APPENDIX B – CHAPTER 2 WILDLIFE RISK ASSESSMENT

1. Introduction

This chapter to the EIS supplement qualitatively evaluates the ecological risks to wildlife from the 17 herbicide product types (as defined by active ingredient or pair of active ingredients) currently used by WSDOT to control vegetation within roadway buffers, as well as 12 additional herbicide product types that have either been added since 2003 or are being assessed for potential future use. The methodologies applied to consider risks to wildlife receptors are similar to the 1993 risk assessment, pursuant to the scope. Thus, the principal focus of this work was to gauge the relative risks and hazards of the updated list of herbicides currently used or considered for future use by WSDOT. Toxicity data published since 1993 were examined, if identifiable through standard literature search and database sources. This assessment should not, however, be construed to represent a critical review of those data sources, as such a task was out of scope of the current effort. Where new data permitted, the assessment was expanded to further consider potential risks from such factors as inert and inactive carriers within the herbicide formulations, adjuvants, and chemical synergism. In practice, however, most toxicity information available referenced only the active ingredient, and therefore our assessment of risks is weighted proportionately to this end. Hazards to reptiles and amphibians were also considered where data allowed, but no estimate of exposure could be formulated for these ecological receptors, so no estimate of relative risks to these animals is conveyed. Finally, while this risk assessment is a useful tool for identifying the relative risks from the range of herbicides used by WSDOT, it does not purport to represent a complete assessment of quantitative risks to wildlife. Our understanding of each herbicide used is far too incomplete to attempt this task on a state-wide scale, and further empirical data would be required to fine-tune toxicity and exposure estimates such that risk characterizations could be represented with a higher degree of confidence.

1.1 Objectives

Ecological risk assessments seek to determine the nature, magnitude, and transience or permanence of observed or expected effects from chemical exposure. 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 most important factors regulating chemical toxicity in animals are the exposure dose, the duration of exposure, and the potency of the chemical. The nutritional, physiological, and genotypic state of an ecological receptor at the time of exposure can significantly modify the toxicity of the chemical or chemical mixture.

The effects of specific contaminants at the broader ecosystem level may also vary significantly among ecosystems based on the physical and chemical properties of the chemicals themselves, and the unique combination of physical, chemical, and biological processes occurring in each ecosystem. Such ecosystem differences can affect differences at the population level of ecological receptors. For example, wildlife populations resident to environments naturally enriched with metals may tolerate a much higher concentration of metal exposure than native

populations. That is, populations of exposed organisms may differ in their response to contaminants depending on their natural tolerance to the chemical, their behavioral and life-history characteristics (*e.g.*, pre-exposure), the dose to which they are exposed, and the duration of exposure. Furthermore, responses may be transient (reversible) or permanent (irreversible).

As indicated, this chapter presents primarily a qualitative assessment of relative hazards and risks to wildlife receptors from the use of herbicides to control roadside vegetation. The analysis represents a non-mathematical examination of the herbicide properties (toxicity, metabolism, environmental fate and transport) that influence the impact that these chemicals will have on wildlife. Our ability to characterize risks for some sectors of wildlife such as amphibians and reptiles is limited for some of the herbicides used because of a lack of basic toxicity and life history information needed to characterize chemical sensitivity and exposure, respectively. Site-specific studies would be required to quantitatively assign risks to ecological receptors with high confidence but the geographic scope of such an effort at the statewide scale was beyond the scope of the current effort. Exposure modeling was therefore conducted in lieu of site-specific work to gauge relative exposure doses based on application rates and delivery mechanisms of relevance to wildlife receptors. Exposure doses were then compared to reference doses from the toxicity literature to gauge the potential risks to relevant ecological receptors that would use habitats within roadway buffers.

1.2 Overview

The first section of this chapter reviews toxicity data for mammals, birds, insects, amphibians and reptiles, as available. The descriptions of toxicity in Table 2-1 are established by the Environmental Protection Agency (U.S. EPA). These descriptions vary slightly depending on the class of animal being tested and the study being performed (*e.g.*, acute oral LD_{50} or dietary LC_{50}). The LD_{50} is the statistical derivation of a value, which is predicted to cause 50% mortality in the population being tested. The 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.

Table 2-1. Toxicity Classifications to Address Wildlife Risk from Herbicide Use (U.S. EPA 1985)

Risk Category	Mammals	Mammals	Avian	Avian
	Acute Oral or Dermal LD_{50} (mg/kg)	Acute Inhalation LC_{50} (ppm)	Acute Oral LD_{50} (mg/kg)	Acute Inhalation LC_{50} (ppm)
Very highly toxic	<10	<50	<10	<50
Highly toxic	10-50	51-500	10-50	50-500
Moderately toxic	51-500	501-1,000	51-500	501-1,000
Slightly toxic	501-2,000	1,001-5,000	501-2,000	1,001-5,000
Practically non-toxic	>2,000	>5,000	>2,000	>5,000

The second section of this report represents the relative exposure assessment. The exposure assessment identifies exposure pathways possible for each of the new herbicides used by WSDOT for the representative ecological receptors. The exposure assessment also considers the environmental fate and transport of the chemicals as discussed, through the inclusion of

persistence parameters in the exposure formula. Similar to the 1993 EIS, we used the rat as a model animal for mammals, and the quail as a model animal for birds. However, where data permitted, exposures to other mammals and birds were computed to improve the range of potential receptors considered that could be found along roadways. Amphibians, reptiles and insects were also considered.

Finally, to characterize relative risks we compared the exposure parameters within each group of ecological receptors based on WSDOT herbicide use practices, and the inherent toxicity of the active ingredients, adjuvants, and inert ingredients. To the extent practicable, the risk characterization considered use practices by physiographic region, based on WSDOT use data in each of the physiographic regions.

In each section, the 17 herbicide formulations in 2003 are presented first, followed by the 12 herbicide formulations examined in 2005. The herbicide formulations examined in 2003 include:

1. 2,4-D
2. Ammonium salt of fosamine
3. Bromacil/Diuron
4. Chlorsulfuron
5. Clopyralid
6. Clopyralid/2,4-D
7. Dicamba
8. Dicamba/2,4-D
9. Dicamba/MCPA
10. Dichlobenil
11. Diuron
12. Glyphosate
13. Metsulfuron Methyl
14. Picloram
15. Oryzalin
16. Sulfometuron Methyl
17. Triclopyr

The 12 herbicide formulations examined in 2005 include:

1. Bromoxynil
2. Diflufenzopyr
3. Flumioxazin
4. Fluroxypyr

5. Imazapyr
6. Isoxaben
7. Norflurazon
8. Oxadiazon
9. Pendimethalin
10. Pyraflufen
11. Sulfentrazone
12. Tebuthiuron

2.0 Herbicide Toxicity to Wildlife Receptors

In this updated risk assessment of WSDOT's herbicide use, we examined data from both acute (short-term) and chronic (long-term) toxicity studies where available. Our literature search referenced each chemical's active ingredient, as well as the trade names of the products that WSDOT currently uses or is considering using. Information on adjuvants, inert ingredients and surfactants was also searched if this information was available. The search engines used included: Aquatic Sciences and Fisheries Abstracts, Biosis, Agricola, Extoxnet, U.S. EPA registration eligibility documents (REDs), and a variety of appropriate texts (*e.g.*, Merck index). Given the nature of the literature searches conducted for this assessment, it is acknowledged that some sources containing potentially relevant information may have been overlooked. In particular, state and federal agency reports that have not been published in peer-reviewed journals but for which defensible data supporting or refuting statements made herein may have been overlooked as they were not generally available from the search modalities exercised. Results from these searches were used to summarize the toxicity of each herbicide used by WSDOT and literature sources were not reviewed critically, although data gaps were identified where found. For example, toxicity data of herbicide formulations on amphibians, reptiles, and insects was generally lacking, as was information on chemical synergism in wildlife receptors.

Data presented to U.S. EPA for the purpose of registering a product have been generated using standardized tests adopted by U.S. EPA. The results of these studies are compared against the criteria previously depicted in Table 2-1 to gauge the toxicity of the active ingredient or product formulation. In using this toxicity classification scheme it becomes possible to compare toxicity values among formulations and species. However, it must be acknowledged that species differences in wildlife receptors may exist that are not predictable from the classification scheme represented in Table 2.1. Some wildlife receptors that may be at risk of exposure to herbicides applied along roadsides are rarely used in toxicity testing for lack of a consistent supply and approved protocols. This deficiency is particularly obvious for omnivorous and carnivorous species such as the raccoon and coyote, but is also an issue for large ungulates such as the black-tailed deer that commonly forage along roadsides, migratory songbirds (passerines), and reptiles and amphibians.

The use of surrogate species with similar dietary and/or behavior patterns has been shown to provide a relatively reliable predictor of toxicity within one half order of magnitude of the toxicity of the most sensitive species for a variety of toxicants tested on fish (Dwyer *et al.*,

2000). It can be reasonably assumed that a similar relationship exists for other wildlife receptors compared against surrogates. However, only site-specific risk assessments would be able to fully quantify risks to resident and migratory wildlife receptors from chemical exposure. This assessment therefore must use surrogate species such as the rat and meadow vole to gauge toxicity to other wildlife that may be more likely to be found using the habitat found along the state-managed roadways of Washington State. The rat provides a reasonable surrogate of an omnivore, the meadow vole an exclusive herbivore, and the quail and duck provide surrogates of upland and wetland bird species, respectively. The data provided below generally reflect U.S. EPA protocols with these standard test species. Toxicity information from other species is provided if it was found. If there were no data available that were generated under U.S. EPA guidelines, then the toxicity values presented are those that were the most conservative.

2.1 2,4-D

2.1.1 *Mammals*

2,4-D is considered to be moderately toxic to mammals based on criteria specified by U.S. EPA (1985). It has a reported acute oral LD_{50} of 375 mg/kg in male rats (USDI 1989) and 370 mg/kg in female rats (USDHHS 1980; see WSDOT 1993). In addition, acute oral tests determined that 2,4-D is slightly toxic to rabbits (lowest published lethal dose/LDL = 800 mg/kg; see WSDOT 1993). Its acute dermal toxicity ranges from 1,400 mg/kg (USDHHS 1980) to 3,980 mg/kg (USDI 1989; see WSDOT 1993) indicating that it is slightly toxic to practically non-toxic through dermal exposures.

Chronic toxicity of 2,4-D, as observed in a two-year diet study in dogs, resulted in death most likely due to inefficiency of excreting organic acids (summarized in EXTOXNET 1996a). A companion paper examined the chronic effects of 2,4-D concentrations in dogs fed 0, 0.5, 1.0, 3.75, and 7.5 mg/kg/day of 2,4-D acid, 2,4-D dimethylamine salt, and 2,4-D 2-ethylhexyl ester (Charles *et al.* 1996). The results indicated a comparable toxicity between the three forms of 2,4-D and a lack of toxic response from all three in dietary tests in dogs. The study established an overall NOEL of 1.0 mg/kg/day in the three subchronic and chronic studies.

2,4-D did not elicit reproductive effects in rats at a 50 mg/kg dietary level, however concentrations of 188 mg/kg resulted in fetuses with abdominal cavity bleeding and increased mortality and concentrations of 150 mg/kg resulted in an increase in skeletal abnormalities such as delayed bone development and wavy ribs (summarized in EXTOXNET 1996a).

Carcinogenicity is not an endpoint that is generally considered in ecological risk assessment; however, in some chronic exposure studies, carcinogenic endpoints are evaluated. In general, however, the lifespans of wildlife species are not long enough to consider cancer endpoints relevant. Chronic levels of 2,4-D administered to mice and rats have also been shown to increase malignant tumors; however direct association of 2,4-D and carcinogenicity is still debatable (summarized in EXTOXNET 1996a, USDA 1995a). A rat oncogenicity study performed on the 2,4-D acid by Charles *et al*. (1996) concluded that 2,4-D does not produce astrocytomas or lymphosarcomas in the rat as a dose of 150 mg/kg/day. An overall NOEL for chronic toxicity effects in rats was established in this study as 5 mg/kg/day.

2.1.2 *Birds*

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 mg/kg indicates that 2,4-D is practically non-toxic to birds when exposed through ingestion (USDI 1989).

2.1.3 *Insects*

The USDI (1989) suggests the LD_{50} of 2,4-D for bees of 11.5-105 µg/bee is essentially non-toxic.

2.1.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.1.5 *Adjuvant and Inert Ingredients in Commercial 2,4-D Formulations*

Adjuvants and inert ingredients in 2,4-D formulations are listed in Table 2-2. As demonstrated in Table 2-2, some formulations of 2,4-D may contain ethylene glycol. Ethylene glycol can result in kidney failure and due to its sweet taste animals have been known to drink the liquid. The LD_{50} for ethylene glycol in rats, guinea pig, and mice has been reported as 5.50, 13.1, and 7.35 ml/kg, respectively (Frank 1967, *as cited in* Verschueren 1983).

Table 2-2. Formulations of Different Commercial Forms of 2,4-D (USDA 1995a)

Commercial Product Name	Concentration of 2,4-D (%)	Concentration of Inert Material (%)
Weedar 64 (liquid)	46.8 of dimethylamine salt of 2,4-D	53.2
HiDep (liquid)	32.3 of dimethylamine salt of 2,4-D + 16.3 of diethanol-amine salt of 2,4-D	10 (including ethylene glycol)

2.2 Ammonium Salt of Fosamine

2.2.1 *Mammals*

The oral and dermal toxicity of the Fosamine is considered practically non- toxic to mammals based on U.S. EPA toxicity criteria (Table 2-1). Toxicity tests conducted by standard U.S. EPA protocols with Krenite, the Dupont product with the ammonium salt of Fosamine, yielded an LD_{50}>5,000 mg/kg for rats through oral exposure, and an LD_{50} of 5,000 mg/kg for rabbits exposed dermally. According to studies conducted by DuPont for product registration, Krenite is not a primary skin irritant when applied as 0.5 ml undiluted formulation to shaved, intact, or

abraded skin of rabbits (DuPont 2002). However, the application of the chemical caused mild to no corneal opacity and temporary severe to moderate conjunctival irritation in the unwashed rabbit eyes. The rabbits' eyes returned to normal within 7 days following exposure (DuPont 2002).

Long-term studies have been conducted to address the potential chronic toxicity of Krenite exposure to mammals administered low doses. During a 90-day rat feeding study, the no-observed-effect-level (NOEL) was considered to be at 1,000 mg/kg with slight effects on the kidneys of male rats at 5,000-10,000 mg/kg (DuPont 2002). During a 6-month dog feeding study, the NOEL was considered to be at 10,000 mg/kg (DuPont 2002). Although the relative stomach weights of the dogs were significantly higher than the control group at the 10,000 mg/kg, this effect was associated with no other clinical or gross pathological changes (DuPont 2002). A generation rat reproduction study with the chemical found no reproductive effects at 5,000 mg/kg, the highest level fed (DuPont 2002). These chronic study results support the oral (acute) toxicity rating of practically non-toxic.

In 4-hour tests for toxicity through inhalation, Fosamine would be considered very highly toxic based on U.S. EPA criteria. The inhalation LC_{50} was 3.30 ppm for male rats and a LC_{50}=2.75 ppm for female rats in the manufacturers studies (DuPont 2002).

2.2.2 *Birds*

An acute oral LD_{50} for the northern bobwhite quail and the mallard duck of >5,000 mg/kg (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). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information

2.2.3 *Insects*

As previously reported (see WSDOT 1993), Fosamine is essentially non-toxic to bees based on a contact LD_{50} of >200 µg/bee (USFS 1989, Meade 1983). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.2.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.2.5 *Adjuvant and Inert Ingredients*

Fosamine is typically applied with an agricultural oil (0.25%) to increase mobility in the soil and promote adsorption by target plant species (ACOE 2003). Krenite contains 41.5%

Fosamine and 58.5% inert ingredients. The inert ingredients are classified by U.S. EPA as having no toxicological effects (*as summarized in* BPA 2000a). However, no specific toxicity studies related to this product were identified for review.

2.3 Bromacil/Diuron

2.3.1 *Mammals*

Bromacil/Diuron is considered to be practically non-toxic or slightly toxic to mammals, based on studies conducted with Krovar, the product manufactured from Bromacil and Diuron (DuPont 1992). Acute oral studies with rats determined Krovar to be practically non-toxic by ingestion (LD_{50}=2,300 mg/kg), and acute dermal studies with rabbits found that dermal applications of concentrated Krovar were not lethal at the highest practical dose tested (LD_{50}>2,000 mg/kg = slightly toxic). However, 4 hours of direct skin contact with the concentrated product resulted in a slight but reversible skin irritation (DuPont 1992). These values differ somewhat from the acute oral and dermal toxicity values previously reported (oral LD_{50} = 3,998 mg/kg; dermal LD_{50} = 2,000 mg/kg).

Application of concentrated Krovar to the eyes produced a moderate irritation, although the eyes were normal within 7 days of treatment (DuPont 1992). Separate 4-hour tests on acute inhalation with rats for each active ingredient resulted in estimates that concentrated Krovar is toxic at an LC_{50} >5 ppm (DuPont 1992). Acute exposure of rats resulted in general signs of distress, rapid and deep respiration, at the highest dose tested, 4.8 ppm (DuPont 2000a).

Bromacil and Diuron have also been evaluated in a number of studies with respect to repeated dietary exposure, both short-term and chronic (DuPont 1992). Daily and excessive exposure of rats and mice to the chemicals have resulted in reduced body weights; increased liver, adrenal, and heart weights; decreased kidney and spleen weights; and slight to minimal microscopic changes in the spleens, thyroids, bladders, and/or livers of one or more of the test species (DuPont 1992, DuPont 2000a). Lifetime exposures resulted in tumors in the livers of mice or the bladders of rats at excessive dietary doses of Bromacil and Diuron, respectively (DuPont 1992). Tumor responses were not observed in other species similarly exposed to excessive doses (DuPont 1992). In addition, an equivocal tumor response was reported for mice at excessive Diuron exposures (DuPont 1992).

No reproductive effects were observed in rats with chronic dietary exposure to Bromacil at 250 mg/kg, and most studies for genetic damage caused by Bromacil to mammalian and bacterial cells in culture were also negative (Dupont 2000a). Testing with some animals has shown Diuron to have some embryotoxic activity, although it is believed that the chemical does not have mutagenic or reproductive effects (Dupont 2000a).

2.3.2 *Birds*

Acute oral and dietary studies with mallard ducks and bobwhite quail were conducted by the manufacturer of Krovar IDF to determine the relative toxicity of Bromacil and Diuron (DuPont 1992). Bromacil was found to be practically non-toxic to bobwhite quail in an acute oral study

(LD_{50}>2,250 mg/kg), and practically non-toxic to both bobwhite quail (LC_{50}>10,000 mg/kg) and mallard ducks (LC_{50}>10,000 mg/kg) in an 8-day sub-chronic dietary study (DuPont 1992). Similarly, in the acute oral study, Diuron was found to be slightly toxic to mallard. Similar results for Bromacil have also been found with several other acute oral and dietary studies (Wellings 1991, Hartley and Kidd 1987).

2.3.3 *Insects*

As previously reported (*see* WSDOT 1993), Bromacil is considered essentially non-toxic to honey bees, a common surrogate species for gauging insect effects (Wellings 1991, Hartley and Kidd 1987). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.3.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.3.5 *Adjuvant and Inert Ingredients*

The inert ingredients in Krovar IDF are not classified by authorities as inert ingredients of toxicological concern to humans or the environment (DuPont 1992). However, no specific toxicity studies related to this product were identified for review.

2.4 Chlorsulfuron

2.4.1 *Mammals*

Acute oral, dermal, and inhalation studies with rats and rabbits were conducted to determine the toxicity of Chlorsulfuron (Dupont 2001, Denny 1991, USDA 1994a). 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 2001, Denny 1991). Additional studies found the acute oral LD_{50} value to be >5,000 mg/kg for both male and female rats (USDA 1994a) and, in a 6-month chronic ingestion study in non-rodents, the no observed adverse effect level (NOAEL) was 18.5 mg/kg/day, while the lowest observed adverse effect level (LOAEL) was 82.3 mg/kg/day based on decreased body weight and weight gain (U.S. EPA 2002a). Chlorsulfuron was also determined to have very low toxicity through acute dermal studies with male and female rabbits (LD_{50}>2,000 and 3,400 mg/kg) (Dupont 2001, USDA 1994a). The acute inhalation LC_{50} for rats was 5.9 ppm (USDA 1994a).

The effects of Chlorsulfuron from repeated exposure have also been evaluated in a number of studies (USDA 1994a). Male and female rats fed dietary doses of Chlorsulfuron for up to 2

years showed no effects at doses of up to 100 mg/kg Chlorsulfuron (USDA 1994a). At doses of 500 or 2,500 ppm Chlorsulfuron there were lung changes, weakness, mild to moderate reduction in body weight and reduced weight gain in male rats (Dupont 2000b, USDA 1994a). During the first year of the study, there were effects on blood components in the 500 and 2,500 mg/kg dose-group of male rats (USDA 1994a).

A similar study of chronic toxicity in dogs placed the NOAEL at 60.6 mg/kg/day and the LOEL at 215 mg/kg/day, based on decreased body-weight gain, erythrocyte counts and hemoglobin levels (U.S. EPA 2002a). A 3-generation reproduction study in rats showed slightly decreased fertility at the highest dietary dose of 2,500 mg/kg (Parental NOEL=125 mg/kg/day, Reproductive LOEL=25 mg/kg/day) (USDA 1994a, U.S. EPA 2002a). Mice and rats fed up to 5,000 ppm per day for 2 years showed no evidence of carcinogenicity (NOAEL=5 mg/kg/day, LOAEL=25 mg/kg/day, based on decreased body weight in males (USDA 1994a, U.S. EPA 2002a). Similarly, tests with pregnant rats and rabbits indicated no evidence of birth defects (USDA 1994a, DuPont 2000b).

2.4.2 *Birds*

As previously reported (*see* WSDOT 1993), avian studies indicate that Chlorsulfuron is practically non-toxic based on acute LD_{50} for the northern bobwhite and the mallard of >5,000 mg/kg (Hinkle 1979). The dietary LC_{50} of >5,000 mg/kg also establishes Chlorsulfuron as practically non-toxic to birds (Hinkle 1979). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.4.3 *Insects*

Chlorsulfuron is considered to be essentially non-toxic to bees based on an acute contact LD_{50} of >25 µg/bee (Meade 1983). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.4.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.4.5 *Adjuvant and Inert Ingredients*

The most common commercial herbicide formulation of Chlorsulfuron is Telar® DF that is a combination of the Chlorsulfuron compound (75%) and inert materials (25%) (*as summarized in* BPA 2000b, USDA 1995b). This formulation is not used on food crops. Chlorsulfuron formulations are also used with surfactants so that the target vegetation better absorbs them. For example, in an experiment testing phytochemical responses to Chlorsulfuron, plants were

sprayed with Glean 20 DF (20% Chlorsulfuron) plus the detergent Citowett (KjΦr *et al*. 2001). Information on the toxicity of the surfactants was not identified in our review, however..

2.5 Clopyralid

2.5.1 *Mammals*

Dermal, oral, and inhalation tests of Transline, the predominant herbicide manufactured with Clopyralid (for non-crop use), showed that the chemical is practically non-toxic to rabbits and rats. In a dermal exposure study with rabbits, the LD_{50} of Transline was established at >5,000 mg/kg, higher than the practicably tested concentration (Dow AgroSciences 1999a). Similarly, in studies of rabbits ingesting Transline, the LD_{50} is >5,000 mg/kg (Dow AgroSciences 1999a). Inhalation studies also produced no ill effects in test animals, when using the maximum practically attainable concentration of Transline (3.0 mg/L for four hours) (Dow AgroSciences 1999a).

A number of studies have also tested the effects of Clopyralid from repeated exposure, and have identified several symptoms, including effects on the liver and kidney and lethargy (Dow AgroSciences 1999a). Clopyralid caused birth defects in test animals, but only at greatly exaggerated doses that were severely toxic to the mothers (Dow AgroSciences 1999a). No birth defects were observed in animals given Clopyralid at doses several times greater than those expected at normal exposure (Dow AgroSciences 1999a). Clopyralid has not been demonstrated to have carcinogenic or reproductive effects on test animals (Dow AgroSciences 1999a).

No effect chronic dietary exposure concentrations of Clopyralid are represented in Table 2-3.

Table 2-3. Response to Chronic Exposures of Clopyralid Concentrations in Mammals (Source U.S. EPA 2000)

Test Species (type of test)	Duration (type of exposure)	Chemical Concentration	Result
Rats (development)	100 days	75 mg/kg/day 0.75 mg/kg/day	NOEL Acute RfD
Rats (chronic carcinogenicity)	100 days	15 mg/kg/day 0.15 mg/kg/day	NOEL Chronic RfD
Rats (development)	1 to 7 days (inhalation)	75 mg/kg/day	NOEL (100% absorption)
Rats (development)	1 week to several months	75 mg/kg/day (inhalation or oral)	NOEL (100% absorption)

2.5.2 *Birds*

The acute oral toxicity LC_{50} concentration for bobwhite quail and mallard ducks is >2,000 mg/kg (*as summarized in* BPA 2000c). The subacute dietary LC_{50} for each of these species is >5,000 mg/kg. These data indicate that Clopyralid is practically non-toxic to birds. A study reported by Tu *et al.* (2001a) looked at the response of bobwhite quail egg hatchability and chick immunocompetence to Clopyralid. The study did not observe any significant effects to bobwhite quail embryos at any of the doses tested.

2.5.3 *Insects*

The reported LD_{50} acute contact toxicity concentration for the honeybee is >100 µg/bee (*as summarized in* BPA 2000c).

2.5.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.5.5 *Adjuvant and Inert Ingredients*

The inert ingredients in Transline include isopropyl alcohol and polyglycol 26-2 (Dow AgroSciences 1999a). The adjuvants and other inert ingredients of Transline, as well as other Clopyralid formulations are represented in Table 2-4. The presence of isopropyl alcohol used as an inert ingredient in some formulations cause the compound to be more volatile. Mixtures of Clopyralid may also create more potent formulations.

Table 2-4 Commercial Formulations of Clopyralid (*as summarized in* BPA 2000c, Cox 1998).

Trade Name	Type	Percent of Active Ingredient	Percent of Inert Ingredients
Transline®	Single Active Herbicide	40.9% Clopyralid amine salt	59.1% isopropyl alcohol and polyglycol 26-2 mixture
Curtail®	Mixture	5.1 % Clopyralid amine salt 39.0% 2,4-D amine salt	55.9%

Other inert ingredients used in Clopyralid formulations include cyclohexanone, triethylamine, and polyethoxylated tallow amines (Cox 1998). Cyclohexanone has a reported oral LD_{50} of 1.62 ml/kg (ppth) in rats (Merck 1989) and would be considered "slightly toxic by criteria expressed in Table 2-1. Currently, WSDOT uses only the Transline formula that contains 59.1% isopropyl alcohol as well as a detergent (polyglycol 26). Generally, the alcohol which serves as a carrier solvent is rapidly evaporated immediately after application. The detergent (surfactant) serves as an adjuvant to facilitate herbicide uptake. Because the exact proporation of adjuvants was not expressed in Clopyralid formulations we examined, it was not possible to ascertain the potential hazards of all the adjuvants relative to the criteria in Table 2-1. However, several of the carriers in Clopyralid may have significant toxicity associated with them, placing a higher degree of uncertainty associated with the risks of applying this herbicide in active formulation.

2.6 Clopyralid/2,4-D

2.6.1 *Mammals*

Dermal and oral studies were conducted to determine the relative toxicity of the Clopyralid/2,4-D formulation to mammals. This herbicide formulation has been shown to cause slight toxicity in some dermal and oral studies. In dermal studies, Clopyralid/2,4-D is practically non-toxic to rabbits, with the LD_{50} for skin absorption in rabbits at >4,000 mg/kg (Dow AgroSciences 2001). Oral studies, using male and female rats, showed a slightly increased toxicity when the chemical was ingested, with an LD_{50} of 3,730 mg/kg for male rats and a LD_{50} of 2,830 mg/kg for female rats (Dow AgroSciences 2001).

Studies have also been conducted with Clopyralid/2,4-D to determine the effects of long-term exposure (Dow AgroSciences 2001). Effects have been reported with test animals on the gastrointestinal tract, kidney, liver, and muscular system (Dow AgroSciences 2001). The chemicals are not known to have any carcinogenic effects, but both chemicals have caused birth defects in laboratory animals when administered at greatly exaggerated doses producing severe toxicity in the mother (Dow AgroSciences 2001).

2.6.2 *Birds*

Toxicity levels for Clopyralid/2,4-D are similar to the original parent compounds. Clopyralid and 2,4-D are both practically non-toxic to birds suggesting no additivity or synergistic toxicity in the formulation (Tu *et al.* 2001b).

