Empowers DOE to implement CERCLA.