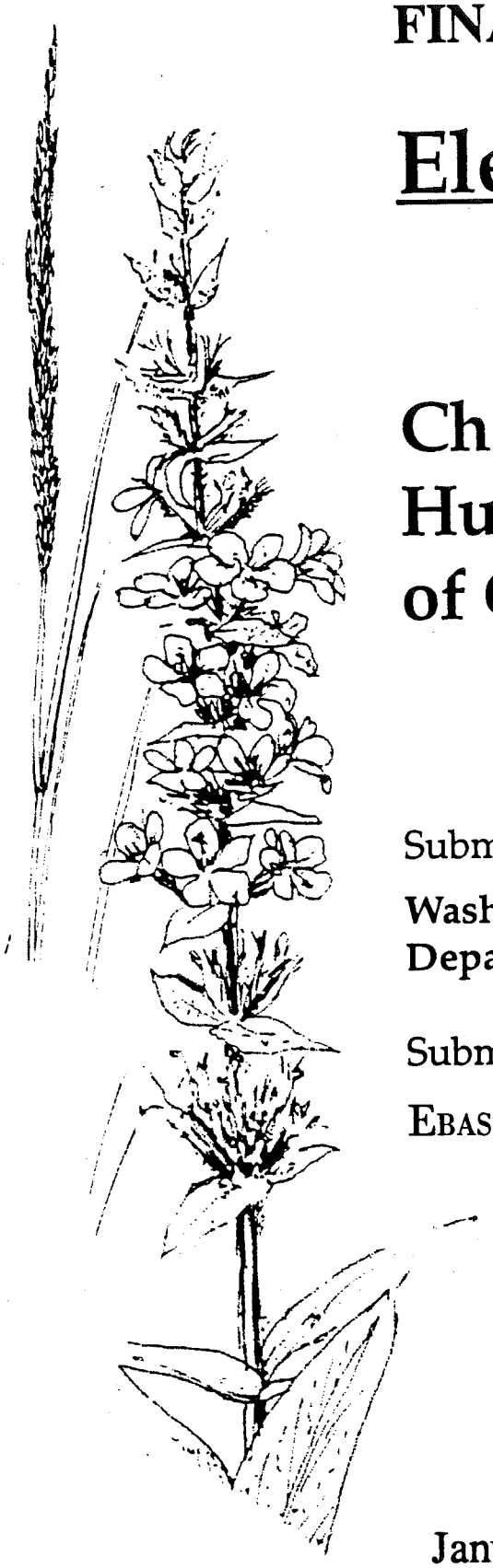
FINAL REPORT

Element F



Chemical Methods Only: Human Health Effects of Glyphosate

Submitted to

Washington State Department of Ecology

Submitted by

EBASCO ENVIRONMENTAL

January 1993

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Submitted to:

Washington State Department of Ecology

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1.0 INTRODUCTION

1.1 PURPOSE

The Washington State Departments of Agriculture, Ecology, Fisheries, Natural Resources, Wildlife, and the Washington State Noxious Weed Control Board, acting as co-lead agencies, have proposed to develop and implement a management plan for noxious emergent plant species in the State of Washington. Species of concern include three species of cordgrass or *Spartina* (*S. patens, S. alterniflora,* and *S. anglica*), purple loosestrife (*Lythrum salicaria* and *Lythrum virgatum*), garden loosestrife (*Lysimachia vulgaris*), giant hogweed (*Heracleum mantegazzianum*), and indigo bush (*Amorpha fruticosa*). These species are included on the Washington State Noxious Weed List (Chapter 16-750 WAC) because they are considered detrimental to the agricultural, aquacultural, and/or natural environments of the state. Their control (eradication, containment, or prevention of seed production) is mandated by inclusion on the Noxious Weed List. The effort required to control a noxious species varies according to its current distribution, likelihood of spread to uninfested areas, and other factors. The lead agencies seek to determine which management alternative or combination of alternatives would provide the most effective management of noxious emergent plants with the least environmental impacts. The ultimate goal of this effort is to develop criteria and approaches for managing infestations of both existing noxious species and new invaders.

The lead agencies have determined that management of these noxious emergent plant species could have a probable significant adverse impacts on the environment. Thus, an environmental impact statement (EIS) is required under RCW (Revised Code of Washington) 43.21C.030(2)(c). The lead agencies, through a public scoping process, have identified topics to be discussed in the EIS, including biology and ecology of problem species, management alternatives, efficacy and impacts of alternatives, and mitigation strategies. Ebasco Environmental was contracted by the nominal lead agency, the Washington State Department of Ecology, to assemble and synthesize available information on the topics of interest for probable inclusion in the EIS.

This report addresses the potential risks to human health from exposure to glyphosate, an herbicide that is currently used to control purple loosestrife and is being considered for *Spartina* control.

1.2 OBJECTIVES

The objectives of this report are to:

(1) review two existing herbicide risk assessments to determine their applicability in characterizing risks associated with human exposures to glyphosate used in controlling *Spartina* and purple loosestrife; and

(2) describe the potential impacts to public health that could occur from application of glyphosate to *Spartina* and purple loosestrife.

The first of the two risk assessments reviewed in this report, entitled "Worst Case Analysis Study on Forest Plantation Herbicide Use," was prepared for the Washington State Department of Natural Resources by Shipp et al. (1986) and will be referred to as the "DNR risk assessment" for the remainder of this report. The other risk assessment evaluated in this report is Appendix D of a Final Environmental Impact Statement entitled "Managing Competing and Unwanted Vegetation," and was compiled for the United States Department of Agriculture by Labat-Anderson Incorporated (1988). This risk assessment will be referred to as the "USDA risk assessment" in this report. Additional sources of information used in this report include published articles and EPA risk assessment guidance documents (e.g., USEPA 1989a, 1989b, 1992a).

Washington State applications of glyphosate are specifically targeted at noxious aquatic vegetation. Both the DNR and USDA risk assessments evaluate potential risks associated with exposures to a variety of pesticides (including glyphosate and 2,4-D) to control forest vegetation. Therefore, this report focuses on potential similarities differences in exposures to glyphosate applied to forest vegetation and exposures to glyphosate applied to control aquatic vegetation.

1.3 REPORT ORGANIZATION

This report is composed of several chapters. Chapter 2 summarizes the potential fate and transport of glyphosate and provides background information on *Spartina* and purple loosestrife. The information in this section is used in subsequent sections to evaluate the applicability of the DNR and USDA risk assessments in assessing risks associated with exposure to glyphosate used to control *Spartina* and purple loosestrife.

Chapter 3 evaluates the exposure assessments in the DNR and USDA risk assessments. Specifically, the methods and assumptions used to estimate glyphosate concentrations and potential human exposure are reviewed. Additionally, given the differences discussed above (e.g., DNR and USDA risk assessments were conducted to evaluate herbicide applications to forest vegetation), acute and chronic exposures are independently calculated using EPA Region 10 Guidance. The results of this evaluation are considered conservative estimates of glyphosate exposures from application to noxious aquatic weeds.

Chapter 4 evaluates the toxicity assessments in the DNA and USDA risk assessments and augments these data with updated glyphosate toxicity information generated after the publication of the two reports. The toxicities associated with glyphosate degradation products; trace impurities generated in the production of glyphosate; surfactants; and other herbicides or pesticides that may be used in areas where glyphosate is applied are also addressed in this chapter.

In Chapter 5, the risk characterization methodology and results of the DNR and USDA risk assessments are evaluated and discussed in terms of their relevancy for the application of glyphosate in the control of *Spartina* and purple loosestrife. Also, acute and chronic risks are calculated using EPA guidance and are compared with the results in the DNR risk assessment. In Chapter 6, a summary of the uncertainties associated with the two risk assessments and the updated calculations using EPA guidance are presented, and conclusions regarding human health implications of glyphosate use for controlling *Spartina* and purple loosestrife are discussed. Chapter 7 provides a list of references cited in the report. Appendix A contains a summary of environmental fate and transport characteristics of glyphosate. Appendix B contains a list of all adjuvants registered for use in Washington State.

2.0 BACKGROUND INFORMATION

This chapter provides summary information upon which the evaluation of the DNR and USDA risk assessments can be based. In Section 2.1, a summary of glyphosate composition, use, and re-registration status is provided. Environmental fate and transport characteristics of glyphosate are summarized in Section 2.2. A brief description of the ecological setting of *Spartina* and purple loosestrife is presented in Section 2.3. Information for this chapter was obtained from Elements A, B, and E of this Report series (Ebasco Environmental 1992a, 1992b, 1992c).

2.1 GLYPHOSATE COMPOSITION, USE, AND RE-REGISTRATION STATUS

Glyphosate is being considered for the control of *Spartina* in coastal Washington and is already being applied to control purple loosestrife in inland waters. Glyphosate is a systemic, nonselective, herbicide that inhibits plant growth by interfering with production of amino acids. Aminophosphonic acid (AMPA) is the primary degradation product of glyphosate (Ebasco Environmental 1992c). The only glyphosate formulation that is commercially available and registered for aquatic use in Washington State is Rodeo®, manufactured by Monsanto Agricultural Products Company. Rodeo® contains 53.5 percent glyphosate as the isoproplyamine salt and 46.5 percent inert ingredients (Monsanto 1990). Additional information concerning glyphosate composition and use can be found in Element E of this Report series (Ebasco Environmental 1992c).

No restrictions exist on the use of water treated with glyphosate for irrigation, recreation, or domestic purposes. However, the Rodeo® label prohibits application within 0.8 km (0.5 mile) upstream of potable water intakes, and requires that at least 24 hours pass before retreating a given area (Ebasco Environmental 1992c).

Data submittal for Rodeo® re-registration is essentially complete. The only remaining study (data gap) is the field soil dissipation study, which will probably be submitted by May 1993. The EPA review of the original field soil dissipation study indicated that it should have included deeper soil samples. All required environmental fate studies have been found acceptable by EPA (Sheila Shooty personal communication 1993). Similarly, all of the wildlife toxicity studies have been submitted and found acceptable by EPA (Ebasco Environmental 1992c).

2.2 GLYPHOSATE ENVIRONMENTAL FATE AND TRANSPORT

This section presents a summary of the environmental fate and transport of glyphosate. More detailed fate and transport data are presented in Appendix A.

The scientific literature indicates that glyphosate degrades or dissipates fairly rapidly in the environment. Degradation occurs almost exclusively through biological activity (i.e.,

biodegradation) and is thus dependent upon factors governing microbial activity in soil and water (i.e., temperature, moisture, pH, etc.).

The half-life of glyphosate in soil and water varies considerably. The half-life in soil ranges from less than 1 day to greater than 249 days but averages about 60 days (Reinert and Rodgers 1987). The half-life in freshwater ranges from 1.5 to 21 days (Goldsborough and Becks 1989, U.S. EPA 1992b), and less than 2 days in estuarine waters (Kroll 1991). Studies (O'Keefe 1985) have demonstrated the persistence of glyphosate (up to 1 year) in some estuarine sediments. Freshwater stream studies (Feng et al. 1990; Newton et al. 1984) have demonstrated that glyphosate and AMPA residues in bottom sediments are persistent when compared to stream water residues, but decrease to below detection over time.

Glyphosate is considered practically nonmobile in soils and sediments by virtue of its rapid and strong adsorption onto soil particles (Sprankle 1974). Thus its leachability through soil is generally low (Torstensson 1985). The soil mobility of glyphosate, as measured by R_f values and soil partition coefficients, is very low (Helling 1971). Soil sorption of glyphosate occurs over a wide range of soil pH values (Nicholls and Evans 1991).

Groundwater contamination by glyphosate has not been reported in the literature. Soil studies (Roy et al. 1989) have demonstrated that more than 95 percent of the total herbicide residue is present in the upper soil organic layer and that the lateral movement of glyphosate in runoff or through subsurface flow was not observed. Based on the physico-chemical characteristics of glyphosate, the possibility for groundwater contamination appears remote.

Glyphosate is considered to be nonvolatile and therefore to have a low potential for damage to nontarget species when used adjacent to agricultural croplands. However, wind drift and spray losses may carry the applied product to non-target plants. Washoff of the herbicide from rainfall or irrigation of treated plants within 2 hours of application may render the treatment ineffective and at the same time disperse the herbicide into the environment.

2.3 ECOLOGICAL SETTING

A brief description of *Spartina* and purple loosestrife is provided below. A more complete evaluation of the biological and ecological aspects of *Spartina* and purple loosestrife can be found in Elements A and B of this Report series (Ebasco Environmental, 1992a, 1992b), from which this summary was taken.

2.3.1 *Spartina*

Three non-native *Spartina* species have been declared noxious in Washington: *S. alterniflora*, *S. patens*, and *S. anglica*. *Spartina* are rhizomatous, deep-rooted, perennial grasses that inhabit a wide range of tidal, salinity, and substrate conditions in intertidal areas. The distribution of *Spartina* in Washington State is shown in Figure 1.

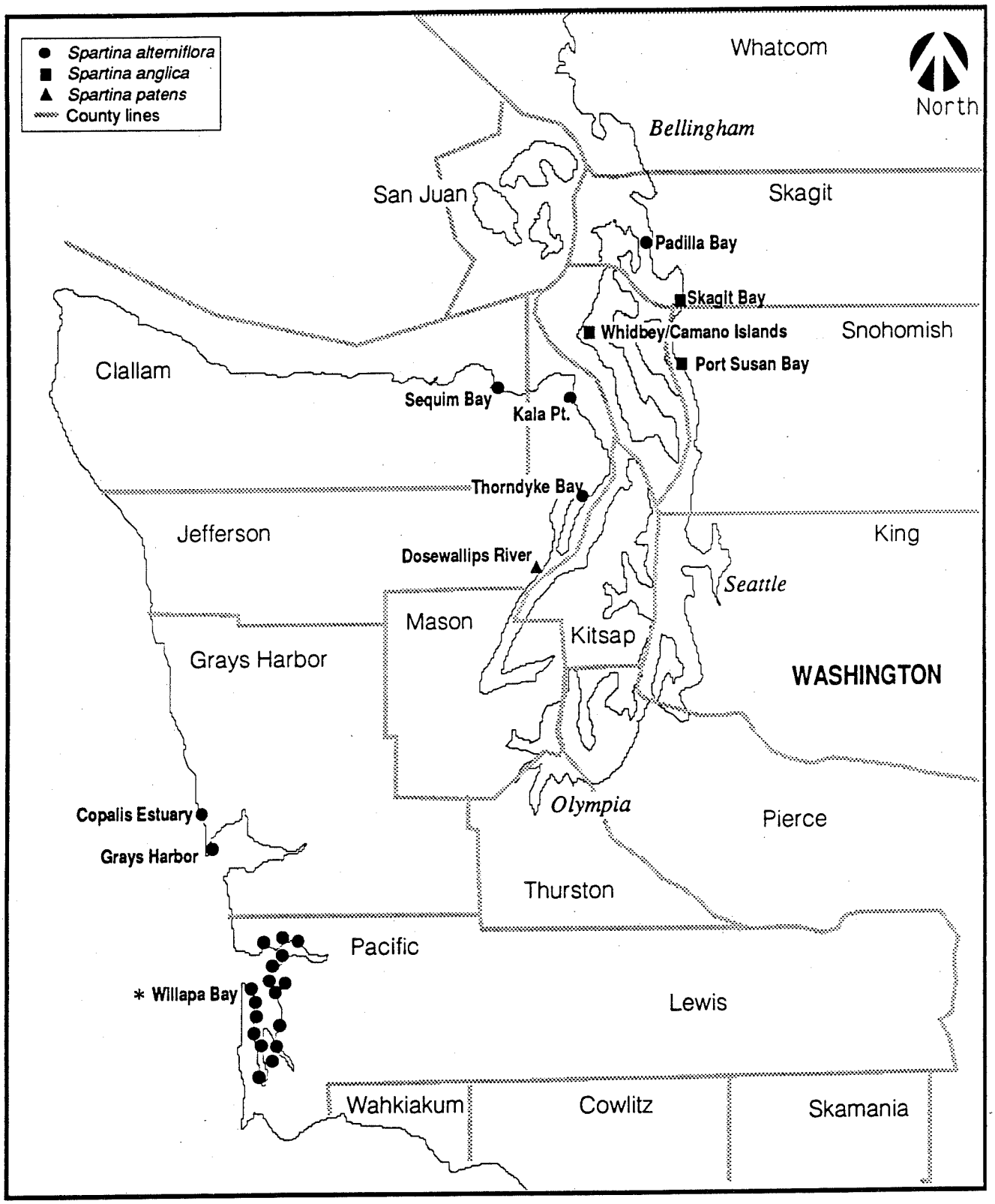

Figure 1. General locations of *Spartina* infestations in Washington. Areal estimates of infestations, if available, are given in text. Redrawn from Aberle (1990).

**Spartina alterniflora* is present throughout Willapa Bay.

S. alterniflora is established in multiple locations in Washington State. According to current areal estimations, *S. alterniflora* occupies between 1,100 and 2,500 acres in Willapa Bay, Washington (Sayce 1990). *S. alterniflora* has also recently established at Damon Point in Grays Harbor (ACOE 1992). Riggs (1992) estimates 48,100 m^2 (11.9 acres) exist in Skagit County, Washington. At three other localities, Thorndyke Bay, Kala Point, and Gibson Spit (Sequim Bay), individual patches of *S. alternifora* covered 350-1300 m^2 (0.1 to 0.3 acres) as of 1984.

The only occurrence of *S. patens* in Washington is a small infestation at Dosewallips State Park at the mouth of the Dosewallips River where it enters Hood canal. In 1984, the patch covered about 150 m^2 (0.04 acre).

S. anglica or a mixture of *S. anglica* and *S.* x *townsendii* occurs in Port Susan Bay, Skagit Bay, and Deer Lagoon on Whidbey Island, Washington. In Skagit Bay, *S. anlica*/x *townsendii* is present at West Pass near the entry of the Stillaguamish River and at the mouth of the north fork of the Skagit River. In 1989, *S. anglica* was discovered at Deer Lagoon on Whidbey Island (Aberle 1990).

2.3.2 Loosestrife

Purple loosestrife is an emergent Eurasian wetland plant. It is a non-rhizomatous, deep-rooted perennial that inhabits a wide range of soil, hydrological, light, and competitive conditions in wetland habitats in Washington. Slow-moving watercourses with broad alluvial deposits provide optimal sites for purple loosestrife colonization (Ebasco Environmental 1992b).

Based on a tentative inventory, purple loosestrife occurs in 30 of Washington's 39 counties. It appears to be colonizing more slowly in western Washington than in eastern Washington. The extensive irrigation canal system in eastern Washington (Winchester and Frenchman Hill wasteways) may also facilitate the spread of this species. At present, the Washington State Departments of Wildlife and Agriculture are compiling a purple loosestrife inventory database, and this information will be used to more accurately map the distribution of purple loosestrife in Washington. Site-specific information is being compiled and a current distribution map will be available upon the completion of that study.

3.0 EXPOSURE ASSESSMENT REVIEW

In this section the exposure assessments presented in the DNR and USDA risk assessments are reviewed and updated using EPA guidance published after these two risk assessments were written. An exposure assessment combines information regarding the concentrations of chemicals from a contaminated area with assumptions about the people who could come into contact with the contamination. The result is an estimation of a person's potential rate of contaminant intake. The intake rates are evaluated in the risk characterization step of a risk assessment to estimate the risks they could pose.

In Section 3.1, the ways in which people could be exposed to glyphosate applied to *Spartina* and purple loosestrife are identified and discussed. In this section, a comparison is made between the exposure scenarios evaluated in the two risk assessments and the exposure scenarios considered possible as a result of glyphosate application to *Spartina* and purple loosestrife. Next, in Section 3.2, the concentrations of glyphosate at locations where people might be exposed are evaluated, and the assumptions used in the two risk assessments concerning people's activities, such as the frequency with which a person could come into contact with glyphosate, are discussed. In Section 3.3, an independent evaluation of exposure to glyphosate was conducted to account for the differences between the application of glyphosate to forest vegetation and application to control aquatic vegetation. Acute and chronic exposures are quantified using conservative assumptions of glyphosate concentrations and human exposures. These values are considered conservative estimates of glyphosate intake resulting from application to control noxious aquatic vegetation.