2.6.3 *Insects*

Toxicity levels for Clopyralid/2,4-D are similar to the original parent compounds. Clopyralid and 2,4-D are both practically non-toxic to insects (Tu *et al.* 2001b).

2.6.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.6.5 *Adjuvant and Inert Ingredients*

Inert ingredients of Curtail include Trissopropanolamine and Ethylenediaminetetraacetic Acid (Dow AgroSciences 2001).

2.7 Dicamba

2.7.1 *Mammals*

Acute oral and dermal studies with rats (Sandoz 1992, USDA 1994b) concluded that Dicamba has a slight to moderate toxicity for mammals. Studies of mammals ingesting the chemical have produced several dissimilar results, including an acute oral LD_{50} of 2,740 mg/kg for rats (USDA 1994b); an oral LD_{50} of 757 to 1,707 mg/kg in rats, 1190 mg/kg in mice, 2,000 mg/kg in rabbits, and 566 to 3,000 mg/kg in guinea pigs (summarized in EXTOXNET 1996b); and an acute oral LD_{50} of 3,512 mg/kg for rats (Syngenta 2000). The acute oral toxicity of Dicamba in rats is reported within the Pesticide Dictionary (1976 *as cited in* Verschueren 1983) as being 2,900 +/- 800 mg/kg.

Acute dermal studies have shown a slight toxicity, with a LD_{50} of >2,000 mg/kg in both rats and rabbits (Syngenta 2000, summarized in EXTOXNET 1996b). A moderate toxicity of the chemical was also evident in acute inhalation tests, with an LC_{50} of >200 mg/L (USDA 1994b, summarized in EXTOXNET 1996b). An additional inhalation study (Syngenta 2000) placed the LC_{50} for rats at >5.3 mg/L (ppm_{air}) for 4 hours. Application of the chemical to the eyes of rabbits produced a corrosive effect, and with dermal application, Dicamba was also a slight skin irritant (USDA 1994b).

The effects of Dicamba from repeated exposure have also been evaluated (summarized in EXTOXNET 1996b). Two-year studies with rats, with doses of 25 mg/kg/day, produced no observable effects on survival, body weight, food consumption, organ weight, blood chemistry, or tissue structure (summarized in EXTOXNET 1996b). A separate two-year study of the chronic toxicity effects of Dicamba in the diet of rats and dogs reported NOEL concentrations of 500 mg/kg in rats and 50 mg/kg in dogs (Hubert 1968 *as cited in* Verschueren 1983). In addition, consumption of the chemical at high levels for a long period of time has been shown to causes changes in the liver and a decrease in body weight in rats (summarized in EXTOXNET 1996b). Some enlargement of liver cells has also occurred in mice (summarized in EXTOXNET 1996b). Dicamba did not affect the reproductive capacity of rats in a 3-generation study (summarized in EXTOXNET 1996b). However, when rabbits were given doses of 0.5, 1, 3, 10, or 20 mg/kg/day of technical Dicamba from days 6 through 18 of pregnancy, the 10 mg/kg dose resulted in slightly reduced fetal weights, toxic effects on the mothers, and increased loss of fetuses (summarized in EXTOXNET 1996b).

2.7.2 *Birds*

Toxicity testing with Dicamba revealed an acute oral LD_{50} of 637 mg/kg for the pheasant and 2009 mg/kg for the mallard (summarized in EXTOXNET 1996b). The results from these acute studies suggest that Dicamba can be slightly toxic in an acute exposure. However, the 8-day dietary LC_{50} for mallards and northern bobwhites is >10,000 mg/kg, indicating that Dicamba is practically non-toxic when birds are exposed through their diet (summarized in EXTOXNET 1996b).

2.7.3 *Insects*

As previously indicated (see WSDOT 1993), Dicamba is considered to be essentially non-toxic based on an acute contact LD_{50} of >100 µg/bee. Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.7.4 *Reptiles and Amphibians*

Toxicity information of Dicamba with regards to reptiles was not available for review. Dicamba is slightly toxic to amphibians, with a reported LC_{50} concentration of >10 ppm (USDA 1995c).

2.8 Dicamba/2,4-D

2.8.1 *Mammals*

Dicamba/2,4-D is considered to be slightly to moderately toxic to mammals, based on studies conducted with $Brash_{tm}$, a herbicide manufactured with the chemicals (Terra 1999). Acute oral studies with rats determined Brash to be slightly toxic by ingestion (LD_{50}>1,150 mg/kg), and acute dermal studies with rabbits found that dermal application of Brash was not lethal at the highest practical dose tested, although it did produce a slight irritation (EC_{50}>2,000 mg/kg) (Terra 1999).

Four-hour acute inhalation tests with the herbicide in rats yielded an LD_{50}>20.3 ppm (Terra 1999). Contact of the chemicals with the eyes of laboratory animals may produce severe irritation and a possible corrosive effect (Terra 1999).

Studies have also been conducted with laboratory animals to evaluate the effects of chronic exposure to Dicamba/2,4-D (Terra 1999). Overexposure to the chemicals has been shown to cause lung congestion, erythemea, and edema (Terra 1999). In addition, rare cases of nerve damage have been reported, but extensive animal studies have failed to substantiate these claims, even at high doses for prolonged periods (Terra 1999).

2.8.2 *Birds*

Toxicity levels for Dicamba/2,4-D are similar to the original parent compounds. Dicamba is considered slightly to practically non-toxic and 2,4-D is considered practically non-toxic to birds (*as summarized in* BPA 2000d).

2.8.3 *Insects*

Toxicity levels for Dicamba/2,4-D are similar to the original parent compounds. Dicamba and 2,4-D are both practically non-toxic to insects (*as summarized in* BPA 2000d).

2.8.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.9 Dicamba/MCPA

2.9.1 *Mammals*

Dicamba/MCPA has been shown to be slightly toxic to mammals, based on several tests conducted with Banvel, a herbicide formerly produced with Dicamba/MCPA as its main ingredient (Syngenta 2001). Currently Vengeance is also produced with a Dicamba/MCPA mixture. Acute oral tests with rats have shown a low toxicity (LD_{50}>2,034 mg/kg), following single doses of the undiluted product, as have acute dermal tests (LD_{50}>8,000 mg/kg), when the undiluted product is applied directly to the skin of the animal (Syngenta 2001). Similarly, inhaling the undiluted product for a 4-hour time period has also proven slightly toxic to rats (LC_{50}>200 mg/L) (Syngenta 2001). Additional tests have indicated the chemicals are an irritant to the eyes and a mild to moderate irritant to the skin (Syngenta 2001).

The effects of chronic exposure to Dicamba/MCPA have also been evaluated with laboratory testing (Syngenta 2001). The chemicals have not demonstrated any carcinogenic, teratogenic, or other reproductive effects, with the exception of slightly reduced body weights and post-implantation issues reported at the Maximum Tolerated Dose Level (Syngenta 2001).

2.9.2 *Birds*

Toxicity levels for Dicamba/MCPA are similar to the original parent compounds. Dicamba is considered slightly toxic and MCPA is moderately toxic to birds (summarized in EXTOXNET 1996b). The combination of the two chemicals lowers the toxicity level of the resultant herbicide. The acute oral LD_{50} of MCPA in bobwhite quail is 377 mg/kg, and the LD_{50} of Dicamba in mallards is 2,009 mg/kg. However, when administered by diet the LC_{50} for Dicamba is >10,000 ppm for mallards (summarized in EXTOXNET 1996b). Sensitivity to Dicamba/MCPA is slightly higher for pheasants than for bobwhite quail or mallards; however the toxicity level is still considered only slightly toxic based upon U.S. EPA criteria in Table 2-1 (*as summarized in* EXTOXNET 1996b).

2.9.3 *Insects*

Toxicity levels for Dicamba/MCPA are similar to the original parent compounds. Dicamba and 2,4-D are both practically non-toxic to insects (*as summarized in* BPA 2000d).

2.9.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.10 Dichlobenil

2.10.1 *Mammals*

Dichlobenil is practically non-toxic through ingestion, based on studies with male and female rats (LD_{50}=4,250 mg/kg) (USDA 1994C). A previous study reported within the Pesticide Dictionary (1976 *as cited in* Verschueren 1983) represents a slightly lower value for the acute oral LD_{50} in rats of 3,160 mg/kg; the same study also reported levels of >2,460 mg/kg in male mice and 150 mg/kg in guinea pigs. Based on these results, Dichlobenil would be considered moderately toxic to guinea pigs, and practically non-toxic to rats and mice. The chemical also has slight toxicity when applied directly to the skin of male rabbits (LD_{50}=1,350 mg/kg) (USDA 1994C, Hubert 1968 *as cited in* Verschueren 1983).

Studies have also been conducted to evaluate the effects of chronic exposure of Dichlobenil (USDA 1994C). A 2-year feeding study with male and female dogs given doses of Dichlobenil of up to 8.75 mg/kg/day produced several physiological changes at the highest dose tested, including increased liver and thyroid weights (USDA 1994C, Hubert 1968 *as cited in* Verschueren 1983). Developmental effects were tested through laboratory tests in pregnant rats, using doses of Dichlobenil of up to 60 mg/kg/day (USDA 1994C). Although this level did not cause birth defects, it did cause toxic effects to the mothers (USDA 1994C). The highest dose tested (180 mg/kg/day) was also associated with maternal toxic effects, and caused a slightly increased frequency of extra ribs in the rat pups born to these mothers (USDA 1994C). The study concluded that Dichlobenil is harmful to fetuses only at levels that are high enough to harm the mothers (USDA 1994C).

A chronic test of Dichlobenil on an oral basis or administered on the skin of mammals resulted in no observable effects below 20 mg/kg for rats (over 2 years), and 50 mg/kg for rats (over 84 days) and pigs (over 3 months) (summarized in EXTOXNET 1996c). A separate feeding study with male and female dogs (for 2 years, at doses up to 8.75 mg/kg/day), physiological changes were evident at the highest doses including increased liver and thyroid weights (USDA 1995d).

2.10.2 *Birds*

Dichlobenil is slightly toxic to birds, with an acute toxic LD_{50} of 1,500 mg/kg (USDA 1994C).

2.10.3 *Insects*

Verschueren (1983) produced a compilation of Dichlobenil exposure affects to a variety of insects (Table 2-5). In general, insects show limited impacts to Dichlobenil after a 96-hour contact exposure period.

Table 2-5. Insect Response to Dichlobenil Exposure.

Test Species	Exposure Period	LC_{50} Concentration (µg/L)
Pteronarcys californica	96 hours	7,000
Tendipendidae	96 hours	7,800
Callibaetes spp.	96 hours	10,300
Limnephilus	96 hours	13,000
Enallegma	96 hours	20,700

Source: Sanders and Cope 1968, Wilson and Bond 1969

2.10.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.11 Diuron

2.11.1 *Mammals*

Studies conducted with Diuron have found the chemical to be practically non-toxic to mammals, through both ingestion and skin absorption (summarized in EXTOXNET 1996d). Acute oral studies with rats have placed the LD_{50} at 3,400 mg/kg, and dermal studies in rats have determined an LD_{50} of >2,000 mg/kg (summarized in EXTOXNET 1996d). This conclusion is consistent with the previous examination of this chemical (see WSDOT 1993).

Studies have also been conducted to determine the effects of chronic exposure, (summarized in EXTOXNET 1996d). The Environmental Health Program of Canada has conducted two chronic feeding studies with Diuron. Diuron was administered for two years through the diet to groups of two male and three female beagle dogs, and 35 rats of each sex (Environmental Health Program 1989). The dogs were dosed at levels corresponding to 0, 0.625, 3.125, 6.25, or 31.25 mg/kg/day, and the rats were dosed at levels corresponding to 0, 1.25, 6.25, 12.5, or 125 mg/kg/day (Environmental Health Program 1989). Traces of abnormal blood pigments were observed in a few animals at 125 mg/kg (3.125 mg/kg/day in dogs and 6.25 mg/kg/day in rats), and hematological alterations, weight loss, haemosiderosis of the liver and erythroid hyperplasia were observed at 250 mg/kg and above (6.25 mg/kg/day in dogs and 12.5 mg/kg/day in rats) (Environmental Health Program 1989). The NOAEL was 125 mg/kg, or 3.125 mg/kg/day in dogs and 6.25 mg/kg/day in rats (Environmental Health Program 1989).

Chronic toxicity experiments with Diuron, reported by the USDA (1996a), resulted in no to low adverse effects. Effects to male rats included changes in spleen (enlargement), bone marrow, blood chemistry, increased mortality, growth retardation, abnormal blood pigment, and anemia. Diuron administered to pregnant rats (days 6 through 15 of gestation) produced no birth defects up to 125 mg/kg. At a dose of 250 mg/kg offspring began to develop wavy ribs, extra ribs, and delayed bone formation. An increase to 500 mg/kg decreased the weight of offspring, but no additional deformations were noted. No other mutagenic or carcinogenic effects were noted in this study.

An additional chronic feeding study found that male rats given extremely high doses of Diuron over a 2-week period, as well as rats given low doses of Diuron over extended periods of time, showed changes in their liver, spleen, and bone marrow (summarized in EXTOXNET 1996d). Other chronic effects due to high levels of exposure to the chemical include changes in blood chemistry, increased mortality, growth retardation, abnormal blood pigment, and anemia (summarized in EXTOXNET 1996d).

Although Diuron does not appear to be mutagenic, it is teratogenic at high doses (summarized in EXTOXNET 1996d). The chemical produced no birth defects when administered to pregnant rats on days 6 through 15 of gestation (at up to 125 mg/kg/day), but doses of 250 mg/kg/day produced wavy ribs, extra ribs, and delayed bone formation in the offspring (summarized in EXTOXNET 1996d). Weight decreases also occurred in the offspring at 500 mg/kg/day (summarized in EXTOXNET 1996d). The LOAEL was therefore 125 mg/kg/day (Environmental Health Program 1989). Pregnant mice given very high doses of Diuron (nearly 2,000 mg/kg/day) exhibited reproductive and embryotoxic effects, and developmental effects were also found in their offspring (summarized in EXTOXNET 1996d). In addition, a 3-generation reproductive study conducted with the female rats being fed small doses of Diuron daily, resulted in significantly decreased body weight of offspring in the second and third litters (summarized in EXTOXNET 1996d).

2.11.2 *Birds*

As previously reported (see WSDOT 1993), Diuron is slightly to practically non-toxic to birds when ingested, as determined with several dietary studies (summarized in EXTOXNET 1996d). The toxicity varies by species: in northern bobwhite, Diuron is slightly toxic (LC_{50}=1,730 mg/kg); and in Japanese quail, ringnecked pheasant, and mallard ducks, it is practically non-toxic (LC_{50}>5,000 mg/kg) (summarized in EXTOXNET 1996d).

2.11.3 *Insects*

As previously reported, Diuron is essentially non-toxic to bees (Wellings 1991). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.11.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.12 Glyphosate

2.12.1 *Mammals*

Glyphosate is considered practically non-toxic to mammals. Tu *et al.* (2001c) report an acute oral LD_{50} in rats of 5,600 mg/kg. In rabbits, the oral LD_{50} has been reported at 3,800 mg/kg, and the dermal LD_{50} is greater than 5,010 mg/kg (U.S. EPA 1988).

During subchronic toxicity tests using rats and mice, responses to dietary administration of Glyphosate included blood and pancreatic effects, and weight gain (Tu *et al.* 2001c). Newton *et al.* (1984 *as cited in* Tu *et al.* 2001c) examined exposure scenarios from an aerial application within a forest in Oregon. Residue levels were comparable to litter and ground cover (<1.7 mg/kg) and were detectable for 55 days at declining levels. The study concluded that carnivores were at lower risk to herbicide exposure than herbivores due to the lower relative visceral weights and a proportionally lower level of food intake.

2.12.2 *Birds*

As previously reported (see WSDOT 1993), Glyphosate is considered practically non-toxic to birds based on acute oral LD_{50} values of >2,000 mg/kg for the mallard and >4,640 mg/kg for the northern bobwhite (Monsanto 2001). The dietary LC_{50} (4,640 mg/kg for both mallards and northern bobwhites) indicates that Glyphosate is slightly toxic for both of these species (Monsanto 2001). An additional study produced similar results in birds (LC_{50}=5,620 mg/kg), further supporting that Glyphosate is practically non-toxic (Monsanto 2001).

A chronic study involving the effects of chicken eggs submerged in a solution of 5 percent Glyphosate found no effect on hatchability (Tu *et al.* 2001c). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.12.3 *Insects*

As previously reported (see WSDOT 1993), Glyphosate is essentially non-toxic to bees based on a LD_{50} of >100 µg/bee (Monsanto 2001). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.12.4 *Reptiles and Amphibians*

No toxicity information on the effect of Glyphosate on reptiles was identified in the literature review. However, the formulation of Glyphosate, Roundup®, has been observed to interfere with cutaneous respiration in frogs and gill respiration in tadpoles (Tu *et al.* 2001c).

2.13 Metsulfuron Methyl

2.13.1 *Mammals*

Metsulfuron methyl is practically non-toxic to mammals based on an acute oral LD_{50} of >5,000 mg/kg in the rat (USDA 1995e). This toxicity rating is supported by results from an acute dermal study where the LD_{50} was >2,000 mg/kg in female rabbits and over 8,000 mg/kg in male rabbits (*summarized in* EXTOXNET 1996e, USDA 1995e).

Acute inhalation exposure was found to be slightly toxic to rats, with a LD_{50} concentration above 5.3 mg/L-air (*summarized in* EXTOXNET 1996e).

Chronic dietary exposure of 50 mg/kg Metsulfuron methyl to rats over an experimental period of one year reduced red blood cell counts, and increased liver weight; whereas a two year feeding study resulted in no effects below concentrations of 50 mg/kg (*summarized in* EXTOXNET 1996e). Shorter experimental periods (90 days) have shown effects at the 250 mg/kg dose level in rats.

Reproductive studies using rats have noted no effects in dietary exposures up to 500 mg/kg within two successive generations of offspring (*summarized in* EXTOXNET 1996e). No birth defects were noted in rats or rabbits fed doses of Metsulfuron methyl up to 750 mg/kg (high dose). No maternal or fetal effects were noted in concentrations up to 300 mg/kg (moderate dose), aside from lower maternal and fetal body weights at concentrations of 250 mg/kg (*summarized in* EXTOXNET 1996e). No carcinogenic effects of Metsulfuron methyl have been observed in either rats or dogs at low to moderate doses after 2- and 1-year studies, respectively (USDA 1995e).

2.13.2 *Birds*

EXTOXNET (1996e) reports the acute oral and dietary LD_{50} values for mallards as >5,000 mg/kg. The USDA (1995e) has provided similar results to establish that Metsulfuron methyl is practically non-toxic to birds (LC_{50}>5,620 ppm) (USDA 1995e).

2.13.3 *Insects*

As previously summarized (WSDOT), the contact LD_{50} for Metsulfuron methyl is >25 µg/bee, which establishes it as being relatively non-toxicity to bees (Meade 1983). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.13.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.14 Oryzalin

2.14.1 *Mammals*

Oryzalin is considered to be slightly toxic to practically non-toxic to mammals based on an acute oral LD_{50} of >5,000 mg/kg for rats and mice, and >1,000 mg/kg in cats, dogs, and chickens (*summarized in* EXTOXNET 1996f). Large oral doses have been observed to cause nausea and vomiting in dogs and cats. Dermal toxicity exposure of technical Oryzalin LD_{50} is reported as >2,000 mg/kg in rabbits, indicating that it causes slight skin and eye irritation in rabbits (*summarized in* EXTOXNET 1996f). Dermal toxicity exposure of guinea pigs did not show any skin sensitization.

Exposure through inhalation, when tested for a 4-hour experimental period, resulted in a LD_{50} of >3.17 mg/L-air in rats (*as summarized in* BPA 2000e). Formulated products of Oryzalin have been tested to show moderate toxicity through oral or inhalation exposure routes, as well as skin and eye irritation properties (*summarized in* EXTOXNET 1996f).

Chronic toxicity tests looking at the response of rats fed a 2.5 mg/kg/day diet of Oryzalin for 2 years; the study resulted in blood changes, increased liver and kidney weights, inhibition of growth, and decreased survival (*summarized in* EXTOXNET 1996f). Other organ toxicity effects included systemic changes on the thyroid, liver, and kidneys. Dogs showed changes in blood cell formation after repeated ingestion of large doses. Mice given a dietary dose of 200 mg/kg/day for 1 year exhibited decreased uterine and ovarian weights, with a NOEL at doses of 75 mg/kg/day (*summarized in* EXTOXNET 1996f).

Studies showed no effects on reproduction within three generations of rats fed concentrations up to 112.5 mg/kg/day, although changes to the fetus were noted at concentrations of 125 mg/kg/day (*summarized in* EXTOXNET 1996f). Teratogenic tests on rats showed no birth defects in the offspring of rats up to dietary concentrations of 112 mg/kg/day or in pregnant rabbits up to dietary concentrations of 125 mg/kg/day. Results of carcinogenic tests with Oryzalin were reported at conflicting levels between studies with rats and mice. Due to these inconsistencies, carcinogenic effects are unknown at this time; however concentrations of Oryzalin have produced mammary gland tumors (*summarized in* EXTOXNET 1996f).

2.14.2 *Birds*

Oryzalin is considered to be slightly to practically non-toxic to birds based on acute oral LD_{50} values of 506.7 mg/kg in northern bobwhite quail and mallards, and >1,000 mg/kg in chickens (*as summarized in* BPA 2000e, *summarized in* EXTOXNET 1996f).

2.14.3 *Insects*

Oryzalin is not considered to be dangerous to bees when used according to label directions. The reported oral LD_{50} of Oryzalin in bees is 11 µg/bee (*as summarized in* BPA 2000e, *summarized in* EXTOXNET 1996f).

2.14.4 *Reptiles*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.14.5 *Adjuvant and Inert Ingredients*

The commercial formulation of Oryzalin (Surflan Herbicide) is 40.4 percent active ingredient, and 59.6% inert ingredients (*as summarized in* BPA 2000e). Formulations of Oryzalin contain carriers (solvents) to increase the product's solubility.

2.15 Picloram

Picloram, a pyridine compound, is a systemic herbicide used to control woody plants as well as a wide range of annual and perennial broad-leafed herbs, mainly in range management programs since most grasses are resistant. Commercially available products that contain the compound include Access, Grazon, Pathway and Tordon. Additionally, Picloram is reportedly used in formulations with other herbicides such as Bromoxynil, Diuron, 2,4-D, Triclopyr and Atrazine, and is compatible with fertilizers (*summarized in* EXTOXNET 1996g). Available formulations include acid, potassium, triisopropanolamine salt, isooctyl ester in soluble concentrates, pellets, or granular formulations (*summarized in* EXTOXNET 1996g). The toxicity information described below is generally based on the technical acid form.

2.15.1 *Mammals*

Picloram is considered slightly to practically non-toxic to mammals via ingestion (*summarized in* EXTOXNET 1996g). This conclusion is based in part on an acute oral LD_{50} of 5,000 to 8,200 mg/kg in rats, 2,000 to 4,000 mg/kg in mice, 2,000 mg/kg in rabbits, and about 3,000 mg/kg in guinea pigs (Hubert 1968, *summarized in* EXTOXNET 1996g, Tu *et al.*, 2001d). The high acute dermal LD_{50} of more than 3,980 mg/kg for the rabbit also indicates the product has practically no dermal toxicity to mammals (Dow/Elanco undated(a) *as cited in* WSDOT 1993).

Although Picloram is not highly toxic to mammals, Tu *et al.* (2001d) cautions some formulations are highly toxic when inhaled. Due to its persistence in the environment, chronic exposure could be a concern to wildlife. For instance, Tu *et al.* (2001d) indicates studies have reported weight loss and liver damage in rabbits exposed to high concentrations (200 to 400 mg/kg per day) over the long term, although no embryotoxic or teratogenic effects were

reported. Liver effects were also seen in rats at high doses (225 mg/kg per day to 3,000 mg/kg per day) over a 90-day period (*summarized in* EXTOXNET 1996g).

Dogs, sheep and beef cattle exhibited no toxic effects after being fed low levels of Picloram over a month time period, although may have additive effects with other herbicides such as 2,4-D (*summarized in* EXTOXNET 1996g).

2.15.2 *Birds*

Bobwhite quail and the mallard duck reportedly have oral LD_{50} values of greater than 5,000 and 2,510 mg/kg, respectively (Tu *et al*. 2001d). An oral LD_{50} value of 6,000 mg/kg is reported for the chicken (USFS 1989 *as cited in* WSDOT 1993). As previously stated in the 1993 EIS (WSDOT) Picloram has an avian LD_{50} of >2,000 mg/kg for the pheasant (USFS 1989). Collectively, these data indicate that Picloram is practically non-toxic to birds.

2.15.3 *Insects*

Picloram is considered to be relatively non-toxic to bees based on an LD_{50} of 14 µg/bee (USFS 1989, U.S. EPA 1988—*as cited in* WSDOT 1993). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.15.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.16 Sulfometuron Methyl

Sulfometuron methyl is a broad-spectrum urea herbicide used to control annual and perennial grasses as well as broad-leafed weeds in non-cropland such as industrial sites, right-of-ways, fencerows and along roadsides. Sulfometuron methyl also has forestry applications to control woody tree species, and is used for conifer site preparation and release (*summarized in* EXTOXNET 1996h; USDA 1995f).

2.16.1 *Mammals*

Sulfometuron methyl is generally considered (*summarized in* EXTOXNET 1996h) to be practically non-toxic with a very low acute oral toxicity. As previously discussed (see WSDOT 1993), this consideration is based on an acute LD_{50} of greater than 5,000 mg/kg for the male rat. Acute dermal LD_{50} values range from over 2,000 mg/kg for the female rabbit to over 8,000 mg/kg for the male rabbit, which also indicates a rating of practically non-toxic. The USDA

(1995f) reported an inhalation toxicity value of 5 mg/L (ppm) after a 4-hour acute exposure to Oust.

Several studies indicate rats have exhibited multiple toxic effects due to chronic dietary exposures to Sulfometuron methyl. For example, rats experienced reduced red blood cell counts and increased liver weight when fed the compound at relatively low doses (LD_{50} of 50 mg/kg) for a one year period (Jordan and Cudney 1987). In a study conducted over a 90-day period, rats exhibited an increased production of white blood cells at 250 mg/kg (the highest dose tested). However, in a two-year feeding study, no effects were found below 50 mg/kg (Retnakaran and Wright 1987 *as cited in summarized in* EXTOXNET 1996h). The formulation Oust produced mild erythema and slight edema for 72 hours in rabbits (USDA 1995f).

A test conducted over two successive generations of offspring concluded no reproductive-related effects were apparent in rats fed up to 500 mg/kg (Retnakaran and Wright 1987 *as cited in summarized in* EXTOXNET 1996h). Birth defects were not reported in respective offspring of a rat study and two rabbit studies in which the mothers were fed up to 750 mg/kg (Jordan and Cudney 1987 *as cited in summarized in* EXTOXNET 1996h). Although no maternal or fetal effects were noted in rabbits fed up to 300 mg/kg, rats fed up to 250 mg/kg exhibited reduced maternal and fetal body weights (U.S. EPA 1993; U.S. EPA 1995 *as cited in summarized in* EXTOXNET 1996h).

2.16.2 *Birds*

Sulfometuron methyl is considered practically non-toxic to birds. A dietary acute LC_{50} value of greater than 5,000 mg/kg is reported for mallards and dietary LC_{50} values of greater than 5,620 mg/kg is reported for the northern bobwhite quail (*summarized in* EXTOXNET 1996h).

2.16.3 *Insects*

As previously reported (see WSDOT 1993), Sulfometuron methyl is considered to be moderately toxic to bees based on a contact LD_{50} of >12.5 µg/bee (USFS 1989). Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.16.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

2.16.5 *Adjuvant and Inert Ingredients*

USDA (1995f) and *summarized in* EXTOXNET (1996h) indicate commercially available Sulfometuron methyl products, such as Oust® (Oust Weed Killer) and DPX 5648 generally contain one or more inert ingredients, although specific ingredient names are not provided.

Oust® Weed Killer generally contains 75% Sulfometuron methyl and 25% inert ingredients. However, no specific toxicity studies related to this product's adjuvant or inert ingredients were identified for review.

2.17 Triclopyr

Triclopyr, a selective systemic herbicide, is used to control both woody and herbaceous broadleaf plants, generally along right-of-ways, within forests, industrial lands, grasslands and parklands (Tu *et al.* 2001e). Triethylamine salt and butoxethyl ester are the two commercially available forms of Triclopyr in the United States (Tu *et al.* 2001e and *summarized in* EXTOXNET 1996i).