One of the main differences between glyphosate exposure evaluated in the two risk assessments and potential exposure to glyphosate applied to *Spartina* and purple loosestrife in Washington is the physical environment into which the glyphosate will be introduced. In the two risk assessments, exposure to glyphosate is evaluated for forest applications. Some differences in the physical environment and the fate and transport of glyphosate are anticipated between the forest setting and the aquatic setting of *Spartina* and loosestrife. These differences will have repercussions in the estimates of environmental concentrations of glyphosate potentially available at points of human exposure and in the types of human exposures anticipated. Additionally, at the time of publication of the two risk assessments, limited information was available concerning environmental concentrations of glyphosate resulting from application of glyphosate in a forest setting. Thus, much of the environmental concentration data used in the DNR and USDA risk assessments are for other herbicides, such as 2,4-D and 2,4,5-T.

3.1 IDENTIFICATION OF EXPOSURE ROUTES

An exposure route describes the ways in which people can be exposed to contamination in a particular area. This section identifies the exposure routes potentially associated with the application of glyphosate to *Spartina* and purple loosestrife. Because Washington State is specifically concerned about the effects of pesticide application on public health, the focus of

this report will be on public exposures. A summary of potential public exposure routes from glyphosate application to control these noxious weeds is provided in Table 1.

Once glyphosate is released into the environment, it can enter a number of media, such as air, surface water, soil, and sediments. People could be exposed to glyphosate if they contact these media. For example, people can be exposed to glyphosate by inhaling fine glyphosate spray droplets or windblown soil particles to which glyphosate is adsorbed. People can also dermally contact airborne glyphosate or glyphosate residues on vegetation, soil, sediments, or surface water. If glyphosate were applied to freshwater, people could potentially come into contact with the herbicide if they were to drink the water. (It is unlikely that glyphosate would migrate to groundwater and cause human exposure via drinking water due to its strong affinity to adsorb to soil particles and low leachability (Ebasco Environmental 1992c)). People could incidentally ingest glyphosate in soil or sediments if they inadvertently swallow the soil, which could occur by activities such as touching dirty hands to their mouths or by placing dirty objects (such as toys) into their mouths. Children are especially prone to exposure to soil because many of their activities, such as playing in dirt and placing their fingers in their mouths, can result in soil ingestion. People can also ingest glyphosate by eating food containing glyphosate residues. Incidental exposure to glyphosate could occur if an individual ingests berries, garden vegetables, wild meat, fowl, fish, or shellfish that have been exposed to glyphosate.

The populations that could come into contact with glyphosate in the soil and water in an area are dictated by the types of land uses at or near a spray site. Land uses in areas where *Spartina* and purple loosestrife are found in Washington State include remote coastal locations where people may be exposed during recreational activities and residential areas where exposure may occur during daily activities. Individuals could be exposed to glyphosate at or near a spray site while engaged in activities such as playing, walking, swimming, and fishing. Nearby residents could be exposed if glyphosate were to drift to their property during application. Also, people who consume plants or wildlife harvested near the spray area could be exposed to glyphosate if it is present in the plant or animal tissues.

3.2 EVALUATION OF DNR AND USDA EXPOSURE ASSESSMENTS

In a human health risk assessment, the possible routes of exposure to contamination are examined to determine if the contamination could pose a threat to a human health. The risks associated with exposure to contamination depend not only on the concentration of the chemical, but also on the frequency and duration of exposure. In this section, the glyphosate concentrations at potential points of human exposure (i.e., exposure point concentrations) and the assumptions regarding the extent of exposure are discussed for the four potential exposure routes evaluated in the DNR and/or USDA risk assessments:

- inhalation,
- dermal contact with soil or vegetation,

Table 1. Potential Exposure Routes for Glyphosate Applied to *Spartina* and Purple Loosestrife

	Included in Risk Assessment?	
Exposure Route	DNR[1]	USDA[2]
Inhalation of Fine Spray Droplets	Yes	No
Dermal Exposure, Terrestrial Site	Yes	Yes
Dermal Exposure, Aquatic Site	No	No
Surface Water Ingestion	Yes	Yes
Soil Ingestion	No	No
Sediment Ingestion	No	No
Ingestion of Wild Meat	Yes	Yes
Ingestion of Wild Fowl	No	Yes
Ingestion of Fish	Yes	Yes
Ingestion of Shellfish	No	No
Ingestion of Garden Vegetables	Yes	Yes
Ingestion of Wild Berries	Yes	Yes

[1] The DNR risk assessment assumed exposures to both adults and children (ages 1 and 10).

[2] The USDA risk assessment assumed exposures to adults only.

- ingestion of surface water, and
- ingestion of food containing glyphosate residues.

This section also includes a discussion the USDA and DNR risk assessments' evaluation of multiple exposure routes, exposure to sensitive subpopulations, and exposure from repeated glyphosate applications. In Section 3.3, intakes associated with all potential exposure routes identified in Table 1 are estimated using the most recent EPA guidance available.

In the DNR and USDA risk assessments, the main evaluation focused on risks to individuals from a single spray event. Except where noted elsewhere in this section (3.2), the exposures discussed herein refer to exposures resulting from a single application of glyphosate. These exposure scenarios are likely to underestimate risks, because glyphosate could be applied twice annually to control noxious aquatic vegetation in Washington State. Therefore, in Section 3.3 of this chapter, potential adult and child acute and chronic exposures resulting from multiple glyphosate applications are addressed.

For each exposure route in the DNR risk assessment, four exposure scenarios were evaluated in order to estimate potential doses associated with a range of possible human exposures. These four scenarios are: single day reasonable exposure; single day worst-case exposure; total reasonable exposure (i.e., multiple exposure to glyphosate from a single spray event); and total worst-case exposure.

In the USDA risk assessment, scenarios were evaluated to represent realistic and worst-case exposure conditions. A worst-case accidental spill scenario was also evaluated for some exposure routes. The main difference between the realistic and the worst-case scenarios evaluated in the USDA risk assessment was the application method that was assumed. In general, realistic scenarios were those with smaller application areas than worst-case scenarios. The application methods evaluated include helicopter spraying, fixed-wing spraying, truck spraying, and back-pack spraying. The terms "reasonable", "realistic", and "worst case" are used in this report to be consistent with the terminology used in the scenarios developed in the DNR and USDA risk assessments. In each section below, an evaluation of the potential conservatism (or lack thereof) of these scenarios with respect to control of noxious aquatic weeds is provided.

3.2.1 Inhalation Exposure

Exposure to glyphosate via inhalation was considered in the DNR risk assessment. Inhalation exposure was not considered in the USDA risk assessment, because the exposures associated with herbicide inhalation were deemed to be negligible based on studies by Draper and Street (1982), Nigg and Stamper (1983), and Libich et al. (1984). An evaluation of the inhalation route exposure assessment provided in the DNR risk assessment is presented below and is summarized in Table 2.

Table 2. Inhalation Exposure Assumptions and Air Concentrations[1/]

Exposure Factor	DNR Reasonable Case	DNR Worst Case	Comments
Exposure Point Concentration	Single Day: based on median 2,4-D concentrations in Lavy et al. (1982)	Single Day: based on maximum single detection 2,4-D concentrations in Lavy et al. (1982).	Glyphosate air concentrations are needed; degree to which 2,4-D and 2,4,5-T data over- or underestimate glyphosate concentration is not known.
	Total Exposure: based on first order decay model using 2,4,5-T ester air monitoring date in Cheney et al. (1978) and single day reasonable concentrations.	Total Exposure: based on first order decay model using 2,4,5-T ester air monitoring data in Cheney et al. (1978) and single day worst case concentrations	
Application Rate	1.5 lbs a.i./acre	3 lbs a.i./acre	For *Spartina* and loosestrife, Rodeo® label application rate specifies 2.7 to 5.1 lbs a.i./ acre; reasonable and worst case assumptions could underestimate risk by less than 1 order of magnitude, assuming 5.1 lbs a.i./acre application rate.
Inhalation Rate	15 m^3/day	20 m^3/day	Appropriate based on EPA Guidance (1989a).
Body Weight	55 kg	55 kg	More conservative than EPA default parameters; could overestimate risks by less than 1 order of magnitude.

1/ Inhalation exposure not evaluated in the USDA risk assessment.

3.2.1.1 Ambient Air Exposure Point Concentrations

In the DNR risk assessment, ambient air concentrations of glyphosate were estimated for four types of exposures: a single day reasonable exposure, a single day worst case exposure, a total reasonable exposure, and a total (i.e., multiple) worst case exposure. Each of these exposures was based on data for 2,4-D or 2,4,5-T because of the lack of glyphosate data.

Single day reasonable and worst case exposure concentrations used in the DNR risk assessment were based on data for 2,4-D indicated in Lavy et al. (1982). In that study, 31 out of 32 air monitors failed to measure detectable levels of 2,4-D. The median level was below the detection limit of 0.05 μg. Using 0.05 μg and the average volume of air sampled in the experiment (21.1 liters), the more reasonable concentration to be encountered by humans in the spray area was assumed to be 0.00118 mg/m^3/lb acid equivalent (a.e.). For worst case single day exposures, the highest air concentration of 2,4-D (i.e., the single detection) by Lavy et al. (1982), 0.0028 mg/m^3/lb a.e./acre, was used. Because glyphosate is less volatile than 2,4-D, exposures based on 2,4-D data may overestimate the exposure point concentration. However, considering that the ambient air concentrations are based on nondetected concentrations, use of these data to estimate glyphosate concentrations may not be appropriate. The degree to which these data over-or underestimate risk is not known.

For total exposures evaluated in the DNR risk assessment, a decay model of the form $A_0 2^{-t/h}$ was fit to the 2,4,5-T ester air monitoring data listed in Cheney et al. (1978). (A_0 = initial concentration of substance at time (t) = 0; t = elapsed time since application; h = half-life of chemical expressed in days). The total concentration of herbicide in ambient air was estimated to be about 2.7 times the concentration estimated for the reasonable single day and worst case single day scenarios. Because 2,4,5-T is more persistent and more volatile than glyphosate, this may be a conservative assumption. However, because the specific data used to arrive at this value were not included in the DNR risk assessment, this calculation was not verified for purposes of this report.

3.2.1.2 Inhalation Exposure Assumptions

The inhalation exposure assumptions used for body weight, inhalation rate, and exposure duration in the DNR risk assessment will likely provide conservative estimates of dose. For body weight, a 55 kg individual was assumed to breathe 15 m^3/day for the reasonable case and 20 m^3/day for the worst case. This body weight is stated to be an average female body weight. According to the data cited in EPA's Exposure Factors Handbook (1989b), 55 kg is a mean body weight for teenage boys and girls. The mean body weight for women age 18 to 75 is 65.4 kg. Therefore, use of a 55 kg body weight is a conservative estimate for most of the adult population. This value is less than one order of magnitude more conservative than the EPA standard default parameter of 70 kg for adults.

An inhalation rate of 15 m^3/day (1.35 m^3/hour) was used in the DNR risk assessment to represent an average breathing rate during moderate activity. The 20 m^3/day (1.8 m^3/hour)

inhalation rate, a reasonable maximum EPA (1991a) assumption, was used to represent a reasonable upper-bound value for adults. Both these hourly inhalation rates fall within the inhalation rates listed in EPA (1989b) for light to moderate activity, and these assumptions seem reasonable. Two exposure durations were considered in the DNR risk assessment: (1) a one day (24 hour) exposure, and (2) repeated exposures until the glyphosate decays.

The 2,4-D and 2,4,5-T data used to estimate glyphosate concentrations were corrected to account for glyphosate application rates. The reasonable and worst case application rates used in the DNR risk assessment may be lower than those expected for the application of Rodeo® to *Spartina* and purple loosestrife. In the DNR risk assessment, median and maximum ambient air concentrations of glyphosate were estimated. Application rates of 1.5 (median) and 3 (maximum) pounds active ingredient per acre (a.i./acre), were used based on data on glyphosate application from the Washington State Department of Natural Resources (1985). However, label instructions for application of Rodeo® to cordgrass specify 4 to 7.5 pints of product per acre for aerial and hand-held broadcast application. The label application rate for purple loosestrife is 4 pints/acre. According to the label, a gallon of Rodeo® contains 5.4 lbs active ingredient per US gallon. Therefore, as shown in Equation 3-1 below, the application rate of Rodeo® would be 2.7 to 5.1 lbs a.i./acre to control *Spartina* and 2.7 lbs a.i./acre for purple loosestrife control. The highest application rate following Rodeo® label directions (5.1 lbs a.i./acre) could exceed the highest application rate addressed in this risk assessment (3.0 lbs a.i./acre), while the median rate could underestimate the application rate. However, the over- or underestimation is expected to be nominal, because the differences in exposure point concentrations based on application rates would be minimal (less than an order of magnitude).

(4 to 7.5 pints/acre)(gallon/8 pints)(5.4 lbs a.i./gallon) = 2.7 to 5.1 lbs a.i./acre (3-1)

3.2.2 Dermal Contact with Vegetation

Dermal exposure can occur from contact with sprayed vegetation or from being directly sprayed by accident, or from skin contact with contaminated soils. In the DNR risk assessment, dermal exposure was evaluated for contact with foliage only. In the USDA risk assessment, dermal exposure was evaluated for skin contact with foliage and berries and for contact from accidental spraying. The exposure point concentrations and exposure assumptions used in the DNR and USDA risk assessments for dermal exposure pathways are discussed below and are summarized in Table 3.

3.2.2.1 Dermal Contact with Vegetation Exposure Point Concentrations

The methodology used to assess concentrations of glyphosate on foliage that could be encountered by a hiker was similar for both risk assessments. In the absence of data specific to glyphosate, both risk assessments used data from a study conducted by Lavy et al. (1980) in which no levels of 2,4,5-T were detected on 100 cm^2 patches on a person walking through a forest following application at 2 lb acid equivalent per acre. For a reasonable exposure

Table 3. Dermal Contact with Vegetation Exposure Assumptions and Exposure Point Concentrations

Exposure Factor	DNR Reasonable Case	DNR Worst Case	USDA Scenario	USDA Accident	Comments
Glyphosate Exposure Point Concentration for Foliage	Single Day: based on 1/2 detection limit of 2,4,5-T concentrations in Lavy et al. (1980). Total Exposure: Based on single day reasonable exposure concentration and first order decay with 30-day half life.	Single Day: based on detection limit of 2,4,5-T in Lavy et al. (1980). Total Exposure: Based on single day worst case concentration and first order decay with 30-day half life.	Based on detection limit of 2,4,5-T data in Lavy et al. (1980) for foliage. (For berries based on unspecified factors reported by Hoerger and Kenaga (1972).)	Not specified.	Need glyphosate data for foliage; DNR concentrations based on nondetected 2,4,5-T values could underestimate risk; not enough specific information provided in USDA scenario for berries or in USDA accident scenario; DNR and USDA could underestimate concentrations due to difference between forest and coxious weed environments; soil half-lives of 70 days have been reported and biodegratation on berry or foliage could occur more slowly thereby underestimating concentration; degree of potential underestimation unknown.
Glyphosate Application Rate	1.5 lbs a.i./acre	3 lbs a.i./acre	Not specifically stated; likely between 1.5 and 5 lbs a.i./ acre.	5 lbs a.i./acre	DNR rates may be low (underestimate risk by less than 1 order of magnitude) if application rates are based on Rodeo® label (2.7 to 5 lbs a.i./acre); USDA worst case not clearly stated; USDA accident scenario reasonable given Rodeo® label application rates.
Skin Surface Area Exposed	0.81 m^2	1.62 m^2	10% of total human surface are exposed, and 40% total human surface area exposed from contact with clothing.	2 square feet of human skin.	DNR worst case parameters likely conservative; USDA worst case, DNR reasonable case, and USDA accident parameters could underestimate dose if more surface area is exposed.
Body Weight	55 kg	55 kg	50 kg	50 kg	More conservative than EPA default parameters; could overestimate risk by less than 1 order of magnitude.
Exposure Duration	Single day, and single day plus once/week until residues gone.	Single day, and single day plus 3 times/week until residues gone.	Exposure on day 1, 30, and 90.	Not specified.	DNR factors reasonable for recreational exposure; DNR factors could underestimate risk from residental exposure by less than 1 order of magnitude; USDA factors require further clarification.

Table 3. Continued.

Exposure Factor	DNR Reasonable Case	DNR Worst Case	USDA Scenario	USDA Accident	Comments
Dermal Absorption	Based on 2% from study of monkeys; totals 3.5% taking into account varying skin absorption efficiencies in humans.	Based on twice the reasonable case; totals 11% taking into account varying skin absorption efficiencies in humans.	10% for bare skin; 30% over 6-hour period for clothed skin.	Not specified.	DNR and USDA worst case factors likely sufficiently conservative.
Transfer Factor	0.25 mg/m^2/lb a.i., based on 2,4,5-T data from Lavy et al. (1980).	0.5 mg/m^2/lb a.i., based in 2,4,5-T data from Lavy et al. (1980).	1600 cm^2/hour	Not specified.	Need transfer factor data specific to glyphosate; degree to which factor over- or underestimates risk is not known.

scenario, the DNR risk assessment used one half the detection limit as the exposure point concentration. Both risk assessments used the detection limit (0.01 mg/100 cm^2) as a worst case approach. Due to the physical differences between forest vegetation and noxious vegetation, dermal exposure concentrations in the forest could be lower than dermal exposure associated with contacting tall, dense, monotypic stands of noxious vegetation. Also, actual glyphosate concentrations could be higher than the nondetected 2,4,5-T concentrations, in which case, risks could be underestimates. However, the potential degree of underestimation is not known.

The USDA risk assessment also evaluated dermal exposure to a berry picker and dermal exposure from an accidental spray. A berry picker was assumed to contact more glyphosate on vegetation than a hiker. The exposure point concentration for a berry picker was assumed to be the concentration of residue on foliage at any point in time after application. No decay was assumed after initial application; this may tend to overestimate risk. Herbicide residues were estimated based on factors reported by Hoerger and Kenaga (1972). The residues for glyphosate were not listed in the risk assessment and calculations showing the equations used were not provided. Therefore, while the general approach used to calculate foliage concentrations seems reasonable, the specific values used were not provided and therefore cannot be verified. Also, the applicability of these concentrations to dermal contact with noxious vegetation may be questionable, due to the physical differences between forest and noxious vegetation. The tall, dense stands of noxious vegetation may result in higher dermal exposure point concentrations.

The exposure point concentration for the accidental spraying exposure in the USDA risk assessment assumed a worst case application rate of 5 pounds active ingredient per acre for aerial, backpack, and truck-spraying. This application rate is approximately the maximum application rate shown on the Rodeo® label for *Spartina* control, and therefore should not be considered conservative for the purposes of controlling noxious emergent vegetation.

3.2.2.2 Dermal Contact with Vegetation Exposure Assumptions

In this section, the dermal exposure assumptions for the DNR hiker scenario, the USDA hiker scenario, the USDA berry picker scenario, and the USDA accidental spraying scenario are discussed.

DNR Risk Assessment:

In the DNR risk assessment, the skin surface area exposed was considered to be one-half a person's body surface for a reasonable case, and a person's whole body surface for a maximum case. It is reasonable to assume that close to a person's whole body could be exposed if the individual is wearing swimming attire. Half a body surface, which was assumed in the DNR assessment, may underestimate risk if the individual is wearing less clothes. Similarly, risks to an individual wearing more clothes could be overestimated. As with the inhalation route, a person was assumed to weigh 55 kg, which is also conservative

for adults given EPA's standard default of 70 kg (USEPA 1989a), but it is consistent with the skin surface area chosen for evaluation.

Exposure durations (the length of time someone is exposed to a chemical) in the DNR risk assessment varied to represent different types of exposures. The doses from single day exposures were assumed to occur immediately following spraying. In the DNR risk assessment, total exposure for a reasonable scenario was estimated by assuming that a person returns to the forest one day a week until all residues are gone. For the worst case total exposure, a person was assumed to return to the forest three days a week until all residues are gone. For a recreational land use scenario, these exposure durations seem appropriate. However, for a nearby resident who might ignore posted warning signs, exposure under worst case conditions could occur daily, in which case the risk may be underestimated.

In the DNR risk assessment, a first order decay model was used to evaluate total exposure, and a 30-day reasonable and 60-day worst case half life for glyphosate was assumed. The 30 day half life was based on information provided in Newton et al. (1984) and the worst case half life was assumed to be double this value. However, biodegradation is the main mechanism of glyphosate degradation. Biodegradation is dependent upon factors governing microbial activity, and therefore, biodegradation of glyphosate on the surface of a berry or on foliage could occur more slowly than biodegradation of glyphosate in soils. Glyphosate half-lives in soils have been shown to be as long as 70 days (Tooby 1985). Therefore, the half-lives used in the DNR risk assessment may tend to underestimate risk. The extent to which the risk could be underestimated as a result of this parameter is not known.

Reasonable case dermal absorption values in the DNR risk assessment for glyphosate were based on absorptions of 2 percent for monkeys (Monsanto Company 1985b). Worst case assumptions used a dermal absorption of twice that amount (4 percent). These rates are similar to those determined in a more recent study of glyphosate absorption (Wester et al. 1991), and are therefore considered appropriate. The dermal absorption values were adjusted to take into account higher absorption in the face, forehead, scalp, and neck. This adjustment resulted in a reasonable case absorption rate of 3.5 percent and a worst-case absorption rate of 11 percent. This approach is appropriate; varying absorption efficiencies for different parts of the skin are discussed in USEPA Dermal Assessment Guidance (1992a). In addition, the EPA Guidance acknowledges that monkey skin has been shown to be a good model for human skin.

The reasonable and worst case transfer factors (0.25 $mg/m^2/lb$ a.i. and 0.5 $mg/m^2/lb$ a.i., respectively) that were used in the DNR risk assessment were based on the Lavy et al. (1980) study of 2,4,5-T. The potential for these factors to over- or underestimate risk is not known. Additional data specific to glyphosate is needed.

As with the inhalation dose, reasonable and worst case herbicide application rates of 1.5 and 3 lb a.i./acre were used in the DNR risk assessment. These values might be slightly low compared with Rodeo® label application directions for *Spartina* and purple loosestrife.

USDA Risk Assessment:

Dermal exposures resulting from vegetation contact for a hiker and a berry picker and from an accidental spraying were also evaluated in the USDA risk assessment. For the evaluation of vegetation contact for a hiker, 10 percent of the total human surface area was considered to contact the foliage, and 40 percent of the total human surface area was assumed to be clothing contacting foliage. This contact rate appears reasonable, but could underestimate exposure if people wear less clothing. A dermal penetration rate of 10 percent was assumed for bare skin. This rate is likely conservative and is close to the worst case glyphosate absorption rates used in DNR risk assessment after a 2 percent absorption rate is doubled and adjusted for varying skin absorption efficiencies. A penetration rate for clothing was assumed to be 30 percent over a 6-hour period, based on work by Newton and Norris (1981). The actual calculations of dose cannot be verified due to the limited information regarding specific methods used in the USDA risk assessment.

Dermal exposure resulting from contact with foliage while picking berries was evaluated in the USDA risk assessment. Exposure was calculated using the unified field model of Popendorf and Leffingwell (1982) and Popendorf (1985). A detailed description of the model and its inputs was not provided in this risk assessment. Therefore, the doses calculated cannot be directly verified. The model input parameters that were briefly discussed in the USDA risk assessment appear reasonable. Doses were evaluated for exposure on day 1, day 30, and day 90 of herbicide application. A residue transfer coefficient of 1600 cm^2/hour was used to estimate doses to berry pickers. This value was derived from data collected for grape harvesting (Popendorf 1985). A body weight of 50 kg was used, which is conservative, based on comparison to EPA standard default of 70 kg (USEPA 1989a). A dermal absorption factor was not provided in the discussion of exposure, but a 10 percent factor was probably used, based on other discussions in that risk assessment; this value is reasonable.

An accidental spraying was evaluated in the USDA risk assessment by assuming that 2 square feet of human skin is sprayed with the worst case application rate. The person is assumed to weight 50 kg, a value more conservative than EPA standard default parameters. Although not explicitly stated, a dermal penetration rate of 10 percent was likely used. The actual skin surface area exposed to glyphosate may be higher if the individual is wearing less clothes (e.g., bathing suit).

3.2.3 Ingestion of Surface Water

Ingestion of surface water from streams receiving a spray drift was evaluated in both risk assessments. For the control of noxious weeds, glyphosate could be applied directly to water bodies within one-half mile of a potable water intake. The degree to which exposure point concentrations from spray drift approximate exposure point concentrations from direct application within one-half mile of a potable water intake is not known.

It is more likely that water ingestion exposure would occur in areas of purple loosestrife spraying, rather than *Spartina* spraying, because purple loosestrife is a freshwater species, while *Spartina* occurs in the marine environment, where people are less likely to use the water as a drinking water source. The exposure point concentrations and exposure assumptions used in these risk assessments to evaluate exposure via drinking water are discussed below and summarized in Table 4.

3.2.3.1 Water Ingestion Exposure Point Concentrations

DNR Risk Assessment:

In the DNR risk assessment, single-day reasonable case exposure point concentrations were based on stream monitoring data provided by the DNR for 12 samples of glyphosate collected in 1981. No detectable levels of glyphosate were found at a 1 ppb detection limit. Therefore, a concentration of 1 ppb was assumed to be the exposure level; this level also coincided with median observed levels of other herbicides monitored. For the worst case single-day exposure scenario evaluated in the DNR risk assessment, a stream concentration of 92 ppb obtained from Newton et al. (1984) was used for glyphosate, based on application of 1 lb/acre. Converting this value to represent a 3 lb a.i./acre glyphosate application rate would result in a value of 276 ppb.

Total reasonable and worst case exposures to glyphosate in surface water used as drinking water were also calculated in the DNR risk assessment. Reasonable total exposure was expected to be the same as the reasonable single-day exposure. Worst case total exposure was calculated using an initial concentration of 92 ppb, a 48-hour concentration of 19.7 ppb, and exponential decay over a seven day period (after which residue is less than 1 ppb).

More recent data reported by Feng et al. (1990) indicate a maximum concentration of 162 ppb in streams intentionally oversprayed with glyphosate, and Westerdahl and Getsinger (1988) recommend a maximum water concentration of 200 ppb, in which case the value used in the DNR risk assessment appears conservative. However, the concentrations of glyphosate observed in the surface waters of prairie pothole wetlands during emergent vegetation control activities ranged from 140 to 600 ppb 12 hours after applications (Henry 1992). If the worst case 600 ppb glyphosate in prairie potholes was applicable to streams, the exposure point concentration used in the DNR risk assessment may be less than half of the maximum exposure concentration. However, the relationship of this concentration to the concentration that could reach a potable water intake is not known.

USDA Risk Assessment:

In the USDA risk assessment, residues in water were calculated by running an unspecified computer program. The water was assumed to be 6 inches deep, and the herbicide spray was assumed to drift directly downwind to the water body over a minimum buffer distance. The buffer strips were assumed to be 50 feet for aerial spraying and 20 feet for ground

Table 4. Drinking water exposure assumptions and exposure point concentrations

Exposure Factor	DNR Reasonable Case	DNR Worst Case	USDA Scenario	USDA Accident	Comments
Glyphosate Exposure Point Concentration	Single Day: 1 ppb, based on DNR sampling. Total Exposure: same as single day reasonable concentration.	Single Day: 276 ppb, based on data from Newton et al. (1984). Total Exposure: based on single day worst case concentration and exponential decay.	Unspecified computer program assuming 6-inch deep water and buffer strips of 50 feet for aerial spraying and 20 feet for ground spraying.	100 gallons dumped from helicopter or 2,000 gallons dumped from truck into pond or reservoir.	Concentrations based on spray drift; glyphosate could be directly applied to water within 0.5 mile of potable intake; 600 ppb is maximum glyphosate concentration in literature (Henry 1992), indicating that concentrations could be underestimated by 2 orders of magnitude for reasonable case and by less than 1 order of magnitude for worst case; USDA scenario does not supply enough information to evaluate.
Surface Water Consumption Rate	1 l/day	2 l/day	1 l/day	Not specified	If water used as source of domestic water, assumptions for DNR worst case are same as EPA reasonable maximum exposure parameters; but DNR reasonable case and USDA scenarios may underestimate risk by a factor of 2 is water used for drinking water.
Body Weight	55 kg	55 kg	50 kg	50 kg	More conservative (by less than 1 order of magnitude) than EPA default parameters.
Application Rate	1.5 lb a.i./acre	3 lbs a.i./acre	Not specifically identified	Not specifically identified	DNR worst case application rate close to anticipated application rate for Rodeo® in freshwater environment; DNR reasonable case application rate could underestimate risk by less than 1 order of magnitude if Rodeo® label directions followed.

spraying. Given the aquatic nature of *Spartina* and purple loosestrife, these assumptions are not appropriate, because glyphosate would be applied in areas immediately adjacent to and directly into water bodies within one-half mile of a potable water intake. The deposition of applied herbicide was calculated for an application rate of 1 lb/acre for six broadcast spray scenarios. A detailed description of the computer program was not provided and therefore the drift values cannot be duplicated or further evaluated.

Under the accident scenario developed in the USDA risk assessment, a person was assumed to ingest water from a pond or reservoir that was contaminated by a dump of 100 gallons of herbicide mix from a helicopter or 2000 gallons of spray mix from a batch truck. These levels represent the largest amount of herbicide carried in these vehicles for applying herbicides in the Pacific Northwest. The pond was assumed to be 1 acre in area and 4 feet deep and to have no inflow or outflow. The reservoir was assumed to be 16 acres in area and 8 feet deep. This scenario is therefore very conservative and should only apply to worst case accidents under these specific exposure conditions.

3.2.3.2 Water Ingestion Exposure Assumptions

DNR Risk Assessment:

In the DNR risk assessment, it was assumed that a person drinks 1 liter of contaminated water per day under a reasonable case scenario, and 2 liters of contaminated water per day under a worst case scenario. A rate of 2 liters/day is used by EPA as a reasonable maximum estimate of domestic water intake for adults. Therefore, this assumption is considered appropriate. A rate of 1.4 liters/day is recommended by EPA as an average adult value (USEPA 1989a). To the extent that the treated water body is used as a drinking water source, the DNR reasonable case intake rate of 1 liter/day may slightly underestimate risk compared to the average intake rate recommended by EPA. The body weight used in the DNR risk assessment was assumed to be 55 kg, which is more conservative than the EPA default value of 70 kg (USEPA 1989a).

A glyphosate maximum application rate of 3 lbs a.i./acre was used in the DNR risk assessment. If Rodeo® label directions were followed, the application rate could be as high as 5.1 lbs a.i./acre for *Spartina* control and 2.7 lbs a.i./acre for purple loosestrife. It is anticipated that if a person were to drink from a stream that had glyphosate residues, it would more likely be in locations where purple loosestrife grows (i.e., freshwater). Therefore, a maximum application rate for this situation (2.7 lbs a.i./acre if Rodeo® label directions were followed) may be similar to the maximum legal application rate used in the DNR risk assessment.

USDA Risk Assessment:

In the USDA risk assessment, people were assumed to ingest 1 liter of contaminated water from a stream. This value may underestimate risk if the treated water body was also used as a source of domestic water. Although not explicitly stated, it was assumed that an individual weighs 50 kg for these scenarios. These assumptions are very similar to the reasonable case DNR water ingestion assumptions.

3.2.4 Ingestion of Food Containing Glyphosate Residues

People could be exposed to glyphosate residues if they consume garden vegetables, berries, wild meat, fish, or shellfish that have been exposed to glyphosate. Exposures associated with consuming each of these potential food sources except shellfish were evaluated in the DNR and/or the USDA risk assessments. Exposure point concentrations and assumptions associated with each of these food sources are discussed below and are summarized in Table 5.

3.2.4.1 Food Exposure Point Concentrations

Garden Vegetables and Berries:

In the DNR risk assessment, data for 2,4-D residues on wild berries and garden vegetables were used to derive potential levels of glyphosate in vegetation. Concentrations used to calculate reasonable and worst case total exposures were the same as single day exposures, but the number of meals eaten was increased, as discussed in Section 3.2.4.2. According to values reported by Erne and Von Haartmann (1973) and described by Norris (1983), a reasonable concentration on berries is 1.81 mg/kg for a 1 lb a.i./acre application rate. The highest observed concentration of phenoxy herbicides on berries (2.47 mg/kg/lb a.i.) was used as a worst case concentration. For garden vegetables, a 0.061 mg/kg/lb a.e./acre concentration was used based on average concentrations of 2,4-D in shrub crown foliage, and the maximum value of 0.082 mg/kg/lb a.e./acre was used as the worst case concentration. Specific data on glyphosate concentrations on berries and vegetables would provide more accurate concentrations. The degree to which these exposure concentrations underestimate or overestimate risk is unknown.

The methods used to calculate residues on garden vegetables in the USDA risk assessment were the same as those used to calculate residues on berries for dermal uptake. Herbicide residues were estimated based on factors reported by Hoerger and Kenaga (1972). Residues in parts per million based on the application rate in pounds per acre were obtained for various classes of plants, such as berries and leafy vegetables. The residues for glyphosate were not listed in the risk assessment and calculations showing the equations used were not provided. Therefore, while the general approach used to calculate foliage concentrations seems reasonable, the specific values used were not provided and therefore cannot be verified.

Table 5. Food Ingestion Exposure Assumptions and Exposure Point Concentrations.

Exposure Factor	DNR Reasonable Case	DNR Worst Case	USDA Scenario	Comments
Glyphosate Exposure Point Concentration	Vegetables: 0.061 mg/kg/lb a.e./acre, based on 2,4-D data.	Vegetables: 0.082 mg/kg/lb a.e./acre, based on 2,4-D data.	Vegetable and berry residues based on unspecified factors reported by Hoerger and Kenaga (1972).	Specific glyphosate data needed for vegetables, berries, and game animals; DNR fish concentrations could underestimate risk by 2 order of magnitude under the reasonable case and less than 1 order of magnitude in the worst case; not enough information provided to adequately evaluate USDA approach.
	Berries: 1.81 mg/kg for 1 lb a.i./acre application rate, based on 2,4-D data.	Berries: 2.47 mg/kg/lb a.i./acre, based on 2.4-D data.		
	Game Animals: Based on atrazine residues in deer tissue (0.036 mg/kg from a 4 lb a.i./acre application rate).	Game Animals: Based on 2,4-D residues (1.73E-03 mg/kg/lb a.i.).	Game Animals: Unspecified computer program.	
	Fish: Based on 1 ppb glyphosate in buffered streams (DNR 1985).	Fish: Based on glyphosate concentration of 276 ppb derived from Newton et al. (1984).	Fish: Equal to residues calculated for water.	
Body Weight	55 kg	55 kg	50 kg	More conservative (by less than 1 order of magnitude) than EPA default parameters.
Exposure Duration	Single Day: 1 meal Total: 20 meals	Single day: 1 meal Total: 20 meals	1 meal	Reasonable assumptions.
Consumption Rate	Berry: 0.125 kg/serving Vegetable: 0.25 kg/serving Deer, Fish: 0.5 kg/serving	Vegetable or Berry: 0.25 kg/serving Deer, Fish: 0.5 kg/serving	Peas or Berries 0.4 kg Deer, Bird Fish: 0.4 kg/serving	More conservative (by less than 1 order of magnitude) compared with EPA (1989a) values.

Game Animals:

In the DNR risk assessment, the reasonable glyphosate concentrations were based on residues of atrazine in deer tissue reported by Newton and Norris (1968). The atrazine was applied at a rate of 4 lb a.i./acre and the resultant tissue concentration was 0.036 mg/kg. This value is likely applicable to concentrations in deer. This value is conservative in that the atrazine residues in deer tissue are higher than those reported for 2,4,5-T in the same study. Also, atrazine and 2,4,5-T generally have higher bioaccumulation or bioconcentration factors than glyphosate. For example, a U.S. Department of Agriculture (1984) study reported a bioaccumulation factor in fish of five for atrazine and one for glyphosate. Most studies of glyphosate report bioconcentration factors of less than one. The highest reported glyphosate bioconcentration factor is 9.6 for mollusks. Bioconcentration factors for the 2-butoxyethyl ester formulation of 2,4-D range from 162 to 408, and bioconcentration factors for the dimethylamine salt of 2,4-D generally range from one to seven (Ebasco Environmental 1992c). Although no specific glyphosate uptake studies in deer have been conducted, an unpublished report found no detectable residue in muscle or fat of cows or pigs fed diets containing up to 75 ppm glyphosate (Monsanto Company 1984); therefore, use of the atrazine data may be reasonable. Worst case glyphosate concentrations were based on maximum 2,4-D concentrations, which were higher than atrazine concentrations. The extent to which these exposure concentrations over or underestimate risk is unknown.

In the USDA risk assessment, the chemical concentrations in a 150-pound deer and a 0.25 pound game bird, such as a quail, were calculated. These animals were assumed to be exposed to glyphosate via the dermal and oral routes. For the dermal route, spray drift was calculated using an unspecified computer program for six broadcast spray scenarios at a 1 lb/acre application rate. Details of the program were not provided, and therefore, a specific critique of the spray drift concentration cannot be provided. The concentration of herbicide in the game meat was calculated by summing the animal's doses from dermal and oral routes and by assuming that 10 percent of that total dose was retained in the meat. This approach appears reasonable in the absence of specific data regarding glyphosate concentrations in birds or deer. It is conservative in its approach to evaluating multiple exposure routes for the animal. However, the glyphosate application rate is less than the rate specified on the Rodeo® label. A more detailed critique is not possible because of the lack of specific information supplied in the risk assessment.

Fish:

In the DNR risk assessment, for the reasonable single day exposure, a median concentration of 1 ppb glyphosate was assumed to be present in buffered streams (DNR 1985). For the worst case single day exposure, glyphosate was assumed to be as high as that reported by Newton et al. (1984), (0.092 mg/kg/lb a.i times 3 lb = 276 ppb). These values could be low, given the discussion of potential concentrations of glyphosate in surface water provided in Section 3.2.4.1. For example, Heydens (1991) reported maximum concentrations of 600 ppb in surface water. For total exposures, the same exposure point concentrations were

used, but the exposure duration was increased from 1 serving to 20 servings. In the DNR risk assessment, a bioaccumulation factor of 1 for an unspecified fish species was obtained from USDA (1984) data. It is possible that fish could receive additional exposure through consumption of contaminated organisms or by incidental ingestion of sediments while feeding.

In the USDA risk assessment, residues in fish were calculated assuming that the fish lived in and were caught from waters 6 inches deep, directly downwind of a treated site, with a minimum buffer strip of 20 feet for ground-based applications and 50 feet for aerial applications. The concentration in the fish was taken to be equal to the concentration of glyphosate in the water. These assumptions may not be appropriate; the buffer strip may be too large considering *Spartina* and purple loosestrife are aquatic plants.

3.2.4.2 Food Exposure Assumptions

Garden Vegetables and Berries:

For the DNR risk assessment, a 55 kg person was assumed to eat 0.125 kg of berries per serving under the reasonable exposure scenario and 0.25 kg of berries per serving under the worst case exposure scenario. In addition, a 55 kg person was assumed to eat 0.25 kg of vegetables under the reasonable and worst case single day exposures. For total exposures, it was assumed that an individual consumes 20 servings of vegetables and berries harvested and frozen on the day that the herbicide was sprayed. For the USDA risk assessment, a 50 kg individual was assumed to consume 0.4 kg (0.9 lbs) of contaminated berries or peas per serving. Other vegetable consumption rates used in both the DNR and USDA risk assessments compared with those specified in USEPA (1989a) are conservative. For example, the 99th percentile of green pea consumption from a three-day dietary recall study was estimated as 0.113 kg/day (USEPA 1989b), which is lower than (but within 1 order of magnitude) the consumption amount specified here.

Game Animals:

For the DNR risk assessment, a 55 kg person was assumed to eat 0.5 kg of deer in a single day. It was assumed that a person freezes 10 kg (twenty 0.5 kg servings) of deer and eventually eats all the meat. For the USDA risk assessment, herbicide doses to humans were calculated by assuming that people eat 0.4 kg of deer meat or 0.4 kg of bird meat per day (0.9 lb). If one assumes that the hunter would consume the meat at a rate similar to the American consumption of beef, the amounts of deer consumed per day specified in the DNR and USA risk assessments are above the national daily average for high beef consumption populations for 70 kg individuals (0.137 kg/day) (USEPA 1989b). Therefore, these assumptions could slightly overestimate risk (within 1 order of magnitude).

Fish:

For the DNR risk assessment, a 55 kg person was assumed to eat 0.5 kg of fish in a single day. It was assumed that a person could obtain as much as 10 kg (twenty 0.5 kg servings) of fish from a single day's catch and that the fish is frozen, no degradation occurs, and the fish is eventually eaten by one person. For the USDA risk assessment, the dose to a 50-kg human was based on consumption of 0.4 kg of fish. The values are conservative (within 1 order of magnitude); they exceed the 95th percentile for fin fish consumption (0.284 kg/meal) provided in USEPA (1989b).