2.17.1 *Mammals*

Triclopyr is considered to be slightly toxic to mammals (Tu *et al.* 2001e). The acute oral LD_{50} values for Triclopyr in rats range from 630 to 729 mg/kg (Kidd and James 1991 *as cited in summarized in* EXTOXNET 1996i). Acute oral LD_{50} values (Triclopyr) listed for the rabbit and guinea pig is 550 mg/kg and 310 mg/kg, respectively. The acute oral LD_{50} concentrations for unspecified amine and ester based products is listed as over 2,000 mg/kg for rats; dermal LD_{50} values are listed as greater than 4,000 mg/kg for rabbits (*summarized in* EXTOXNET 1996i).

Findings of chronic toxicity effects of Triclopyr indicate that rats experienced no adverse effects when administered an oral dose between 3 and 30 mg/kg per day. However, decreased liver weight was apparent in male mice at dietary doses of approximately 60 mg/kg per day; decreased body (and liver) weight and increased kidney weight resulted at doses of 100 mg/kg of Triclopyr per day. Monkeys fed Triclopyr at doses of 20 mg/kg per day reportedly showed no ill effects.

Triclopyr does not appear to cause reproductive or teratogenic toxicity in rats or rabbits. Rabbits fed doses of 25, 50 and 100 mg/kg per day (during days 6 through 18 of the gestation period) reportedly had no effect on maternal body weight, litter size or fetal body weight (see WSDOT 1993). Offspring experienced no teratogenic effects when pregnant rabbits were fed doses between 10 and 25 mg/kg per day (*summarized in* EXTOXNET 1996i). Fertility rates reportedly showed no impact during a three-generation study in which rats were fed Triclopyr (at doses of 3, 10 and 30 mg/kg per day for an 8 to 10 week period prior to breeding). Mild fetotoxicity with no birth defects was reported in offspring of rats fed 50 to 200 mg/kg per day during days 6 though 15 of the gestation period. Additionally, *summarized in* EXTOXNET (1996i) concludes Triclopyr is unlikely to be mutagenic or carcinogenic but does cause organ toxicity in the kidneys and liver.

The U.S. EPA presents an evaluation of available toxicity data for Triclopyr in the Federal Register Environmental Documents (U.S. EPA 2002b). Information within Table 2-6 displays NOEL and LOEL values for rabbits, dogs and rodents that are identified in studies considered by the U.S. EPA to be valid, complete and reliable.

Table 2-6 Toxicity Profile of Triclopyr for Rodents, Dogs and Rabbits

Study Type	Mammal	NOEL	LOEL
90-day oral toxicity with acid	Rat	5 mg/kg/day in males and females	20 mg/kg/day, in males and females based on degeneration of the proximal tubules of the kidneys
90-day oral toxicity with ester	Rat	7 mg/kg/day in males and <7 mg/kg/ rodents with ester - rat day in females	28 mg/kg/day in males, 7 mg/kg/day based on increased relative kidney weight (M) and decreased red blood cell content, hemoglobin content, and packed cell volume (F). Degeneration of the proximal tubules of the kidneys was seen in males at 70 and 350 mg/kg/day and females at 350 mg/kg/day highest dose tested (HDT).
183-day oral toxicity	Dog	<= 2.5 mg/kg/day (HDT) in males and rodents - dog females	>2.5 mg/kg/day in males and females based on toxicologically non-significant decreased rate of phenolsulfothalein (PSP) due to competition between Triclopyr and PSP for renal excretion.
21-Day dermal toxicity	Rabbit	1,000 mg/kg/day (males and females)	>1,000 mg/kg/day. Decreased alkaline phosphatase in both sexes of rabbits at 1,000 mg/kg/day and increased absolute and relative liver weight in males at 1,000 mg/ kg/day were considered marginal and not of toxicological significance.
Prenatal developmental with ester	Rats	Maternal = 100 mg/kg/day Developmental =100 mg/kg/day	Maternal =300 mg/kg/day based on mortality, clinical signs, necropsy findings, decreased body weight gains, decreased food consumption, increased water consumption, and increased relative kidney and liver weight. Developmental = 300 mg/kg/day based on increased incidence of hydrocephalus, cleft palate, microphthalmia/anophthalmia, retinal folds, thin diaphragm/protrusion of the liver, decreased fetal weight and visceral and skeletal anomalies and variants.
Prenatal developmental with ester	Rabbits	Maternal = 30 mg/kg/day Developmental = 30 mg/kg/day	Maternal = 100 mg/kg/day based on mortality Developmental = 100 mg/kg/day based on decreased total live fetuses and increased total fetal deaths, as well as increased fetal and/or litter incidence of skeletal anomalies and variants
Prenatal developmental with salt	Rabbit	Maternal = 30 mg/kg/day Developmental = 30 mg/kg/day	Maternal = 100 mg/kg/day based on mortality, abortions, decreased body weight gain, decreased food efficiency, increased liver and kidney weight Developmental = 100 mg/kg/day based on decreased live fetuses and increased embryonic deaths due to abortions.
Prenatal developmental with salt	Rat	Maternal = 100 mg/kg/day Developmental = 100 mg/kg/day	Maternal = 300 mg/kg/day based on mortality Developmental = 300 mg/kg/day based on decreased fetal weight, increased fetal and litter incidence of skeletal anomalies, increased fetal incidence of unossified sternebrae.

Study Type	Mammal	NOEL	LOEL
Prenatal developmental with acid	Rat	Maternal NOEL = <50 mg/kg/day Developmental NOEL = 100 mg/kg/day	Maternal = 50 mg/kg/day based on increased clinical signs Developmental = 200 mg/kg/day based on increase incidence of fetuses and litters with retarded ossification of skull bones, and two litters (one fetus per litter) with cleft palate and brachycephaly.
Reproduction and fertility in effects with acid	Rat	Parental/Systemic NOEL = 5 mg/kg/day males and in females Reproductive/ Offspring = 5 mg/kg/day in males and females	Parental/Systemic = 25 mg/kg/day in males and females based on increased incidence of proximal tubular degeneration in male and female P1 and P2 rats. Reproductive/Offspring = 25 based on increased incidence of F2 pups with exencephaly and ablepharia.
228-Day toxicity study - acid	Dogs	10 mg/kg/day in males and females	20 mg/kg/day in males and females based on decreased body weight gain (M), decreased hematological parameters (M), changes in clinical chemistry (both sexes), and liver histopathology (both sexes).
Chronic toxicity (1 year) - acid	Dogs	<= 5 mg/kg/day in males and females	>5 mg/kg/day in males and females based on changes in clinical chemistry which are due not to toxicity, but a physiologic response of the dog based on limited ability of the dog to excrete organic acids at higher plasma concentrations.
Chronic/ carcinogenicity acid	Rats	12 mg/kg/day in males, <= 36 mg/kg/ day- in females	36 in males, >36 mg/kg/day in females based on marginal increases in proximal tubular degeneration at 6 months. Increase in adrenal gland pheochromocytoma in males and significant trend (<0.05) for mammary gland adenocarcinomas in females.
Carcinogenic- acid	Mice	84 mg/kg/day in males, 109.5 mg/kg/ day in females	143 mg/kg/day in males, 135 mg/kg/ day in females based on decreased weight gain No evidence of carcinogenicity in males, but females had a significant trend (<0.05) for mammary gland adenocarcinomas

2.17.2 *Birds*

Triclopyr is practically non-toxic to birds. Mallard ducks and bobwhite quail reportedly have oral LD_{50} values of 1,698 mg/kg and 2,935 mg/kg, respectively (*summarized in* EXTOXNET 1996i). After being fed Triclopyr for 8 days, the avian dietary LC_{50} values for the mallard, the northern bobwhite quail and Japanese quail are reported to be >5,600 and 2,935, and 3,278 ppm, respectively (*summarized in* EXTOXNET 1996i). These data indicate exposure of Triclopyr through diet is practically non-toxic for birds (Dow/Elanco undated(b) *as cited in* WSDOT 1993).

Weight loss and behavioral alterations were recorded in zebra finches after the addition of sub-lethal doses of Triclopyr ester to their diets (Holmes *at el.,* 1994 as cited Tu *et al.,* 2001e).

2.17.3 *Insects*

Triclopyr is considered to be relatively non-toxic based on an oral LD_{50} of >60.4 µg/bee. Review of recent literature current through January 2003 did not reveal other research results that differ from these toxicity values or expand on this basic toxicity information.

2.17.4 *Reptiles and Amphibians*

No toxicity information on reptiles or amphibians was identified in a review of recent literature current through January 2003. Toxicity information may exist in unpublished sources, government reports, or privately supported research that was not identified in our reviews of standard ecological toxicity data sources.

New Herbicides Evaluated in 2005

2.18 Bromoxynil

Common forms of Bromoxynil include Bromoxynil phenol, Bromoxynil octanoate and Bromoxynil heptanoate. Bromoxynil octanoate was found to be generally more toxic to test species (U.S. EPA 1998) and is the form that is present in the herbicide Buctril 2EC®.

2.18.1 *Mammals*

Bromoxynil is classified by U.S. EPA as moderately toxic to small mammals based on results from acute oral studies with rats. Bromoxynil octanoate exposure resulted in an acute oral LD_{50} of 238 mg/kg for female rats and 400 mg/kg for male rats (U.S. EPA 1998).

Long-term studies included a two-generation reproductive study in which a NOEL of 250 ppm was reported for reproductive effects and a LOEL of >250 ppm was reported based on body weight reduction in female rats. Another two-generation reproductive study using Bromoxynil phenol reported a NOEL of 50 ppm based on reduced body weight in offspring (U.S. EPA 1998).

2.18.2 *Birds*

The overall toxicity to birds from Bromoxynil is considered slight to moderate based on acute toxicity studies. Acute oral LD_{50} values of 148 mg/kg and 193 mg/kg were reported for bobwhite quails exposed to 87.3% Bromoxynil octanoate and 89.3% Bromoxynil phenol, respectively (Fletcher 1981 *as cited in* U.S. EPA 1998). An LD_{50} of 359 mg/kg was reported for bobwhite quails exposed to 94.8% Bromoxynil heptanoate (Campbell 1993 *as cited in* U.S. EPA 1998). The LD_{50} for mallard ducks exposed to 87.3% Bromoxynil octanoate was 2050 mg/kg (Fletcher 1981 *as cited in* U.S. EPA 1998).

2.18.3 *Insects*

Bromoxynil is considered practically non-toxic to honeybees. In an acute contact study, an LD_{50} of 14.5 µg a.i. per bee was reported for Bromoxynil octanoate (U.S. EPA 1998).

2.18.4 *Reptiles and Amphibians*

No information on toxicity to reptiles or amphibians was identified.

2.18.5 *Adjuvant and Inert Ingredients*

The label for Buctril® herbicide indicates that it contains 66.6% inert ingredients consisting of "xylene range/petroleum distillates" (Bayer 2002a). The MSDS further states that by weight, Buctril® contains a minimum concentration of the following chemicals: trimethyl benzene (14.8%), xylene (10%), and ethyl benzene (2.3%) (Bayer 2002b).

2.19 Diflufenzopyr

2.19.1 *Mammals*

Diflufenzopyr is considered practically non-toxic to small mammals based on acute oral toxicity studies. For technical grade Diflufenzopyr, LD_{50} values were >5,000 mg/kg for both male and female rats. Acute oral LD_{50} values for formulated product were 4,800 and 3,300 mg/kg for male and female rats, respectively. Acute dermal LD_{50} values in male and female rabbits were >5,000 mg/kg for both technical grade and formulated product. Acute inhalation studies in rats resulted in an LC_{50} value of >2,930 mg/m^3 for technical grade Diflufenzopyr and >5,210 mg/m^3 (381 ppm) for manufacturing use product (U.S. EPA 1999), which makes it highly toxic via this route.

Additional studies reviewed by U.S. EPA indicated that technical grade Diflufenzopyr was non-irritating to rabbits while the manufacturing use product was very slightly irritating, but irritation resolved within 24 hours. Neither technical grade nor manufacturing grade Diflufenzopyr exhibited dermal sensitization in guinea-pigs. Exposure to technical grade Diflufenzopyr resulted in mild eye irritation in rabbits, while manufacturing grade Diflufenzopyr resulted in slight to mild eye irritation (U.S. EPA 1999).

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Diflufenzopyr. In a subchronic study in rats administered technical grade Diflufenzopyr in their diet, a NOAEL of 352 mg/kg/day for males and 431 mg/kg/day was reported based on decreased body weight and food efficiency. A 2-generation reproductive study in which technical Diflufenzopyr was administered in the diet of rats resulted in a systemic LOAEL range of 113-176 mg/kg/day based on changes in body weight, increased food consumption, and increased seminal vesicle weight. In a developmental study in which rats were administered technical Diflufenzopyr by gavage, maternal NOAEL and LOAEL values were 300 and 1,000 mg/kg/day, respectively, based on decreases in food consumption and weight

gain. Developmental effects in offspring, including decreased body weight and skeletal variations, were seen at 1,000 mg/kg/day (U.S. EPA 1999). In another study in which rats were exposed to 98.1% Diflufenzopyr, developmental abnormalities of the musculoskeletal system were noted at 10,000 mg/kg (MDL 2002).

In a combined chronic toxicity/carcinogenicity study in which rats were fed technical grade Diflufenzopyr, NOAEL values were 236 mg/kg/day and 323 mg/kg/day based on systemic toxicity. No evidence of oncogenic potential was found at any of the dose levels tested (U.S. EPA 1999).

2.19.2 *Birds*

Diflufenzopyr is practically non-toxic to birds based on an acute oral LD_{50} >2,250 mg/kg for unspecified avian species (U.S. EPA 1999).

2.19.3 *Insects*

Diflufenzopyr is considered practically non-toxic to honey bees based on an LD_{50} >25 µg a.i. per bee (U.S. EPA 1999).

2.19.4 *Reptiles and Amphibians*

No information on toxicity to reptiles or amphibians was identified.

2.19.5 *Adjuvant and Inert Ingredients*

The label for Overdrive® herbicide indicates that this product contains 23.6% inert ingredients. The specific ingredients were not provided on the label (BASF 2003).

2.20 Flumioxazin

2.20.1 *Mammals*

Acute toxicity studies conducted with Flumioxazin have found the product to be practically non-toxic to rats both by the oral and dermal routes. An LD_{50} of >5,000 mg/kg was reported for rats exposed to technical grade Flumioxazin in an acute oral exposure study. Via the dermal route, an LD_{50} of >2,000 mg/kg was reported for rats. An LD_{50} of 3,930 mg/m^3 (377 ppm) was reported for rats exposed to Flumioxazin via inhalation (U.S. EPA 2001), making it highly toxic via this route.

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Flumioxazin. In separate 90-day oral studies of rats, LOAEL values ranged from 197 - 244 mg/kg/day based on changes in blood parameters. An oral rat study of developmental effects reported a LOAEL of 10 mg/kg/day based on cardiovascular effects, including ventrical septal defects (U.S. EPA 2001).

Another chronic rat study reported changes in body weight following exposure to 300 mg/kg/day Flumioxazin for 10 days; changes in liver weight, spleen and blood parameters were reported in rats exposed to 1,620 mg/kg for 90 days (MDL 2004).

2.20.2 *Birds*

Flumioxazan is practically non-toxic to avian species according to studies conducted by the manufacturer. Acute oral LD_{50} values for bobwhite quail were >2,250 mg/kg. A dietary LD_{50} of >5,620 ppm was reported for the mallard duck (U.S. EPA 2001, Valent 2003).

In longer term studies, a NOEL for reproductive effects (effects on egg production) in mallard ducks was reported as 250 ppm. In bobwhite quails, the NOEL for reproductive effects was >500 ppm technical grade Flumioxazan in the diet (Valent 2003).

2.20.3 *Insects*

Flumioxazan is reported to be practically non-toxic to bees with a LC_{50} >105 µg/bee (U.S. EPA 2001).

2.20.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.20.5 *Adjuvant and Inert Ingredients*

The label for Payload® herbicide indicates that this product contains 49% "other ingredients" aside from the active ingredient Flumioxizan (Valent 2003). The MSDS further indicates that Payload® contains by weight the following ingredients: kaolin clay (16%) titanium dioxide (<1%), crystalline silica (<1%).

2.21 Fluroxypyr

2.21.1 *Mammals*

Fluroxypyr was found to be practically non-toxic to small mammals based on acute oral and dermal toxicity studies. An acute oral LD_{50} of >5,000 mg/kg was reported for rats (U.S. EPA 1998). Elsewhere, an acute LD_{50} of 2,405 was reported (MDL 1994a). Via the dermal route, an LD_{50} of >2,000 mg/kg was reported in rats. Via inhalation, an LC_{50} of >2,000 mg/m^3 (192 ppm) was reported for rats (U.S. EPA 1998). Elsewhere, an LC_{50} of >296 mg/m^3 (28.4 ppm) was reported for rats exposed via inhalation for 4 hours (MDL 1994a), making this compound very highly toxic via this route.

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Fluroxypyr. In a 90-day feeding study in rats, LOAEL values of 750 and 1,000 mg/kg/day

were reported for male and female rats, respectively, based on kidney effects and death. In a 2-generation reproductive study in rats, a LOAEL of 1,000 mg/kg/day was reported based on pup body weight gain and slightly decreased survival. In a developmental toxicity study in rats, a maternal LOAEL of 250 mg/kg/day was reported based on clinical signs and a developmental LOAEL of 500 mg/kg/day was reported based on reduced ossification. A combined chronic toxicity/carcinogenicity study in rats resulted in a LOAEL of 5,000 mg/kg/day based on increased kidney weight and chronic glomerulopathy, increased incidence of atrophy, and adipose tissue changes (mesenteric tissues) (U.S. EPA 1998).

2.21.2 *Birds*

Fluroxypyr is practically non-toxic to avian species based on acute exposure of bobwhite quail and mallard ducks that resulted in LD_{50} values >2,000 mg/kg. Following a subacute 5-day exposure, an LC_{50} of >5,000 mg/kg was reported (U.S. EPA 1998).

2.21.3 *Insects*

Fluroxypyr was reported to be practically non-toxic to honey bees with an LD_{50} >25 µg/bee (U.S. EPA 1998).

2.21.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.21.5 *Adjuvant and Inert Ingredients*

The MSDS for Vista® herbicide indicates that this product contains 73.8% inert ingredients aside from the active ingredient Fluroxypyr. Although the complete list of these inerts is not included, the following chemicals are listed: 1-methyl-2-pyrrolidinone and petroleum solvent, which includes naphthalene (Dow AgroSciences 2004).

2.22 Imazapyr

2.22.1 *Mammals*

Imazapyr is practically non-toxic to mammals based on an acute oral LD_{50} of >5,000 mg/kg in rats. Acute dermal toxicity of >2,000 mg/kg was reported in rabbits (MDL 1996, USDA 1995).

2.22.2 *Birds*

Imazapyr is practically non-toxic to birds. Oral LD_{50} values of >2,150 were reported for both quail and duck (MDL 1996).

2.22.3 *Insects*

Imazapyr was reported to have low toxicity to bees with a reported LD_{50} of >100 µg/bee (USDA 1995).

2.22.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.22.5 *Adjuvant and Inert Ingredients*

The labels for Habitat® and Arsenal® herbicides indicate that these two herbicides contain 71.3% inert ingredients in addition to 28.7% Imazapyr (BASF 2000, BASF 2004a). The MSDS for Arsenal® indicates that the formulation contains 46.9% inert ingredients and 54.1% Imazapyr (BASF 2000b). The specific inert ingredients were not provided by the manufacturer. However, Grisolia *et al*. (2004) indicate that the surfactant nonylphenol ethoxylate is used with Imazapyr; they report on the toxicity of this compound in mice (see Chapter 2, Section 2). Information on the toxicity of the surfactant nonylphenol ethoxylate to fish and aquatic invertebrates was not available among the reference sources searched (Section 1.2).

2.23 Isoxaben

2.23.1 *Mammals*

Isoxaben is practically non-toxic to mammals based on an acute oral LD_{50} of >10,000 mg/kg in rats and mice. Isoxaben is moderately toxic via the dermal route, based on an LD_{50} of >200 mg/kg reported in rabbits. Via inhalation, an LD_{50} >1,990 mg/m^3 (146 ppm) was reported in rats (MDL 1994b), making it highly toxic via this route.

2.23.2 *Birds*

Isoxaben is practically non-toxic to birds based on an acute oral LD_{50} of >2,000 mg/kg reported in quail (MDL 1994b).

2.23.3 *Insects*

No information on toxicity to insects was identified.

2.23.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.23.5 *Adjuvant and Inert Ingredients*

The MSDS for Gallery* 75® Dry Flowable herbicide indicates that it contains 25% "other ingredients" including Kaolin and crystalline silica in Kaolin (Dow AgroSciences 2003). Information on these particular compounds was not identified among the reference sources searched (See Section 2.0).

2.24 Norflurazon

2.24.1 *Mammals*

Norflurazon is practically non-toxic to small mammals based on an acute oral LD_{50} of 9300 mg/kg reported for rats (U.S. EPA 1996b).

2.24.2 *Birds*

Norflurazon is practically non-toxic to slightly toxic to birds. Formulated product with 80% active ingredient was slightly toxic to the mallard duck and bobwhite quail with reported LD_{50} values of >1,000 mg/kg (Shellberger 1971 *as cited in* U.S. EPA 1996b). An LD_{50} of >2,510 mg/kg was reported for mallard ducks exposed orally to technical grade Norflurazon (Fink 1972 *as cited in* U.S. EPA 1996b).

In subacute dietary studies, Norflurazon was practically non-toxic to both bobwhite quails and mallard ducks with LC_{50} values of >10,000 mg/kg. In chronic reproductive studies, NOEC and LOEC dietary concentrations of 40 ppm and 200 ppm, respectively, were reported for bobwhite quails for reduced hatchling success (Beavers *et al.*, 1992 *as cited in* U.S. EPA 1996b).

2.24.3 *Insects*

Norflurazon is classified as practically non-toxic to insects based on an acute toxicity study conducted with honey bees with exposure to two different dose levels. Exposure to 97.6% Norflurazon resulted in an LD_{50} of >235 µg a.i./bee, while exposure to 80% Norflurazon resulted in an LD_{50} of >90 µg a.i./bee (Atkins 1985 *as cited in* U.S. EPA 1996b).

2.24.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.24.5 *Adjuvant and Inert Ingredients*

The label for Predict® herbicide indicates that this product contains 21.4% "other ingredients" aside from the active ingredient Norflurazon (Syngenta 2001). The specific inert ingredients were not provided by the manufacturer.

2.25 Oxadiazon

2.25.1 *Mammals*

Oxadiazon is practically non-toxic to mammals based on studies evaluated by U.S. EPA (2003a). An LD_{50} was reported for rats exposed orally to >5,000 mg/kg (U.S. EPA 2003a). In another study of rats exposed orally, an LD_{50} of 3,500 mg/kg was reported. An LD_{50} of 5,200 mg/kg was reported following dermal exposure in rats. In rabbits, a dermal LD_{50} of >2,000 mg/kg was reported (MDL 2003a).

Chronic exposure to 200 ppm in the diet resulted in a LOAEL of $\geq$38 mg/kg/day for inactive mammary tissue and fetal pup death (U.S. EPA 2003).

2.25.2 *Birds*

An oral acute LD_{50} of 1,040 mg/kg was reported for mallard ducks exposed to Oxadiazon via acute oral exposure. In another study reviewed by U.S. EPA, LD_{50} values of >5,000 ppm were reported for quail and mallard ducks following dietary exposure (U.S. EPA 2003a). Elsewhere, an LD_{50} of 6,000 mg/kg was reported for quail exposed orally to Oxadiazon; for ducks, an LD_{50} of 1,000 mg/kg was reported (MDL 2003a). Based on the acute oral study of quails evaluated by U.S. EPA, Oxadiazon is slightly toxic to birds.

2.25.3 *Insects*

No information on toxicity to insects was identified.

2.25.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.25.5 *Adjuvant and Inert Ingredients*

The label for Ronstar 50 WSP® herbicide indicates that this product contains 50% "inert ingredients" aside from the active ingredient Oxadiazon (Bayer 2004). The specific inert ingredients were not provided by the manufacturer.

2.26 Pendimethalin

2.26.1 *Mammals*

Pendimethalin is considered to be slightly toxic to small mammals by the oral route based on an acute oral rat study in which LD_{50} values were reported as 1,050 mg/kg for females and 1,250 mg/kg for males (U.S. EPA 1997a).

Chronic effects on reproduction were reported for a two-generation rat study evaluated by U.S. EPA. In this study, a reproductive NOEL of 2,500 ppm and a LOEL of 5,000 ppm were reported.

2.26.2 *Birds*

Pendimethalin was found to be slightly toxic to the mallard duck following an acute oral LD_{50} of 1,421 mg/kg (Fink 1976 *as cited in* U.S. EPA 1997a). In a subacute dietary study, LC_{50} values of 4,187 ppm and 4,640 ppm were reported for bobwhite quail and mallard duck, respectively (Fink 1973 *as cited in* U.S. EPA 1997a).

Long-term avian exposure has not been adequately characterized and U.S. EPA reports that there is a concern related to potential reproductive effects due to ingestion of fish that might bioaccumulate Pendimethalin (U.S. EPA 1997a).

2.26.3 *Insects*

Pendimethalin was reported to be practically non-toxic to honey bees based on an LD_{50} value of >49.7 µg/bee (Atkins 1974 *as cited in* U.S. EPA 1997a).

2.26.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.26.5 *Adjuvant and Inert Ingredients*

The two Pendimethalin-containing herbicide formulations suggested for use by WSDOT contain varying degrees of inert ingredients. Pendulum® WDG contains 40% inert ingredients that are not specified by the manufacturer (BASF 2001); Pendulum 3.3 EC contains 62.6% inert ingredients that include petroleum distillates (BASF 2004b).

2.27 Pyraflufen

2.27.1 *Mammals*

A summary report presented by the European Commission (EC) indicates that Pyraflufen is practically non-toxic to mammals based on an acute oral LD_{50} value of >5,000 mg/kg in rats. A dermal LD_{50} of >2,000 mg/kg was reported for rats. Via the inhalation route, an LD_{50} of >5,030 mg/m^3 (298 ppm) was reported in rats (EC 2002), making it highly toxic via this route.

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Pyraflufen. In a 90-day oral toxicity study in rats, a LOAEL range of 1,489-1,503 mg/kg/day was reported based on death and clinical signs associated with liver function and

erythrocytes. In a reproductive study, a LOAEL range of 721-844 mg/kg/day (10,000 ppm) was reported based on changes in body weight (U.S. EPA 2003b).

2.27.2 *Birds*

Pyraflufen is practically non-toxic to birds according to a European Commission (EC) summary that reported an acute LD_{50} of >2,000 mg/kg for birds. Dietary toxicity to birds was reported as an LC_{50} > 5,000 mg/kg food. A NOEC for avian reproductive toxicity was reported as 50 mg/kg food. The species tested in these studies were not provided in this EC summary report (EC 2002).

2.27.3 *Insects*

No information on toxicity to insects was identified.

2.27.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.27.5 *Adjuvant and Inert Ingredients*

The label for Edict® IVM herbicide indicates that this product contains 97.5% "other ingredients" aside from the active ingredient Pyraflufen (Nichino America 2004). The specific inert ingredients used in this product were not provided by the manufacturer.

2.28 Sulfentrazone

2.28.1 *Mammals*

Sulfentrazone is practically non-toxic to small mammals based on an acute oral LD_{50} of 2,855 mg/kg for rats. Formulated product reportedly had similar toxicity to the active ingredient. Via the dermal route, an LD_{50} of >2,000 mg/kg was reported in rats. Following inhalation exposure, an LC_{50} of >4,130 mg/m^3 (261 ppm) was reported by U.S. EPA (1997b), making it highly toxic via this route.

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Sulfentrazone. In a subchronic 90-day feeding study, LOEL values of 65.8 and 78.1 mg/kg/day were reported for male and female rats, respectively. NOEL values of 19.9 and 23.1 mg/kg/day were reported for male and female rats, respectively. In a dietary 2-generation reproductive study in rats, systemic and reproductive/developmental NOEL values of 14 and 16 mg/kg/day were reported for males and females, respectively. A developmental toxicity study in rats reported a maternal LOEL of 50 mg/kg/day and a NOEL of 25 mg/kg/day based on effects to the spleen. The developmental (fetal) LOEL and NOEL values for this study were reported as 25 and 10 mg/kg/day, respectively, based on decreased body weight and retardation

of skeletal development. No evidence of carcinogenicity was reported in a 2-year carcinogenicity feeding study of rats. This study reported LOEL values of 82.8 mg/kg/day in males and 67 mg/kg/day in females, and NOEL values of 40 mg/kg/day in males and 36.4 mg/kg/day in female (U.S. EPA 1997b).

2.28.2 *Birds*

U.S. EPA's review of toxicity data indicates Sulfentrazone is practically non-toxic to birds with an LD_{50} >2,250 mg/kg following acute oral exposure. An additional dietary study reported an LD_{50} >5,620 ppm. Bird species tested were not identified (U.S. EPA 1997b).

2.28.3 *Insects*

No information on toxicity to insects was identified.

2.28.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.28.5 *Adjuvant and Inert Ingredients*

The label for Portfolio® herbicide indicates that this product contains 25% inert ingredients aside from the active ingredient Sulfentrazone (Wilbur-Ellis 2005). The specific inert ingredients were not provided by the manufacturer.

2.29 Tebuthiuron

2.29.1 *Mammals*

Tebuthiuron is slightly to moderately toxic to small mammals based on acute toxicity studies. An acute oral LD_{50} value of 388 mg/kg has been reported for rats (U.S. EPA 1994). Elsewhere, reported oral LD_{50} values were 644 mg/kg in rats, 579 mg/kg in mice, and 286 mg/kg in rabbits (EXTOXNET 1996, MDL 2003b). Via the dermal route, Tebuthiuron toxicity ranges from practically non-toxic to moderately toxic based on an LD_{50} >5,000 mg/kg in rats (MDL 2003) and an LD_{50} of >200 mg/kg in rabbits (EXTOXNET 1996). Via inhalation, an LC_{50} >3,696 mg/m^3 (396 ppm) was reported based on ataxia, effects on seizure threshold, and dyspnea (MDL 2003), making it highly toxic via this route. Elsewhere, it was reported that inhalation of technical grade Tebuthiuron for 4 hours did not result in toxicity (EXTOXNET 1996).

Longer-term toxicity studies have been developed to evaluate chronic exposure to lower doses of Tebuthiuron. In a 3 month chronic toxicity study in rats, a dose of 125 mg/kg/day resulted in weight gain, changes in blood cell counts, and effects on the pancreas (EXTOXNET 1996). In a 2-generation reproductive study, a LOEL of 200 mg/kg/day was reported along with a NOEL of 100 mg/kg/day (U.S. EPA 1994). Elsewhere, it was reported that dietary concentrations up

to 56 mg/kg/day did not result in reproductive effects or abnormalities in offspring (EXTOXNET 1996).

2.29.2 *Birds*

Tebuthiuron is practically non-toxic to birds based on an LD_{50} >2,000 mg/kg for mallard ducks exposed to 98% Tebuthiuron in an acute oral exposure study. In subacute oral exposure studies, LD_{50} levels were >5,000 ppm for quail and mallard ducks exposed to 99.1% Tebuthiuron. In chronic reproductive studies with inoculated food, NOELs of 100 ppm in food were reported for these same species (U.S. EPA 1994).

2.29.3 *Insects*

Tebuthiuron is practically non-toxic to honey bees based on an LD_{50} value of >100 µg/bee exposed to 99.1% material (U.S. EPA 1994). Elsewhere, an LD_{50} of >30,000 µg/bee was reported, although the concentration of active ingredient used in this test was not specified (EXTOXNET 1996).

2.29.4 *Reptiles and Amphibians*

No information on toxicity to reptiles and amphibians was identified.

2.29.5 *Adjuvant and Inert Ingredients*

The label for Spike® 80 DF herbicide indicates that this product contains 20% inert ingredients aside from the active ingredient Tebuthiuron (Dow AgroSciences 1999b). Specific inert ingredients used in this product were not provided by the manufacturer.

3.0 Wildlife Exposure Assessment and Risk Characterization

3.1 Methodology for Mammals and Birds

Similar to the original 1993 Draft EIS (WSDOT 1993), this supplemental wildlife exposure assessment and risk characterization relies heavily on toxicity data from the rat and quail to qualitatively gauge risks. The rat is a representative omnivore that is relatively abundant, particularly on the western side of the state. The quail is particularly abundant in eastern Washington and commonly occupies edge habitats along roadways. There are substantial toxicity data available for these species; however, they may not represent the wildlife receptors at greatest risk because of their life history and/or dietary patterns. Thus, in addition to these sentinel animals, we examined exposures to several other surrogate species to provide perspective on the potential doses consumed by wildlife receptors reflective of the different physiographic regions in Washington State.

Exposure parameters were acquired from the Wildlife Exposure Handbook for the marsh wren, American Robin, deer mouse, and meadow vole. The marsh wren is primarily a wetland species that consumes a diet almost exclusively of animals and insects, and is particularly common in coastal areas. The American robin is a migratory passerine that consumes a mixed diet of soil-associated invertebrates and fruit, and is very common in the Puget Sound lowland. The meadow vole represents the wildlife receptor most likely to receive the highest dietary dose based on its high food consumption rate of a diet exclusively of herbs and shoots, and its lack of migratory behavior. The deer mouse consumes a diet of seeds and nuts primarily and is found throughout the state and at higher altitudes than any of the other species considered.

The route of exposure considered in this assessment is limited to ingestion through the consumption of grain treated with the active ingredient of the herbicide formulation. Although other routes of exposure are acknowledged as possible (*e.g.*, dermal and inhalation), the current application practices of WSDOT and the life-history behaviors of the wildlife receptors potentially exposed preclude these exposure routes from substantive or significant additional exposure. Trucks with focused application equipment create noise that will cause birds and diurnal mammals to avoid the immediate area. Similarly, nocturnal animals such as the rat and meadow vole would largely avoid dermal and inhalation exposure because WSDOT applies herbicides only during daylight hours. Defining the quantitative doses of additional dermal or inhalation exposures cannot be considered further without site-specific studies that are beyond the scope of this qualitative risk assessment.

Although the inhalation and dermal exposure routes are not considered in this assessment, the assumptions of the dietary exposure route considered in this assessment presume extreme exposure conditions such that these other exposure routes are sufficiently accounted for. For example, we have assumed that the entire home range of the wildlife receptors has received herbicide application. The assessment also considers that the diets of the receptors are exclusively wheat grain, and that the surface area of application is completely covered with available wheat for forage.

A variety of other assumptions should be recognized in this exposure assessment, as originally detailed (WSDOT 1993). First, although the grains eaten by mammals and birds 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 was used as an estimate of the surface area and weight of other grains. Second, average weights and average daily consumption for the wildlife receptors considered were used to represent exposure. However, these numbers can exhibit a great deal of inter- and intra-species variation. Third, when toxicity information for an exposed receptor was not available, as was the case for almost all of the additional receptors considered in this supplemental EIS (*i.e.*, the robin, meadow vole, deer mouse and marsh wren), the herbicide sensitivity of test animals with the closest dietary patterns to the other receptors was considered. Finally, it must be recognized that the wildlife exposure assessment and risk characterization provided here considers the consumption of wheat grain, but the environmental fate data that are generally referenced are the half-lives in soil. Wheat grain is not likely to remain on the ground 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. These assumptions should provide a “worst case” exposure scenario for considering risk.

The values used to calculate consumption of wheat utilized the average consumption pattern of each wildlife receptor represented in Table 3-1. The surface area and weight of one grain of

wheat and the weights and daily food consumption for rats and quail are based on those presented in the original EIS. Thus, the surface area of one grain of wheat was 1.0×10^{-5} m^2, and the weight of a typical grain of wheat was considered to be 40 mg (4×10^{-2} g/grain). Although several of the species were omnivorous, their diet was considered to be exclusively grain-based for this assessment, adding another element of conservatism in the interpretation of risks.

This assessment of exposure was performed using the data in Table 3-1 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, was determined.

   No. of grains/acre = 4047 m^2/acre ÷ 1.0×10^{-5} m^2

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

   Chemical/grain (mg) = (# lbs/acre ÷ # grains/acre) x 453,592.4 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)

Results of the acute dietary assessment are presented in Table 3-2, along with conservative estimates of LD_{50} for rats, bobwhite quail, and mallard ducks (as available). Qualitative estimation of risk was made by dividing the LD_{50} for each species by the estimated dietary dose of potential exposure according to WSDOT's roadside application rates (Table 3-3).

Table 3-1. Exposure Parameters for Addressing Risks from WSDOT's Herbicide Applications

Species	Body Weight (grams)	Food Intake (g/day)	Water Intake (ml/day)	Inhalation Rate (m3/day)	Surface Area (cm2)	Diet Preference	Relevant Life History Characteristics to Exposure from WSDOT Herbicides
Bobwhite quail	B: 190	15	20	F: 0.10 M: 0.11	F: 298 M: 320	Plants and insects. Max insects 20% in summer	Breeding in April-July; hatching May to August; Non-migratory; annual mortality rate of approx. 80%
Marsh Wren	B: 11.25	8	3	No data	F: 45 M: 48	Insects, spiders, mollusks, and crustaceans	Breed in April; hatch in May; Migration in fall and spring
American robin	B: 84.9	77	12	No data	182	Insects 71 to 83%,fruit 17-29% (summer & spring)	Breed in April; hatch in May, molt in fall; migrate in September and Feb-March.
Deer Mouse	B:21	9 (lactating)	7	F: .025 M: 023	F: 86 M: 91	Mixture of nuts, seeds, and insects	Breed several times during the year
Cottontail Rabbit	B: 1,286	No data	125	0.63	1,254	Grasses, shrubs, woody plants	Breed several times during the year
Norway Rat	B: 300	15	No data	No data	No data	Omnivorous	Breed several times during the year
Meadow Vole	B: 36.7	12	8	0.052	F: 143 M: 161	Vegetation, shoots; grasses, seeds	Breed in early April; Disperses in fall/winter

Table 3-2. WSDOT Herbicide Use and Estimated Doses to Terrestrial Wildlife Receptors

Chemical Name	Active Ingredient Applied/Acre (mg/m^2)	Estimated Rat Dietary Dose (mg/Kg)	Estimated Quail Dose (mg/Kg)	Estimated Deer Mouse Dose (mg/Kg)	Estimated Meadow Vole Dose (mg/Kg)	Estimated Marsh Wren Dose (mg/Kg)	Estimated Robin Dose (mg/Kg)	Rat LD_{50} (mg/Kg)	Quail LD_{50} [Duck LD_{50}] (mg/Kg)
2,4-D (Weedar 64, Amine 4)	426	5.3	8.48	45.6	34.8	75.4	96.6	370	472 (pheasant) [2,000]
Ammonium Salt of Fosamine (Krenite)	897	11.2	17.7	96.1	73.3	158.8	203.4	5,000	5,000 (both)
Bromacil/Diuron (Krovar)	717	8.9	14.2	76.8	58.6	126.9	162.6	2,300	2,250 (both)
Chlorsulfuron (Telar)	16	0.2	0.3	1.7	1.3	2.8	3.6	2,341	5,000 (both)
Clopyralid (Transline)	42	0.5	0.8	4.5	3.4	7.4	9.5	5,000	2,000 (both)
Clopyralid/2,4-D (Curtail)	133	1.7	2.6	14.3	10.9	23.5	30.2	2,830	2,000 (est.)
Dicamba (Vanquish)	112	1.4	2.2	12.0	9.2	19.8	25.4	2,740	637 (pheasant) [2,009]
Dicamba/2,4-D (Weedmaster)	244	3.1	4.8	26.1	19.9	43.2	55.3	1,150	2,000 (est.)
Dicamba/MCPA (Vengeance)	210	2.6	4.1	22.5	17.1	37.2	47.6	2,034	377 [2,009]
Dichlobenil (Casoron)	448	5.6	8.8	48.0	36.6	79.3	101.6	3,160	1,500 (both)
Diuron Karmex, Diuron, Direx)	538	6.7	10.6	57.6	44.0	95.2	122.0	3,400	1,730 [5,000]
Glyphosate (Roundup, Rodeo, Aquamaster)	224	2.8	4.4	24.0	18.3	39.6	50.8	5,600	4,640 [2,000]
Metsulfuron methyl (Escort)	8	0.1	0.2	0.9	0.7	1.4	1.8	>5,000	5,000
Oryzalin (Surflan)	448	5.6	8.8	48.0	36.6	79.3	101.6	>5,000	507
Picloram (Tordon)	112	1.4	2.2	12.0	9.2	19.8	25.4	5,000	5,000 [2,510]

Chemical Name	Active Ingredient Applied/Acre (mg/m^2)	Estimated Rat Dietary Dose (mg/Kg)	Estimated Quail Dose (mg/Kg)	Estimated Deer Mouse Dose (mg/Kg)	Estimated Meadow Vole Dose (mg/Kg)	Estimated Marsh Wren Dose (mg/Kg)	Estimated Robin Dose (mg/Kg)	Rat LD_{50} (mg/Kg)	Quail LD_{50} [Duck LD_{50}] (mg/Kg)
Sulfometuron methyl (Oust)	26	0.3	0.5	2.8	2.1	4.6	5.9	5,000	5,620 [5,000]
Triclopyr (Garlon)	448	5.6	8.8	48.0	36.6	79.3	101.6	630	2,935 [1,698]
New Herbicides Evaluated in 2005									
Bromoxynil	56	0.70	1.1	6.0	4.6	10	13	238	148
Diflufenzopyr	39	0.49	0.77	4.2	3.2	7.0	8.9	5,000	2,250 [5,620]
Flumioxazin	28	0.35	0.55	3.0	2.3	5.0	6.4	5,000	2,250
Fluroxypyr	28	0.35	0.55	3.0	2.3	5.0	6.4	2,405	2,000
Imazapyr	112	1.40	2.2	12	9.2	20	25	5,000	2,150
Isoxaben	112	1.40	2.2	12	9.2	20	25	10,000	2,000
Norflurazon	440	5.5	8.7	47	36	78	100	9,300	1,000
Oxadiazon	224	2.8	4.4	24	18	40	51	5,000	5,000
Pendimethalin	336	4.2	6.6	36	27	60	76	1,050	[1,421]
Pyraflufen	0.45	0.01	0.009	0.048	0.037	0.080	0.10	5,000	2,000 (unspecified species)
Sulfentrazone	42	0.53	0.83	4.5	3.4	7.5	9.5	2,855	2,250
Tebuthiuron	269	3.4	5.3	29	22	48	61	388	[2,000]

Table 3-3. Qualitative Risk Characterization of Potential Dietary Herbicide Exposure.

Ratio of Exposure Dose vs. Dietary LD_{50}*	Risk Characterization
0 to 10	High
11 to 24	Moderate

25 to 250	Low
>250	Insignificant

*this ratio is the "hazard quotient", determined by dividing dietary LD_{50} by estimated exposure dose.

3.2 Methodology for Evaluating Insects, Reptiles and Amphibians

3.2.1 *Insects*

Exposure for insects involves direct contact due to spray techniques. An estimate of exposure could not be projected from our current understanding of WSDOT's practices and the publicly available toxicity literature; thus, risk characterization for insects was only evaluated on a qualitative level based on acute toxicity data.

3.2.2 *Reptiles and Amphibians*

Reptiles and amphibians can be exposed to herbicides through dietary consumption, inhalation and direct skin contact. Amphibians may be susceptible to additional exposure in run-off waters. Exposure parameters have not been developed to accurately gauge reptile or amphibian exposures.

No toxicity levels have been reported for reptiles and amphibians to the herbicides used by WSDOT, and exposure parameters have not been fully developed. Thus, risks to reptiles and amphibians cannot be fully assessed. Life history behaviors and WSDOT application practices would suggest that exposures to herbicides would be low under most circumstances. Roadside areas near aquatic habitats where amphibians and some reptiles (*e.g.*, garter snakes and turtles) might be found will be protected by the minimum 60 ft buffer that will be employed by WSDOT. Reptiles in Washington's arid climates will generally avoid disturbance zones around roadways. In addition, these arid reptiles are mostly nocturnal in their behavior and would therefore largely avoid dermal contact. However, snakes would be susceptible to dermal contact to perhaps a greater extent than any other animal potentially found in the roadside vegetation management zone of the state's highways.

3.3 2,4-D

3.3.1 *Mechanism of Action and Environmental Fate 2,4-D*

2,4-Dichlorophenoxyacetic acid (2,4-D) is a chlorinated phenoxy compound used as a systemic herbicide to control broadleaf weeds (*summarized in* EXTOXNET 1996a). 2,4-D is the first successful selective herbicide developed (1942, marketed by 1944) and is now the third most widely used herbicide in the United States and Canada, and the most widely used herbicide worldwide (Pesticides News 2003, Industry Task Force II 2002). The most commonly used form of 2,4-D is an acid; other common derivatives include esters, amines, and salts, which vary in solubility and volatility (*summarized in* EXTOXNET 1996a).

2,4-D controls plant growth by stimulating nucleic acid and protein synthesis and affects enzyme activity, respiration, and cell division (USDA 1995a). 2,4-D acts as a plant

growth regulator because the structure of 2,4-D is similar to the plant specific hormone indole acetic acid, and it can take the place of the hormone in processes listed above (Charles *et al.* 1996). It is distributed aerially in fields, in spot locations for more direct application, along basal bark positions and injected (tree root-collar). The plant absorbs the compound through the leaves, stems, and roots, and translocates it throughout the plant where it eventually accumulates in the growing tips (USDA 1995a).

It has a half-life between <1 day to 30 days in soil depending upon soil characteristics (temperature, moisture, microbes), and 10 to >50 days in surface water (Spectrum 2003, *summarized in* EXTOXNET 1996a, USDA 1995a). Microbes in the soil are the primary form of degradation when the compound binds to organic matter in the soil, however when organic content is low a porous soil with high water content will tend to leach out the compound from soils into ground water sources in approximately 22 weeks (*summarized in* EXTOXNET 1996a, USDA 1995a). The only reported amounts of 2,4-D in ground water sources are from spills or other point sources. Although the compound is mobile to highly mobile in sand, silt, loam, clay loam, and sandy loam, it is degraded so rapidly that it does not reach ground water sources before it is broken down (USDA 1995a). Products of biodegradation of 2,4-D include 2,4-dichlorophenol, 4-chlorophenol, and other harmless hydroxylic aromatics and polymeric acids (Spectrum 2003, USDA 1995a). Rate of evaporation depends on the formulation used, with acids, inorganic salt, amines and long chain esters having the least tendency to evaporate (USDA 1995a).

3.3.2 *Exposure Assessment 2,4-D*

3.3.2.1 Mammals

Mammalian wildlife can be exposed to 2,4-D through inhalation, ingestion, and direct skin contact. 2,4-D is readily absorbed in the lungs and through the skin. After ingestion, nearly all of a dose is excreted in the urine, and it has not been shown to accumulate to any significant level in mammals (*summarized in* EXTOXNET 1996a). The half-life in living organisms is estimated to be 10 to 20 hours after adsorption (*summarized in* EXTOXNET 1996a). Concentrations of 2,4-D have been found in the blood, liver, kidney, lungs, and spleen with lower concentrations in muscle and brain after administering concentrations of 1 mg/kg over six and eight days to rats; however the compound was undetected after 24 hours from the last administered dose (*summarized in* EXTOXNET 1996a).

Based on the exposure parameters provided in Table 3-1, the deer mouse and meadow vole would have the highest potential exposure to 2,4-D from WSDOT's herbicide application rates. The deer mouse could experience a dose of up to 46 mg/kg, and the meadow vole a dose of up to 35. mg/kg. Given the conservative factors brought into this assessment, these values are probably largely exaggerated but they provide an index for qualitative ecological risk assessment nonetheless.

3.3.2.2 Birds

Avian wildlife are primarily exposed to 2,4-D through ingestion, and inhalation. As demonstrated in Table 3-1, the American Robin would have the highest potential for exposure based on the exposure parameters depicted in Table 3-1 (96.59 mg/kg). However, given the species' mobile behavior, the estimated exposure dose of 96.59 mg/kg is highly unlikely. The estimated exposure to quail (8.41 mg/kg) is nearly an order of magnitude lower, and is more realistic given this species' behavior and smaller home range.

3.3.2.3 Adjuvant and Inert Ingredients

Some commercial formulations of 2,4-D include ethylene glycol (antifreeze) at up to 10 percent. Weedar$_{tm}$ the formulation used by WSDOT does not apparently contain ethylene glycol, but it does contain up to 53.2 percent inert materials. The exposure to these inert ingredients is not considered a significant risk.

3.3.3 *Risk Characterization 2,4-D*

The following discussion qualitatively summarizes potential risks to Washington State wildlife based on estimated exposures to the herbicide applications represented in Table 3-2. In addition to direct-action toxicities principally evaluated here, wildlife may be indirectly affected by the use of herbicide due to loss of vegetation that supplies habitat for various species. However, it must be recognized that herbicide use is a critical component within an integrated pest management protocol that WSDOT must exercise to control invasive, non-native plants that will colonize roadside habitat due to the lack of competition by native plants in these buffer zones. Thus, the potential loss of habitat must be considered in context with the preservation of native plant species that WSDOT is mandated to consider.

3.3.3.1 Mammals

As discussed in section 2.2, the acute oral toxicity of 2,4-D to mammals is rated as moderately toxic, based on the criteria outlined in Table 2-1. The LD_{50} concentrations for 2,4-D range from 375 to 666 mg/kg in rats, 370 mg/kg in mice, and 320 to 1,000 mg/kg in guinea pigs (*summarized in* EXTOXNET 1996a). The maximum doses estimated from an exclusive hypothetical grain diet to rats, mice, and meadow vole are 113, 13, and 17-fold lower than the doses found to elicit acute toxicity in the laboratory surrogates for these wildlife species, respectively (Table 3-2). Given the conservative factors included in the exposure assessment such as the assumption that animals will utilize the roadside vegetation management zone exclusively (and therefore their dose will not be diluted with other dietary sources), the wildlife risks from WSDOT's applications of 2,4-D must be considered low for rats, and moderate for mice and meadow voles.

3.3.3.2 Birds

The USDA (1995b) reports 2,4-D forms ranging between practically non-toxic to moderately toxic to birds based on toxicity criteria outlined in Table 2-1. 2,4-D is slightly toxic to wildfowl (i.e. mallards, pheasants, quail, and pigeons), with acute toxicity LD_{50} concentrations ranging from 272 (pheasants) to 1,000 mg/kg (mallards) (*summarized in* EXTOXNET 1996a). The 2,4-D butyl ester is practically non-toxic to birds on an acute and chronic basis (USDA 1995a). Maximum exposures were calculated for the American robin and marsh wren, however, these species' diets are more omnivorous than that of the quail, and thus the actual dosage received would be likely less than that predicted in Table 3-2. Based on the more conservative acute LD_{50} values represented in Table 3-2 (LD_{50}=472 mg/kg for pheasant and 2,000 mg/kg for mallard duck), the acute dietary exposures to quail, marsh wren and American robin from WSDOT's current application practices would be 56, 6, and 5-fold lower than the acute dietary LD_{50} for pheasants, and 238, 27, and 21-fold lower than the acute dietary LD_{50} for mallard ducks. Based on the LD_{50} for pheasants, estimated dietary exposure risk is considered low for quail, and high for wren and American robin. Based on the LD_{50} for mallard ducks, estimated dietary

exposure risk is considered low for quail and wren, and moderate for American robin.

3.3.3.3 Insects

2,4-D is relatively non-toxic to honeybees, with ester formulations as the least toxic. Moderate exposure concentrations of the compound resulted in impaired brood production (*summarized in* EXTOXNET 1996a). Risks to insects cannot be fully characterized with existing data.

3.3.4 *Uncertainties and Data Gaps*

The potential effects of 2,4-D on amphibians, reptiles and a variety of bird species has not been investigated thoroughly. A detailed exposure assessment is for these species would require further study.

3.4 Ammonium Salt of Fosamine

3.4.1 *Mechanism of Action and Environmental Fate of Fosamine*

Fosamine is an organophosphonate used as a selective, post-emergent herbicide to control woody and herbaceous plants (e.g. maple, birch, alder, blackberry, vine maple, ash, and oak) by preventing dormant tissue from becoming active (Tu *et al.* 2001f, *as summarized in* BPA 2000a). Fosamine is applied one to two months before autumn leaf-drop to inhibit budding the following spring (Swift *et al.* 2002, Tu *et al.* 2001f). Some non-deciduous species that do not have a budding period (e.g. pine and bindweed) may develop immediate reactions to Fosamine exhibited in brown leaves. The specific mechanism of action is not fully understood, however there is some evidence that it inhibits mitosis in susceptible plants (Tu *et al.* 2001f).

Fosamine is highly soluble in water and binds readily to some soils (high in organic content). Soil mobility is low due to binding with soil particles, with reported soil adsorption coefficients (Kd) ranging from 0.22 to 350 for low organic sandy barns and silt barns, respectively (Swift *et al.* 2002, Tu *et al.* 2001f). Fosamine is degraded mostly through microbial activity, with an average half-life of 8 days (range of one to two weeks in soils high in temperature, moisture and organic content, but can be as much as 6 weeks without these conditions) in soils; degradation by abiotic chemical reactions or photolysis is slow, however low pH (i.e. below 5) and high temperatures aid in the ability to breakdown the product (Swift *et al.* 2002, Tu *et al.* 2001f, *as summarized in* BPA 2000a). Persistence in water is high, however the compound will bind to suspended particles, sink, and degrade in aquatic sediments (Tu *et al.* 2001f).

Fosamine does not extensively leach into ground water sources. A study by Han (1979 as cited in Tu *et al.* 2001f), collected 93% of the residual radio-labeled carbon form Fosamine in the top 10 cm of the soil after one year and 165 cm of rainfall; in fine sands the leaching rate increased slightly and 62% of the radio-labeled carbon was found in the top 10 cm after six months and 40 cm of precipitation. This slow leaching process is due in part to Fosamine forming insoluble salts or complexes with soil minerals (Swift *et al.* 2002, Tu *et al.* 2001f).

Microbial metabolism degrades Fosamine to the metabolites cabamoylphosphonic acid (CPA), carboxylphosphonic acid, and carbon dioxide within several weeks (Tu *et al.*

2001f, *as summarized in* BPA 2000a). CPA is the only metabolite known to have toxic affects, and has a reported half-life of three to six weeks (Tu *et al.* 2001f).

3.4.2 *Exposure Assessment—Fosamine*

3.4.2.1 Mammals

Exposure for mammals involves ingestion of affected prey or direct contact of the ground after application to soils. Predicted maximum dosages to representative species potentially exposed by WSDOT application practices are depicted in Table 3-2, based on dietary exposure parameters outlined in Table 3-1. Based on consumption patterns, the deer mouse would be expected to have the highest body burden of Fosamine delivered via diet, with a projected exposure of 96.1 mg/kg. The omnivorous rat would be expected to attain a burden of approximately 11.21 mg/kg, and the herbivorous meadow vole a burden of approximately 73.3 mg/kg.

3.4.2.2 Birds

Exposure for birds involves ingestion of affected invertebrate or direct contact of the ground after application to soils. Predicted dosages to representative species potentially exposed by WSDOT application practices are depicted in Table 3-2. The estimated exposures to the robin and marsh wren are two orders of magnitude greater than the quail, with dietary exposures up to 203, 158.5 and 17.7 mg/kg for the robin, wren and quail, respectively. Given the large proportion of animal and insect components to diets of the robin and wren, the projected doses for these species are likely overestimated.

3.4.3 *Risk Characterization—Fosamine*

3.4.3.1 Mammals

Fosamine is considered practically non-toxic to mammals, based on criteria provided in Table 2-1. The most conservative acute dietary LD_{50} reviewed was approximately 5,000 mg/kg (Table 3-2). However acute dermal toxicity could increase the level to slightly toxic when combined with dietary exposure (Tu *et al.* 2001f). Other reports indicate the acute oral LD_{50} in rats at 24,400 mg/kg (Tu *et al.* 2001f, Swift *et al.* 2002, ACOE 2003). Dermal toxicity is slightly higher for mammals than oral toxicity and is listed as the second most severe level of acute toxicity (Category II).

Subchronic concentrations administered to rats resulted in weight loss and effects to the kidney and bladder. Fosamine was reported to be eliminated from the rats' bodies within 72 hours with an average of 79% of the herbicide was excreted unchanged, while 13% was excreted as a hydrolyzed metabolite (Tu *et al*. 2001f). A similar study in rats reported Fosamine distribution (a combination of Fosamine ammonium and CPA) as 86% in feces and 11% in urine after 48 hours (Swift *et al.* 2002).

Potential dietary exposures to rats, mice and meadow voles modeled for this assessment, based on WSDOT application rates, are 446, 52, and 68-fold lower than the acute dietary toxicity value of 5,000 mg/kg, depending on species' consumption patterns considered in Table 3-1. Based on this finding, the risks to mammalian wildlife from the current application practices are considered insignificant for rats, and low for mice and meadow voles.

3.4.3.2 Birds

Fosamine is practically non-toxic to birds, based on criteria in Table 2-1. An acute toxicity study, involving mallard ducks and bobwhite quail, administered doses of 0, 312.5, 625, 1,250, 2,500, and 5,000 mg/kg. The LD_{50} was reported as greater than 5,000 mg/kg in both species (Swift *et al.* 2002, *as summarized in* BPA 2000a).