3.2.5 Multiple Exposure Routes

Scenarios depicting risks associated with exposure via multiple routes were not considered in the DNR risk assessment, except for sensitive subgroups, as discussed in Section 3.2.6 below. However, in the USDA risk assessment, exposures were evaluated for multiple exposure routes. People were assumed to receive simultaneous doses representing the sum of individual exposure routes. While the likelihood of such exposure occurring is not known, this sort of approach is thought to be conservative because a series of upper-bound assumptions about people's activities are added together.

In the USDA risk assessment, exposure from repeated pesticide application is evaluated, assuming that glyphosate is applied once per year for thirty years and that all single application exposure assumptions are valid. This evaluation could also underestimate risks if glyphosate were applied twice annually.

3.2.6 Exposure to Sensitive Subgroups

In order to account for possible adverse effects to sensitive individuals within a subpopulation, the no-observed effects level (NOEL) in animals is reduced by a safety factor of 100 to determine an acceptable daily human intake. This safety factor is considered sufficient to ensure that most people will experience no toxic effects. However, in general, unusually sensitive individuals may experience effects even when the risk characterization results indicate that noncarcinogenic health effects are not anticipated. In the USDA risk assessment, effects on sensitive individuals are qualitatively addressed. In the DNR risk assessment, risks associated with exposure of glyphosate to pregnant women and the elderly are addressed qualitatively, while risks to individuals exposed to glyphosate as children are addressed quantitatively.

In the DNR risk assessment, reasonable and worst case exposure estimates for a one-year old child and a ten-year old child were developed. Specific child exposure assumptions are shown in Table 6. The exposure point concentrations for children were assumed to be the same as those estimated for adults. Thus, the uncertainties associated with these concentrations with respect to noxious weed control have been discussed previously in this chapter. The potential routes of exposure considered for children at age one were inhalation,

Table 6. Child Exposure Assumptions in the DNR Risk Assessment.

Exposure Factor	One-Year Old	Ten-Year Old	Comments
Inhalation Rate	Reasonable: 0.043 m^3/hour Worst Case: 0.188 m^3/hour	Reasonable: 0.227 m^3/hour Worst Case: 0.800 m^3/hour	May be less than an order of magnitude underestimate risk by, based on USEPA (1989b).
Body Weight	9 kg	32 kg	Reasonable, given values specified in USEPA (1989b).
Skin Surface Area	0.40 m^2	1.10 m^2	For 10-year old child, 95th percentile surface area is 1.48 m^2 (USEPA 1992a)
Drinking Water Intake Rate	NC[a]	1 liter/day	Reasonable for child.
Wild Meat Consumption Rate	NC	0.25 kg/day	Greater than twice the daily self consumption rate for 70 kg person (USEPA 1989b).
Fish Consumption Rate	NC	0.25 kg/day	Order of magnitude higher than upper 95th percentile fish consumption rate for 10-19 years olds.
Berry Consumption Rate	NC	0.125 kg/day	Three times higher than reasonable worst caase homegrown fruit consumption (USEPA 1989b).
Vegetable Consumption	0.05 kg/day	0.125 kg/day	Rate for 1-year old represents typical adult homegrown vegetable rate. Rate for 10-year old is well above reasonable worst case value specified in USEPA (1989b).

[a] NC = not considered

terrestrial dermal absorption, and ingestion of garden vegetables. It is possible that one-year olds could be exposed by other exposure routes, as well, such as dermal contact and ingestion of surface water. At age ten, children are assumed to be exposed via inhalation, terrestrial dermal contact, water ingestion, and ingestion of wild meat, fish, berries, and garden vegetables. It is possible that ten-year olds could also be exposed from other routes, such as shellfish consumption and dermal contact with surface water.

The child inhalation rates may be low, given the values specified in EPA (1989b). The body weights, drinking water intake rate, and one-year old skin surface area appear reasonably conservative. The ten-year old skin surface area may be slightly low, but the wild meat, fish, berry, and vegetable consumption rates are conservative compared with the values specified in USEPA (1989b). Child exposures are addressed in more detail in Section 3.3 of this report.

3.2.7 Exposures From Repeated Pesticide Applications

Exposures from repeated pesticide applications were addressed in both risk assessments. In the DNR risk assessment, three scenarios were developed. The first two scenarios were based on the assumption that an individual could be exposed three times in a lifetime; this coincides with data indicating two sprays occur for each harvest rotation of 64 years. The third scenario estimated exposures occurring six times in a lifetime. Glyphosate spraying to control noxious weeds could occur as frequently as twice per year. Therefore, the exposure frequency used in the DNR risk assessment could underestimate risk. Additionally, the first two DNR scenarios assumed exposure to 2,4-D at age one, glyphosate at age 10, atrazine as an adult, and then the risks for these age groups are added together. The third scenario assumed exposure to 2,4-D and picloram at age one, exposure to glyphosate and 2,4-D at age ten, and exposure to atrazine and triclopyr as an adult. Of these herbicides, glyphosate has the lowest cancer slope factor, and therefore, risks will be overestimated on the basis of toxicity values. Chronic exposures are addressed in more detail in Section 3.3 of this report.

3.3 UPDATE OF EXPOSURES BASED ON EPA GUIDANCE

In this section, acute and chronic exposures for all potential exposure routes identified in Table 1 are estimated using EPA Region 10 Guidance (USEPA 1991a). The exposures are then compared to the exposures estimated in the DNR risk assessment. (The DNR risk assessment was chosen because all calculations and assumptions are clearly stated.) Acute exposures are short-term exposures that could result from a single spraying event. Chronic exposures are repeated, long-term exposures that could result from semi-annual spraying events over a number of years.

The updated exposure assumptions are based on EPA reasonable maximum exposure parameters for residential intake of contaminants. These assumptions will therefore result in conservative risk estimates. The exposure point concentrations are based on a variety of sources. Due to the limited data available for glyphosate, 2,4-D concentrations were used as

surrogates for some exposure routes. For acute exposure associated with these routes, the uncertainty associated with use of 2,4-D is not known. However, for chronic exposures, all exposure point concentrations could be overestimates because no degradation of glyphosate is assumed. All acute and chronic exposure assumptions are presented in Table 7; the exposure point concentrations are shown in Table 8; and the acute and chronic exposure equations are listed in Table 9. The acute and chronic intake rates associated with non-carcinogenic effects and the chronic intake rates estimated for carcinogenic effects are shown in Table 10.

The acute and chronic intake rates estimated in this section are compared with worst case intake rates estimated in the DNR risk assessment in Table 10. In general, EPA-based acute intake rates are slightly different from DNR acute intake rates, due to differences in exposure point concentrations, body weight, and intake rates. EPA chronic daily intake rates are estimates based on a 30-year exposure duration averaged over the 30-year exposure duration for noncarcinogenic and over a 70-year lifetime for carcinogens. The chronic intake rates are 0.956 times the acute rates because the exposure is averaged for 350 out of 365 days per year. DNR chronic exposures are estimates of total exposure (not a daily intake rate) and thus are not directly comparable to EPA chronic daily intake rates.

Although exposures to children were considered in the DNR risk assessment, the applicability of the assumptions to exposures associated with noxious aquatic weed control is questionable. Therefore, child intake rates for acute and chronic noncarcinogenic effects are estimated below. Assumptions and intake rates for exposures to children are summarized in Table 11.

Acute child intake rates are estimated to range from 2 to 9 times the rate of adult intake due to the differences in body weights and intake rates between children and adults. Like adult chronic intake rates for noncarcinogens, child chronic exposure rates will be 0.956 times the child acute exposure rate to account for exposures 350 days out of 365 days per year. Child intake rates for carcinogens were not calculated because adult intake rates are more conservative. Adult chronic daily intake rates for carcinogenic effects pro-rate a 30-year exposure duration over a 70-year lifetime for carcinogenics while child intake rates pro-rate a 6-year exposure over a 70-year lifetime. In Chapter 5 of this report, the adult and child intake rates are used to characterize the risks associated with these exposures to glyphosate.

Table 7. EPA Acute and Chronic Assumptions Based on Adult Residential Reasonable Maximum Exposure[a].

Exposure Route	Intake Rate (RME)	Body Weight (Avg.)	Dermal-Specific Assumptions[b] (RME)	Acute Exposure Duration	Chronic Exposure Frequency (RME)	Chronic Exposure Duration (RME)	Chronic Averaging Time[c]
Inhalation	15 m^3/day-child 20 m^3/day-adult	15 kg-child 70 kg-adult	NA	1 day	350 days/year	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years
Dermal Contact with Vegetation	NA	15 kg-child 70 kg-adult	Skin Surface Area: 8,023 cm^2-child; 5,800 cm^2-adult Dermal Absorption: 11% Adherence Rate: 1 mg/cm^2event	1 day	350 days/year	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years
Dermal Contact with Surface Water	NA	70 kg	Skin Surface Area: 8,023 cm^2-child 23,000 cm^2-adult Permeability Coefficient (kp): $1x10^{-5}$ cm/hour Conversion Factor: 1 liter/1,000 cm^3	1 day	1 hour/event 1 event/day 150 days/year	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years
Ingestion of Surface Water	1 liter/day-child 2 liters/day-adult	15 kg-child 70 kg-adult	NA	1 day	350 days/year	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years Care: 365 days/year, 70 years
Ingestion of Wild Meat, Fish, Shellfish	0.25 kg/meal-child[d] 0.5 kg/meal-adult	15 kg-child 70 kg-adult	NA	1 meal	260 meals/year[e]	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years Care: 365 days/year, 70 years

Table 7. Continued.

Exposure Route	Intake Rate (RME)	Body Weight (Avg.)	Dermal-Specific Assumptions[b] (RME)	Acute Exposure Duration	Chronic Exposure Frequency (RME)	Chronic Exposure Duration (RME)	Chronic Averaging Time[c]
Ingestion of Berries, Garden Vegetables	0.125 kg/meal[d] 0.25 kg/meal	15 kg-child 70 kg-adult	NA	1 meal	260 meals/year[e]	6 years-child 30 years-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years Care: 365 days/year, 70 years
Ingestion of Soil	200 mg/day-child 100 mg/day-adult	15 kg-child 70 kg-adult	NA	1 day	350 days/year	6 years-child 30 years (24 as adult and 6 as child)-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years Care: 365 days/year, 70 years
Ingestion of Sediments	200 mg/day-child 100 mg/day-adult	15 kg-child 70 kg-adult	NA	1 day	350 days/year	6 years-child 30 years (24 as adult and 6 as child)-adult	NC: 365 days/year, 30 years (adult), 6 years (child) Carc: 365 days/year, 70 years Care: 365 days/year, 70 years

[a] Based on USEPA Region 10 Supplemental Risk Assessment Guidance for Suerfund (1991a), except where otherwise noted.
[b] Source: USEPA (1992a, 1989a, 1989b).
[c] "NC" = noncarcinogenic effects; "Carc" - carcinogenic effects.
[d] Professional Judgement: Slightly more conservative than EPA parameters; coincides with DNR use assessment parameters.
[e] Professional Judgement: Corrsepondence to 5 meals per week.

Table 8. Environmental Concentrations Used to Calculate Acute and Chronic Exposures Using EPA Guidance.

Exposure Rate	Environmental Concentration	Basis	Comments
Inhalation	0.014 mg/m^3	2,4-D concentration of 0.0056 mg/m^3/2 lb a.i./acre (Lavy et al. 1980) corrected for 5 lb a.i./acre glyphosate application rate.	Based on 2,4-D data used in DNR risk assessment; glyphosate data needed.
Dermal Contact with Vegetation	12.35 mg/kg	2,4-D concentration of 2.47 mg/kg/lb a.i. (Siltanen et al. 1981) corrected for 5 lb a.i./acre glyphosate application rate.	Based on 2,4-D data used in DNR risk assessment for berry ingestion; glyphosate data needed.
Dermal Contact with Surface Water	0.6 mg/L	Maximum surface water glyphosate concentration reported (Henry 1992) for prairie pothole wetlands.	Maximum surface water concentration in literature.
Ingestion of Surface Water	0.6 mg/L	Maximum surface water glyphosate concentration reported (Henry 1992) for prairie pothole wetlands.	Maximum surface water concentration in literature.
Ingestion of Fish	0.6 mg/kg	Based on maximum surface water glyphosate concentrations reported by Henry (1992), corrected for bioaccumulation factor of 1.	Use of maximum concentration and bioaccumulation factor of 1 likely conservative.
Ingestion of Shellfish	2.4 mg/kg	Based on Heydens (1991) report of mollusks exposed for 35 days to an initial water concentration of 0.54 mg/L glyphosate, after 42-day depuration period.	Most conservative shellfish concentration in literature.

Table 8. Continued.

Exposure Rate	Environmental Concentration	Basis	Comments
Ingestion of Wild Meat	0.95 mg/kg	Based on 2,4-D concentration in cattle fed 2,4-D; converted to equivalent concentration in deer feeding for 28 days on foliage with glyphosate residues; assumes 5 lb a.i./acre application rate.	Based on 2,4-D data in DNR risk assessment; glyphosate data needed.
Ingestion of Berries	12.35 mg/kg	2,4-D concentration of 2.47 mg/kg a.i. (Siltanen et al. 1981) corrected for 5 lb a.i./acre glyphosate application rate.	Based on 2,4-D data used in DNR risk assessment; glyphosate data needed.
Ingestion of Garden Vegetables	0.41 mg/kg	2,4-D concentration estimated from 0.082 mg/kg a.e. (DNR risk assessment) corrected for 5 lb a.i./acre glyphosate application rate.	Based on 2,4-D data used in DNR risk assessment; glyphosate data needed.
Ingestion of Soil	1,953 mg/kg	Based on Roundup™ concentration at day 0 of 707 mg/Kg (Roy et al. 1989) applied at 1.8 lb/acre and corrected for 5 lb/acre application rate.	Upper end of glyphosate concentrations reported in literature.
Ingestion of Sediments	6.8 mg/kg	Based on sediment data reported by Ferg et al. (1990) in oversprayed stream on Vancouver Island, B.C.	Upper end of glyphosate concentrations reported in literature.

Table 9. Equations Used for Estimating Intake Rates

EXPOSURE ROUTE	EQUATION	PARAMETER DEFINITIONS
All Routes, Acute	$Intake = \frac{EC \times IR \times CF}{BW}$	EC = Exposure Point Concentration (mg/kg or mg/m^3) IR[a] = Intake Rate (mg/day or m^3/day) BW = Body Weight (kg) CF = Conversion Factor, if needed (10^{-6}kg/mg)
Inhalation, Chronic	$CDI = \frac{CA \times IR \times EF \times ED}{BW \times AT}$	CA = Concentraction Main (mg/m^3) IR = Inhalation Rate (m^3/day) EF = Exposure Frequency (days/year) ED = Exposure Duration (years) BW = Body Weight (kg) AT = Averaging Time (days/year x years)
Dermal Contact with Surface Water, Chronic	$CDI = \frac{CW \times SA \times PC \times EF \times ED \times CF}{BW \times AT}$	CW = Concentration in Water (mg/L) SA = Skin Surface Area (cm^2) PC = Permeability Coefficient (cm/hr) EF = Exposure Frequency (hr/year) ED = Exposure Duration (years) CF = Conversion Factor (L/cm^3) BW = Body Weight (kg) AT = Averaging Time (days/year)
Dermal Contact with Soil, Chronic	$DAD = \frac{CS \times AB \times CF \times AF \times EF \times ED \times A}{BW \times AT}$	CS = Concentration in Soil (mg/kg) AB = Fraction of Concentration Absorbed (unitless) CF = Unit Conversion factor (10^{-6} kg/mg) AF = Adherence Factor (mg/cm^2 - event) EF = Exposure Frequency (days/year) ED = Exposure Duration (years) A = Skin Surface Area (cm^2) BW = Body Weight (kg) AT = Averaging Time (days/year x years)
Surface Water Ingestion, Chronic	$CDI = \frac{CW \times IR \times EF \times ED}{BW \times AT}$	CW = Concentration in Water (mg/liter) IR = Ingestion Rate (liters/day) EF = Exposure Frequency (days/year) ED = Exposure Duration (years) BW = Body Weight (kg) AT = Averaging Time (days/year x years)

Table 9. Continued.

EXPOSURE ROUTE	EQUATION	PARAMETER DEFINITIONS
Ingestion of Wild Meat, Fish, Shellfish, Berries, Garden Vegetables; Chronic	$CDI = \frac{CF \times IR \times EF \times ED}{BW \times AT}$	CF = Concentration in food (mg/kg) IR = Ingestion Rate (kg/meal) EF = Exposure Frequency (meals/year) ED = Exposure Duration (years) BW = Body Weight (kg) AT = Averaging Time (days/year x years)
Soil/Sediment Ingestion, Chronic	Adult: $CDI = CS \times CF \times [\frac{(IR_c \times EF \times ED_c)}{BW_c \times AT} + \frac{(IR_a \times EF \times ED_a)}{BW_a \times AT}/AT_a]$ Child: $CDI = \frac{CS \times AB \times IR_c \times CF \times EF \times ED_c}{BW_c \times AT}$	CS = Concentration in Soil/Sediment (mg/kg) CF = Unit Conversion factor (10^{-6} kg/mg) IR_c = Child Ingestion Rate (mg/day) IR_a = Adult Ingestion Rate (mg/day) EF = Exposure Frequency (days/year) ED_c = Child Exposure Duration (years) ED_a = Adult Exposure Duration (years) BW_c = Child Body Weight (kg) BW_a = Adult Body Weight (kg) AT = Averaging Time (days/year) AT_a = Adult Averaging Time (days/year)

[a] For dermal contact with vegetation, IR is substituted with: (surface area cm^2) x (adherence rate mg/cm^2-event) x (absorption factor). For aquatic dermal contact, IR is substituted with surface area cm^2) x (permeability coefficent cm/hour) x (conversion factor liter/cm^3).

Table 10. EPA Adult Acute and Chronic Intake Rates.

Exposure Route	Acute Intake Rate (mg/kg/day)		Chronic Intake Rate (mg/kg/day) Noncarcinogenic Effects		Chronic Intake Rate [b] Carcinogenic Effects		Comments
	EPA	DNR	EPA	DNR	EPA (mg/kg/day)	DNR (mg/kg)	
Inhalation	4.0 E-03	6.6 E-03	3.8 E-03	NA	1.7 E-03	1.7 E-02	For all routes:
Dermal Contact with Vegetation	1.1 E-04	4.8 E-03	1.1 E-04	NA	4.7 E-05	1.8 E-01	Acute intake rates using EPA guidance are different from DNR acute intakes, due to differences in exposure concentrations, body weight, and contaminant ingestion, inhalation, and/or dermal contact rate assumptions.
Dermal Contact with Surface Water	2.0 E-06	NA	8.1 E-07	NA	2.4 E-05	NA	
Surface Water Ingestion	1.7E-02	1.0 E-02	1.6 E-02	NA	7.1 E-03	1.9 E-02	
Fish Ingestion	4.3 E-03	2.5 E-03	4.1 E-03	NA	1.8 E-03	5.0 E-02	
Shellfish Ingestion	1.7 E-02	NA	1.6 E-02	NA	7.1 E-03	NA	
Wild Meat Ingestion	6.8 E-03	5.2 E-03	6.5 E-03	NA	2.8 E-03	2.9 E-02	EPA chronic intake rates for carcinogenic effects are based on daily intake for 30 years averaged over a 70-year lifetime. DNR reported total intake estimate for single spray event. The DNR total intake values and EPA chronic intake rates for carcinogenic effects are not directly comparable.
Berry Ingestion	4.4 E-02	3.4 E-02	4.2 E-02	NA	1.8 E-02	6.7 E-01	
Garden Vegetable Ingestion	1.4 E-03	1.1 E-02	1.4 E-03	NA	6.0 E-04	2.2 E-02	
Soil Ingestion	2.8 E-03	NA	7.1 E-03	NA	3.1 E-03	NA	
Sediment Ingestion	9.7 E-06	NA	2.5 E-05	NA	1.1 E-06	NA	

[a] EPA acute and chronic exposures based on EPA Region 10 Supplemental Risk Assessment Guidance (1991a). EPA exposures based on adult residential reasonable maximum assumptions. DNR exposures based on worst case single day acute exposures.

[b] DNR chronic exposures for carcinogenic effects based on worst case total exposure to single spray event (i.e, initial concentration plus exposure as glyphosate decays). This value is not a daily intake rate, but rather, a total intake estimate.