A subacute toxicity study administered doses of 0, 625, 1,250, 2,500, 5,000 and 10,000 mg/kg orally to mallard ducks and bobwhite quail for eight days. The subacute oral LD_{50} for bobwhite quail and mallard ducks was reported as greater than 10,000 mg/kg (Swift *et al.* 2002, Tu *et al.* 2001f).

Based on the more conservative acute LD_{50} values represented in Table 3-2 (LD_{50}=5,000 mg/kg for both bobwhite quail and mallard duck), the acute dietary exposures to quail, marsh wren and American robin from WSDOT's current application practices would be 282, 31, and 25-fold lower than the acute dietary LD_{50}. Estimated dietary exposure risk is considered insignificant for quail, and low for wren and American robin.

3.4.3.3 Insects

Fosamine is listed as practically non-toxic to microorganisms and invertebrates (Swift *et al.* 2002, *as summarized in* BPA 2000a). The acute dermal LC_{50} for a Fosamine ammonium salt formulation (42%) was reported as 10,000 mg/L for bees. The herbicide fact sheet produced by *as summarized in* BPA (2000d) reported an acute contact toxicity to honey bees as >200 µg/bee. Based on these data and application practices of WSDOT, the risks to insects are considered insignificant.

3.4.3.4 Adjuvant and Inert Ingredient Hazards

Fosamine is typically applied with an agricultural oil (0.25%) to promote adsorption by target plant species (ACOE 2003). The Krenite formulation of Fosamine contains 41.5% Fosamine and 58.5% inert ingredients not classified as having toxicological effects by the U.S. EPA (*as summarized in* BPA 2000a). The inert ingredients are primarily surfactants of proprietary nature. The surfactants apparently increase the toxicity level of Fosamine but do not alter the reported risk category (practically non-toxic). The oral LD_{50} in male rats for Fosamine plus surfactant (41.1 to 42% active ingredient) was reported as 7,295 mg/kg compared to 24,400 mg/kg of the formulated product without surfactant (Swift *et al.* 2002).

3.4.4 *Uncertainties and Data Gaps*

Like virtually all of the herbicides marketed today, the body knowledge on the effects of the compounds on reptiles and amphibians is scant to non-existent.

3.5 Bromacil/Diuron (Krovar$_{tm}$)

Bromacil and Diuron are both broad-spectrum herbicides used for brush control in non-cropland areas (USDA 1995g, U.S. EPA 1996a).

3.5.1 *Mechanism of Action and Environmental Fate—Bromacil/Diuron*

Bromacil is introduced via ground broadcast application by WSDOT via mechanically controlled metering systems. The compound migrates into the root zone of a plant through irrigation or rainfall where they are taken up by the plant. Bromacil inhibits photosynthesis, thereby disrupting plant growth.

Hung and Mackay (1997) developed a study that modeled the distribution of ^{14}C-labeled Bromacil into soybean plants. They created a model that was based upon the uptake of a hydroponic solution of ^{14}C-labeled Bromacil introduced to the roots. The experiment that the model was based upon reported an accumulation of Bromacil mainly in the foliage due to its low vapor pressure (4.1x10-5 Pa) and its slow foliage-air exchange rate ($7.80x10^{-9}$ µmol/h). The transport process from stem to leaf occurred at a rate of 10x10-12 mol/h, with an overall accumulation in the leaf of approximately 7.7x10-12 mol/h. The slowest transport occurred in the phloem where the rate was approximately 0.5 to 1.0x10-12 mol/h.

Potential mobility of a herbicide within the soil depends both on the chemical properties of the herbicide compound as well as the chemical and physical properties of the soil (Futch and Singh 1999). Soil mobility defines how quickly that compound will infiltrate to plants outside the direct exposure area, to ground water supplies, and to surface water of surrounding rivers, lakes, or wetlands. The leaching properties of Bromacil and Diuron differ significantly from each other. In a study to identify the rates of leaching for Bromacil and Diuron, Futch and Sing (1999) applied herbicide at a rate of 7.167 kg/ha with an associated irrigation amount of 3.2, 6.4, 9.6 and 12.8 cm. The specific soil used in this study was Chandler fine sand common in Florida citrus farms. This particular soil, under 3.2 to 12.8 cm of irrigated water, exhibited mobility rates in soil of 8.36 to 16.10 cm for Diuron, and 34.72 to 106.68 cm for Bromacil. The same general pattern of mobility in soil was also confirmed by Gomez *et al.* (1996) in soils from citrus farming in Spain. Other studies have stressed the need to understand soil characteristics when applying Bromacil or Diuron (Li *et al.* 2001, Indelman *et al.* 1998, Miles and Pfeuffer 1997).

Bromacil is degraded in anaerobic soil by microbial activity and in water under alkaline conditions (U.S. EPA 1996a). If there is not enough organic material in soils, and permeability is high, the compound will be able to penetrate to groundwater supplies before they are degraded. Degradation is slower for Bromacil than Diuron; the half-life of Bromacil is an average of 275 days verses an average of 90 days (with a range of one month to one year) for Diuron (*as summarized in* BPA 2000f, 2000g, USDA 1996b).

3.5.2 *Exposure Assessment—Bromacil/Diuron*

3.5.2.1 Mammals

Mammalian wildlife can be exposed to Bromacil/Diuron through dermal, oral or inhalation routes, although the dietary route is considered predominant (USDA 1995g). Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Based on the exposure parameters provided in Table 3-1, and application rates considered in Table 3-2, the deer mouse would receive the highest dietary dose (76.82 mg/kg) and the rat would

receive the lowest (8.96 mg/kg).

3.5.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. The robin would have a projected exposure of 162.6 mg/kg verses the quail's exposure of 14.15 mg/kg.

3.5.2.3 Adjuvant and Inert Ingredient Exposure

Bromacil is used in commercial herbicides that contain a percentage of the chemical and an inert ingredient (Table 3-4; *as summarized in* BPA 2000f). Inert ingredient exposure is considered insignificant.

Table 3-4. Concentrations of Bromacil in commercial herbicides.

Commercial Herbicide	Chemical	Concentration (%)
Hyvar X (wettable powder)		
	Bromacil	80.0
	Inert	20.0
Hyvar X-L (water soluble liquid)		
	Bromacil	21.9
	Inert	78.1

3.5.3 *Risk Characterization—Bromacil/Diuron*

3.5.3.1 Mammals

Acute toxicity of Bromacil was noted as being low to moderate in dry vs. liquid formulations, respectively (USDA 1995g). The compound causes eye and throat irritation, but the risk is low for intact skin. The lowest threshold concentration of Bromacil that impacted dogs through oral administration was 100 mg/kg, which caused vomiting, watering of the mouth, muscular weakness, excitability, diarrhea, and dilation of the pupils (USDA 1995g).

The estimated exposure doses to the deer mouse and rat is 30 and 257-fold lower than the reported acute oral LD_{50} for rats. The estimated exposure for the meadow vole is 39-fold lower than the acute LD_{50}. Based on this qualitative modeling, the risk to mammalian receptors from the current WSDOT application practice is considered low to insignificant.

3.5.3.2 Birds

Projected exposure values for the robin and quail are 14 and 159-fold lower than the quail acute dietary LD_{50}. The risk to avian receptors is therefore considered to be moderate to low.

3.5.3.3 Insects

Bromacil is reported as practically non-toxic to microorganisms and invertebrates (Swift *et al.* 2002, *as summarized in* BPA 2000f). Acute dermal LC_{50} for a Bromacil formulation (42%) was reported as

10,000 mg/L for bees. These data indicate that insects are at low to insignificant risk.

3.5.3.4 Adjuvant and Inert Ingredient Hazards

The inert ingredients in the Bromacil/Diuron formulation are not considered to result in additive risk to the use of the compound.

3.5.4 *Uncertainties and Data Gaps*

Knowledge on toxicity and exposure parameters for amphibians and reptiles is lacking. Further understanding of the specific components in the inert ingredients is desirable.

3.6 Chlorsulfuron

3.6.1 *Mechanism of Action and Environmental Fate--Chlorsulfuron*

Chlorsulfuron is a systemic, selective pre- and post-emergent herbicide for most broadleaf weeds and some annual grass weeds that works by inhibiting acetolactate synthase (ALS), thereby inhibiting cell division in the root tips and shoots of sensitive plants (Purdue 2003, *as summarized in* BPA 2000b, USDA 1995b).

Chlorsulfuron is applied to soils through aerial or direct spraying distribution. Plants absorb the compound through the soils (USDA 1995b). The primary form of degradation for Chlorsulfuron is through non-microbial hydrolysis, but microbial degradation is also significant. The half-life of Chlorsulfuron in soil ranges from 28 to 42 days (*as summarized in* BPA 2000b, PMEP 1985). Higher temperatures, lower pH levels, higher oxygen (aerobic conditions), and higher moisture content of soils increases the rate of degradation.

Chlorsulfuron is highly soluble in water (31,800 mg/L at a pH of 7), however formulations of Chlorsulfuron (*e.g.*, Telar®) form suspensions in moving water (*as summarized in* BPA 2000b, USDA 1995b). Some formulations of Chlorsulfuron (e.g. Glean Weed Killer) is intended for use in soils with a pH of 7.5 or lower, due to the fact that less leaching occurs at pH levels below 6.0 (PMEP 1985). The reason for using the formulation in soils with a pH less than 7.5 is to minimize persistence. Chlorsulfuron is not known to have any major metabolites or degradation products that are of significant toxicological concern.

3.6.2 *Exposure Assessment--Chlorsulfuron*

3.6.2.1 Mammals

Exposure for mammals involves ingestion of affected plant matter or prey, direct contact with the ground after application to soils, or inhalation during application. Estimated exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 0.2, 1.71, and 1.31 mg/kg, respectively (Table 3-2).

3.6.2.2 Birds

Exposure for birds involves ingestion of affected invertebrate or direct contact of the ground after application to soils. Estimated exposures based on WSDOT application rates for the quail, marsh wren and robin are 0.32, 2.83 and 3.63 mg/kg, respectively (Table 3-2).

3.6.2.3 Adjuvant and Inert Ingredient Exposure

The most common commercial herbicide formulation of Chlorsulfuron is Telar® DF that is a combination of the Chlorsulfuron compound (75%) and inert materials (25%) (*as summarized in* BPA 2000b, USDA 1995b). Specific information on the surfactants used in Telar was not available to gauge potential effects.

3.6.3 *Risk Characterization--Chlorsulfuron*

There is no potential found for bioaccumulation of Chlorsulfuron in any species (*as summarized in* BPA 2000b). Chlorsulfuron is rapidly absorbed through oral exposure, metabolized, and excreted primarily through urine (58% to 72%) and secondarily feces (20-35%) (U.S. EPA 2002a). Chlorsulfuron is reported as being not hazardous due to inhalation (Dupont 2001).

3.6.3.1 Mammals

Chlorsulfuron is reported as practically non-toxic to mammals, based on Table 2-1 criteria. The acute oral toxicity LD_{50} of Chlorsulfuron >5,000 mg/kg for male rats and >6,000 for female rats has been reported by a variety of agencies (*as summarized in* BPA 2000b, USDA 1995b, PMEP 1985). Chlorsulfuron has been tested for dermal toxicity as a primary skin irritant and eye irritant. The LD_{50} for acute dermal toxicity for rabbits is >2,000 mg/kg for formulations of 75% active ingredient for an exposure of 21 hours, and as high as 3,400 mg/kg for technical Chlorsulfuron (USDA 1995b, PMEP 1985). Tests have shown that Chlorsulfuron is not an irritant to skin and a moderate irritant to eyes (*as summarized in* BPA 2000b, USDA 1995b, PMEP 1985). Inhalation is the least sensitive pathway for toxicity, with an LC_{50} of >5.9 mg/L was reported in rats.

No chronic toxicity levels were reported on a chronic basis in mammals based on carcinogenicity, developmental/reproductive, or mutagenicity at exposure concentrations ranging from 100 to 5,000 ppm for rats and mice over 3 months to 2 years (*as summarized in* BPA 2000b, USDA 1995b).

Based on the more conservative acute LD_{50} values represented in Table 3-2 (LD_{50}=2,341 mg/kg), the acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 11,705, 1,366 and 1,789-fold lower than the acute dietary LD_{50}. This estimated exposure is considered insignificant for all of these species.

3.6.3.2 Birds

Chlorsulfuron is reported as practically non-toxic to birds, based on Table 2-1, with an acute oral toxicity LD_{50} of >5,000 mg/kg for both mallard ducks and bobwhite quail (*as summarized in* BPA 2000b, PMEP 1985). The subacute dietary toxicity LD_{50} is >5,620 mg/kg for bobwhite quail and >5,000 mg/kg for mallard ducks (*as summarized in* BPA 2000b). Based on the LD_{50} estimate of 5,000 mg/kg as provided in Table 3-2, the estimated exposures to the quail, marsh wren and American robin, would be 15,822, 1,766, and 1,377-fold lower than the acute dietary LD_{50} for the

quail and mallard duck. This estimated exposure is considered insignificant for all of these species.

3.6.3.3 Insects

Chlorsulfuron is reported as practically non-toxic to microorganisms (*as summarized in* BPA 2000b). Acute contact LC50 for Chlorsulfuron was reported as >25 µg/bee. Current application rates should yield similar non-toxic results.

3.6.3.4 Adjuvant and Inert Ingredient Hazards

Chlorsulfuron formulations are also used with surfactants so that they are better absorbed through the soil. No information was acquired to enable a characterization of the risks from the surfactants. The effects on wildlife receptors are not expected to be significant, but indirect effects from the surfactants may need to be considered.

3.6.4 *Uncertainties and Data Gaps*

Amphibian and reptile toxicity information is lacking as is sufficient exposure data to ascertain drinking water effects in mammals (U.S. EPA 2002a).

3.7 Clopyralid

3.7.1 *Mechanism of Action and Environmental Fate--Clopyralid*

Clopyralid is auxin-mimic type herbicide (similar to Triclopyr, Picloram and 2,4-D) used to selectively control annual and perennial broadleaf weeds (e.g. sunflower, legume, nightshade, knotweed and violet), but has little effect on grasses and other monocots (Tu *et al.* 2001a, Cox 1998). A paper by Tu *et al.* (2001a) tested the reaction of different concentrations of Clopyralid in plants. Clopyralid works by mimicking the plant growth hormone auxin (indole acetic acid), which causes uncontrolled and disorganized plant growth. The uncontrolled growth acidifies the cell wall and leads to cell elongation and ultimately death. Low concentrations of Clopyralid produce disorganized growth and uncontrolled cell division from stimulated RNA, DNA, and protein synthesis that leads to vascular tissue destruction. High concentrations of Clopyralid inhibit cell division and growth.

The active ingredient of Clopyralid is used at a rate of approximately 0.1 percent of that of atrazine, the most widely used herbicide in the U.S. (Cox 1998). Even with this low use pattern, USGS detected concentrations of Clopyralid within two of twenty river basins sampled within the Central Columbia Plateau (Washington and Idaho) and the Trinity River Basin.

Clopyralid is primarily distributed aerially as well as ground broadcast, and spot and localized application (*as summarized in* BPA 2000c). The compound is absorbed through the leaves, so application timing is generally limited to emergent plants. Clopyralid does not bind well to soils (*i.e.* adsorption coefficient of 6) and potential mobility within soil is high. The average half-life of Clopyralid in soils in laboratory studies ranges from 14 to 56 days, and one to two months in the field with a maximum value of 1 year (Tu *et al.* 2001a, Terra 1999). Clopyralid is resistant to degradation by sunlight, hydrolysis or other chemical actions. It is decomposed almost entirely through slow microbial metabolism. Clopyralid disassociates after adsorption by a plant

to an anion form that is highly soluble in water but not in soil. This disassociation makes the mobility in the soil high (measured at a depth of 180 cm 20 days after application) however, even though persistence within soils is high and mobility is potentially high, no evidence has been found of excessive concentrations in ground water sources (Tu *et al.* 2001a).

Critical concentrations of Clopyralid have recently been measured in compost material of both immature compost (477 to 1,550 ppb) and mature or finished compost (nondetect to 182 ppb) within eastern Washington (BioCycle 2002). The western Washington region also found concentrations of greater than 20 ppb in 12 of 14 compost samples. Critical concentrations in this scenario are in relation to sensitive crops (e.g. tomatoes, potatoes, sunflower, beans, peas) that are affected at concentrations of 10 ppb. This contamination originated primarily from yard and grass clippings and represents how persistent Clopyralid is in treated vegetation. However, Washington State University (BioCycle 2002) estimated that the average concentration of Clopyralid in grass clippings mowed ten weeks after application was less than one percent of the initial concentration (approximately 150 ppb).

Clopyralid is degraded primarily through microbial activity into primarily hydrogen chloride, with traces of chlorinated pyridine (Terra 1999). Under fire conditions it degrades into trace elements of nitrogen oxides. All three products are considered hazardous.

3.7.2 *Exposure Assessment--Clopyralid*

3.7.2.1 Mammals

Exposure for mammals involves ingestion of affected prey or inoculated vegetation, direct contact during ground application (especially to the eyes), and inhalation of vapors via burning or airborne particles after aerial application. There is little or no potential for bioaccumulation of Clopyralid in exposed terrestrial organisms (*as summarized in* BPA 2000c).

Based on the assumed exposure parameters detailed in Table 3-1, rats, deer mice and meadow vole could be exposed to 0.525, 4.50, and 3.43 mg/kg dietary Clopyralid in settings where WSDOT applies Clopyralid at their maximum projected application rate.

3.7.2.2 Birds

Exposure for birds involves ingestion of affected invertebrate, direct contact during ground application (especially to the eyes), and inhalation of vapors via burning or airborne particles after aerial application. Based on the assumed exposure parameters detailed in Table 3-1, quail, marsh wren and American robin could be exposed to 0.829, 7.43, or 9.5 mg/kg dietary Clopyralid in settings where WSDOT applies Clopyralid at their maximum projected application rate (Table 3-2)

3.7.3 *Risk Characterization--Clopyralid*

3.7.3.1 Mammals

Clopyralid is practically non-toxic to mammals, based on criteria specified in Table 2-1., with an acute oral LD_{50} concentration in rats of 4,300 to 5,000 mg/kg (Tu *et al.*2001a, *as summarized in* BPA 2000c). After exposure, rats have been observed to rapidly eliminate almost all of the compound in urine after 24 hours, with some residual amounts retained in the liver and kidney (Tu *et al.*

2001a, Terra 1999). Acute dermal toxicity is considered low, with an LD_{50} of >5,000 mg/kg for rabbit for the compound Transline (*as summarized in* BPA 2000c). Acute inhalation exposure is more dangerous at excessive concentrations, with an LD_{50} of >3.0 mg/L in rats. Incidental contact of the eye with Clopyralid may result in corneal injury and lead to blindness.

Chronic oral exposures have affected liver and kidney organs in mammal (Cox 1998). A 2-year exposure of Clopyralid to rats resulted in hyperplasia of the stomach lining and an increase in cells within the stomach lining that produced enlargement of the lining. Dogs fed Clopyralid for one year resulted in an enlarged liver and decreased number of red blood cells in all but the lowest concentration administered (Cox 1998). Chronic toxicity has also affected the stomach, liver, blood, and body weight of test species (Cox 1998). Inhalation is a potential hazard under these conditions. However, there is little or no potential for bioaccumulation within any organism (*as summarized in* BPA 2000c).

Birth defects have been observed with severe toxic concentrations to the pregnant female, but not observed in lower doses that are still 3-4 times higher than natural potential exposure during standard release of the compound (Terra 1999). Chronic dermal and inhalation exposure to skin or eyes is considered more toxic than any acute exposure.

Based on the WSDOT application rates of Clopyralid (Table 3-2) the dietary doses to rats, mice and meadow vole are 8,190, 955, 1,254-fold lower than the LD_{50} of 4,300 mg/kg. These doses can be characterized as presenting insignificant mammalian wildlife risk. Bioaccumulation through the food chain does not appear to be a concern, however, the persistence of the compound in the environment could result in a low-level exposure over a chronic period under some exposure scenarios. The meadow vole would appear more susceptible to this type of exposure due to its forage base and small home range. Because small mammals would be less mobile than avian wildlife, the additional toxicity of the adjuvants added to typical Clopyralid mixtures elevates the potential risk to mammalian wildlife to low.

3.7.3.2 Birds

The compound Clopyralid itself is considered slightly toxic to birds on an acute basis, with an acute oral LD_{50} concentration of 2,000 mg/kg for both mallard ducks and bobwhite quail (*as summarized in* BPA 2000c, Terra 1999). Subacute dietary toxicity LD_{50} concentrations are reported at 5,000 mg/kg for both mallard ducks and bobwhite quail (Tu *et al*. 2001a, *as summarized in* BPA 2000, Terra 1999). The commercial formulation Transline is considered practically non-toxic to birds on an acute oral basis. Chronic concentrations of Clopyralid did not cause significant effects to bobwhite quail embryos (Tu *et al.* 2001a).

Based on the application rates, the estimated dietary doses to quail, marsh wren and the American robin are 2,412, 269, and 210-fold less than the acute dietary LD_{50} for the quail and duck. Provided significant additional dermal and/or inhalation exposure is prevented through avoidance, as would be expected with avian receptors, the risks from the use of Clopyralid appear insignificant. In conjunction with the adjuvants commonly added to the mixture, however, the risk must be elevated to low.

3.7.3.3 Insects

Clopyralid is listed as practically non-toxic to microorganisms (*as summarized in* BPA 2000c, Terra 1999). Acute contact toxicity LC50 for a Clopyralid is >100 µg/bee (*as summarized in* BPA 2000c). Clopyralid is practically non-toxic to soil invertebrates and microbes (Tu *et al.*

2001a). Risks to insects and other invertebrates would appear to be insignificant from current WSDOT application practices.

3.7.3.4 Adjuvant and Inert Ingredient Hazards

Inert ingredients used in Clopyralid formulations can cause exacerbated symptoms due to toxic exposures beyond what the active chemical produces. For instance, the combination of Clopyralid with the inert ingredient isopropyl alcohol may make the commercial formulation more volatile, rendering exposure via inhalation more likely. Isopropyl alcohol has also been shown to affect the kidney and/or developed tumors in rats (Terra 1999). Isopropyl alcohol exposure has been found to yield birth defects in rat fetuses at extremely high concentrations.

Four more inert ingredients (cyclohexanone, triisopropanolamine, triethylamine, and polyethoxylated tallow amines) cause reactions in organisms. Cyclohexanone (found in Curtail M) produces tearing and burning of the eyes, vomiting, diarrhea, and dizziness (Cox 1998). Triisopropanolamine (found in Curtail) produces eye and skin irritation, irritation of the respiratory tract if inhaled, and cause spasms, inflammation, and fluid accumulation in the lungs (Cox 1998). Triethylamine (found in Confront, Confront Weedstick, Confront F) is a severe eye irritant (including a "blue haze") and can cause chemical pneumonia after inhalation. Polyethoxylated tallow amines (found in Confront-fertilizer combinations) cause eye burns, and nausea (Cox 1998).

3.7.4 *Uncertainties and Data Gaps*

Basic toxicity information of the effects of Clopyralid on amphibians and reptiles is lacking. Synergistic effects of Clopyralid with its inert ingredients should be evaluated further.

3.8 Clopyralid/2,4-D

3.8.1 *Mechanism of Action and Environmental Fate Clopyralid/2,4-D*

Clopyralid/2,4-D is a specific mixture of Clopyralid and 2,4-D (39.0% 2,4-D, triisopropanolamine salt and 5.1% Clopyralid, monoethanolamine salt) with other inert materials (55.9%) in the commercial herbicide Curtail®. Curtail is a selective herbicide for broadleaf weeds. Clopyralid 2,4-D controls growth in the same way as formulations of Clopyralid and 2,4-D (see previous discussion). The compound is adsorbed through the leaves of the target plant species.

The primary degradation pathway for 2,4-D is through microbial activity in the soil. More than 40% of the compound was biodegraded under aerobic static laboratory conditions, however Clopyralid biodegradation under the same conditions was below detectable limits (<2.5%) (Dupont 2001). Migration through soils is high, which creates a potential to leach into ground water sources when applied to soils with high permeability and areas with shallow aquifers (Dow AgroSciences 2001).

3.8.2 *Exposure Assessment--Clopyralid/2,4-D*

3.8.2.1 Mammals

Exposure for mammals involves ingestion of affected prey or inoculated vegetation, direct contact during ground application (especially to the eyes), and inhalation of vapors via burning or airborne particles after aerial application. Dietary exposure alone from WSDOT application practices was estimated to yield doses of 1.66, 14.25 and 10.87 mg/kg for the rat, deer mouse, and meadow vole, respectively.

3.8.2.2 Birds

Exposure for birds involves ingestion of affected invertebrate, direct contact during ground application (especially to the eyes), and inhalation of vapors via burning or airborne particles after aerial application. Dietary exposure alone from WSDOT application practices was estimated to yield doses of 2.63, 23.54 and 30.15 mg/kg for the quail, marsh wren, and robin, respectively.

3.8.3 *Risk Characterization--Clopyralid/2,4-D*

Symptoms of acute toxic dermal exposure to Clopyralid/2,4-D include severe eye irritation with corneal injury and skin irritation (Dupont 2001). Exposure through inhalation is not reported as hazardous in all test species. Overall acute oral toxicity is considered low, however chronic oral toxicity symptoms of exposure include gastrointestinal tract, kidney, liver, and muscular system, coupled with nausea and/or vomiting, abdominal cramps and/or diarrhea with excessive exposure (Dupont 2001).

3.8.3.1 Mammals

Clopyralid/2,4-D is considered practically non-toxic to mammals, based on criteria in Table 2-1 (*as summarized in* BPA 2000c). As previously indicated, the acute oral LD_{50} for male rats is 3,730 mg/kg and for female rats is 2,830 mg/kg (Dupont 2001). Reactions to dermal exposure, especially near the eyes, are more severe in mammals than oral exposure to Clopyralid/2,4-D. The acute dermal LD_{50} for rabbits is >4,000 mg/kg (Dupont 2001).

Chronic test on mammals have shown a variety of physiological responses. Clopyralid/2,4-D has caused birth defects such as decreased weight of the fetus in rabbits at low and high doses, and skeletal abnormalities in rabbits at all doses tested (Cox 1998). Birth defects are reported as being linked to zinc deficiency due to chelation (Dupont 2001).

Based on WSDOT application rates (Table 3-2), the estimated doses of active ingredient potentially received by rats, deer mouse and meadow vole, are 1,705, 199, and 260-fold lower than the acute oral LD_{50} for rats. These values discount the potential additive toxicity of some of the adjuvants. For this reason it is concluded that the use of Clopyralid/2,4-D poses a low (as opposed to insignificant) risk to mammalian wildlife using habitat found along roadway corridors.

3.8.3.2 Birds

Clopyralid/2,4-D, similar to both Clopyralid and 2,4-D chemicals separately, is considered practically non-toxic to birds when applied according to manufacturer specifications (*as summarized in* BPA 2000c). However, we did not identify specific numeric criteria in our reviews of the Clopyralid/2,4-D mixture, so the avian toxicity indicated is based on an estimate of Clopyralid toxicity alone (LD_{50}=2,000 mg/kg). Based on this criteria, the estimated doses of active ingredient to quail, marsh

wren and robin are 760, 85, and 66 fold lower the acute dietary LD_{50}. Given the potential for additive toxicity from inert ingredients in this formulation, the herbicide is conservatively considered to present low to moderate risks to avian wildlife based on current WSDOT practices.

3.8.3.3 Insects

Clopyralid/2,4-D, similar to both Clopyralid and 2,4-D chemicals separately, is practically non-toxic to microorganisms and invertebrates when applied according to manufacturer specifications (*as summarized in* BPA 2000c). The WSDOT generally applies these herbicides at a rate lower than that recommended; therefore, we characterize the risks to insects from the use of this herbicide formulation as insignificant.

3.8.3.4 Adjuvant and Inert Ingredient Hazards and Synergism

The inert ingredients of triisopropanolamine and cyclohexanone found in Curtail causes eye irritation and possibly skin irritation. Inhalation of the chemical during aerial distribution can irritate the respiratory tract, and may be fatal as a result of spasms, inflammation, and fluid accumulation in the lungs (Cox 1998). This condition would be considered highly unlikely to occur, but cannot be discounted for small mammals with limited ability to remove them from an application area.

3.8.4 *Uncertainties and Data Gaps*

The same uncertainties and data gaps apply for the Clopyralid/2,4-D mixture as they did for Clopyralid alone.