Table 11. EPA Child Acute and Chronic Intake Rates[1]

Exposure Route	Acute Intake Rates (mg/kg/day)	Chronic Intake Rates (mg/kg/day) Noncarcinogenic Effects
Inhalation	1.4 E-02	1.3 E-02
Dermal Contact with Vegetation	7.2 E-04	6.8 E-04
Dermal Contact with Surface Water	3.2 E-06	1.6 E-05
Surface Water Ingestion	3.9 E-02	3.7 E-02
Fish Ingestion	9.9 E-03	9.5 E-03
Shellfish Ingestion	3.9 E-02	3.7 E-02
Wild Meat Ingestion	1.6 E-02	1.5 E-02
Berry Ingestion	1.0 E-01	9.7 E-02
Garden Vegetable Ingestion	3.2 E-03	3.1 E-03
Soil Ingestion	2.6 E-02	2.5 E-02
Sediment Ingestion	9.1 E-05	8.7 E-05

[1] EPA acute and chronic exposures based on EPA Region 10 Supplemental Risk Assessment Guidance (1991a). EPA exposures based on child residential reasonable maximum assumptions.

4.0 TOXICITY REVIEW

In Section 4.1 of this chapter, the toxicity of glyphosate is reviewed. In Section 4.2, the toxicities associated with surfactants, glyphosate degradation products and impurities, and other herbicides or pesticides used in areas where glyphosate may be applied are addressed.

4.1 TOXICITY REVIEW

In this section, a summary review of the potential carcinogenic and noncarcinogenic effects associated with human exposure to glyphosate is provided. This section includes information presented in the DNR and USDA risk assessments, as well as updated information resulting from more recent toxicity studies.

4.1.1 Carcinogenic Effects

Available oncogenicity data for glyphosate include one chronic study in mice and two chronic studies in rats. The chronic feeding/oncogenicity study in mice tested dosages of 1000, 5000, and 30,000 ppm glyphosate for 24 months. On the basis of increased incidence of renal tumors in mice, glyphosate was originally classified as a Group C chemical, or possible human carcinogen. However, following an independent review of the slides, the classification was changed to Group D (not classifiable due to inadequate evidence of carcinogenicity), because no statistically significant differences between renal tubule adenomas in the control mice and the dosed mice were observed (USEPA 1992a, Monsanto Company 1991).

In the two rat studies, carcinogenic effects were not observed. In the earlier rat study, (a 26-month feeding study), male and female rats were dosed up to 31 mg/kg/day and 34 mg/kg/day, respectively. No treatment-related carcinogenic effects were observed (Monsanto Company 1991). However, this study needed to be repeated because a maximally tolerated dose (MTD) that might produce additional tumors was not reached (USEPA 1992d). In the more recent study, completed in 1990, rats were dosed with glyphosate at dietary levels of 0, 2000, 8000, and 20000 ppm for 24 months. Results indicated reduced body weight gains in females and cataracts in males at the high dose levels, but no treatment-related carcinogenic effects were found (Monsanto Company 1991, EPA 1992d). Based on these results, glyphosate has been reclassified as a Group E chemical (evidence of noncarcinogenicity for humans) (USEPA 1992a).

4.1.2 Noncarcinogenic Effects

Unlike carcinogenic effects, noncarcinogenic health effects are thought to have a dose threshold, below which adverse health effects are not expected to occur. This level is known as a "no observed effects level" or "NOEL". The lowest level at which effects are observed is termed the "lowest effect level" or "LEL". Different threshold levels may exist for various critical effects. In this section, the toxicity associated with acute, subacute, systemic, developmental, reproductive, and mutagenic effects are discussed.

4.1.2.1 Acute and Subacute Effects

Intraperitoneal:

Daily intraperitoneal injections of 15, 30, 45, or 60 mg/kg in rats for 28 days resulted in reduced daily body weight gain, decreased blood hemoglobin, red blood cell count and hematocrit values, and elevated levels of serum glutamic-pyruvic transaminase and leucineamino peptidase during the study period (USEPA 1992d).

Dermal:

No dermal irritation was observed with technical glyphosate (99 percent pure; 0.5 ml of 25 percent strength solution) and the isoproplyamine salt of glyphosate (0.5 ml of undiluted material) applied to the intact and abraded skin of rabbits for 24 hours (FAO 1986). A slight degree of dermal irritation was observed at the site of application in rabbits receiving 5,000 mg/kg/day dermal doses of glyphosate for 6 hours/day, 5 days/week for 3 weeks.

Eye Irritation:

Technical glyphosate (99 percent pure; 0.1 ml of 25 percent strength solution) instilled into the eye of rabbits produced conjunctival redness, chemosis, and corneal opacity/ulceration. All eyes were normal within 7 days (FAO 1986). Under the same conditions, the isopropylamine salt produced no irritation (FAO 1986).

Inhalation:

In an acute inhalation study in rats the four hour nominal LC_{50} was 25 mg Roundup™/L of air (Monsanto 1984). A second four-hour study using a 2 percent vol/vol spray resulted in a nominal concentration of 4.89 mg solution/L air with no signs of toxicity in the rats (Monsanto 1984). A subchronic inhalation study in rats exposed to aerosols of up to 0.36 mg aqueous Roundup/L of air showed only mild nasal irritation.

Oral:

In a 90-day feeding study with rats and dogs with doses of glyphosate up to 100 mg/kg/day and 60 mg/kg/day, respectively, no adverse effects on body weight, behavior, mortality, hematology, blood chemistry, or urinalysis were observed. Similarly, no adverse effects were observed in another 90-day feeding study in rats dosed at levels up to 1000 mg/kg/day (USEPA 1992c).

Glyphosate was administered for 3 months at dietary levels of 0, 5000, 10000, and 50000 ppm to mice and 0, 1000, 5000, and 20000 ppm to rats. Decreased body weight gains were observed in the high-dose group mice. No treatment-related effects in pathologic or

histopathologic evaluations were observed. The no-effect level was considered to be 10000 ppm in mice and 20000 ppm in rats (Monsanto Company 1991).

An oral LD_{50} in rats is 5,600 mg/kg, and a dermal LD_{50} in rabbits exceeds 5,000 mg/kg (USEPA 1992c). Acute oral LD_{50} values of 4,873 mg/kg in the rat and 1,568 mg/kg in the mouse were reported by Bababunmi et al. (1978), as reported in USEPA (1992c).

In a recent study conducted in China, Talbot et al. (1991) evaluated 93 cases of acute poisoning in humans due to intentional ingestion of herbicides containing glyphosate and surfactant (Roundup™). Seven deaths were reported, and the average amount non-survivors ingested was 184 +/- 70 ml (1,260 mg a.i./kg for 70 kg person). Deaths occurred after the victim exhibited hypotension, unresponsiveness to intravenous fluids or vasopressor drugs, and sometimes pulmonary edema, in the presence of normal central venous pressure. Some patients were reported to have ingested as much as 500 ml and exhibited only mild to moderate symptoms. Accidental ingestion resulted in erosion of the gastrointestinal tract, sore throat, dysphagia, and gastrointestinal hemorrhage (Talbot et al. 1991). The portion of these effects attributable to the surfactant in Roundup™ is not known.

4.1.2.2 Reproductive, Teratogenic, and Mutagenic Effects

In a three-generation rat study, dietary concentrations of glyphosate were administered at dose levels of 0, 3, 10, and 30 mg/kg/day. No treatment-related effects on fertility were noted, and no systemic effects in adult rats were apparent. Males from the F3b mating of the high dose group (30 mg/kg/day) showed an increase in the incidence of unilateral renal tubular dilation. Since the animals were exposed to glyphosate both in utero and as pups, these effects were classified as systemic rather than teratogenic. The systemic NOEL for this study is 10 mg/kg/day and the systemic LEL is 30 mg/kg/day based on renal effects observed. The authors concluded that the highest dose tested (30 mg/kg/day) had no adverse reproductive effects (EPA 1992d, EPA 1992b).

In a 2-year study, rats were fed glyphosate at dietary levels of 1, 30, 100, and 300 ppm. No effects on clinical signs, body weights, mortality, clinical pathology, or organ pathology were apparent. The NOEL for systemic effects is 300 ppm (31 mg/kg/day for males; 34 mg/kg/day for females), the highest dose tested (EPA 1992d).

A 1-year feeding study in beagle dogs dosed at 0, 20, 100, and 500 mg/kg/day found a decrease in absolute and relative pituitary weights in mid- and high dose male dogs. Based on these findings, the NOEL and LEL for systemic toxicity are 20 and 100 mg/kg/day (EPA 1992d).

Groups of pregnant rats were dosed orally by gavage at 0, 300, 1000, and 3500 mg/kg/day on days 6 through 19 of gestation. In the high dose group, mean maternal body weight gain was reduced, and a statistically significant increase in the mean number of early resorptions resulted in a slight increase in mean postimplantation loss. A statistically significant decrease

in the mean number of total implantations, viable fetuses, and mean fetal body weight, and a slight decrease in the mean number of corpora lutea was also noted in the high dose group. In addition, an increase in the number of litters and fetuses with unossified sternebrae was noted in the high dose group. The NOEL and LEL for maternal toxicity are 1000 and 3500 mg/kg/day, respectively. This NOEL and LEL also apply to developmental toxicity (EPA 1992d, Monsanto Company 1991).

Developmental toxicity was also evaluated in pregnant rabbits dosed orally by gavage at 0, 75, 175, and 350 mg/kg/day. An increase in the incidence of soft stool and diarrhea was noted in the 175 mg/kg/day group and a definite increase in these signs and nasal discharge were noted in the 350 mg/kg/day group. The NOEL and LEL for maternal toxicity are 175 and 350 mg/kg/day, respectively. Although structural malformations were observed in two fetuses in the 175 mg/kg/day dose group and one fetus in the 350 mg/kg/day dose group, the incidences were not statistically significantly greater than the control. The NOEL for developmental effects is equal to or greater than 350 mg/kg/day, because no dose-related developmental toxicity effects were noted at any dose tested (EPA 1992d, Monsanto Company 1991).

Glyphosate was not mutagenic or genotoxic for Salmonella, E. coli, or Chinese hamster ovary cells. In addition, no evidence of mutagenicity was observed in DNA repair assays in Bacillus subtilis and hepatocyte cultures (EPA 1992d, Monsanto Company 1991).

4.1.3 Toxicity Parameters Used to Evaluate Glyphosate

To characterize risks, toxicity factors are compared with estimates of chemical intake. In the DNR and USDA risk assessments, the glyphosate cancer slope factor was developed, based on the rate of renal tumor formation in male mice in the 24-month feeding study. The upper 95 percent limit of the cancer potency of glyphosate calculated from the kidney tumor data using a multistage model was 2.6×10^{-5} $(mg/kg/day)^{-1}$. This approach is highly conservative; two reviewing pathologists judged that the renal tumors were not treatment-related. Additionally, EPA has recently reclassified glyphosate in Group E (evidence of non-carcinogenicity in humans). EPA guidance suggests that chronic and acute risks associated with exposure to chemicals classified as Groups D and E need not be evaluated in a quantitative risk assessment (EPA 1989a).

In the DNR risk assessment, a NOEL of 10 mg/kg/day was selected for use in evaluating systemic effects associated with glyphosate exposure, and a NOEL of 30 mg/kg/day was used to evaluate reproductive effects. These values are based on the three-generation rat study described under "Reproductive Effects", above. In addition, a NOEL of 350 mg/kg/day for fetotoxic effects and a NOEL of 75 mg/kg/day for teratogenic effects were also used for evaluating noncarcinogenic effects of glyphosate. Use of these values to evaluate the potential for noncarcinogenic health effects is appropriate. In the USDA risk assessment, a systemic NOEL of 31 mg/kg/day and a reproductive/teratological NOEL of 10 mg/kg/day

were used. This conclusion was apparently based on their interpretation of the renal tubular dilation in the three-generation rat study as a teratogenic rather than systemic effect.

To estimate risks using updated EPA guidance, acute exposures were evaluated using a NOEL of 10 mg/kg/day. Chronic noncarcinogenic exposures were evaluated using an EPA reference dose of 0.1 mg/kg/day. A reference dose is an estimate of a lifetime daily dose to humans that is likely to be without appreciable deleterious noncarcinogenic effects. (EPA (1992d) divided the 10 mg/kg/day NOEL obtained from the three-generation rat study by a safety factor of 100 to estimate the reference dose of 0.1 mg/kg/day.) The cancer slope factor of 2.6×10^{-5}, which was derived in the DNR risk assessment, was used to evaluate cancer risks associated with EPA's exposure assumptions.

In this evaluation, toxicity criteria for evaluating potential risks associated with dermal contact with glyphosate were corrected to account for the dose that is absorbed, because the toxicity criteria are based on the dose administered. The absorbed dose is usually more conservative than the administered dose, because it indicates that a smaller amount of chemical than was administered could cause an adverse health effect. An oral reference dose (RfD) of 0.1 mg/kg/day based on an administered dose was corrected for a 30 percent absorption factor (USEPA 1992a) using the methods specified in EPA (1992a) to obtain chronic RfD of 0.03 mg/kg/day based on an absorbed dose. Similarly, the cancer slope factor specified in the DNR risk assessment was corrected for a 30 percent absorbed dose and estimated at 8.67×10^{-5} $(mg/kg/day)^{-1}$. For the evaluation of acute exposures, the NOEL of 10 mg/kg/day was corrected for a 30 percent absorption factor to obtain a NOEL of 3 mg/kg/day.

4.2 INTERACTIONS WITH OTHER CHEMICALS

In addition to the toxicity associated with exposure to glyphosate, other factors may influence human health if glyphosate were applied to control *Spartina* and purple loosestrife. In this section, the toxicities associated with surfactants, glyphosate degradation products, trace impurities generated in the production of glyphosate, and other herbicides or pesticides that may be used in areas where glyphosate is applied are addressed.

4.2.1 Surfactants

The EPA registration label requires that Rodeo® be used with a surfactant. Surfactants reduce the surface tension of water, thereby increasing the ability of water to "wet" the plants waxy cuticle and allow herbicides to readily enter the plant. The label specifies surfactant concentrations ranging from 0.25 to 0.5 percent of the total spray volume. Currently, 183 surfactants are registered by the Washington State Department of Agriculture (See Appendix A). Any non-ionic surfactant from this list may be used with Rodeo®. However, the majority of Rodeo® applications in Washington State are used with R-11, X-77, and LI-700. R-11 is estimated to be used in 90 to 95% of the applications in Washington (Ebasco Environmental 1992a). R-11 is manufactured by Wilbur Ellis, which is the sole

distributor of Rodeo® in Washington State. Thus, R-11 and Rodeo® are commonly sold together (Mary Gilmore personal communication 1992).

Mammalian oral and dermal acute toxicity data for R-11, X-77, and LI-700 are summarized in Table 12. All three adjuvants are classified as practically nontoxic based on rat and rabbit studies. Although no product-specific data are available, chronic toxicity studies using other alkylphenol ethoxylates administered at doses of 1,000 mg/kg/day over a 2-year period to rats and dogs showed no significant toxicity (Sivak et al. 1980). Both X-77 and LI-700 are rated as system corrosive based on eye irritation in rabbits. No eye irritation rating is provided for R-11. LI-700 is also rated corrosive based on dermal irritation in rabbits.

A recent study by Martinez and Brown (1991) suggests that the surfactant polyoxyethyleneamine (POEA) used with the herbicide Roundup™ has pulmonary toxicity following oral administration. However, when used in combination with glyphosate (as in the commercial herbicide Roundup™), the toxicity is enhanced. While POEA did not produce any significant pulmonary injury or death in rats orally dosed with up to 1.03 g/kg, the Roundup™ combination containing the same amount of POEA produced 100 percent death within 24 hours. This study also cites a recent paper by Tai et al. (1990) in which cardiac depression after Roundup™ injection was considered mostly due to the POEA surfactant and that the glyphosate active ingredient actually opposed this toxic effect. In summary, evidence suggests that polyoxyethyleneamine surfactant may cause pulmonary toxicity, but additional research is needed to determine toxicity to humans.

4.2.2 Degradation Products

Glyphosate metabolized in plants can form aminomethylphosphonic acid (AMPA). AMPA can range as high as 28 percent of the total residue on the plant. No data regarding potential mutagenic, reproductive, oncogenic, or chronic effects of AMPA in animals are available. However, available data indicate that AMPA is irritating to the skin and eye. A 90-day subchronic feeding study indicated urinary bladder irritation (hyperplasia of the cells lining the bladder) in rats treated with 1200 mg/kg/day, the LEL for this study. Epithelial hyperplasia of the renal pelvis was observed in the high dose rats (4800 mg/kg/day). The NOEL for this effect was 400 mg/kg/day (EPA 1986). Results of a rat metabolism study suggest that AMPA is rapidly excreted as the parent compound, and no evidence for bioaccumulation was noted (EPA 1986). Additional information regarding toxicity of AMPA is required in order to more completely address its potential effects in humans.

4.2.3 Trace Impurities

N-nitroso-glyphosate (NNG) is a contaminant in technical glyphosate at levels less than or equal to 0.1 ppm. EPA requires oncogenicity testing of nitroso contaminants only in those cases where concentrations of nitroso compounds exceed 1 ppm. The 1 ppm level appears to have been chosen because it represents a practical level of detection for all types of volatile and non-volatile nitroso contaminants (45 FR 42854, June 25, 1980). A chronic toxicity

Table 12. Toxicity Characteristics Adjuvants Used With Aquatic Herbicides

	R-11	LI-700	X-77
CAS Number	9036-19-5[1/]		
Manufacturer	Wilbur-Ellis	Loveland Ind.	Valent U.S.A.
Application Rate (qt/100 gal)	2	0.5-2	0.25-2
Mammals			
Oral			
Rat	NA	>5000	>5000
Rabbit	5840[2/]	NA	NA
Toxicity Rating	Practically nontoxic	Practically nontoxic	Practically nontoxic
Dermal			
Rat	NA	5000	NA
Rabbit	13000[2/]	NA	>5000
Primary Irritation Index	NA	6.2	4

1/ For octylphenoxypoly(ethoxyethanol)
2/ For isopropyl alcohol

study on NNG in dogs indicated an apparent treatment-related increase in absolute and relative kidney weights and in the blood glucose in high-dose (30 mg/kg/day) females. The NOEL for this apparent effect was 10 mg/kg/day. In a subchronic 90-day study of oral toxicity of NNG in rats, a dose-related decrease in survival, food consumption, and body weight gain was noted in the lowest dose tested (3000 mg/kg/day). For various reasons, the dog and rat studies were judged by EPA to be "supplementary" (i.e., do not fulfill guideline requirements). No acceptable studies for mutagenic or reproductive effects are available at present for NNG. Unlike glyphosate, NNG in rats is rapidly absorbed and excreted via the kidneys. Glyphosate absorption from the gut is poor and the majority of excretion occurs in the feces. EPA is not requiring repeat toxicity studies of NNG because its concentration in glyphosate is less than 1 ppm (USEPA 1986). In order to evaluate the carcinogenic and noncarcinogenic effects of NNG in humans, additional acute, subchronic, and chronic toxicity testing would be needed.

4.2.4 Herbicides/Pesticides

Other herbicides or pesticides could be applied in areas where glyphosate is applied. These include (but are not limited to): carbaryl (trade name Sevin), Bti (Bacillis thuringiensis israilensis), malathion, 2,4-D, metsulfuron (trade name Escort), triclopyr (trade name Garlon 3A), copper, acrolein, xylene, endothall, and fluridone (Diane Dolstad personal communication 1992; Kari Rokstad personal communication 1993). Similar toxic endpoints between chemicals may indicate areas of potential concern if exposure to multiple chemicals were to occur. However, both data and methodology for assessing the additive and synergistic impacts of combined pesticides are lacking. Given these limitations, a review of the major known human health effects was conducted, and areas where additive effects could potentially occur were summarized. A summary of the major known effects and potential interaction between glyphosate and some herbicides are provided below.

As discussed in Section 4.1, potential noncarcinogenic effects associated with glyphosate exposure include slight skin irritation in rabbits, eye irritation, renal tubular dilation, and deaths and GI irritation in accidental human poisonings. Additionally, no reproductive effects in animals and no teratogenic effects have been observed from exposure to glyphosate. The results of one chronic mouse study and two chronic rat studies suggest that glyphosate is not a carcinogen.