3.9 Dicamba, Dicamba/2-4D & Dicamba/MCPA

3.9.1 *Exposure Assessment--Dicamba*

3.9.1.1 Mammals

Exposure for mammals can occur through ingestion of exposed plant matter or prey, direct contact with the ground after application to soils, or inhalation during application. Estimated exposures of Dicamba based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 1.40, 12.00, and 9.16 mg/kg, respectively (Table 3-2). Estimated exposures of Dicamba/2,4-D based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 3.05, 26.14, and 19.95 mg/kg, respectively (Table 3-2). Estimated exposures of Dicamba/MCPA based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 2.63, 22.50, and 17.17 mg/kg, respectively (Table 3-2).

3.9.1.2 Birds

Exposure for birds can occur through ingestion of exposed invertebrates and plant materials or direct contact of the ground after application to soils and/or inhalation. Estimated exposures of Dicamba based on WSDOT application rates for the quail, marsh wren and robin are 2.21, 19.82 and 25.40 mg/kg, respectively (Table 3-2). Estimated exposures of Dicamba/2,4-D based on WSDOT application rates for the quail, marsh wren and robin are 4,82, 43.19 and 55.32 mg/kg,

respectively (Table 3-2). Estimated exposures of Dicamba MCPA based on WSDOT application rates for the quail, marsh wren and robin are 4.15, 37.17 and 47.62 mg/kg, respectively (Table 3-2).

3.9.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Dicamba, Dicamba/2,4-D or Dicamba/MCPA was not addressed in this revision.

3.9.2 *Risk Characterization—Dicamba*

There is no potential for bioaccumulation of Dicamba in any species (*as summarized in* BPA 2000d). Dicamba is rapidly absorbed into the bloodstream from the gastrointestinal tract (*summarized in* EXTOXNET 1996b). When administered orally to rats, the compound is excreted primarily through urine (90 to 99%) in an unmetabolized form and secondarily through feces (1 to 4%) (*summarized in* EXTOXNET 1996b). When administered to mice, rats, rabbits, and dogs, Dicamba is excreted through urine (85%) in an unmetabolized form within 48 hours (*summarized in* EXTOXNET 1996b). The mixtures of Dicamba/2,4-D or Dicamba MCPA, react in similar fashion to Dicamba formulations. When oral doses were discontinued, the compound was observed to be quickly eliminated from the storage organs.

3.9.2.1 Mammals

Dicamba is reported as slightly toxic to mammals by ingestion, inhalation, and dermal exposure, based on Table 2-1 criteria. The acute oral toxicity LD_{50} of Dicamba is >757 to 1707 mg/kg for rats (*summarized in* EXTOXNET 1996b). The LD_{50} for acute dermal toxicity for rabbits is >2000 mg/kg (*summarized in* EXTOXNET 1996b). Inhalation is probably the most sensitive exposure pathway, with an LC50 for Dicamba in rats of >200 mg/L.

No chronic toxicity levels were reported in mammals based on carcinogenicity, developmental/reproductive, or mutagenicity at exposure concentrations ranging from 0.5 to 25 mg/kg/day for rats exposed over 2 years (*summarized in* EXTOXNET 1996b).

Based on the more conservative acute LD_{50} values for Dicamba, Dicamba/2,4-D and Dicamba MCPA represented in Table 3-2 (LD_{50}=2,740, 1,150, and 2,034 mg/kg, respectively), the acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 1,957, 228 and 299-fold lower than the acute dietary LD_{50} for Dicamba. Because of the higher risk seen in mice, the use of Dicamba poses a low risk to mammalian wildlife using habitat found along roadway corridors.

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 377, 44 and 58-fold lower than the acute dietary LD_{50} for Dicamba/2,4-D. Because of the higher risk seen in mice and meadow voles, the use of Dicamba/2,4-D poses an insignificant to low risk to mammalian wildlife using habitat found along roadway corridors.

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 775, 90 and 119-fold lower than the acute dietary LD_{50} for Dicamba/MCPA.

Because of the higher risk seen in mice and meadow voles, the use of Dicamba/MCPA poses an insignificant to low risk to mammalian wildlife using habitat found along roadway corridors.

3.9.2.2 Birds

Dicamba is reported as practically non-toxic to birds, based on criteria in Table 2.1, with an acute oral toxicity LD_{50} of 2,009 mg/kg for mallard ducks (*summarized in* EXTOXNET 1996b). The 8-day dietary toxicity LD_{50} is >10,000 mg/kg for bobwhite quail (*summarized in* EXTOXNET 1996b). The most conservative acute LD_{50} values for Dicamba (LD_{50} for pheasants = 637 mg/kg), Dicamba/2,4-D and Dicamba/MCPA for bobwhite quail (LD_{50}=2,000, and 377 mg/kg, respectively), and for Dicamba and Dicamba MCPA for mallard duck (LD_{50}=2009 for both) are represented in Table 3-2.

The acute dietary exposures of Dicamba to quail, marsh wren and American robin from WSDOT's current application practices would be 288, 32 and 25-fold lower than the acute dietary LD_{50} for pheasants. The acute dietary exposures of Dicamba to quail, marsh wren and American robin from WSDOT's current application practices would be 909, 101 and 79-fold lower than the acute dietary LD_{50} for mallard ducks. Because of the higher risk seen in marsh wren and American robin using the more sensitive LD_{50} for pheasants, the use of Dicamba poses a low risk to avian species using habitat found along roadway corridors.

The acute dietary exposures of Dicamba/2,4-D to quail, marsh wren and American robin from WSDOT's current application practices would be 415, 46 and 36-fold lower than the acute dietary LD_{50} for mallard ducks. Because of the higher risk seen in marsh wren and American robin, the use of Dicamba/2,4-D poses a low risk to avian species using habitat found along roadway corridors.

The acute dietary exposures of Dicamba/MCPA to quail, marsh wren and American robin from WSDOT's current application practices would be 91, 10 and 8-fold lower than the acute dietary LD_{50} for bobwhite quail. The acute dietary exposures of Dicamba/MCPA to quail, marsh wren and American robin from WSDOT's current application practices would be 485, 54 and 42-fold lower than the acute dietary LD_{50} for mallard ducks. Because of the higher risk seen in marsh wren and American robin using the more sensitive LD_{50} for bobwhite quail, the use of Dicamba/MCPA poses a high risk to avian species using habitat found along roadway corridors.

3.9.2.3 Insects

Dicamba is reported as practically non-toxic to microorganisms and insects (*summarized in* EXTOXNET 1996b). Current application rates should yield similar non-toxic results.

3.10 Dichlobenil

3.10.1 *Mechanism of Action and Environmental Fate—Dichlobenil*

Dichlobenil is a commonly used urban pesticide to control weeds in gardens, ornamental trees, lawns, and invasive aquatic species (e.g. cattail, purple loosestrife). Dichlobenil is a broad-spectrum herbicide that inhibits meristem growth in roots and shoots, seed germination, and cellulose synthesis (ACOE 2003, *as summarized in* BPA 2000h, and USDA 1995d). The compound is absorbed within the water column of soils by the roots and travels to the leaves through translocation. Dichlobenil does not appear to be persistent in plants that are inoculated through soil with the

herbicide; however, it is known to leach into ground water sources when present in coarse-textured soils low in organic matter and drain into surface water sources through runoff. Dichlobenil may dissipate quickly from surface waters through volatilization (U.S. EPA 1998a). Dichlobenil is reported as being extremely mobile and persistent in anaerobic soils where microbial activity does not break down the compound (U.S. EPA 1998a). Under natural conditions, the compound has an average half-life of 60 days, with a range between 1.5 and 12 months in soils (*summarized in* EXTOXNET 1996c, *summarized in* BPA 2000h).

3.10.2 *Exposure Assessment—Dichlobenil*

3.10.2.1 Mammals

The most common exposure pathway is through water; its solubility is 21.2 mg/L in water at a pH of 7.0 and temperature of 25C (*as summarized in* BPA 2000h). Exposure through inhalation is also possible due to the moderately volatility of Dichlobenil at temperatures of 20 C and atmospheric pressure of 0.88 Pa (*as summarized in* BPA 2000h, U.S. EPA 1998a). Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 5.60, 48.00, and 36.62 mg/kg, respectively (Table 3-2).

3.10.2.2 Birds

The most common exposure pathway of avian species is through diet, inhalation and direct contact with aerial-dispersed amounts of Dichlobenil. Estimated exposures based on WSDOT application rates for the quail, marsh wren and robin are 8.84, 79.29 and 101.58 mg/kg, respectively (Table 3-2).

3.10.2.3 Adjuvant and Inert Ingredient Exposure and Synergism

There are different formulations of Dichlobenil that are used for urban, highway, and agricultural purposes. It is used in a granular form (1-10 % active ingredient [a.i.]), liquid-ready to use (0.5 % a.i.), soluble concentrate (0.5 % a.i.), and wettable powders (0.55 % a.i.) (U.S. EPA 1998a). Examples of a commercial formulation of Dichlobenil and other ingredients are expressed in Table 3-5.

Table 3-5. Contents of Eight Dichlobenil Formulations (*as summarized in* BPA 2000h, USDA 1995d)

Commercial Herbicide	Dichlobenil Content (%)	Inert Content (%)
Barrier 50W	50	50
Casoron 2G	2	98
Casoron 4G	4	96
Dyclomec 4G	4	96
Norosac 4G	4	96
Casoron 10G	10	90
Norosac 10G	10	90
Casoron 50W	50	50 (including crystalline silica)

Dichlobenil has two main metabolites: 2,6-dichlorobenzamide (BAM) and 2,6-dichlorobenzoic acid (*as summarized in* BPA 2000h, U.S. EPA 1998a, USDA 1995d). BAM is the only degradate that is slightly toxic through oral pathways, and is listed as Toxicity Category III (U.S. EPA 1998a). The metabolite is generated at a general rate of 13.1% at 50 weeks through aerobic soil metabolism,

although this rate depends upon soil type.

3.10.3 *Risk Characterization—Dichlobenil*

In general, the compound is considered to be of low acute toxicity, and is labeled as Toxicity Category III (the second lowest of four) (U.S. EPA 1998a).

3.10.3.1 Mammals

On an acute basis, Dichlobenil is listed as slightly toxic to mammals according to LD_{50} values represented in Table 2-1 (acute oral LD_{50} for rat is 4,250 mg/kg, acute dermal toxicity LD_{50} for rabbit is >2,000 mg/kg). The more conservative LD_{50} values presented in Table 3-2 for acute dietary exposure to Dichlobenil is 3,160 mg/kg for rats.

Based on WSDOT application rates (Table 3-2) and the more conservative LD_{50} values (Table 3-2), the estimated doses of active ingredient potentially received by rats, deer mouse and meadow vole, are 564, 66, and 86-fold lower than the acute oral LD_{50} for rats. The use of Dichlobenil poses an insignificant risk to rats, and a low risk to deer mice and meadow vole that may use habitat found along roadway corridors.

3.10.3.2 Birds

The overall toxicity for avian species is reported as practically non-toxic to slightly toxic (*as summarized in* BPA 2000h). Acute oral toxicity LD_{50} values range from 683 mg/kg for bobwhite quail to >2,000 mg/kg for a mallard duck (*as summarized in* BPA 2000h). The subacute dietary toxicity LD_{50} for avian species is approximately 5,200 mg/kg for both the bobwhite quail and mallard duck (*as summarized in* BPA 2000h). The most conservative values for LD_{50} based on acute dietary exposure to bobwhite quail and mallard ducks is 1,500 mg/kg for both species (Table 3-2).

Dichlobenil formulations can modify the toxicity of the herbicide. For example, granular formulations of Dichlobenil with greater than 10% granules (10 G) may create a potential acute risk at the 6 lb. a.i./A or greater application rate to unincorporated soils (U.S. EPA 1998a). Some endangered species of birds may be affected by 4G to 10G granules at all unincorporated rates at the 10-lb. ai./A or higher incorporated rates (U.S. EPA 1998a). Chronic risks to avian species have not been assessed for Dichlobenil.

The acute dietary exposures of Dichlobenil to quail, marsh wren and American robin from WSDOT's current application practices would be 170, 19 and 15-fold lower than the acute dietary LD_{50} for bobwhite quail and mallard ducks, according to the more conservative LD_{50} values for acute dietary exposure (Table 3-2). The use of Dichlobenil poses an low risk to quail, and a moderate risk to marsh wren and American robin that may use habitat found along roadway corridors.

3.10.3.3 Insects

Overall toxicity of Dichlobenil to insects (e.g. honeybee) is practically non-toxic. Acute toxicity LD_{50} of honeybees from direct contact is >120 µg/bee (*as summarized in* BPA 2000h).

3.11 Diuron

3.11.1 *Mechanism of Action and Environmental Fate—Diuron*

Diuron is used as broad-spectrum herbicides for brush control in non-cropland areas (BPA 2000g). Diuron is introduced via ground broadcast application and via aerial broadcast system (2000g). The compound migrates into the root zone of a plant through irrigation or rainfall where is taken up by the plant. Diuron, similar to Bromacil, inhibits photosynthesis, thereby disrupting plant growth.

Potential mobility of a herbicide within the soil depends both on the chemical properties of the herbicide compound as well as the chemical and physical properties of the soil (Futch and Singh 1999). Soil mobility defines how quickly that compound will infiltrate to plants outside the direct exposure area, to ground water supplies, and to surface water of surrounding rivers, lakes, or wetlands. Leaching mobility of Diuron is significantly slower than the more viscous associated herbicide Bromacil. In a study to identify the rates of leaching, Futch and Sing (1999) applied different herbicides at a rate of 7.167 kg/ha with an associated irrigation amount of 3.2, 6.4, 9.6 and 12.8 cm. The specific soil used in this study was Chandler fine sand common in Florida citrus farms. This particular soil, under 3.2 to 12.8 cm of irrigated water, exhibited mobility rates in soil of 8.36 to 16.10 cm for Diuron vs. 34.72 to 106.68 cm for Bromacil. The same general pattern of mobility in soil was also confirmed by Gomez *et al.* (1996) in soils from citrus farming in Spain. These and other studies have stressed the need to understand soil characteristics when applying Bromacil or Diuron (Li *et al.* 2001, Indelman *et al.* 1998, Miles and Pfeuffer 1997).

Diuron is degraded in anaerobic soil by microbial activity and in water under alkaline conditions (U.S. EPA 1996a). If there is not enough organic material in soils, and permeability is high, the compounds will be able to penetrate to groundwater supplies before they are degraded. Diuron degrades quickly in soils; the half-life of Diuron is an average of 90 days (with a range of one month to one year) (*as summarized in* BPA 2000g, USDA 1996b).

3.11.2 *Exposure Assessment—Diuron*

3.11.2.1 Mammals

Three exposure routes are possible for Diuron: inhalation, dermal, and oral (USDA 1996b). Wildlife species are most predominantly exposed to the contaminants through bioaccumulation in plants and the transfer of the contaminants through natural food chains. Estimated dietary exposures to the rat, deer mouse and meadow vole are 6.73, 57.64, and 43.98 mg/kg, respectively, based on WSDOT application rates (Table 3-2).

3.11.2.2 Birds

Exposure for birds can occur through ingestion, inhalation after aerial application, or via direct contact of the ground after application to soils. Estimated dietary exposures for the quail, marsh wren and robin are 10.62, 95.22 and 121.99 mg/kg, respectively, based on WSDOT application rates (Table 3-2).

3.11.2.3 Adjuvant and Inert Ingredient Exposure and Synergism

Diuron is used in commercial herbicides that contain a percentage of the chemical and proprietary inert ingredients (Table 3-6; as summarized in BPA 2000g). Potential synergism with inert ingredients is unknown.

Table 3-6. Concentrations of Diuron in Commercial Herbicides.

Commercial Herbicide	Chemical	Concentration (%)
Diuron 4L		
	Diuron	40.0
	Inert	60.0
Diuron 80 DF		
	Diuron	80.0
	Inert	20.0
Karmex DF		
	Diuron	80.0
	Inert	20.0

3.11.3 *Risk Characterization—Diuron*

3.11.3.1 Mammals

Acute toxicity of Diuron is considered low (USDA 1996b). The rat oral LD_{50} ranges from 1,017 to 3,750 mg/kg and is associated with nervous system depression at the highest concentrations (USDA 1996b).

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 506, 59, and 77-fold lower than the acute dietary LD_{50} for Diuron, as represented in Table 3-2 (LD_{50}=3,160 mg/kg). This estimated exposure is considered an insignificant risk to rats, and a low risk to deer mice and meadow voles.

3.11.3.2 Birds

Diuron is reported as being slightly toxic to birds (USDA 1996b). For example, the bobwhite quail dietary LC50 is 1,730 ppm, whereas for Japanese quail, ring-necked pheasant, and mallard ducks it is greater than 5,000 ppm (USDA 1996b). The acute dietary LD_{50} values reported for bobwhite quail and mallard duck are 1,730 and 5,000 mg/kg, respectively (Table 3-2).

The acute dietary exposures of Diuron to quail, marsh wren and American robin from WSDOT's current application practices would be 163, 18, and 14-fold lower than the acute dietary LD_{50} for bobwhite quail. The estimated exposure is considered a low risk to quail, and a moderate risk to wren and American robin based on the LD_{50} value.

The acute dietary exposures of Diuron to quail, marsh wren and American robin from WSDOT's current application practices would be 471, 53, 41-fold lower than the acute dietary LD_{50} for mallard, and compared to acute dietary LD_{50} values. The estimated exposure is considered an insignificant risk to quail, and a low risk to wren and American robin based on the LD_{50} value.

3.11.3.3 Insects

Diuron is reported as essentially non-toxic to microorganisms and invertebrates (see WSDOT 1993).

3.12 Glyphosate

3.12.1 *Exposure Assessment—Glyphosate*

3.12.1.1 Mammals

Exposure for mammals involves ingestion of affected plant matter or prey, direct contact with the ground after application to soils, or inhalation during application. Estimated exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 2.80, 24.00, and 18.31 mg/kg, respectively (Table 3-2).

3.12.1.2 Birds

Exposure for birds occurs through diet, inhalation, or via direct contact of the ground after application. Estimated exposures based on WSDOT application rates for the quail, marsh wren and robin are 4.42, 39.65 and 50.79 mg/kg, respectively (Table 3-2).

3.12.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Glyphosate was not addressed in this revision.

3.12.2 *Risk Characterization—Glyphosate*

3.12.2.1 Mammals

Based on the more conservative acute LD_{50} values for Glyphosate represented in Table 3-2 (LD_{50}=5,600 mg/kg), the acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 2,000, 233, and 306-fold lower than the acute dietary LD_{50} for Glyphosate (Table 3-2). The estimated exposure is considered an insignificant risk to rats, and a low risk to deer mice and meadow voles.

3.12.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail and mallard duck are 4,640 and 2,000 mg/kg, respectively (Table 3-2).

The acute dietary exposures of Glyphosate to quail, marsh wren and American robin from WSDOT's current application practices would be 105, 117, and 91-fold lower than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values. The estimated exposure is considered low risk to all species.

The acute dietary exposures of Glyphosate to quail, marsh wren and

American robin from WSDOT's current application practices would be 452, 50, and 39-fold lower than the acute dietary LD_{50} for mallard, and compared to acute dietary LD_{50} values. The estimated exposure is considered an insignificant risk to quail, and a low risk to wren and American robin based on the LD_{50} value for mallard ducks.

3.12.2.3 Insects

Glyphosate is reported as essentially non-toxic to microorganisms (see WSDOT 1993). Acute contact LC50 for Chlorsulfuron was reported as >100 µg/bee. Current application rates should yield similar non-toxic results.

3.13 Metsulfuron Methyl

3.13.1 *Exposure Assessment—Metsulfuron methyl*

3.13.1.1 Mammals

Exposure for mammals can occur by ingestion of affected plant matter or prey, direct contact after application, or inhalation during application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 0.10, 0.86, and 0.65 mg/kg, respectively (Table 3-2).

3.13.1.2 Birds

Exposure for birds can occur via diet, inhalation, or via direct contact of the ground after application. Estimated dietary exposures based on WSDOT application rates for the quail, marsh wren and robin are 0.16, 1.42 and 1.81 mg/kg, respectively (Table 3-2).

3.13.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Metsulfuron methyl was not addressed in this revision.

3.13.2 *Risk Characterization—Metsulfuron methyl*

There has been no potential found for the bioaccumulation of Metsulfuron methyl (*as summarized in* BPA 2000i). Metsulfuron methyl is rapidly absorbed through oral exposure, metabolized, and excreted within 9 to 16 hours for low doses and 23 to 29 hours for high doses (*summarized in* EXTOXNET 1996e).

3.13.2.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 50,000, 5,834, and 7,645-fold lower than the acute dietary LD_{50} for Metsulfuron methyl (>5,000 mg/kg). This estimated exposure is considered insignificant risk for all of these species.

3.13.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail and mallard is 5,000 mg/kg for both species (Table 3-2). The acute dietary exposures of Metsulfuron methyl to quail, marsh wren and American robin from WSDOT's current application practices would be 31,646, 3,531, 2,756-fold lower than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values. This estimated exposure is considered an insignificant risk for all avian species evaluated.

3.13.2.3 Insects

Metsulfuron methyl is reported as practically non-toxic to microorganisms (*as summarized in* BPA 2000i). Acute contact LC_{50} for Chlorsulfuron was reported as >25 µg/bee. Current application rates should yield similar non-toxic results.

3.14 Oryzalin

3.14.1 *Exposure Assessment—Oryzalin*

3.14.1.1 Mammals

Exposure for mammals can occur via diet, direct contact with the ground or plants after application to soils, or inhalation during application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 5.60, 48.00, and 36.62 mg/kg, respectively (Table 3-2).

3.14.1.2 Birds

Exposure for birds can occur through diet, inhalation, or direct contact with the ground or plants after application. Estimated exposures based on WSDOT application rates for the quail, marsh wren and robin are 8.84, 79.29 and 101.58 mg/kg, respectively (Table 3-2).

3.14.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Oryzalin was not addressed in this revision.

3.14.2 *Risk Characterization—Oryzalin*

There is small potential found for bioaccumulation of Oryzalin (*as summarized in* BPA 2000e). Oryzalin is moderately well absorbed from the gastrointestinal tract, metabolized, and excreted primarily through urine (40%) and secondarily feces (40%) in rats within 3 days (*summarized in* EXTOXNET 1996f). Results for rabbits, steer, and Rhesus monkeys were observed to be similar to that of rats.

3.14.2.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from WSDOT's

current application practices would be 893, 104, 137-fold lower than the acute dietary LD_{50} for Oryzalin. The estimated dietary exposure is considered an insignificant risk to rats, and a low risk to mice and meadow voles.

3.14.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail is 507 mg/kg, respectively (Table 3-2). The acute dietary exposures of Oryzalin to quail, marsh wren and American robin from WSDOT's current application practices would be 57, 6, and 5-fold lower than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values. The estimated exposure is considered a low risk to quail, and a high risk to wren and American robin based on the LD_{50} value for mallard ducks.

3.14.2.3 Insects

Oryzalin is reported as practically non-toxic to microorganisms and insects when used according to label directions (*summarized in* EXTOXNET 1996f). Acute contact LC_{50} for Chlorsulfuron was reported as 11 µg/bee. Current application rates should yield similar non-toxic results.

3.15 Picloram

3.15.1 *Exposure Assessment—Picloram*

3.15.1.1 Mammals

Exposure for mammals occurs via diet, direct contact after application, or inhalation during application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 1.40, 12.00, and 9.16 mg/kg, respectively (Table 3-2).

3.15.1.2 Birds

Exposure for birds occurs via diet, inhalation, or direct contact after application. Estimated dietary exposures based on WSDOT application rates for the quail, marsh wren and robin are 2.21, 19.82 and 29.40 mg/kg, respectively (Table 3-2).

3.15.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Picloram was not addressed in this revision.

3.15.2 *Risk Characterization—Picloram*

There has been no potential found for the bioaccumulation of Picloram (*as summarized in* BPA 2000j). Picloram is rapidly absorbed through the gastrointestinal tract and excreted primarily through urine in an unchanged form (*summarized in* EXTOXNET 1996g). Residues were reduced by half within 1 day of exposure.

3.15.2.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 3,571, 417, and 546-fold lower than the acute dietary LD_{50} for Picloram (LD_{50} 5,000 mg/kg). This estimated exposure is considered insignificant risk for all of these species.

3.15.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail and mallard duck are 5,000 and 2,510 mg/kg, respectively (Table 3-2).

The acute dietary exposures of Picloram to quail, marsh wren and American robin from WSDOT's current application practices would be 2,261, 252, and 197-fold lower than the acute dietary LD_{50} for bobwhite quail. The estimated dietary exposure is considered an insignificant risk to quail and wren, and a low risk to American robin.

The acute dietary exposures of Picloram to quail, marsh wren and American robin from WSDOT's current application practices would be 1,135, 127, and 99-fold lower than the acute dietary LD_{50} for mallard ducks. The estimated dietary exposure is considered an insignificant risk to quail, and a low risk to wren and American robin.

3.15.2.3 Insects

Picloram is reported as relatively non-toxic to microorganisms and insects (*as summarized in* BPA 2000j). Acute contact LC_{50} for Picloram was reported as >14 µg/bee. Current application rates should yield similar non-toxic results.

3.16 Sulfometuron Methyl

3.16.1 *Exposure Assessment—Sulfometuron methyl*

3.16.1.1 Mammals

Exposure for mammals can occur via ingestion, inhalation, or direct contact after application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 0.33, 2.79, and 2.13 mg/kg, respectively (Table 3-2).

3.16.1.2 Birds

Exposure for birds can occur via ingestion, inhalation or direct contact after application. Estimated dietary exposures based on WSDOT application rates for the quail, marsh wren and robin are 0.51, 4.60 and 5.90 mg/kg, respectively (Table 3-2).

3.16.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Sulfometuron was not addressed in this revision.

3.16.2 *Risk Characterization— Sulfometuron methyl*

Sulfometuron methyl is readily adsorbed through the gastrointestinal tract and rapidly broken down and excreted within 48 hours, depending on the dose. Sulfometuron does not bioaccumulate in organisms (*summarized in* EXTOXNET 1996h).

3.16.2.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 15,385, 1,795, and 2,535-fold lower than the acute dietary LD_{50} for Sulfometuron methyl (5,000 mg/kg). This estimated exposure is considered insignificant for all of these species.

3.16.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail and mallard duck are 5,620 and 5,000 mg/kg, respectively (Table 3-2).

The acute dietary exposures of Sulfometuron methyl to quail, marsh wren and American robin from WSDOT's current application practices would be 10,955, 1,221, and 953-fold lower than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values. The estimated dietary exposure is considered an insignificant risk.

The acute dietary exposures of Sulfometuron methyl to quail, marsh wren and American robin from WSDOT's current application practices would be 9,747, 1,086, and 848-fold lower than the acute dietary LD_{50} for mallard ducks, and compared to acute dietary LD_{50} values. These estimated exposures are considered insignificant risk.

3.16.2.3 Insects

Sulfometuron methyl is considered to be moderately toxic to bees based on a contact LD_{50} of >12.5 µg/bee (USFS 1989). No other toxicological analyses were preformed for insects.

3.17 Triclopyr

3.17.1 *Exposure Assessment*

3.17.1.1 Mammals

Exposure for mammals can occur via ingestion, direct contact after application, or inhalation during application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 5.60, 48.00, and 36.62 mg/kg, respectively (Table 3-2).

3.17.1.2 Birds

Exposure for birds can occur via ingestion, inhalation, or direct contact after application. Estimated dietary exposures based on WSDOT application rates for the quail, marsh wren and robin are 8.84, 79.29 and 101.58 mg/kg, respectively (Table 3-2).

3.17.1.3 Adjuvant and Inert Ingredient Exposure

Further investigation of the work presented in the original EIS regarding how adjuvant and inert ingredients affect the toxicity of Triclopyr was not addressed in this revision.

3.17.2 *Risk Characterization*

There has been no potential found for the bioaccumulation of Triclopyr (*as summarized in* BPA 2000k). Triclopyr is rapidly absorbed through the gastrointestinal tract and excreted primarily through urine in an unchanged form, and secondarily through feces as the adsorption capacity of the intestine is exceeded (*summarized in* EXTOXNET 1996i). The compound was excreted within 14 hours for dogs and <24 hours for monkeys.

3.17.2.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 113, 13, and 17-fold lower than the acute dietary LD_{50} for Triclopyr (630 mg/kg). The estimated dietary exposure is considered a low risk to rats, and a moderate risk to mice and meadow voles.

3.17.2.2 Birds

Acute dietary LD_{50} values reported for bobwhite quail and mallard duck are 2,935 and 1,698 mg/kg, respectively (Table 3-2). The acute dietary exposures of Triclopyr to quail, marsh wren and American robin from WSDOT's current application practices would be 332, 37, and 29-fold lower than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values. The estimated dietary exposure is considered an insignificant risk to quail, and a low risk to wren and American robin.

The acute dietary exposures of Triclopyr to quail, marsh wren and American robin from WSDOT's current application practices would be 192, 21, and 17-fold lower than the acute dietary LD_{50} for mallard, and compared to acute dietary LD_{50} values. The estimated dietary exposure is considered a low risk to quail, and a moderate risk to wren and American robin.

3.17.2.3 Insects

Triclopyr is reported as practically non-toxic to microorganisms (*summarized in* EXTOXNET 1996i). Current application rates should yield similar non-toxic results.

New Herbicides Evaluated in 2005

3.18 Bromoxynil

3.18.1 *Mechanism of Action and Environmental Fate*

Bromoxynil is a nitrile herbicide that acts by inhibiting photosynthesis in target species of plant. Its primary use is for control of post-emergent broadleaf weeds (EXTOXNET 1996j).

Bromoxynil was found to be mobile in sand, sandy loam and and loam soils but is nonpersistent. It is broken down in the environment by photolytic degradation, abiotic hydrolysis

and microbial-mediated metabolism under both aerobic and anaerobic conditions. Studies of Bromoxynil octanoate indicate that the hydrolysis of this herbicide is base-catalyzed. Respective half lives of 34.1, 11.5, and 1.7 days were reported for Bromoxynil octanoate at pH of 5, 7 and 9. Under conditions of soil photolysis bromoxynil octanoate had a reported half life of 2.6 days, which was reportedly comparable to aqueous photolysis. In two field experiments, half-lives were 14 days at a site in California and 1 day at a site in North Carolina. Bromoxynil octanoate was reported to have a K_d of 7.0 ml/g (K_{oc} of 1,003) in soils with 1.2% organic matter. However, U.S. EPA notes that additional information has requested that mobility under various conditions be reported (U.S. EPA 1998b).

3.18.2 *Exposure Assessment Bromoxynil*

3.18.2.1 Mammals

Mammalian wildlife can be exposed to Bromoxynil through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 0.7, 6.0, and 4.6 mg/kg, respectively (Table 3-2).

3.18.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on WSDOT application rates for the quail, marsh wren and robin are 1.1, 10 and 13 mg/kg, respectively (Table 3-2).

3.18.3 *Risk Characterization Bromoxynil*

3.18.3.1 Mammals

The estimated acute dietary exposures to rats, mice and meadow vole based on maximum label use would be 340, 40 and to 52-fold lower, respectively, than the acute dietary LD_{50} for Bromoxynil (Table 3-7). The estimated dietary exposure is considered an insignificant risk to rats and a low risk to mice and meadow voles (Table 3-3).

3.18.3.2 Birds

The estimated acute dietary exposures of Bromoxynil to quail, marsh wren and American robin based on maximum label application rates would be 134, 15, and 12-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered a low risk to quail and a moderate risk to wren and robin (Table 3-3).

3.19 Diflufenzopyr

3.19.1 *Mechanism of Action and Environmental Fate Diflufenzopyr*

Diflufenzopyr provides control of postemergent noxious broadleaf weeds by inhibiting the plant's ability to spread auxins, which normally play a key role in plant development and cell growth. Diflufenzopyr is absorbed through a plant's leaves, roots and shoots and is translocated to the growing parts of the plant (BASF 2003). The stability of Diflufenzopyr varies in the environment. In aerobic soil, the half-life ranged from 8-10 days; in an aerobic aquatic environment, the half life ranged from 5-26 days. In an anaerobic aquatic environment, the half-life was 20 days. Both the parent compound and its metabolites are considered very mobile in soil (U.S. EPA 1999).

Diflufenzopyr can be applied by ground broadcast or by spot spray (BASF 2003). Diflufenzopyr can enhance the effect of Dicamba when the two herbicides are applied concurrently (U.S. EPA 1999).

3.19.2 *Exposure Assessment Diflufenzopyr*

3.19.2.1 Mammals

Mammalian wildlife can be exposed to Diflufenzopyr through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 0.49, 4.2, and 3.2 mg/kg, respectively (Table 3-2).

3.19.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 0.77, 7.0 and 8.9 mg/kg, respectively (Table 3-2).

3.19.3 *Risk Characterization Diflufenzopyr*

3.19.3.1 Mammals

The estimated acute dietary exposures to rats, mice and meadow vole based on maximum label use would be 6,900, 810 and 1,100-fold lower, respectively, than the acute dietary LD_{50} for Diflufenzopyr (Table 3-7). The estimated dietary exposure is considered an insignificant risk to each of these species (Table 3-3).

3.19.3.2 Birds

The estimated acute dietary exposures of Diflufenzopyr to quail, marsh wren and American robin based on maximum label application rates would be 2,900, 320 and 250-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered an insignificant risk to quail and wren and a low risk to the robin (Table 3-3).

3.20 Flumioxazin

3.20.1 *Mechanism of Action and Environmental Fate Flumioxazin*

Flumioxazin is a selective control herbicide used to control preemergent and postemergent grasses and broadleaf weeds. Flumioxazin interferes with heme and chlorophyll biosynthesis, resulting in a buildup of photo-toxic porphoryns. Flumioxazin is relatively unstable in the environment but its metabolites APF and THPA are persistent and have the potential to leach into groundwater (U.S. EPA 2001).

Application techniques include broadcast, band and tank mix (Valent 2003).

3.20.2 *Exposure Assessment Flumioxazin*

3.20.2.1 Mammals

Mammalian wildlife can be exposed to Flumioxazin through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on WSDOT label application rates are 0.35, 3.0, and 2.3 mg/kg for rat, deer mouse and meadow vole, respectively (Table 3-2).

3.20.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for quail, marsh wren and robin are 0.55, 5.0 and 6.4 mg/kg, respectively (Table 3-2).

3.20.3 *Risk Characterization Flumioxazin*

3.20.3.1 Mammals

The estimated acute dietary exposures to rats, mice and meadow vole from WSDOT's current application practices would be 14,000, 1,700 and 2,200-fold lower than the acute oral LD_{50} for rats (Table 3-7). The estimated dietary exposure is considered insignificant for each of these species (Table 3-3).

3.20.3.2 Birds

The acute dietary exposures of Flumioxazin to quail, marsh wren and American robin from WSDOT's current application practices would be 4,100, 450 and 350-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered insignificant for each of these species (Table 3-3).

3.21 Fluroxypyr

3.21.1 *Mechanism of Action and Environmental Fate Fluroxypyr/Vista*

Fluroxypyr is a selective herbicide used to control annual and perennial broadleaf weeds and woody brush post emergence. Application methods include broadcast and spot treatment. Fluroxopyr binds to auxin receptor sites causing inhibition of plant growth and cellular processes. It also interferes with the processes of enzyme production and nitrogen metabolism in target plants (U.S. EPA 1998c).

U.S. EPA's evaluation of laboratory studies suggests that Fluroxypyr is mobile to very mobile in soil. Field studies, however, suggest that potential hydrolysis and microbial degradation act to limit its leaching potential and persistence. Fluroxypyr is resistant to photolysis in water with a half-life of 197 to 429 days at pH 5. In soil the half-life due to photolysis is 119 days. It is also stable to hydrolysis with an estimated half-life of 454 days at pH 7. In soil, the half-life from bacterial metabolism under aerobic conditions was 23 days. Metabolism under aerobic and anaerobic aquatic conditions resulted in a half-lives of 14 and 8 days, respectively (U.S. EPA 1998c).

3.21.2 *Exposure Assessment Fluroxypyr*

3.21.2.1 Mammals

Mammalian wildlife can be exposed to Fluroxypyr through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 0.35, 3.0, and 2.3 mg/kg, respectively (Table 3-2).

3.21.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 0.55, 5.0 and 6.4 mg/kg, respectively (Table 3-2).

3.21.3 *Risk Characterization Fluroxypyr*

3.21.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from maximum label application rates would be 6,900, 800 and 1,100-fold lower, respectively, than the acute dietary LD_{50} for Fluroxypyr (Table 3-7). The estimated dietary exposure is considered insignificant for each of these species (Table 3-3).

3.21.3.2 Birds

The acute dietary exposures of Fluroxypyr to quail, marsh wren and American robin from WSDOT's current application practices would be 3,600, 400 and 320-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered insignificant for quail and low for wren and robin (Table 3-3).

3.22 Imazapyr

3.22.1 *Mechanism of Action and Environmental Fate Imazapyr*

Imazapyr acts by interfering with cell growth, protein synthesis and DNA synthesis. After it is absorbed through either the leaves or the roots, Imazapyr moves quickly through the plant and accumulates in the meristem region. Imazapyr is used to control broadleaf weeds, vines and brush as well as annual and perennial grasses (USDA 1995h).

Imazapyr can be applied either preemergence or postemergence by several different methods including aerial application, hand held sprayers, boom equipment and tree injection. Imazapyr adsorbs strongly to soils and can remain active in soils for up to 2 years. Imazapyr has a low potential for leaching into groundwater. In surface water, Imazapyr breaks down quickly with a reported half-life of about four days (USDA 1995h).

3.22.2 *Exposure Assessment Imazapyr*

3.22.2.1 Mammals

Mammalian wildlife can be exposed to Imazaypyr through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 1.4, 12 and 9.2 mg/kg, respectively (Table 3-2).

3.22.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 2.2, 20 and 25 mg/kg, respectively (Table 3-2).

3.22.3 *Risk Characterization Imazapyr*

3.22.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from maximum label application rates would be 3,600, 420 and 550-fold lower, respectively, than the acute dietary LD_{50} for Imazaypyr (Table 3-7). The estimated dietary exposure is considered insignificant for each of these species (Table 3-3).

3.22.3.2 Birds

The acute dietary exposures of Imazaypyr to quail, marsh wren and American robin from WSDOT's current application practices would be 970, 110, and 85-fold lower than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered insignificant for quail and low for wren and robin (Table 3-3).

3.23 Isoxaben

3.23.1 *Mechanism of Action and Environmental Fate Isoxaben*

Isoxaben is a preemergence herbicide used to control certain broadleaf weeds. Isoxaben is somewhat persistent in the environment. Under aerobic soil conditions, Isoxaben has a reported half life of 4.3-10.6 months. Under photolysis conditions in soil, the half-life was 248 days. Breakdown occurs more rapidly in water under photolysis conditions, with a reported half-life of 6 days (Dow AgroSciences 2003)

3.23.2 *Exposure Assessment Isoxaben*

3.23.2.1 Mammals

Mammalian wildlife can be exposed to Isoxaben through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 1.4, 12 and 9.2 mg/kg, respectively (Table 3-2).

3.23.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 2.2, 20 and 25 mg/kg, respectively (Table 3-2).

3.23.3 *Risk Characterization Isoxaben*

3.23.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from maximum label application rates would be 7,100, 830 and 1,100-fold lower, respectively, than the acute dietary LD_{50} for Isoxaben (Table 3-7). The estimated dietary exposure is considered insignificant for each of these species (Table 3-3).

3.23.3.2 Birds

The acute dietary exposures of Isoxaben to quail, marsh wren and American robin from WSDOT's current application practices would be 900, 100, and 79-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered insignificant for quail and low for wren and robin (Table 3-3).

3.24 Norflurazon

3.24.1 *Mechanism of Action and Environmental Fate Norflurazon*

Norflurazon is a selective herbicide used to control germinating annual grasses and broadleaf weeds. It can be applied either by aerial application, chemigation or soil treatment. Norflurazon is considered to be a persistent compound. Photodegradation in either water or soil is the main way that Norflurazon is broken down. Photodegradation in soil and water results in a half life of 2-3 days and 12-15 days, respectively. By comparison it is resistant to hydrolysis. Under aerobic soil conditions, a half-life of 130 days was reported. In an aerobic aquatic environment, Norflurazon reportedly broke down to desmethyl Norflurazon with a half-life of 6-8 months. A similar half-life of 8 months was reported under anaerobic conditions. The main break down product of Norflurazon is desmethyl Norflurazon, which is also persistent under both aerobic and anaerobic conditions (U.S. EPA 1996b).

Norflurazon is mobile in soil and may leach into groundwater. Contamination of surface water is a concern for drift applications (U.S. EPA 1996b).

3.24.2 *Exposure Assessment Norflurazon*

3.24.2.1 Mammals

Mammalian wildlife can be exposed to Norflurazon through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 5.5, 47 and 36 mg/kg, respectively (Table 3-2).

3.24.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 8.7, 78 and 100 mg/kg, respectively (Table 3-2).

3.24.3 *Risk Characterization Norflurazon*

3.24.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole based on maximum label application rates would be 1,700, 200 and 260-fold lower, respectively, than the acute dietary LD_{50} for Norflurazon (Table 3-7). The estimated dietary exposure is considered insignificant for rats and meadow vole and low for mice (Table 3-3).

3.24.3.2 Birds

The acute dietary exposures of Norflurazon to quail, marsh wren and American robin based on

maximum label application practices would be 120, 13 and 10-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated dietary exposure is considered low for quail, moderate for wren, and high for robin (Table 3-3).

3.25 Oxadiazon

3.25.1 *Mechanism of Action and Environmental Fate Oxadiazon*

Oxadiazon is a selective herbicide used to provide preemergent and early postemergent annual grasses and broadleaf weeds (Cornell University 2001). Oxadiazon works by interfering with the chlorophyll production pathway (U.S. EPA 2003a).

Granular formualations of Oxadiazon can be applied using mechanical spreaders (*i.e.*, belly grinder, push type spreader) or tractor-drawn spreaders. Application methods used to apply other formulations include: rights-of-way sprayer, handgun sprayer, backpack sprayer, low pressure handwand, high pressure handwand, and lawn handgun and groundboom (U.S. EPA 2003a).

Under typical terrestrial environment conditions, Oxadiazon would be stable and persistent according to an U.S. EPA review of environmental fate studies. Aqueous photolysis is a more effective mechanism of breakdown compared with soil photolysis or hydrolysis. Microbial degradation of parent compound occurs only to a limited extent. Due to its low mobility and affinity for soil, Oxadiazon is likely to be available for transport by surface runoff (U.S. EPA 2003a).

3.25.2 *Exposure Assessment Oxadiazon*

3.25.2.1 Mammals

Exposure for mammals can occur via diet, direct contact with the ground or plants after application to soils, or inhalation during application. Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 2.8, 24 and 18 mg/kg respectively (Table 3-2).

3.25.2.2 Birds

Exposure for birds can occur through diet, inhalation, or direct contact with the ground or plants after application. Estimated exposures based on maximum label application rates for the quail, marsh wren and robin are 4.4, 40 and 51 mg/kg, respectively (Table 3-2).

3.25.3 *Risk Characterization Oxadiazon*

3.25.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from maximum label application rates would be 1,800, 210 and 270-fold lower, respectively, than the acute dietary LD_{50} for Oxadiazon (Table 3-7). The estimated dietary exposure is considered insignificant for rats and meadow vole and low for

mice (Table 3-3).

3.25.3.2 Birds

The acute dietary exposures of Oxadiazon to quail, marsh wren and American robin from maximum label application rates would be 1,100, 130 and 98-fold lower, respectively, than the acute dietary LD_{50} value selected to represent avian species (Table 3-7). This risk is considered insignificant for quail and low for marsh wren and robin (Table 3-3).

3.26 Pendimethalin

3.26.1 *Mechanism of Action and Environmental Fate Pendimethalin*

Pendimethalin is selective herbicide used to control annual grasses and certain broadleaf weeds at both preemergent and early post emergent stages (EXTOXNET 1996k). The mechanism of action is microtubule disruption and application methods include broadcast, chemigation, direct spray and soil incorporation (U.S. EPA 1997a).

Pendimethalin is a persistent herbicide that readily binds to soil. Once bound to soil, Pendimethalin becomes highly immobile. For the purpose of exposure assessment, U.S. EPA selected a representative half-life of 172 days for Pendimethalin exposed to aerobic microbial soil metabolism (the reported range of values was 42-1,322 days). Half-lives due to photodegradation in water were 16.5 days at pH 5, 7, and 9, respectively, and 21 days at pH 7 (U.S. EPA 1997a). A half-life of 40 days was reported in a field study (EXTOXNET 1996k).

Concentrations of Pendimethalin would normally be limited in surface waters due to the high affinity for soil and sediment sorption; however, spray drift or runoff from rainfall events could both contribute to elevated concentrations of Pendimethalin in surface waters. Pendimethalin breaks down into many minor degradates, which generally consistent of an intact benzene ring coupled with alkyl groups. Studies have shown Pendimethalin to bioaccumulate at high levels in sunfish fish with bioaccumulation factors of 1,400 for edible tissue and 5,100 for whole fish (U.S. EPA 1997a).

3.26.2 *Exposure Assessment Pendimethalin*

3.26.2.1 Mammals

Exposure for mammals can occur via diet, direct contact with the ground or plants after application to soils, or inhalation during application. Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 4.2, 36, and 27 mg/kg, respectively (Table 3-2). Although Pendimethalin is persistent in soil it is not readily taken up by plants. When it is absorbed by plant roots, translocation of Pendimethalin is limited and the chemical tends to break down by oxidation (EXTOXNET 1996k). These factors would tend to limit the potential for dietary exposure among herbivorous mammals.

3.26.2.2 Birds

Exposure for birds can occur through diet, inhalation, or direct contact with the

ground or plants after application. Estimated exposures based on maximum label application rates for the quail, marsh wren and robin are 6.6, 60 and 76 mg/kg, respectively (Table 3-2).

3.26.3 *Risk Characterization Pendimethalin*

3.26.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from the maximum label application rate would be 250, 29, and 38-fold lower, respectively, than the acute dietary LD_{50} for Pendimethalin (Table 3-7). The estimated risk from dietary exposure is considered low for each of these species (Table 3-3).

3.26.3.2 Birds

The acute dietary exposures of Pendimethalin to quail, marsh wren and American robin from the maximum label application rate would be 210, 24 and 19-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The estimated risk is low for quail and moderate for wren and robin (Table 3-3).

3.27 Pyraflufen

3.27.1 *Mechanism of Action and Environmental Fate Pyraflufen*

Pyraflufen is a non-selective herbicide used to control broadleaf weeds. Broadcast spray techniques are the primary application method (Nichino America 2004).

3.27.2 *Exposure Assessment Pyraflufen*

3.27.2.1 Mammals

Mammalian wildlife can be exposed to Pyraflufen through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 0.01, 0.05 and 0.037 mg/kg, respectively (Table 3-2).

3.27.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 0.009, 0.08, and 0.1 mg/kg, respectively (Table 3-2).

3.27.3 *Risk Characterization Pyraflufen*

3.27.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from the maximum label application rate would be 890,000, 100,000, and 140,000-fold lower, respectively, than the acute dietary LD_{50} for Pyraflufen (Table 3-7). The estimated risk from exposure to Pyraflufen is considered insignificant for each of these species (Table 3-3).

3.27.3.2 Birds

The acute dietary exposures of Pyraflufen to quail, marsh wren and American robin from the maximum label application rate would be 230,000, 25,000 and 20,000-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values (Table 3-7). The estimated risk from Pyraflufen exposure is considered insignificant for each of these species (Table 3-3).

3.28 Sulfentrazone

3.28.1 *Mechanism of Action and Environmental Fate Sulfentrazone*

Sulfentrazone acts by inhibiting protoporphyrinogen oxidase (PPO). Following soil application, Sulfentrazone is primarily taken up by plant roots. Contact with leaves causes dessication and necrosis of plant tissue. Treated plants become necrotic and die following exposure to light (U.S. EPA 1997b).

U.S. EPA characterizes Sulfentrazone as highly mobile and persistent with a strong potential to leach into groundwater and migrate off site. Under aerobic soil conditions, Sulfentrazone was found to have a half-life of 1.5 years; under anaerobic conditions, the half-life was 9 years. Sulfentrazone is resistant to hydrolysis but readily susceptible to photolysis (U.S. EPA 1997b).

3.28.2 *Exposure Assessment Sulfentrazone*

3.28.2.1 Mammals

Mammalian wildlife can be exposed to Sulfentrazone through dermal, oral or inhalation routes. Wildlife species are predominantly exposed to the herbicide through plant forage and the transfer of the chemical through natural food chains.

Estimated dietary exposures based on maximum label application rates for the rat, deer mouse and meadow vole are 0.53, 4.5 and 3.4 mg/kg, respectively (Table 3-2).

3.28.2.2 Birds

Exposure for birds involves ingestion of exposed invertebrates or plant matter or direct contact with the ground after application to soils. Estimated dietary exposures based on maximum label application rates for the quail, marsh wren and robin are 0.83, 7.5 and 9.5 mg/kg, respectively (Table 3-2).

3.28.3 *Risk Characterization Sulfentrazone*

3.28.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole from the maximum label application rate would be 5,400, 630, and 830-fold lower, respectively, than the acute dietary LD_{50} for Sulfentrazone (Table 3-7). The estimated risk from Sulfentrazone exposure is considered insignificant for each of these species (Table 3-3).

3.28.3.2 Birds

The acute dietary exposures of Sulfentrazone to quail, marsh wren and American robin based on the maximum label application rate would be 2,700, 300 and 240-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail, and compared to acute dietary LD_{50} values (Table 3-7). Thus, the estimated risk from Sulfentrazone exposure is considered insignificant for quail and wren, and low for robin (Table 3-3).

3.29 Tebuthiuron

3.29.1 *Mechanism of Action and Environmental Fate Tebuthiuron*

Tebuthiuron is registered as a general use pesticide and is a relatively non-selective herbicide that affects plant growth via inhibition of photosynthesis. This herbicide becomes activated in soil where it is readily absorbed by plant roots (U.S. EPA 1994, USDA 1995h). Target plants include broadleaf weeds, grasses and woody brush. Tebuthiuron can be applied by broadcast or spot application to soil. Tebuthiuron accumulates in plants and moves to the stems and leaves after getting taken up by the root system. It may take 1 to 3 years for a plant to die from Tebuthiuron treatment (USDA 1995h).

Tebuthiuron is a persistent herbicide. In soil it can remain active for more than 3 years. A half-life of 35 months was estimated based on a study in which sandy loam was treated with Tebuthiuron in a laboratory setting. In field tests, half-lives in soil were estimated at between 12 – 15 months in areas receiving over 40 inches of annual rainfall. U.S. EPA has concluded that Tebuthiuron is persistent in both aerobic and anaerobic soil conditions (U.S. EPA 1994).

Tebuthiuron is also resistant to photodegradation in water. In continuously irradiated water Tebuthiuron did not degrade in 33 days. U.S. EPA has concluded that Tebuthiuron is persistent in both aerobic and anaerobic aquatic environments (U.S. EPA 1994).

Degradation products formed by break down of Tebuthiuron have low toxicity (USDA 1995h)

3.29.2 *Exposure Assessment Tebuthiuron*

3.29.2.1 Mammals

Exposure for mammals can occur via diet, direct contact with the ground or plants after application to soils, or inhalation during application. Estimated dietary exposures based on maximum WSDOT application rates for the rat, deer mouse and meadow vole are 3.4, 29 and 22 mg/kg, respectively (Table 3-2).

3.29.2.2 Birds

Exposure for birds can occur through diet, inhalation, or direct contact with the ground or plants after application. Estimated exposures based on WSDOT application rates for the quail, marsh wren and robin are 5.3, 48 and 61 mg/kg, respectively (Table 3-2).

3.29.3 *Risk Characterization Tebuthiuron*

3.29.3.1 Mammals

The acute dietary exposures to rats, mice and meadow vole based on maximum label application rates would be 120, 13 and 18-fold lower, respectively, than the acute dietary LD_{50} for Tebuthiuron (Table 3-7). The estimated risk from Tebuthiuron exposure is considered low to rats but moderate to mice and meadow vole (Table 3-3)

3.29.3.2 Birds

The acute dietary exposures of Tebuthiuron to quail, marsh wren and American robin from WSDOT's current application practices would be 380, 42, and 33-fold lower, respectively, than the acute dietary LD_{50} for bobwhite quail (Table 3-7). The risk from Tebuthiuron exposure is considered insignificant for quail and low for wren and robin (Table 3-3).

Table 3-7. Relative Risk for Herbicides Evaluated in 2005.

Chemical	Rat risk ratio	Deer Mouse risk ratio	Vole risk ratio	Quail risk ratio	Wren risk ratio	Robin risk ratio
2,4-D	70	8	11	56	6	5
Ammonium Salt of Fosamine	450	52	68	280	31	25
Bromacil/Diuron	260	30	39	160	18	14
Chlorsulfuron	12000	1400	1800	17000	1800	1400
Clopyralid	10000	1100	1500	2500	270	210
Clopyralid/2,4-D	1700	200	260	770	85	66
Dicamba	2000	230	300	290	32	25
Dicamba/2,4-D	370	44	58	420	46	36
Dicamba/MCPA	780	90	120	92	10	8
Dichlobenil	560	66	86	170	19	15
Diuron	510	59	77	160	18	14
Glyphosate	2000	230	310	1100	120	91
Metsulfuron methyl	50000	5600	7200	25000	3600	2800
Oryzalin	890	100	140	58	6	5
Picloram	3600	420	540	2300	250	200
Sulfometuron methyl	17000	1800	2400	11000	1200	950
Triclopyr	110	13	17	330	37	29
New Herbicides Evaluated in 2005						

Chemical	Rat risk ratio	Deer Mouse risk ratio	Vole risk ratio	Quail risk ratio	Wren risk ratio	Robin risk ratio
Bromoxynil	340	40	52	130	15	12
Diflufenzopyr	6,900	810	1,100	2,900	320	250
Flumioxazin	14,000	1,700	2,200	4,100	450	350
Fluroxypyr	6,900	800	1,100	3,600	400	320
Imazapyr	3,600	420	550	970	110	85
Isoxaben	7,100	830	1,100	900	100	79
Norflurazon	1,700	200	260	120	13	10
Oxadiazon	1,800	210	270	1,100	130	98
Pendimethalin	250	29	38	210	24	19
Pyraflufen	890,000	100,000	140,000	230,000	25,000	20,000
Sulfentrazone	5,400	630	830	2,700	300	240
Tebuthiuron	120	13	18	380	42	33

3.30 Uncertainties, Data Gaps and Mitigation Measures

Adjuvants and Inert Ingredients

Much remains to be understood regarding the specific components within each herbicide that are reported as inert ingredients. These ingredients are understood to have no toxicological (herbicidal, in this case) effects on the target organisms, but their effects on other organisms remain poorly understood. In cases where more than one herbicide may be applied, the potential for synergistic actions cannot be discounted. Adjuvants, as explained subsequently in Chapter 3, also require further study, as in some cases their toxicity to non-target ecological receptors may exceed the herbicidal toxicity, yielding additive effects as well. Current regulations do not require manufacturers to report toxicity in combination with adjuvants, and this information is therefore rarely reported. Thus, more focused work will be required to ascertain the true exposures and risks from the use of adjuvants applied with formulated herbicides, and the potential toxicological significance, if any, of the inert ingredients registered with the U.S. EPA as part of the herbicide products licensing.

Insect Risks

Exposure for insects involves direct contact due to spray techniques. An estimate of exposure to insects could not be projected from our current understanding of WSDOT's practices and the publicly available toxicity literature.

Reptiles and Amphibian Risks

Reptiles and amphibians can be exposed to herbicides through dietary consumption, inhalation and direct skin contact. Amphibians may be susceptible to additional exposure in run-off waters. No toxicity levels have been reported of reptiles and amphibians to the herbicides used by WSDOT, and exposure parameters have not been fully developed. Thus, risks to reptiles and amphibians cannot be fully assessed. Life history behaviors and WSDOT application practices would suggest that exposures to herbicides would be low under most circumstances. Roadside areas near aquatic habitats where amphibians and some reptiles (*e.g.*, garter snakes and turtles) might be found will be

protected by the minimum 60 ft buffer that will be employed by WSDOT. Reptiles in Washington's arid climates will generally avoid disturbance zones around roadways. In addition, these arid reptiles are mostly nocturnal in their behavior and would therefore largely avoid dermal contact. However, snakes would be susceptible to dermal contact to perhaps a greater extent than any other animal potentially found in the roadside vegetation management zone of the state's highways. Further study is necessary to establish exposure parameters necessary to estimate reptile or amphibian exposures to any degree of accuracy.

The following measures should be considered when herbicides are applied to minimize potential exposure and risks to wildlife. Measures specific to certain herbicides or their formulations are called out where appropriate.

- Do not overspray or apply the herbicides to adjacent non-target areas (*as summarized in* BPA 2000g).

- Control drift/runoff of the chemicals from target areas by observing weather conditions and not spraying during potentially high rainfall events (*as summarized in* BPA 2000g).

- Do not spray droplets less than 150 microns (*as summarized in* BPA 2000g).

- Formulate knowledge of the rate and extent of chemical exchange processes among plants, soil, water, and the atmosphere through monitoring and modeling. This information will aid in deciding where to distribute the chemical, at what times, and in what concentrations to avoid contamination of the environment or risk to wildlife, water quality, and/or aquatic species.