Acute humans poisonings from carbaryl have resulted in cholinesterase inhibition and delayed neurotoxicity (Dichoff et al. 1987). Limited information exists on the long-term effects of humans exposed to carbaryl (Branch and Jacqz 1986). There is inadequate evidence of carbaryl carcinogenicity in animals and no human data currently exist (IARC 1987).

BTI is not thought to be a significant human pathogen (de Barjac et al. 1990) but specific polypeptides of BTI did cause cardiac toxicity in mice and GI lesions (Mayes et al. 1989). Mortality in rats was observed only with intraperitoneal injections of high concentrations of BTI.

Maternal toxicity in rats due to exposure to triclopyr has been observed, but no teratogenic effects have been noted (NRC 1977). In rabbits, triclopyr exposure led to decreased maternal weight gain but no teratogenic effects. No reproductive effects were noted in a 3 generation rat study. In workers exposed to triclopyr, increased incidence of cirrhosis was noted (Cook et al. 1987). Triclopyr has also caused skin irritation and nasal and respiratory irritation (Doull et al. 1986, Morgan 1982). Additionally, increased incidence of soft tissue sarcomas and lymphoma in humans was noted (Smith et al. 1984, Hardell et al. 1981).

Malathion is a cholinesterase inhibitor. Cardiac toxicity has been noted from acute exposures to malathion (NIOSH 1976). Large exposures are required to produce symptoms because it is metabolized in liver to an inactive form. Malathion causes irritation of nose, eyes, and skin (Gosselin et al. 1984). It also causes reversible renal dysfunction (Ellenhorn and Barceloux 1988). The elimination half-life of malathion was increased by simultaneous administration of carbaryl in rats (Waldron and Abdel-Rahman 1986). No carcinogenic data for malathion exists for humans, and there is inadequate evidence in animals (IARC 1987).

Exposure to 2,4-D has resulted in skin irritation in rabbits. Renal tubular degeneration and GI and liver pathology has been noted in dogs exposed to 2,4-D. Teratogenic effects and skeletal abnormalities were observed in rats dosed with 2,4-D (Shipp et al. 1986). In humans, peripheral neuropathy has been observed (Labat-Anderson Incorporated 1988). EPA has placed 2,4-D in Weight of Evidence Group D - "not classifiable as to human carcinogenicity."

Since there is so little evidence for human toxicity from glyphosate, it is doubtful that there would be significant additive effects with these other chemicals. However, potential areas where there could be some additive effects between glyphosate and the five other herbicides evaluated in this section would be skin and eye irritation with malathion and triclopyr, and skin irritation with 2,4-D. Renal effects (if the three-generation rat study renal effect could possibly occur in humans) may be possible from a combination of glyphosate, malathion, and/or 2,4-D.

5.0 RISK CHARACTERIZATION REVIEW

In a risk assessment, the estimated rate at which a person incidentally intakes a chemical is compared with information about the toxicity of the chemical to estimate the potential risk to human health. This step is known as the risk characterization. In Section 5.1 of this chapter, the risk characterization methodology used in the DNR and USDA risk assessments is discussed. In Section 5.2, a discussion of the risk characterization results for exposure to glyphosate is provided. This section includes the results of the independent characterization of risks, based on the conservative estimates of acute and chronic exposures calculated in Section 3.3 of this report.

5.1 RISK CHARACTERIZATION METHODOLOGY

Cancer health risks are evaluated separately from noncancer health threats. The methods used in the DNR and USDA risk assessments for assessing cancer and noncancer risks are discussed below.

5.1.1 Cancer Risk Characterization Methods

Evaluation of a Group E chemical for carcinogenic effects is a very conservative approach and is not recommended in EPA risk assessment guidance (USEPA 1989a). EPA has indicated that chronic toxicity data suggest glyphosate is not a human carcinogen.

The cancer risks associated with exposure to glyphosate were obtained by multiplying a person's daily intake rate (i.e., dose) by the chemical's cancer slope factor. A cancer risk is expressed as a probability that a person will develop cancer from exposure to a chemical. For example, a risk of 1×10^{-6} indicates a 1 in 1,000,000 probability that a person could contract cancer due to exposure to a carcinogen. Risk levels of 10^{-4} (1 in 10,000) to 10^{-6} (1 in 1,000,000) are often used as regulatory benchmarks.

The cancer slope factors used in the DNR and USDA assessments (2.56×10^{-5} $(mg/kg/day)^{-1}$) were derived from results of the three-generation rat study discussed in Chapter 4 of this report. For the DNR risk assessment, reasonable cancer estimates were derived using the maximum likelihood estimates from the multistage model. Worst case cancer estimates for the DNR and USDA risk assessments were calculated using the 95 percent upper-bound estimates of the linearized multistage dose response model. Although worst case estimates of risk obtained using this model are thought to be fairly uncertain, use of the 95 percent upper confidence limit is unlikely to underestimate the risk from low exposures, such as those anticipated from pesticide use. EPA considers use of the 95 percent upper confidence limit obtained from the multistage model an appropriate approach for evaluating risks from carcinogen exposure (USEPA 1989a).

In the DNR risk assessment, lifetime public exposures were derived by assuming a person is exposed to pesticides from a single spray event. Additionally, potential carcinogenic effects

associated with repeated exposures to herbicides were evaluated. Risk estimates for adults were multiplied by a factor of 1.3 to reflect equivalent risk from short-term exposure at age 20 to a given total dose, as opposed to an average daily dose over a 70-year lifetime. For children, risks were multiplied by a factor to reflect equivalent risk from short-term exposure at age one and at age 10.

In the USDA risk assessment, lifetime public exposures were derived by assuming a realistic estimate would be exposure from a single pesticide application once per lifetime in each of the public exposure scenarios. The USDA risk assessment also presented risk estimates for one exposure to glyphosate per year for 30 years. Lifetime exposures were averaged over a 70-year lifetime. Unlike the DNR approach, the cancer estimates in the USDA risk assessment were not multiplied by 1.3 to account for a single exposure. While this may lead to a slight underestimation in risk, this factor alone is not expected to appreciably underestimate the risks.

In the evaluation of chronic daily intake rates estimated using EPA guidance, a cancer slope factor of 2.56×10^{-5} $(mg/kg/day)^{-1}$ was used. This slope factor was derived in the DNR risk assessment; no EPA cancer slope factors for glyphosate are available. To evaluate dermal contact risks, the cancer slope factor was corrected to reflect an absorbed dose, as described in Chapter 4.

5.1.2 Noncancer Risk Characterization Methods

The potential for noncarcinogenic effects to occur is evaluated using methods that differ from carcinogenic risk evaluations, because noncarcinogens are thought to have exposure thresholds below which a dose is assumed safe. While both cancer and noncancer risk estimates indicate the potential for adverse health effects to occur, a cancer risk estimate is not directly comparable to a noncancer risk estimate. A cancer risk estimate is expressed as a probability of occurrence, while a noncancer estimate provides a value above or below a threshold level that is assumed safe.

5.1.2.1 Margin of Safety Approach

In the DNR and USDA risk assessments, a margin of safety (MOS) approach was used to determine the potential for noncarcinogenic effects from exposure to glyphosate. The margins of safety were calculated for each exposure route by dividing the NOEL in the most sensitive species by the maximum estimated daily human exposure. The MOS is not a measure of dose, but a factor that denotes the relationship between the maximum daily human exposure estimate and the NOEL. The MOS indicates the number of times lower the estimated human exposure is than the animal NOEL. A MOS of 100 or greater indicates that the estimated human dose is at least 100 times lower than the NOEL. If the MOS is 100 or greater, the risk is generally considered minimal. The factor of 100 is thought to be conservative because it takes into account variations in intraspecies and interspecies sensitivity.

For the DNR risk assessment, margins of safety were calculated assuming an individual is exposed to glyphosate from a single spray event. A NOEL of 10 mg/kg/day was used to assess systemic effects, and a NOEL of 75 mg/kg/day was used to evaluate reproductive, fetoxic, and teratogenic effects.

For the USDA risk assessment, a systemic NOEL of 31 mg/kg/day and a reproductive/teratologic NOEL of 10 mg/kg/day, both derived from a long-term 3-generation rat study, were used. It may be more appropriate to label the systemic NOEL as 10 mg/kg/day and the reproductive/teratologic NOEL as 31 mg/kg/day, as discussed in Chapter 4. If doses exceeded NOELs, they were compared with the LD_{50} for the rat (4,320 mg/kg/day) to evaluate risk of immediate, severe effects including fatalities.

In this assessment, a NOEL of 10 mg/kg/day for systemic effects was used to evaluate acute intake rates for adults and children estimated using EPA Guidance. This value was adjusted to account for absorbed doses for evaluating the dermal routes. This NOEL was used because it is the most conservative NOEL available in the glyphosate toxicity literature.

5.1.2.2 Reference Dose Approach

Noncancer health threats associated with chronic daily intakes are estimated by comparing the daily intake rate (estimated in Section 3.3) with an intake level at which no adverse health effects are expected to occur for a long period of exposure (i.e., the reference dose listed in Chapter 4). Intake rates and reference doses are compared by dividing the intake rate by the reference dose (USEPA 1989a). The resulting value is known as a hazard quotient. If a person's daily intake rate is less than the reference dose (i.e., if the hazard quotient is less than one), the chemical is considered unlikely to pose a noncarcinogenic health hazard to individuals under the given exposure conditions.

5.2 DISCUSSION OF RISK CHARACTERIZATION RESULTS

A summary of the main results from the DNR and USDA risk assessments is provided below in Section 5.2.1. In Section 5.2.2, risk characterization results for acute and chronic intake rates estimated using EPA guidance are presented and compared to the DNR risk assessment results.

5.2.1 DNR and USDA Risk Characterization Results

All cancer risks estimated in the DNR and USDA risk assessments for exposures from a single spray event were less than the risk range of 10^{-4} to 10^{-6}, which is often used by agencies as a regulatory benchmark. Cancer risks calculated in the DNR risk assessment were very small. The largest risk, 9×10^{-10} (9 in ten billion), was estimated for the worst case ingestion of wild berries. Similarly, in the USDA risk assessment, cancer risks for public exposure to glyphosate for all routine (non-accidental) scenarios were also small (less than 10^{-7}). The largest risk to the public from a non-accident exposure scenario was

estimated at 4×10^{-8}, which was due to doses to a berry-picker exposed from multiple pathways after glyphosate is applied over a large area (400 acres) by fixed wing (i.e., a worst case application). The largest risk from an accident scenario (3.3×10^{-7}) was also estimated for a berry-picker exposed to glyphosate from multiple pathways.

In the USDA risk assessment, risks from glyphosate exposure are compared with other common risks. The highest risk level for public exposure (3×10^{-7}) is similar to the risk of death from an animal bite or sting (2×10^{-7}). This risk is less than many common risks, such as the risk of death from lightning, fires, falls, and motor vehicle accidents.

Both risk assessments also indicate little potential for noncarcinogenic health effects associated with public exposure to glyphosate from a single herbicide application. In the DNR risk assessment, all margins of safety corresponding to systemic, reproductive, and teratogenic effects exceeded 100. This indicates that no adverse noncarcinogenic health effects are anticipated under the specific exposure conditions described in Chapter 3 for the DNR risk assessment. In the USDA risk assessment, all margins of safety for all non-accident scenarios are greater than 100, with the exception of the multiple exposure scenario for the berry picker. For this scenario, the margin of safety is 95, based on a NOEL of 10 mg/kg/day. This value is only slightly below 100 and thereby indicates a slight potential for adverse health effects to occur under one multiple exposure scenario.

Margins of safety for accidental spill and spray scenarios evaluated in the USDA risk assessment indicate the potential for adverse systemic and reproductive effects via a variety of single or multiple exposure routes. These accident scenarios represent worst-case spills and accidental sprays, and are not estimates of risk anticipated from a normal spray operation.

Risks from exposure to repeated spray events were also evaluated in the DNR and USDA risk assessments. In the DNR assessment, three repeated exposure scenarios were developed. However, as described in Section 3.2.7, these scenarios are not applicable to the application of glyphosate to control noxious aquatic weeds. The USDA risk assessment reported risks associated with exposure from one glyphosate spray event per year for 30 years. All risks from 30-year exposures were at or below 10^{-7}. Given that glyphosate could be applied twice yearly to control aquatic noxious weeds, the assumption of exposure from one spray event per year could underestimate risk.

Risks to sensitive subgroups were considered qualitatively in the USDA risk assessment. Risks to the elderly and pregnant women were considered qualitatively in the DNR risk assessment, and risks to children were quantified in the DNR risk assessment. According to the USDA risk assessment, risks to sensitive individuals are possible, but because sensitive individuals comprise only a fraction of the population, the risk assessment concludes that the likelihood of exposure of a sensitive individual is low. In the DNR risk assessment, it is concluded that there is no evidence available that indicates the elderly or pregnant women are

at any greater risk of cancer than the general population as a whole as a result of exposure to glyphosate. Risks to children were estimated at 10^{-9} or less.

5.2.2 Risk Characterization Results Using EPA Guidance

After publication of the DNR and USDA risk assessments, EPA issued additional human health risk assessment guidance documents (e.g., USEPA 19989a, 1989b, 1991, 1992a). In these guidance documents, risk assessment methods and default exposure assumptions are recommended. To account for the updated information presented in these guidance documents, acute and chronic daily intake rates were calculated using the updated assumptions to estimate reasonable maximum residential exposures (see Section 3.3). Intake rates were calculated for both adult and child exposures for the following exposure routes:

- inhalation;
- dermal contact with vegetation;
- dermal contact with surface water;
- surface water ingestion;
- fish ingestion;
- shellfish ingestion[1];
- wild meat ingestion;
- berry ingestion;
- garden vegetable ingestion;
- soil ingestion; and
- sediment ingestion.

Of these exposure routes, dermal contact with surface water, shellfish ingestion, soil ingestion, and sediment ingestion were not evaluated in either the DNR or USDA risk assessments. The results of the evaluation are shown in Table 13.

The risk characterization results suggest that no glyphosate intake rates estimated in Section 3.3 are of concern to human health. Margins of safety for acute adult and child exposures are 100 or greater, all hazard quotients are less than 1, and all cancer risks are at or less than 10^{-7}.

[1] Potential contamination of shellfish beds from application of glyphosate and subsequent public consumption is an important exposure route. The U.S. Food and Drug Administration (FDA) typically sets "action levels" for common fish and shellfish contaminants, above which consumption is considered unsafe and the seafood is not allowed to be sold commercially. Glyphosate does not have an action level because it is not considered by the FDA to be a common or expected contaminant. If glyphosate were to contaminate shellfish, the FDA would first determine if the level of contamination poses a potential risk to human health (Tom Piekarski personal communication 1993). The results of this evaluation suggest that consumption of shellfish would not result in significant adverse effects to human health.

Table 13. Risk Characterization Using EPA Guidance[1].

	Margin of Safety[2]			Hazard Quotient[3]		Excess Cancer Risk[4]	
Exposure Route	**EPA, adult**	**EPA, child**	**DNR**	**EPA, adult**	**EPA, child**	**EPA, adult**	**DNR**
Inhalation	2,500	714	1,522	0.038	0.13	4E-08	2.3E-011
Dermal Contact with Vegetation	27,273	4,167	2,070	0.0037	0.0023	4E-09	2.3E-10
Dermal Contact with Surface Water	1,500,000	937,500	NA[5]	0.000027	0.00053	2E-09	NA
Surface Water Ingestion	588	256	990	0.16	0.37	2E-07	2.5E-11
Fish Ingestion	2,326	1,010	3,984	0.041	0.095	5E-08	6.5E-11
Shellfish Ingestion	588	256	NA	0.16	0.37	2E-07	NA
Wild Meat Ingestion	1,471	625	1,927	0.065	0.15	7E-08	2.6E-11
Berry Ingestion	227	100	298	0.42	0.97	5E-07	8.8E-10
Garden Vegetable Ingestion	7,143	3,125	8,928	0.014	0.031	2E-08	2.9E-11
Soil Ingestion	3,571	385	NA	0.71	0.25	8E-08	NA
Sediment Ingestion	1,030,900	109,890	NA	0.00025	0.00087	3E-11	NA

[1] DNR risk assessment results shown for comparison.
[2] Margin of safety = NOEL (10 mg/kg/day) divided by acute intake rate estimated in Section 3.3. An MOS of 100 or greater indicates estimated human intake rate is at least 100 times lower than NOEL.
[3] Hazard Quotient = chronic daily intake rate divided reference dose. Hazard quotient less than 1 indicates noncarcinogenic effects not likely.
[4] Cancer risk = chronic daily intake rate multiplied by cancer slope factor.
[5] NA = not applicable

6.0 SUMMARY AND CONCLUSIONS

In this chapter, the uncertainties associated with the results of the two glyphosate risk assessments are discussed (Section 6.1), additional information needs are listed (Section 6.2), and conclusions are presented regarding potential impacts to public health due to exposure to glyphosate applied to control purple loosestrife and *Spartina* (Section 6.3).

6.1 UNCERTAINTIES

Risk characterization results are estimates of potential risk that have uncertainties associated with them. Uncertainties in a risk assessment can result in overestimations or underestimations of risk. In this section, the uncertainties associated with the DNR and USDA risk assessments and the calculations conducted for this report are addressed, and the applicability of these risk results in approximating the risks associated with glyphosate use to control *Spartina* and purple loosestrife are addressed.

Risk characterizations are estimated by combining site data, assumptions about the ways and extent to which people are exposed, and toxicity values. The characterizations are thus limited by the uncertainties associated with each of these steps of a risk assessment. The uncertainties in the two risk assessments are addressed below.

6.1.1 Site Data

The USDA and DNR risk assessments were written to address herbicide application to forests in Washington and Oregon. Certain assumptions about the size of areas to which herbicides are applied, the methods and application rates of herbicides, and the distinct physical characteristics of the forest have been made in these risk assessments. The degree to which the physical environment differs from what is projected in the risk assessment may influence the applicability of the risk numbers. For example, if a small pond with no inflows or outflows is not present near the application area, the USDA accident scenario depicting a spill into such a pond would be inapplicable.

Spartina occurs in a marine environment and purple loosestrife occurs in a freshwater environment. A number of differences exist between these environments and the forest setting used in the DNR and USDA risk assessments. For example, noxious vegetation grows in tall, dense, monotypic stands that might lead to more dermal exposure than exposure to forest vegetation.

Assumptions have also been made regarding the concentrations of glyphosate in the environment available for human uptake. These exposure point concentrations are discussed further in Section 6.1.2.

6.1.2 Exposure Assumptions

A number of uncertainties typically occur in an exposure assessment. Uncertainties can arise from the types of exposures examined, the points of potential human exposure, the concentrations of glyphosate at the points of human exposure, and the intake assumptions. Each of these factors and how they contribute to the risk estimations are discussed below.

6.1.2.1 Exposure Routes Examined

The selection of exposure routes is a process, often based on best professional judgment, which attempts to identify the harmful exposure scenarios that are most likely to occur. In a risk assessment, it is possible that risks are not calculated for all of the exposures that may occur, possibly causing some underestimation of risk. In the two risk assessments, risks associated with the inhalation, dermal, water ingestion, and food ingestion pathways were considered. Risks associated with soil/sediment ingestion, shellfish consumption, and dermal contact while swimming in treated water were not considered in either risk assessment. To account for this omission, the potential risks from shellfish consumption, dermal contact while swimming, and soil/sediment ingestion were considered in this report. Considering the types of exposures possible, it is probable that the most likely exposure routes were addressed in this report.

6.1.2.2 Human Exposure Assumptions and Concentrations of Chemicals at Points of Exposure

Two more sources of uncertainty in the exposure assessment are the assumptions made regarding the locations where people could be exposed to the contaminants at a site and the concentrations of chemicals at the points of exposure. In this assessment, assumptions are made to indicate the locations where nearby residents and recreationalists could come into contact with glyphosate. Chemical concentrations at the point of potential human contact were modeled or were derived from relevant literature. The uncertainties associated with exposure points, concentrations at points of exposure, and modeling are presented below.

Inhalation Exposure

The degree to which exposure point concentrations used to evaluate inhalation exposure in the DNR risk assessment and used in this evaluation over- or underestimate risk is not known. Data for 2,4-D and 2,4,5-T applied to forests were used as glyphosate exposure point concentrations. However, even if the true glyphosate exposure point concentrations were 10 times the concentration used in the DNR risk assessment, all margins of safety evaluated in the DNR risk assessment would still be a safe level (i.e., above 100) and cancer risks would still be well below 10^{-6}. Similarly, cancer risks, hazard indices, and margins of safety for adults would still be below levels of concern. Margins of safety and hazard indices for children might be of concern if the exposure point concentration were an order of magnitude higher.

The exposure assumptions used in the DNR risk assessment to evaluate inhalation of glyphosate applied to forests are applicable to glyphosate applied to control *Spartina* and purple loosestrife and are generally conservative. The worst case evaluation, for example, assumes a 20 m^3/day breathing rate, which is a reasonable maximum exposure assumption (USEPA 1989a) and averages exposure over a body weight of 55 kg, rather than the standard default of 70 kg (USEPA 1989a). Further, the exposure is assumed to occur until glyphosate decays, thereby capturing the maximum possible length and exposure. A slightly lower glyphosate application rate is used in the DNR risk assessment than is recommended on the Rodeo® label, which could lead to a slight underestimation of risk.

For the intake rates calculated in Section 3.3 of this evaluation, the EPA exposure assumptions used to represent reasonable maximum residential exposures. It is not likely that these assumptions would underestimate risk.

Terrestrial Dermal Exposure

The exposure point concentrations in the DNR and USDA risk assessments were derived from data for 2,4,5-T (Lavy et al. 1980). These data could underestimate the risks for glyphosate because 2,4,5-T was not detected above the detection limit. Similarly, while the USDA conservatively assumed that no decay would occur, the residue concentrations on foliage evaluated for the berry-picker exposure were not provided, and therefore, the degree to which these concentrations underestimate risk is unknown. However, even if all non-accident dermal exposure point concentrations in the USDA and DNR risk assessments were 10 times higher, the margins of safety would still be above 100 (with the exception of the highly unlikely scenario in which 400 acres are sprayed by fixed wing). Similarly, all cancer risks would still be well below 10^{-6}. The USDA accidental spraying scenario assumes a worst case application rate of 5 pounds active ingredient per acre, which exceeds the Rodeo® application rate specified on the label and is, therefore, slightly conservative if label directions are followed.

In this evaluation, data for 2,4-D on berries (obtained from the DNR risk assessment) was used to evaluate risks. The degree to which glyphosate concentrations differ from this amount is not known.

The terrestrial dermal exposure assumptions could underestimate risks for some parameters. For example, the reasonable case assumption that one-half of the body is exposed and available for dermal contact with glyphosate might not be conservative for individuals wearing swimming attire. Glyphosate absorption efficiencies appear reasonable, as does 55 kg body weight. A 30 or 60 day half-life could slightly underestimate risks, given empirical data indicating up to 70-day half-lives in soil (Tooby 1985). The application rate used in the risk assessment is less than that specified on the Rodeo® label and may slightly underestimate the risks. Similarly, a maximum exposure of three times a week may slightly underestimate exposure if a person is exposed daily.

An evaluation of the individual exposure assumptions used in the USDA risk assessment is difficult, given the lack of specific information regarding the modeling conducted to arrive at the estimated human doses. However, the dose could be ten times greater and all non-accident scenarios would still be within acceptable risk levels and margins of safety with the exception of the scenario in which 400 acres are sprayed by fixed wing. In this evaluation, EPA exposure assumptions used to estimate intake rates represent reasonable maximum residential exposures. It is not likely that these assumptions would underestimate risk.

Aquatic Dermal Exposure

The aquatic dermal exposure point concentration was 600 ppb, the highest concentration of glyphosate measured in a surface water body. Aquatic dermal exposure assumptions were also conservative upperbound estimates provided in USEPA (1992a). Because very low risks and hazard quotients under these conservative assumptions were estimated for this scenario, it is thought to not represent a significant human exposure route.

Water Ingestion Exposure

The degree to which exposure point concentrations developed in the DNR and USDA risk assessment for estimating glyphosate doses to people who drink surface water over or underestimate the risk is not known. In these risk assessments, a buffer zone around the surface water was assumed. However, glyphosate could be applied directly to water for the control of noxious vegetation, but application is restricted for locations within one-half mile of a drinking water source. Also, the DNR worst case scenario is the only scenario for which multiple day exposure is evaluated. The degree to which people are exposed beyond seven days will depend on the initial concentration of glyphosate at the drinking water intake location and the rate of glyphosate decay in the surface water. However, even if the true exposure point concentration is an order of magnitude higher, the margins of safety and risks are expected to remain within safe levels (with the exception of the USDA worst case 400 acre fixed wing spraying scenario). In Section 3.3 of this evaluation, a surface water concentration of 0.6 mg/L was assumed for calculating intake rates using EPA assumptions. This is the highest concentration found in the glyphosate literature and is likely conservative, especially because no degradation was assumed.

If people are using the surface water as their source of domestic water, the two liter per day intake assumption used in the DNR risk assessment coincides with EPA's reasonable maximum default value. The one liter per day assumption is slightly below EPA's average case default value of 1.4 liters per day, and could, therefore, slightly underestimate the risk. The 50 to 55 kg body weight is more conservative than the standard EPA default value.

In Section 3.3 of this evaluation, EPA exposure assumptions used to estimate intake rates represent reasonable maximum residential exposures. It is not likely that these assumptions would underestimate risk.

Food Ingestion Exposures

The degree to which garden vegetable, berry, wild game, and fish concentrations specified in the USDA risk assessment may over- or underestimate the risk is not known because specific details regarding the various modeling approaches were not provided. However, in the USDA concentrations of glyphosate in fish may not be accurate, due to the assumptions regarding buffer zones. In the DNR risk assessment, exposure point concentrations were based on values of 2,4-D, 2,4,5-T, and glyphosate reported in the literature for vegetables, wild game, and fish, respectively. The degree to which these concentrations may over- or underestimate the risk is unknown. However, chemical concentrations in fish could be underestimated by a factor of 2.2 if the surface water concentration of glyphosate was 600 ppb, the maximum value reported in the literature. If the concentrations of glyphosate in vegetables, berries, wild game, and fish were increased by a factor of 10, all margins of safety and risks in the DNR risk assessment would be within levels considered safe, with the exception of the worst case wild berry ingestion scenario. Similarly, all margins of safety and risks in the USDA risk assessment for single exposure routes would still be within acceptable levels if exposure point concentrations were increased by a factor of 10, with the exception of the worst case 400-acre fixed wing spraying scenario. The glyphosate concentration of 0.276 mg/kg in fish corrected for bioaccumulation factor of approximately 10 yielded an exposure point concentration of 2.76 mg/kg, which closely approximates the glyphosate concentration of 2.4 mg/kg in marine mollusks exposed to 540 ppb of glyphosate in water (Heydens 1991).

The fish and shellfish exposure concentrations used to estimate intake rates in Section 3.3 are likely conservative. The fish concentration was based on a maximum surface water concentration of 0.600 mg/L and a bioaccumulation factor of 1. The shellfish concentration is the most conservative concentration in the literature. The degree to which the meat, vegetable, or berry concentrations over or underestimate risk is not known because 2,4-D data were used as surrogates to glyphosate.

Exposure assumptions for vegetable, berry, game, fish, and shellfish consumption appear conservative when compared with upperbound percentiles of consumption amounts for these food items, as discussed in Section 3.2.4.2. Also, the food ingestion exposure assumptions are considered conservative in the DNR risk assessment, because an individual is also assumed to be exposed 20 times (i.e., the individual consumes 20 meals of each food item that has been frozen and no glyphosate decay occurs). Overall, it is likely that the food ingestion exposure assumptions overestimate the risks characterized in the DNR and USDA risk assessments.

In Section 3.3 of this evaluation, EPA exposure assumptions used to estimate food intake rates represent reasonable maximum residential exposures. It is not likely that these assumptions would underestimate risk.

Soil and Sediment Ingestion

Soil and sediment exposure point concentrations used to estimate intake rates in Section 3.3 were upper-end glyphosate concentrations reported in the literature, and no degradation was assumed. Therefore, these values likely overestimate risks.

In Section 3.3 of this evaluation, EPA exposure assumptions used to estimate intake rates represent reasonable maximum residential exposures. It is not likely that these assumptions would underestimate risk.

Multiple Exposure Pathways and Accident Scenarios

The multiple exposure and accident scenarios evaluated in the USDA risk assessment could be highly conservative and likely overestimate the risks associated with routine glyphosate applications. Generally, multiple exposure pathways are considered conservative and unlikely because they are quantified by adding together a series of conservative parameters from more than one exposure route to create a scenario that is unlikely to occur.

Repeated Spray Events

The repeated spray event exposure scenarios in the USDA risk assessment assume one spray per year, which could underestimate risks by a factor of two if two spray events occurred each year. The repeated exposure scenarios developed in the DNR risk assessment may not be good indicators of repeated sprayings of glyphosate, because only three sprays per lifetime were assumed and exposure to a number of herbicides were evaluated collectively.

The chronic carcinogenic and noncarcinogenic intake rates estimated in Section 3.3 using EPA guidance are likely over-estimates, because exposure for 350 out of 365 days per year for 30 years is assumed. Also, no glyphosate degradation is assumed.

Sensitive Subgroups

The uncertainties associated with the exposure point concentrations used to evaluate childhood exposures in the DNR risk assessment are the same as those indicated for each exposure route. As discussed in Section 3.2.6, child exposure assumptions vary in conservatism (e.g., food intake rates) or lack thereof (e.g., inhalation rates). The main uncertainty associated with these scenarios, however, is that they assume children are not repeatedly exposed to glyphosate from multiple spray events, a scenario that is quite possible.

In this evaluation, risks for child exposures to glyphosate were characterized for all exposure routes using EPA's reasonable maximum residential exposure assumptions and the same exposure point concentrations used for adults. Use of the reasonable maximum exposure

assumptions is likely to result in conservative risk estimates. However, the degree to which the exposure point concentrations over- or underestimate risk is generally not known.

6.1.3 Toxicological Data and Dose-Response Extrapolations

The availability and quality of toxicological data is another source of uncertainty in the risk assessment. Extrapolation of toxicological data from animal tests is one of the largest sources of uncertainty in risk assessments. There may be important, but unidentified, differences in uptake, metabolism, and distribution of chemicals in the body between the test species and humans. Also, animals are typically administered high doses of a chemical in a standard diet. Humans, on the other hand, may be exposed to much lower doses in a highly variable diet. In these studies, animals, usually laboratory rodents, are exposed daily to the chemical agent for various periods of time up to their 2-year lifetime. Humans have an average 70-year lifetime and may be exposed either intermittently or regularly for an exposure period ranging from hours to a full lifetime.

Toxicological uncertainties are accommodated through use of conservative assumptions in establishing the toxicity criteria. In this evaluation, the uncertainty associated with glyphosate toxicity was approached conservatively. The limited carcinogenicity information available was used to establish a cancer slope factor, which typically was the 95 percent upper confidence limit of the dose response curve. Similarly, the lowest (and thus most conservative) NOEL for the noncarcinogenic effect in question was used to evaluate noncarcinogenic risks. Further, a conservative margin of safety of 100 was used to take into account intraspecies and interspecies variations.

Additional uncertainty is introduced in this analysis because the toxicities of the adjuvants that could be used in combination with the glyphosate have not been fully characterized. Similarly, toxicities associated with the degradation products of glyphosate and the N-nitroso impurities in Rodeo® have not been quantified in this evaluation. Also, interactions between glyphosate and other herbicides applied in locations where glyphosate is applied could enhance or mitigate glyphosate toxicity. However, information concerning these potential interactions was not available.

6.1.4 Combinations of Sources of Uncertainty

Uncertainties from different sources are compounded in the risk assessment. For example, if a person's daily intake rate for a contaminant is combined with a cancer slope factor to determine potential health risks, the uncertainties in the concentration measurements, exposure assumptions, and toxicities will all be expressed in the result. To ensure that human health is adequately protected, many conservative assumptions and approaches that are unlikely to underestimate risk when these factors are combined were incorporated into the two risk assessments.

6.2 INFORMATION NEEDS

While both the DNR and USDA risk assessments are helpful in evaluating potential public health risks associated with exposure to glyphosate, a number of information needs still exist. For example, information needs are present in the exposure assessment portions of the risk assessments. Concentrations of glyphosate at potential points of human exposure for some exposure routes were estimated from other chemicals or by chemical modeling. Actual glyphosate environmental concentrations resulting from spraying noxious aquatic weeds would provide more accurate data for the risk evaluation. Data regarding glyphosate concentrations and degradation in soils, sediments, surface water, fish, shellfish, ambient air, berries, and vegetables would help verify concentrations used in this report or would indicate where exposure concentrations used in this report over- or underestimate risk. Also, more accurate information concerning the activities of populations potentially exposed to glyphosate would result in more accurate risk estimations. To ensure conservatism in the risk estimates, residential reasonable maximum exposure scenarios were evaluated.

Additional toxicity data would also be useful for improving the accuracy of the risk characterization. Data gaps in toxicity information exist for glyphosate. Information is needed to verify or refute the potential carcinogenicity of glyphosate. Information regarding the toxicity of adjuvants used in combination with glyphosate is also needed. Similarly, the potential interactions between glyphosate and other pesticides that could be applied in locations where glyphosate is applied is not known.

6.3 CONCLUSIONS

While the DNR and USDA risk assessments were written to evaluate risks specific to herbicide applications in a forest setting, they do provide information needed to evaluate most potential types of public exposures to glyphosate. To account for new EPA guidance and potential routes of exposure not evaluated in the DNR and USDA risk assessments, acute and chronic exposures based on EPA Guidance were evaluated in this report. A conservative reasonable maximum residential exposure scenario was used in this evaluation (e.g., exposures 350 days per year for 30 years). Also, no glyphosate degradation was assumed, which leads to conservative results. However, in some instances, the degree to which the exposure point concentrations approximate true glyphosate concentrations is unknown, due to lack of data or lack of adequate modeling details. Therefore, some exposure point concentrations may over- or underestimate risks.

The toxicity values used in the risk characterizations are likely conservative. Worst case cancer risks were evaluated using the 95 percent upper confidence limit of the dose response curve, based upon a study where the incidence of renal tumors in rats was not even considered to be treatment related. The lowest NOELs available from toxicity studies conducted for glyphosate were also used in these risk assessments. However, toxicity associated with adjuvants, trace impurities, and degradation products were not addressed

quantitatively in either risk assessment. Also, potential interactions of other herbicides and glyphosate are not known.

The USDA and DNR risk characterizations for non-accident, single-route exposures from single spray events in both risk assessments indicate a very low probability of cancer risks (well below a 10^{-6} risk level), and a very low potential for adverse health effects (most scenarios well above a margin of safety of 100). All margins of safety, hazard quotients, and risks associated with EPA exposure assumptions are also below levels of concern, as shown in Table 14, for adults and children.

Table 14. Summary Risk Characterization Results Based on EPA Guidance[1].

	Margin of Safety[2]		Hazard Quotient[3]		Excess Cancer Risk[4]
Exposure Route	**EPA, adult**	**EPA, child**	**EPA, adult**	**EPA, child**	**EPA, adult**
Inhalation	2,500	714	0.038	0.13	4E-08
Dermal Contact with Vegetation	27,273	4,167	0.0037	0.0023	4E-09
Dermal Contact with Surface Water	1,500,000	937,500	0.000027	0.00053	2E-09
Surface Water Ingestion	588	256	0.16	0.37	2E-07
Fish Ingestion	2,326	1,010	0.041	0.095	5E-08
Shellfish Ingestion	588	256	0.16	0.37	2E-07
Wild Meat Ingestion	1,471	625	0.065	0.15	7E-08
Berry Ingestion	227	100	0.42	0.97	5E-07
Garden Vegetable Ingestion	7,143	3,125	0.014	0.031	2E-08
Soil Ingestion	3,571	385	0.071	0.25	8E-08
Sediment Ingestion	1,030,900	109,890	0.00025	0.00087	3E-11

[1] As discussed in Section 3.3, this independent evaluation was conducted to address exposures to glyphosate applied to control aquatic vegetation. Given the data voids, limitations of these estimates were compensated for by using conservative exposure point concentrations and reasonable maximum residential exposure assumptions listed in EPA Region 10 Guidance (1991).
[2] Margin of safety = NOEL (10 mg/kg/day) divided by acute intake rate estimated in Section 3.3. An MOS of 100 or greater indicates estimated human intake rate is at least 100 times lower than NOEL.
[3] Hazard Quotient = chronic daily intake rate divided reference dose. Hazard quotient less than 1 indicates noncarcinogenic effects not likely.
[4] Cancer risk = chronic daily intake rate multiplied by cancer slope factor.

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APPENDIX A

ENVIRONMENTAL FATE AND TRANSPORT OF GLYPHOSATE

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In this Appendix, aspects of the environmental fate and transport of glyphosate, such as biodegradation and bioaccumulation, are presented. This information is summarized from Element E of this report series (Ebasco Environmental 1992c).

Biodegradation

The degradation of glyphosate occurs aerobically or anaerobically through the action of microorganisms present in soil, water, hydrosoil, and activated sludge (Sprankel et al. 1975b; Quilty and Geoghan 1976; Rueppel et al. 1977; Torstensson and Aamisepp 1977; Balthazar and Hallas 1986). Such microflora typically include bacteria, fungi, algae, and protozoans. Because these organisms are indigenous to water and soil environments, similar degradation processes take place in soil/water and wetland/water habitats where glyphosate use is proposed.

Aqueous biodegradation half-lives of glyphosate in field and laboratory studies are reported to range from 2 to 15 days (Hunter et al. 1984; Sacher 1978) and from 7 to 10 weeks in nonflowing natural freshwater ponds and wetland systems (Ghassemi et al. 1981).

U.S. EPA (1992b) pesticide fate summary data indicate that glyphosate has a half-life of 7 days in aerobic silty, clay-loam sediment. Anaerobic aquatic metabolism is slower, with a half-life of 5 weeks at pH 4.2 and 7 weeks at pH 6.3.

Depending on the soil type, the rate of glyphosate degradation is variable and dependent upon the level of microbial activity in the soil (Muller et al. 1981) or the strength of adsorption, which regulates the availability of the herbicide for degradation (Torstensson 1985). The U.S. EPA (1992b) reports that glyphosate exposure to aerobic soils containing indigenous microflora results in rapid biodegradation, with a half-life of less than 1 day in sandy-loam soils and a half-life of 1 to 3 days in silty-loam soils. Sacher (1978) reports the half-life of glyphosate to vary from 3 to 27 days depending on the soil type, with almost complete biodegradation occurring in 112 days.

Persistence studies of glyphosate by Muller et al. (1981) in Finnish agricultural fields revealed that, after observations lasting 249 days over the winter period, concentrations of glyphosate decreased to levels of 10 to 53 percent of the initial (17 mg/kg) concentration in loam and silt soils, respectively. Mean monthly air temperatures during the study ranged from 10.3°C to -12.0°C. pH in the two soils was 5.1 to 5.5 while organic carbon was 44 and 1.5 percent in the loam and silt, respectively. The researchers note that no significant accumulation of AMPA occurred (maximum of 3.2 mg/kg) and that glyphosate was degraded even at low temperatures. Soil respiration activity was positively correlated with the rate of glyphosate degradation. Soil nitrification, denitrification, and nitrogen fixation activity were not affected by the application of glyphosate to these soils.

Studies of glyphosate persistence in Canadian forest soils (Feng and Thompson 1990) indicate that residues of glyphosate dissipated to 13-18 percent of initial levels (31-40 mg/kg) within 360 days of initial application. The estimated time to 50 percent dissipation was 45-60 days.

Studies have examined the potential for the formation of nitrosoamines in aerobic soils treated with glyphosate. Some nitrosoamines may cause carcinogenic, mutagenic, and teratogenic effects and toxicity at low levels. Khan and Young (1977) demonstrated that when different soils were treated at 25°C with sodium nitrate and glyphosate at high rates (20 mg/kg and 740 mg/kg, respectively), the formation of N-nitrosoglyphosate occurred (i.e., <1 ppm in Granville sandy loam to 20 mg/kg in Fox sandy loam soils). Clay content of these soils was 20.0 and 5.1 percent, respectively. The researchers note that the high levels of glyphosate employed in these experimental conditions are not likely to be encountered in typical agricultural applications. Furthermore, treatment of these soils with typical application levels of glyphosate (5 mg/kg) and sodium nitrate (2 mg/kg) did not result in the formation of N-nitrosoglyphosate. Khan and Young state that at these levels of application the formation of N-nitrosoglyphosate in soil is not expected.

No information is currently available on the anaerobic metabolism of glyphosate in soils.