- Model how herbicides move in the soil layers (*e.g.,* see The Chemical Movement in Layered Soils (CMLS) model developed by the University of Florida) (Futch and Singh 1999). Diuron and Bromacil, for example, are effective herbicides that can migrate slowly through soils (depending upon soil characteristics [*i.e*. saturated conductivity, amount of saturation, porosity, organic matter]). Because of this mobility, there have been reported problems with leaching into nearby ground water resources (Li *et al.* 2001, Futch and Singh 1999, Indelman *et al.* 1998, Miles and Pfeuffer 1997, Powell *et al.* 1996). Therefore, avoid the use of Diuron and Bromacil in areas where porosity is high, organic material is low, and a water source can be easily accessed through leaching or runoff (Futch and Singh 1999).

- Monitor the persistence rate in treated vegetation to avoid cross contamination of non-target areas if mowing, vegetation removal, or composting is included within the mitigation measures.

- Do not use plant residues, including hay or straw from treated areas, or manure from animals that have grazed or consumed forage from treated areas for composting or mulching where susceptible plants may be grown the following season (Dow AgroSciences 2001).

- Avoid drift to non-target species. Clopyralid/2,4-D, for example, could be transported by wind in a dust form (Dow AgroSciences 2001). Use drift control or suitable deposition agent when dispersing the compound.

- Select formulations for use that minimize or avoid mixing with toxic adjuvants such as cyclohexanone and triisopropanolamine, which are adjuvants in some Clopyralid formulations.

- Do not apply aerially when an air temperature inversion exists. Air

temperature inversion is characterized by little or no wind and lower air temperature near the ground than at higher levels (Dow AgroSciences 2001).

- Do not treat powdery, dry soil and light, sand soil when rain is not predicted soon after application (USDA 1995b)
- Do no use on walkways, roads, frozen surfaces or other impermeable surfaces to reduce runoff or drift to non-target areas and aquatic environments (*as summarized in* BPA 2000b, USDA 1995b).
- Treated soil should remain undisturbed (*as summarized in* BPA 2000b).
- For Bromacil, reduce maximum application from 32 lbs a.i./A to 12 lbs a.i./A (except for toxic waste holding ponds at 25 lbs a.i./A) (U.S. EPA 1996a).
- Develop training materials to reduce potential contamination of water sources (U.S. EPA 1996a).
- Standardize use rates for certain weed control situations (U.S. EPA 1996a).
- Change labels to specify the time of application (U.S. EPA 1996a).
- Create Pesticide Management Zones (PMZ) (U.S. EPA 1996a).
- For Fosamine, the minimum recommended rates for dense stands of brush greater than six feet high is 8 lb (2 gal)/acre. A rate of 3 gal/acre may be best for taller brush and tough to control species (Tu *et al*. 2001f).
- Foliage should be dry at time of application (Tu *et al.* 2001f).
- To minimize inhalation exposures, do not apply with hollow cone-type insecticide or other nozzles that produce fine spray droplets (USDA 1995a).
- To reduce drift, do not apply on windy days or when wind is blowing toward desirable plants (USDA 1995a).
- Formulations with ethylene glycol as a carrier solvent should be avoided (sometimes seen with 2,4-D)
- Incorporate Dichlobenil granules into soils when using the 10% granular formulation. Incorporation of the compound into soils reduces exposure risks to wildlife (U.S. EPA 1998).
- For Dichlobenil, reduce application rates to 10 lbs. a.i./A (U.S. EPA 1998).
- Also for Dichlobenil, do not apply to St. Lucie fine sand, Arzell fine sand, or other light sandy soil (USDA 1995d).

4.0 Conclusions

Risks to terrestrial wildlife, including amphibians and reptiles, from WSDOT's use of herbicides to control roadside vegetation were examined to supplement the original EIS with current information (WSDOT 1993). Potential dietary doses of each herbicide were modeled by assuming the maximum amount of active ingredient applied per acre (as specified by WSDOT), and body weight and food consumption parameters derived from the Wildlife Exposure Handbook. Ecological receptors evaluated included the rat, quail, deer mouse, meadow vole, marsh wren and robin. The deer mouse, meadow vole and robin were generally found to have the highest potential exposure based on the modeling exercise. Based on exposure parameters used in this evaluation, the robin was the most sensitive species considered, as summarized in Table 3-7. However, the lack of species-specific toxicity data for the robin, as well as other species considered in this evaluation, adds additional uncertainty to the characterization of risks. Among mammals, the rat was the least sensitive species considered; while estimated risks for the deer mouse were the highest. Avian species were generally more sensitive than mammals to the herbicides evaluated. For avian species, quail were the least sensitive and robins were the most sensitive species [Note: in table 3-7, values are expressed in terms of toxicity value to estimated exposure; however, the discussion below uses the inverse ratio to compare risk among herbicides]. For mammals, estimated doses were at least two orders of magnitude less than the reported acute dietary toxicity for rats for all herbicides except 2,4-D in the deer mouse. Among avian species there were three herbicides for which the estimated dose for the American robin was less than an order of magnitude below the reported acute toxicity level for birds. These herbicides are: 2,4,-D, Dicamba/MCPA and Oryzalin. The estimated dose to wren was also less than an order of magnitude below the reported avian toxicity for 2,4-D and Oryzalin.

These results suggest that the maximum application rates suggested by WSDOT for most of the herbicides evaluated in this report do not pose a significant risk to small mammals or birds. However, the potential toxicity of certain herbicides, in particular 2,4-D, appears to pose an unacceptable toxicity risk to both mammals and birds at the maximum application rates suggested by WSDOT. In addition, the estimated toxicity risk presented by Dicamba and Oryzalin exceeds the acceptable threshold for at least one of the bird species evaluated. Among the herbicides evaluated in 2005, none appear to present a significant risk to mammals or avian species. Bromoxynil and Norflurazon appear to have the highest potential for toxicity if applied at maximum application rates suggested by WSDOT.

This evaluation has a number of important limitations including those described above. For instance, the toxicity data base is limited for adequately gauging potential effects from these herbicides to birds and mammals other than typical test species (*i.e.* rat and quail); and for reptiles, amphibians and insects to a slightly lesser extent there is far less information available for conducting a quantitative risk assessment. Amphibians, in particular, may be more susceptible to excessive exposure due to their dermal absorption capacity. More work is needed in this area to adequately gauge risks to these ecological receptors. Based on the results from this evaluation, it is recommended that the use of 2,4-D be examined closely to determine if there are areas where over-application may occur, or could be minimized. Given that 2,4-D is among the most common of herbicides applied in residential settings, and that the estimated doses to birds yields the smallest margin of safety relative to its toxicity, it is prudent to seek means to minimize exposure through reduced application. Caution is also suggested for application of Dicamba/MCPA and Oryzalin.

Chapter 2 References

Army Corps of Engineers (ACOE). 2003. L. Adjuvant Use – Fosamine – Willows. Obtained online at www.wes.army.mil/el/emrrp/emris/emrishelp4a/l_adjuvant_use_fosamine_willows.htm.

Bayer Crop Science. Buctril® Herbicide. Product label. U.S. EPA Registration No. 264-437

Bayer Crop Science. Buctril® Brand Herbicide. Material Safety Data Sheet (MSDS). MSDS Version 2.2. MSDS No. 000000000029

Bayer, 2004. Ronstar® 50 WSP product label. U.S. EPA Registration No. 432-893

BASF. 2000. Arsenal ® herbicide product label. U.S. EPA Registration No. 241-346

BASF. 2000b. Arsenal ® herbicide. Material Safety Data Sheet (MSDS). Product No.: 579611

BASF. 2001. Pendulum ® WDG herbicide product label. U.S. EPA Registration No. 241-340

BASF. 2003. Overdrive® herbicide. Product label. NVA 2002-04-078-0138.

BASF. 2004a. Habitat® herbicide product label. U.S. EPA Registration No. 241-426

BASF. 2004b. Pendulum ® 3.3 EC herbicide product label. U.S. EPA Registration No. 241-341

BioCycle. 2002. Prevalence and Fate of Clopyralid in Compost. *Journal of Composting and Organics Recycling*. February, page 57. Obtained online at http://www.jgpress.com/BCArticles/2002/020257.html.

Bonneville Power Administration (*as summarized in* BPA). 2000a. Fosamine Ammonium: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000b. Chlorsulfuron: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000c. Clopyralid: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000d. Dicamba: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000e. Oryzalin: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000f. Bromacil: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000g. Diuron: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000h. Dichlobenil: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000i. Metsulfuron methyl: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000j. Picloram: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Bonneville Power Administration (*as summarized in* BPA). 2000k. Triclopyr: Herbicide Fact Sheet. U.S. Department of Energy. March 2000.

Charles J.M., Bond D.M, Jeffries T.K, Yano B.L., Scott W.T., Johnson K.A., Cunny H.D., Wilson R.D. and Bus J.B.1996. Chronic Dietary Toxicity/Oncogenicity Studies on 2,4-Dichlorophenoxyacetic Acid in Rodents. *Fund. Appl. Toxicol.* 33: 166-172.

Cox C. 1998. Clopyralid: Herbicide Factsheet. *Journal of Pesticide Reform* 18: 15-19.

Cornell University. 2001. Oxadiazon (Ronstar) herbicide profile 2/85. Pesticide Management Education Program. http://pmep.cce.cornell.edu/

Denny J.W. 1991. DuPont Agricultural Products. Personal Communication.

Dow AgroSciences Canada Inc. (Dow AgroSciences). 1999a. Transline. Herbicide Material Safety Data Sheet (MSDS). Calgary, Alberta, Canada.

Dow AgroSciences. 1999b. Spike® 80DF specialty herbicide product label. U.S. EPA Registration No. 62719-107

Dow AgroSciences Canada Inc. (Dow AgroSciences). 2001. Material Safety Data Sheet (MSDS): Curtail Herbicide. Calgary, Alberta, Canada.

Dow AgroSciences. 2003. Gallery® 75 Dry Flowable herbicide. Material Safety Data Sheet (MSDS). MSDS: 003994

Dow AgroSciences. 2004. Vista® Specialty Herbicide. Material Safety Data Sheet. MSDS: 006301

Dow/Elanco. Undated(a). The Toxicology of Picloram. Form # 137-1943-789.

Dow/Elanco. Undated(b). Triclopyr: Technical Information Guide. Dow/Elanco Form Number 137-1973-88-B&L.

Dupont Inc. (DuPont). 1992. Product Information Bulletin: Krovar IDF. Mississauga, Ontario, Canada.

Dupont Inc. (DuPont). 2000a. Krovar IDF: Chemical Product and Company Information. Mississauga, Ontario, Canada. Obtained online at http://www.dupont.com/msds/95_05_gpa00081.html.

Dupont Inc. (DuPont). 2000b. Telar Toss-N-Go Bags: Chemical Product and Company Information. Mississauga, Ontario, Canada. http://www.dupont.com/msds/95_05_gpa00081.html.

Dupont Inc. (DuPont). 2001. Chlorsulfuron. Herbicide Material Safety Data Sheet (MSDS). MSDS Number: GPA00026. Obtained online at http//:www.dupont.ca/ag.

Dupont Inc. (DuPont). 2002. Material Safety Data Sheet (MSDS): DuPont "Krenite" Brush Control Agent. Mississauga, Ontario, Canada. Obtained online at http://www.dupont.com/msds/95_05_gpa00081.html.

Dwyer, F.J., D.K. Hardesty, C.G. Ingersoll, J.L. Kunz, and D.W. Whites. 2000. Assessing contaminant sensitivity of American shad, Atlantic sturgeon, and shortnose sturgeon. Action plan project Hudson River Estuary. United States Geological Survey.

Environmental Health Program. 1989. Diuron. HECS Publishing, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario, Canada.

European Commission. 2002. Review report for the active substance pyraflufen-ethyl. Health and Consumer Protection Dictorate-General. SANCO/3039/00-Final. July 2

Extension Toxicology Network (EXTOXNET). 1996a. 2,4-D: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/*summarized in* EXTOXNET/pips/24-D.htm.

Extension Toxicology Network (EXTOXNET). 1996b. Dicamba: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/ EXTOXNET/pips/Dicamba.htm.

Extension Toxicology Network (EXTOXNET). 1996c. Dichlobenil: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/*summarized in* EXTOXNET/pips/dichlobenil.htm.

Extension Toxicology Network (EXTOXNET). 1996d. Diuron: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/EXTOXNET/pips/diuron.htm.

Extension Toxicology Network (EXTOXNET). 1996e. Metsulfuron methyl: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/EXTOXNET/pips/metsulfuronmethyl.htm.

Extension Toxicology Network (EXTOXNET). 1996f. Oryzalin: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/ EXTOXNET/pips/Oryzalin.htm.

Extension Toxicology Network (EXTOXNET). 1996g. Picloram: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/EXTOXNET/pips/picloram.htm.

Extension Toxicology Network (EXTOXNET). 1996h. Sulfometuron methyl: Pesticide Information Profiles. Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/EXTOXNET/pips/sulfomet.htm.

Extension Toxicology Network (EXTOXNET). 1996i. Triclopyr: Pesticide Information Profiles. . Revised June 1996. Obtained online at http://ace.ace.orst.edu/info/EXTOXNET/pips/Triclopyr.htm.

Extension Toxicology Network (EXTOXNET). 1996j. Bromoxynil: Pesticide Information Profiles. Revised June 1996. Obtained online at http://extoxnet.orst.edu/pips/bromoxyn.htm

Extension Toxicology Network (EXTOXNET). 1996k. Pendimethalin: Pesticide Information Profiles. Revised June 1996. Obtained online at http://extoxnet.orst.edu/pips/pendimet.htm.

Extension Toxicology Network (EXTOXNET). 1996l. Tebuthiuron: Pesticide Information Profiles. Revised June 1996. Obtained online at http://extoxnet.orst.edu/pips/tebuthiu.htm.

Futch S.H. and Singh M. 1999. Herbicide Mobility Using Soil Leaching Columns. *Bull. Environ. Contam. Toxicol.* 62: 520-529.

Gomez de Barreda D., Lorenzo E., Gamon M., Walker A., Ramos C., Saez A., Carbonell E.A., Garcia de la Cuadra J., Munoz N., del Busto A. and Lidon A.L. 1996. Persistence and Leaching of Some Residual Herbicides in Uncropped Soils. *Bull. Environ. Contam. Toxicol*. 56: 219-224.

Grisolia CK, Bilich MR, and Formigli LM. 2004. A comparative toxicologic and genotoxic study of the herbicide arsenal, its active ingredient imazapyr, and the surfactant nonylphenol ethoxylate. *Ecotoxicology and Environmental Safety* 59 (2004) 123–126

Hartley D. and Kidd H., editors. 1987. *The Agrochemicals Handbook, 2nd edition*. Royal Society of Chemistry, Unwin Brothers Ltd. Old Woking, Surrey.

Hinkle S. 1979. Avian Dietary Toxicity (LC_{50}) in Bobwhite Quail and Mallard Ducks. H-12361. HLO-0706-79 and HLO-0565-79.

Holmes S.B., Thompson D.G., Wainio-Deizer K.L., Capell S.S. and Staznik B.1994. Effects of lethal and sublethal concentrations of the herbicide Triclopyr butoxyethyl ester in the diet of Zebra finches. *J. Wildlife Dis.* 30: 319-327.

Hung H. and Mackay D. 1997. A Novel and Simple Model of the Uptake of Organic Chemicals by Vegetation from Air and Soil. *Chemosphere* 35: 959-977.

Indelman P., Touber-Yasur I., Yaron B. and Dagan G. 1998. Stochastic analysis of water flow and pesticides transport in a field experiment. *J. Contaminant Hydrol.* 32: 77-97.

Jordan L.S. and Cudney D.W. 1987. Herbicides. In Biggar J.W. and Seiber J.N., editors. *Fate of Pesticides in the Environment,*. Agricultural Experiment Station, Division of Agriculture and Natural Resources, University of California, CA..

Kidd H. and James D.R., editors. 1991. *The Agrochemicals Handbook, 3rd edition.* Royal Society of Chemistry Information Services, Cambridge, UK, 1991 (as updated).

KjΦr C., Elmegaard N., Pederson M.B., Damgaard C. and Nielsen J.K. 2001. *Phytochemical responses to herbicide exposure and effects on herbivorous insects: Section 4 Phytochemical responses to Chlorsulfuron treatment under laboratory and field conditions* (Pesticide Research No. 55). Miljρstyrelsen: Danish Environmental Protection Agency. .

Li Q.X., Hwang E.C. and Guo F. 2001. Occurrence of Herbicides and their Degradates in Hawaii's Groundwater. *Bull. Environ. Contam. Toxicol*. 66: 653-659.

Meade A.B. 1983. Honeybee Test. G/F-1.

MDL Information Systems. 1994a. RTECS Profile for Fluroxypyr. DialogWeb™ RTECS Number: AF2500000

MDL Information Systems. 1994b. RTECS Profile for Isoxaben. DialogWeb™ RTECS Number: CV4370300

MDL Information Systems. 1996. RTECS Profile for Imazapyr. DialogWeb™ RTECS Number: US5682500

MDL Information Systems. 2002. RTECS Profile for Diflufenzopyr. DialogWeb™ RTECS Number: US5648943

MDL Information Systems. 2003a. RTECS Profile for Oxadiazon. DialogWeb™ RTECS Number: RO0874000

MDL Information Systems. 2003b. RTECS Profile for Tebuthiuron. DialogWeb™ RTECS Number: YS4250000

MDL Information Systems. 2004. RTECS Profile for Flumioxazin. DialogWeb™ RTECS Number: NR3399382

Miles C.J. and Pfeuffer R.J. 1997. Pesticides in Canals of South Florida. *Arch. Environ. Contam. Toxicol*. 32: 337-345.

Monsanto Company (Monsanto). 2001. Roundup. Original Herbicide Material Safety Data Sheet (MSDS). St. Louis, Missouri.

Nichino America. 2004. Edict® IVM herbicide product label. U.S. EPA Registration No. 71711-7

PMEP. 1985. Chlorsulfuron (Glean Weed Killer) Herbicide Profile. Cornell University. March 1985. Obtained online at http://pmep.cce.cornell.edu/profiles/herb-growthreg/cacodylic-cymoxanil/Chlorsulfuron/herb-prof-Chlorsulfuron.html.

Purdue University (Purdue). 2003. HORT640C – Metabolic Plant Physiology: Inhibition of Branched Chain Amino Acid Biosynthesis by Chlorsulfuron. Department of Horticulture and Landscape Architecture. Obtained online at http://www.hort.purdue.edu/rhodcv/hort640c/branch/br00005.htm.

Reno F.E. 1979. Acute Oral LD_{50} in Bobwhite Quail and Mallard Ducks, H-11, 627. Project Number 201-515 and 201-517.

Retnakaran A. and Wright J.E., editors. 1987. Control of Insect Pests with Benzoylphenyl Urea. In *Chitin and Benzoylphenyl Ureas* (Series Entomologica Volume 38) Dordrecht, Netherlands: Kluwer Academic Publishers Group.

Sandoz Crop Protection Corporation (Sandoz). 1992. Toxicology Information Summary – Dicamba.

Spectrum. 2003. Chemical Fact Sheet: 2,4-D. Spectrum Laboratories – Cas#94757. Obtained online at http://www.speclab.com/compound/c94757.htm.

Swift J. 2002. Fosamine Ammonium. The Commonwealth of Massachusetts Executive Office of Environmental Affairs Department of Food and Agriculture. Boston, MA. May 2, 2002. Obtained online at http://www.mass.gov/dfa.

Syngenta. 2000. Vanquish. Herbicide: Material Safety Data Sheet (MSDS). Registration Number: 26980. Formulation Number: A-10182 A. Guelph, Ontario, Canada.

Syngenta. 2001. Banvel. Herbicide Material Safety Data Sheet (MSDS). Syngenta Crop Protection Pty Limited, Pendle Hill, NSW, Australia.

Syngenta. 2001. Predict® Herbicide. Product label. U.S. EPA Registration No. 100-849

Terra International Inc. (Terra). 1999. Brash (Dicamba/2,4-D). Herbicide Material Safety Data Sheet (MSDS). CAS #: 2300-66-5 Dicamba, 94-75-7 DMA Salt of 2,4-D. May 30, 1999.

Tu, M., C. Hurd, and J.M. Randall. 2001a. *Weed Control Methods Handbook: Clopyralid*. The Nature Conservancy. Section 7B. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

Tu, M., C. Hurd, and J.M. Randall. 2001b. *Weed Control Methods Handbook: Clopyralid/2,4*-D. The Nature Conservancy. Section 7C. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

Tu, M., C. Hurd, and J.M. Randall. 2001c. *Weed Control Methods Handbook: Glyphosate*. The Nature Conservancy. Section 7E. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

Tu, M., C. Hurd, and J.M. Randall. 2001d. *Weed Control Methods Handbook: Picloram*. The Nature Conservancy. Section 7I. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

Tu, M., C. Hurd, and J.M. Randall. 2001e. *Weed Control Methods Handbook: Metsulfuron* methyl. The Nature Conservancy. Section 7J. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

Tu, M., C. Hurd, and J.M. Randall. 2001f. *Weed Control Methods Handbook: Fosamine*. The Nature Conservancy. Section 7D. Obtained online at http://tncweeds.ucdavis.edu/handbook.html.

U.S. Department of Agriculture (USDA). 1989. *Final Environmental Impact Statement: Vegetation Management in the Coastal Plain/Peidmont. Volume II. Appendices*. Management Bulletin R8-MB-23. U.S. Department of Agriculture, Forest Service Southern Region, Atlanta, Georgia.

U.S. Department of Agriculture (USDA). 1994a. Chlorsulfuron: Pesticide Fact Sheet. Prepared for the U.S. Department of Agriculture, Forest Service by Information Ventures, Inc. http://infoventures.com/e-hlth/pesticide/chlorsul.html.

U.S. Department of Agriculture (USDA). 1994b. Dicamba: Pesticide Fact Sheet. Prepared for the U.S. Department of Agriculture, Forest Service by Information Ventures, Inc. http://infoventures.com/e-hlth/pesticide.Dicamba.html.

U.S. Department of Agriculture (USDA). 1994c. Dichlobenil: Pesticide Fact Sheet. Prepared for the U.S. Department of Agriculture, Forest Service by Information Ventures, Inc. http://infoventures.com/e-hlth/pesticide/dichlobe.html.

U.S. Department of Agriculture (USDA). 1995a. 2,4-D: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pesticide/24d.html.

U.S. Department of Agriculture (USDA). 1995b. Chlorsulfuron: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pestcide/chlorsul.html.

U.S. Department of Agriculture (USDA). 1995c. Dicamba: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pestcide/Dicamba.html.

U.S. Department of Agriculture (USDA). 1995d. Dichlobenil: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pesticide/24d.html.

U.S. Department of Agriculture (USDA). 1995e. Metsulfuron methyl: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pesticide/metsulf.html.

U.S. Department of Agriculture (USDA). 1995f. Sulfometuron methyl: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pesticide/sulfomet.html.

U.S. Department of Agriculture (USDA). 1995g. Bromacil: Pesticide Fact Sheet. Prepared by Ventures, Inc. November 1995. Obtained online at http://infoventures.com/e-hlth/pesticide/bromacil.html.

U.S. Department of Agriculture (USDA). 1995h. Tebuthiuron: Pesticide Fact Sheet. Prepared for the U.S. Department of Agriculture, Forest Service by Information Ventures, Inc. http://infoventures.com/e-hlth/pestcide/tebuth.html

U.S. Department of Health and Human Services (USDHHS). 1980. Registry of Toxic Effects of Chemical Substances.

U.S. Department of the Interior (USDI). 1989. 2,4-dichlorophenoxyacetic acid. Herbicide Material Safety Data Sheet (MSDS). http://physchem.ox.ac.uk/MSDS/DI/2,4-dichlorophenoxyacetic_acid.html.

U.S. Environmental Protection Agency (U.S. EPA). 1988. Pesticide Fact Sheet: Picloram. No. 48.1.

U.S. Environmental Protection Agency (U.S. EPA). 1993. Sulfometuron methyl – Evaluation of two-generation, four-litter reproduction study in rats. Office of Pesticides and Toxic Substances. Memo from R.F. Fricke, Toxicology Branch, to L. DeLuise, Registration Div. Washington, D.C., February 23, 1993.

U.S. Environmental Protection Agency (U.S. EPA). 1994. Reregistration Eligibility Decision for Tebuthiuron. U.S. EPA Case 0054. http://www.epa.gov/oppsrrd1/REDs/old_reds/tebuthiuron.pdf

U.S. Environmental Protection Agency (U.S. EPA). 1996a. *Bromacil: Reregistration Eligibility Decision (R.E.D.) Facts*. Prevention, Pesticides and Toxic Substances Division. EPA-738-F-96-013. August 1996.

U.S. Environmental Protection Agency (U.S. EPA). 1996b. Registration Eligibility Decision Norflurazon: List A Case 0229. Office of Pesticide Programs: Special Review and Reregistration Division. http://www.epa.gov/oppsrrd1/REDs/0229.pdf

U.S. Environmental Protection Agency (U.S. EPA). 1997a. Reregistration Eligibility Decision for Pendimethalin. EPA document No 738-R-97-007. http://www.epa.gov/oppsrrd1/REDs/0187red.pdf

U.S. Environmental Protection Agency (U.S. EPA). 1997b. Sulfentrazone: Pesticide Fact Sheet Office of Prevention, Pesticides and Toxic Substances (7501C). February 27.

U.S. Environmental Protection Agency (U.S. EPA). 1998a. *Dichlobenil: Reregistration Eligibility Decision (R.E.D.) Facts.* Prevention, Pesticides and Toxic Substances Division. EPA-738-F-98-005. October 1998.

U.S. Environmental Protection Agency (U.S. EPA). 1998b. Bromoxynil: Reregistration Eligibility Decision (RED) Facts (EPA-738-F-98-011). Office of Prevention, Pesticides and Toxic Substances. September.

U.S. Environmental Protection Agency (U.S. EPA). 1998c. Fluroxypyr: Pesticide Fact Sheet Office of Prevention, Pesticides and Toxic Substances (7501C). September 30, 1998.

U.S. Environmental Protection Agency (U.S. EPA). 1999. Diflufenzopyr: Pesticide Fact Sheet Office of Prevention, Pesticides and Toxic Substances (7501C). January 28, 1999.

U.S. Environmental Protection Agency (U.S. EPA). 2000. Clopyralid: Pesticide Tolerance for Emergency Exemptions. Federal Register Environmental Documents. September 27, 2000. Volume 65, Number 188. Obtained online at http://www.epa.gov/fedrgstr/EPA-PEST/2000/September/Day-27/p24320.htm.

U.S. Environmental Protection Agency (U.S. EPA). 2001. Flumioxazin: Pesticide Fact Sheet Office of Prevention, Pesticides and Toxic Substances (7501C). April 12, 2001.

U.S. Environmental Protection Agency (U.S. EPA). 2002a. Chlorsulfuron: Pesticide Tolerance. Federal Register Environmental Documents. August 14, 2002. Volume 67, Number 157. Obtained online at http://www.epa.gov/fedrgstr/EPA-PEST/2002/August/Day-14/p20229.htm.

U.S. Environmental Protection Agency (U.S. EPA). 2002b. Federal Register Environmental Documents: Triclopyr and Pesticide Tolerance. From the Federal Register: August 14, 2002 (Volume 67, Number 157). http://www.epa.gov/fedrgstr/EPA-PEST/2002/August/Day-14/p20229.htm.

U.S. Environmental Protection Agency (U.S. EPA). 2003a. Reregistration Eligibility Decision for Oxadiazon. EPA document No. 738-R-04-003. http://www.epa.gov/oppsrrd1/REDs/oxadiazon_red.pdf

U.S. Environmental Protection Agency (U.S. EPA). 2003b. nPyraflufen-ethyl; Pesticide Tolerance. DocID:fr30ap03-8. Federal Register Volume 68, No. 83. April 30.

U.S. Forest Service (USFS). 1988. *Managing Competing and Unwanted Vegetation.* Pacific Northwest Region. Final Environmental Impact Statement.

U.S. Forest Service (USFS). 1989. *Vegetation Management in the Appalachian Mountains*. Vol. I, FEIS, July 1989.

Valent. 2003. Payload ® Herbicide. Material Safety Data Sheet (MSDS) VC No. 1420

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

Wellings I. 1991. DuPont Agricultural Products. Personal Communication.

Wilbur-Ellis. 2005. Portfolio® herbicide product label. EPA Registration No. 279-3282-2935

Wilson D.C. and Bond C.E. 1969. The effects of the herbicides diquat and Dichlobenil (Casoron) on pond invertebrates. Part I. Acute toxicity. *Trans. Am. Fishery. Soc*. 98: 438-443.

Washington Department of Transportation (WSDOT). 1993. *Roadside Vegetation Management: Final Environmental Impact Statement*. December.