AMPA also biodegrades in the soil, but at a slower rate than glyphosate, thus resulting in accumulation in some soils (Rueppel et al. 1977). A maximum concentration of 0.21 ppm AMPA was observed in sandy loam soil studies in Iowa, while in aerobic aquatic studies the concentration of AMPA increased with time to 23 percent of the radiolabeled percent glyphosate applied (USEPA 1992b). Aerobic soil studies have shown levels of AMPA at day 14 to reach 26 to 28 percent of the parent glyphosate applied. The measurement of radiolabeled carbon dioxide (CO_2) evolved after 12 months in these studies amounted to approximately 71 percent of the theoretical maximum applied.

Anaerobic aquatic studies of AMPA showed 31 percent of the applied concentration remaining at day 15, and 14 to 24 percent remaining at up to 365 days. The amount of CO_2 evolved from the degradation of glyphosate was 35 percent of that applied after 1 year. Aerobic aquatic studies showed 19 to 25 percent of that applied remaining after 7 to 30 days. CO_2 evolved was 23 percent of the initial concentration after 30 days (USEPA 1992b).

Soil Adsorption

Glyphosate adsorbs strongly to soil particles (Sprankle 1974), and thus its leachability through soil is generally low (Torstensson 1985). Soil adsorption of glyphosate is correlated with the unoccupied phosphate sorption capacity of the soil. This binding to soil particles occurs rapidly within the first hour, decreasing slowly thereafter (Sprankle et al. 1975b). Glyphosate binding to soils is strongest in soils having low pH, high organic matter, and the highest phosphate-binding capacity (Ching et al. 1975). Soil pH was reported earlier by some researchers to have little effect on adsorption of glyphosate (Sprankle et al. 1975a, 1975b). However, recent studies of field soils indicate that the sorption of glyphosate shows

a strong dependence on soil pH. Nicholls and Evans (1991) have demonstrated that glyphosate is sorbed very strongly at pH values near 4.0. This very strong sorption is attributed to ligand exchange interactions which occur over a wide range of soil pH values.

Adsorption studies of glyphosate in nine different soil types indicate that glyphosate adsorption is correlated with unoccupied phosphate sorption capacity of the soil, thus suggesting that inorganic phosphate competes with glyphosate for sorption sites in the soil (Hance 1976). Hance (1976) has concluded that the low activity (phytotoxicity) of glyphosate in soil is a result of the combination of moderate adsorption and low intrinsic toxicity of the herbicide when made available to the root system of plants.

R_f (soil mobility) values have been developed by Helling (1971) using thin-layer chromatography to evaluate pesticide mobility in soils. As soil pH increases, so do R_f values. The following R_f values describe general soil mobility:

R_f	Mobility
0.0-0.09	Immobile
0.10-0.34	Low mobility
0.35-0.64	Intermediately mobile
0.65-0.89	Mobile
0.90-1.0	Very mobile

R_f values for glyphosate ranging from 0.04 to 0.20 were observed by Sprankle et al. (1975b). Based on these values, glyphosate would be considered practically immobile in soil. Rueppel et al. (1977) reported R_f values of 0.09 to 0.18 for glyphosate and classified the herbicide as immobile in soil.

AMPA, the primary degradation product of glyphosate, was classified as slightly mobile by Rueppel et al. (1977). Runoff studies of glyphosate summarized by Brønstad and Friestad (1985) also indicate that the mobility of glyphosate in soil is low. Studies of glyphosate applied to Canadian boreal forest soils indicate no evidence of lateral movement through subsurface flow (Roy et al. 1989).

Soil-water partition coefficients (Kd) identified for glyphosate (USEPA 1992b) show the following soil type variation: Drummer silt-clay-loam, 62; Roy silt, 90; Spinks sandy-loam, 70; and Lintonia sandy-loam, 22. Generally, compounds with Kd values less than 5 are considered highly mobile in soil. Thus, glyphosate is generally considered to have very low mobility in soils.

In freshwater sediments, glyphosate is reported to be fairly immobile, being rapidly adsorbed by cations, in the first hour after application (Torstensson 1985; Brønstad and Friestad 1985).

Environmental Half-Lives

The U.S. EPA (1992b) reports that field dissipation studies of glyphosate in loam-sand and silt-clay-loam soils indicate a 50 percent dissipation in less than 1.5 weeks and 3 weeks, respectively. Dissipation of glyphosate in forest soils in Michigan and Georgia indicate a half-life of less than 1 day; in Oregon, less than 14 days. Glyphosate or its primary degradation product AMPA did not leach into the soil below a depth of 6 inches.

The dissipation rate of glyphosate in flowing water was examined by Comes et al. (1976) in two irrigation canals located in the Yakima Valley, Washington. At application rates ranging from 1.7 to 2.2 kg/ha (1.5 to 1.95 lb/ac), glyphosate loss was reported to be 28 to 30 percent in the initial 1.6 km of each canal, with only about an additional 12 percent loss in the next 6.4 km to 12.8 km. The reason for the observed difference in loss was unknown. Initial glyphosate concentrations ranged from approximately 130 mg/L to 160 mg/L. Canal flow rates ranged from 1.7 m^3/sec to 2 m^3/sec, and water temperature ranged from 9°C to 12°C. The researchers state that dilution alone did not account for the reduction in glyphosate concentrations observed and cite the need for additional studies to identify those factors responsible for the glyphosate loss pattern observed.

Laboratory studies by Rueppel et al. (1977) indicate that the dissipation of glyphosate was nearly 90 percent after 14 days in Ray silt loam soils and after 80 days in Drummer silt—clay—loam soils. Soil temperatures ranged from 26°C to 32°C during the study. The half-life of glyphosate in Ray and Drummer soils at 4 mg/kg was 3 days and 27 days, respectively; at 8 mg/kg the half-life was 3 days and 25 days, respectively. These results suggest that the rate of glyphosate degradation may be independent of the initial concentration, although higher rates of application were not examined in this study. Others report that the half-life of glyphosate ranges from 2 to 10 weeks in biologically active soils and hydrosoils where microbial degradation occurs (Tooby 1985).

The dissipation of glyphosate was observed to be rapid in four small Canadian (Manitoba) boreal forest ponds ranging in depth from 0.25 meters to 1.5 meters (Goldsborough and Beck 1989). The half-life of glyphosate in water ranged from 1.5 days to 3.5 days based on an initial application rate of 0.89 kg active ingredient per hectare (0.79 lb/ac) for each freshwater pond. While water temperature of the ponds was not measured, other physico-chemical properties were recorded during the August 1986 study: specific conductance, 44-502 uS/cm (at 25°C); pH, 7.0-8.1; alkalinity, 20-260 mg/L. Samples of pond water collected in the spring of the year following the glyphosate treatment did not contain detectable glyphosate.

USEPA (1992b) pesticide fact sheet data for glyphosate indicate that dissipation studies in pond water show a half-life of 14 to 21 days with no glyphosate detectable after 129 days. Pond sediment concentrations of glyphosate increased from 190 μg/kg at day 7 to 6,800 μg/kg at day 127.

Bioaccumulation

The USEPA (1992b) reports that the glyphosate bioconcentration factor in bluegill sunfish exposed to 12 ppm for 35 days is 0.38 in edible tissue, 0.63 in nonedible tissue, and 0.52 in whole fish.

Sacher (1978) conducted fish metabolism studies with glyphosate and reported a bioconcentration factor of less than 0.18. Fourteen-day exposure studies to 10 mg/L glyphosate in three fish species (channel catfish, largemouth bass, and rainbow trout) resulted in maximum whole tissue concentrations of 0.55 mg/kg, 0.12 mg/kg, and 0.11 mg/kg, respectively.

Marine mollusks (*Rangia cumenta*) exposed for 35 days to an initial water concentration of 0.54 mg/L glyphosate exhibited an average soft tissue concentration of 2.4 mg/kg following a 42-day depuration period (Heydens 1991). The maximum bioconcentration factor for soft mollusk tissues was reported to be 9.6, suggesting no significant bioconcentration of glyphosate in marine mollusks. Similar studies were conducted with crayfish (*Procambarus simulans*). After exposure for 28 days to an initial concentration of 0.53 mg/L glyphosate, and following a 44-day depuration period, the average concentration of glyphosate in edible tissue was 0.052 mg/kg, yielding a bioconcentration factor of 0.27 (Heydens 1991).

Transport Mechanisms

This section describes the potential transport of glyphosate via groundwater, surface water, and air.

Because glyphosate binds strongly with soil particles and has not been shown to leach, the potential for groundwater contamination is low. There are presently no studies demonstrating the contamination of groundwater by glyphosate.

Glyphosate is soluble in water and therefore may be transported via runoff or direct contact with surface waters. Direct application of glyphosate to surface waters may result in the direct export of this material from the site. Precipitation or irrigation of an application site may result in runoff that is contaminated with glyphosate (Edwards et al. 1980). Product label information (Monsanto 1990) indicates that if rainfall or irrigation occurs within 6 hours of application, the effectiveness of the herbicide may be reduced.

Glyphosate exhibits a negligible vapor pressure, and therefore transfer from water to the atmosphere is negligible. Thus, effects due to volatilization on other plants or agricultural crops are not expected. However, wind drift and spray losses of glyphosate during application may occur.

Field Test Results

Field studies investigating the application of glyphosate to aquatic systems are limited. This section summarizes the results of those studies that have examined the fate of glyphosate when applied to aquatic systems or to uplands subject to runoff. Some of these field studies have been conducted in the Pacific Northwest, while others were conducted in latitudes where climatic conditions may be considered at least comparable to those in the Pacific Northwest.

Studies by Kroll (1991) examined the fate and transport of glyphosate used to control *Phragmites* in a tidal marsh system (i.e., Fishing Bay, Maryland). Following low-tide glyphosate application to isolated patches of *Phragmites* at two tidal marsh sites, little or no glyphosate ($\leq$5 ppb) was observed to be transported away from the application areas. The glyphosate application rate varied between 47 and 58 ounces of active ingredient per acre (3.6-4.5 kg/ha; 3.21-4.02 lb/acre). When applied directly at the same rate to adjacent tidal ponds, glyphosate, or its primary metabolite, AMPA, was not observed to persist in the ambient pond water. These observations were made during October 1989 through January 1990. During this time water temperatures steadily decreased from approximately 23°C to 1°C.

Highly variable persistence in tidal marsh sediments and *Phragmites* thatch was observed, ranging from 348 μg/kg to 1,273 μg/kg through 91 days in sediment and thatch, respectively. The results for the tidal-marsh sediments were unexpected, and Kroll (1991) reports that while rapid microbial degradation was anticipated for glyphosate in the estuarine sediments, the results of this field study indicate that glyphosate can persist in certain tidal pond sediments.

Degradation of glyphosate in saline sediments has been studied, and results indicate that while breakdown occurs, the herbicide tends to persist even up to 1 year (O'Keefe 1985). These studies, following application of glyphosate for the control of *Scirpus moritimus* in saline mudflats, resulted in initial residues of 2.6 mg/kg glyphosate in the top 5 cm of sediment. Half-life of the residue was 30 days. Tidal effects were said to likely increase the rate of herbicide dissipation through dilution.

Similar results were obtained by Torstensson et al. (1989) in studies of Swedish soils where the average accumulation of AMPA was observed to be 8 percent of the theoretical maximum applied (i.e., 2 kg active ingredient/ha; 1.78 lb/acre) after 2 years. This occurred in forest soils located at the arctic circle, while forest soils in more temperate regions of Sweden contained only 1 percent of the theoretical maximum applied AMPA after 1 year.

Three-year field studies at the North Appalachian Experimental Watershed (Edwards et al. 1980) examined the transport of glyphosate in runoff. These studies showed that the greatest export of applied glyphosate was 0.165 kg/ha (0.147 lb/acre) or 1.85 percent of the amount applied. Typically, transport in runoff was less than 1 percent of that applied. The

concentration of glyphosate in runoff was influenced by the application rate and time elapsed between herbicide application and runoff (i.e., precipitation event). At normal use rates (1.12-3.36 kg/ha; 1.0-3.0 lb/acre), runoff was affected for less than 2 months when levels decreased to <2 mg/L.. At the highest application rate (8.96 kg/ha; 8.0 lb/acre), glyphosate was detected at 5,200 μg/L in runoff and at 2 μg/L in runoff 4 months after treatment. At normal use rates, the maximum concentration of glyphosate in runoff was less than 100 μg/L.

Studies by Goldsborough and Beck (1989) examined the dissipation of glyphosate applied to the water surface of four small Canadian boreal forest ponds and six *in situ* microcosms over periods up to 255 days. Glyphosate added at a rate of 0.89 kg of active ingredient per hectare (0.79 lb/acre) was shown to dissipate rapidly from all ponds, with half-lives ranging from 1.5 days to 3.5 days. Glyphosate (2.5 kg/ha as Roundup®) remained at or above treatment levels in those microcosms containing only water but decreased rapidly (mean half life of 5.8 days) in those with sediment. Levels of AMPA were consistently low in ponds (<2.2 mg/L) and microcosms (<20 mg/L). Glyphosate residues in sediments of the treated microcosms generally increased over a 30-day period (i.e., maximum increases of 0.02 to 0.06 mg/kg). These results confirm the rapid dissipation of glyphosate from surface waters of lentic systems and suggest that sediment adsorption or biodegradation represent the major losses of glyphosate from the water column.

Feng et al. (1990) studied glyphosate and AMPA residues in oversprayed and buffered streams on the west coast of Vancouver Island. Maximum glyphosate residues were observed in two intentionally oversprayed tributaries (stream water, 162 μg/L; sediments, 6.80 mg/kg dry weight; suspended sediments, <0.03 μg/L). These levels dissipated to <1 μg/L within 96 hours after application. Glyphosate residues were primarily associated with stream sediments rather than the stream water, suggesting that sediments act as a primary sink for these compounds. While trace levels of glyphosate (<1 μg/L) were detected occasionally in the main stream channel and two oversprayed tributaries, no quantifiable residues (<1 μg/L) of glyphosate or AMPA were detected in any stream water samples associated with storm events. Also, biweekly samples from the main stream channel and tributaries during the long-term monitoring period (196-364 days) did not show the presence of detectable residues (limits of detection = 0.1 μg/L) after treatment. Glyphosate and AMPA residues in bottom sediments (<0.1-1.92 μg/g) were persistent compared to stream water residues (<0.1 μg/L) but declined over time so that residue concentrations were <0.2 μg/g by the end of the long-term monitoring period (i.e. day 196-364).

The concentrations of glyphosate observed in the surface waters of prairie pothole wetlands during emergent vegetation control activities ranged from 140 to 600 ppb 12 hours after applications (Henry 1992). Eight days after glyphosate treatment, concentrations had fallen considerably, ranging from 35 to 490 ppb.

Studies of the persistence, movement, and degradation of glyphosate in Canadian forest soils were conducted after the addition of glyphosate (Roundup®) at a rate of 2 kg of active

ingredient per hectare (1.8 lb/acre) (Roy et al. 1989). Soils at three depths (surface organic layer [SOL], SOL-15 cm, and 15 cm-30 cm) were analyzed for glyphosate and AMPA. More than 95 percent of the total herbicide residue (ranging from 707 μg/g at day 0 to <0.05 μg/g at day 691 and 762) was present in the upper organic layer throughout the study period (762 days). No evidence of lateral movement of glyphosate in runoff water or through subsurface flow was observed.

Newton et al. (1984) examined glyphosate herbicide residues and metabolites in Oregon forest foliage, litter, soil, stream water, sediments, and wildlife for 55 days following aerial application (3.3 kg/ha; 2.94 lb/acre). The half-life of glyphosate observed in forest foliage and litter ranged from 10.4 to 26.6 days and was twice as long in forest soils. Forest stream concentrations reached a maximum of 0.27 mg/L and decreased to below detection after 7 days. Sediment concentrations (0.55 mg/kg) were higher than water concentrations and persisted up to 55 days (0.15 mg/kg) or more. AMPA was observed at low levels in stream sediment (0.10 mg/kg) and to below detection within 55 days.

Detectable amounts of glyphosate did not accumulate in coho salmon fingerlings. All species of mammalian herbivores, carnivores, and omnivores examined had visceral and body contents of glyphosate at levels below those observed in groundcover and litter, indicating no accumulation at higher trophic levels.

APPENDIX B

ADJUVANTS REGISTERED FOR USE IN WASHINGTON STATE

Surfactants Registered by Washingto.. State Department of Agriculture.

#	REGISTRANT	NAME	ACTIVE INGREDIENTS	EPA
1	AMERICAN CYANAMID COMPANY	SUN-IT II SPRAY ADJUVANT	METHYLATED SEED OIL	AC11656-50093
2	AMWAY CORPORATION	ARSA-80 ALL PURPOSE SPRAY ADJUVANT	FREE FATTY ACIDS; NONIONIC SURFACTANT (ALKYL ARYL ALKOXYLATE)	AW11656-70005
3	AMWAY CORPORATION	ALL-PURPOSE SPRAY ADJUVANT	ALKYLARYL POLYALKOXYLATED ALCOHOLS	AW2935-70012
4	ATOCHEM NORTH AMERICA/DEC	DECCO 311 BUFFER CONC.	SODIUM PHOSPHATE; MONO SODIUM	AW47528-50002
5	ATOCHEM NORTH AMERICA/DEC	DECCO 239 CAUSTIC SODA BUFFER	SODIUM HYDROXIDE	AW802-70001
6	BASF CORPORATION	CASH	PROPRIETARY BLEND OF SURFACTANTS	AW99972-70006
7	BIOPLUS MANUFACTURING, IN	BIOPLUS SS 100	BINARY AND TERTIARY CO-POLYMER ALCOHOLS; DIMETHICONE; PVP-VA CO-POLYMER; HYDROXYTRICARBALLYLIC ACID	AW5905-70007
8	BLACK LEAF PRODUCTS CO.	DOMINANT SPRAY	PARAFFINIC OIL	AW5905-70006
9	CCT CORPORATION/WEBB WRIG	COMX - INSECT FEEDING STIMULANT	DISACCHARIDE; PHARMAMEDIA COTTONSEED FLOUR; VEGETABLE LIPID OIL	AW5905-70005
10	CHAS. H. LILLY CO.	LILLY MILLER SPRAY AID	ALKYLPHENOXY POLYETHOXY ETHANOLS; COTTONSEED OIL; IPA	AW5905-70004
11	CONKLIN COMPANY INC.	SATURALL 85	ACETYLENI DIOL; ALKYLARYL POLYOXYETHYLENE GLYCOL; DIMETHYLPOLYSILIOXANE; FATTY ACIDS; GLYCOL ETHERS; POLYOXYPROPYLENE POLYOXYEHTYLE	AW2792-70001
12	CONKLIN COMPANY INC.	KOMBIND COMPATIBILITY	PHOSPHATE ESTER; ISOPROPYL ALCOHOL	AW241-50001
13	CONKLIN COMPANY INC.	WEX	ALCOHOL ETHOXYLATES; DIMETHYLPOLYSILOXANE; PROPYLENE GLYCOL	904-7001
14	CREATIVE MARKETING & RESE	NO FOAM A	FATTY ACIDS; ISOPROPANOL; NONYLPHENOXY POLYETHOXY ETHANOLS	99968-70001
15	CREATIVE MARKETING & RESE	CMR SPREADER STICKER	ALKYLPHENOXY POLYETHOXY ETHANOLS; COTTONSEED OIL; IPA	AC707-50044
16	CREATIVE MARKETING & RESE	NO FOAM ADJUVANT	NONYL PHENOXY POLYETHOXY ETHANOL; POLYDIMETHYLSILOXANE	AC707-50043
17	CREATIVE MARKETING & RESE	NO FOAM B	BUFFERING ACIDS; COCONUT AMINE CONDENSATE; ISOPROPANOL; LINEAR ALKYL SULFONATE; OCTYL PHENOXY POLYETHOXY ETHANOL	AW11656-70004
18	CREATIVE MARKETING & RESE	CMR HERBICIDE ACTIVATOR	ALKYLPHENOXY POLYETHOXY ETHANOLS; PETROLEUM DISTILLATE	AC707-50042
19	CREATIVE MARKETING & RESE	CMR ORGARNIC OIL ADJUVANT	ALKYLARYL POLYETHER-ETHANOL; COTTONSEED OIL	AC707-50041
20	CUSTOM CHEMICIDES	ENTICE FEEDING STIMULENT FOR INSECTS	DISACCHARIDE; ETHOXY LATED ESTER; PHARMAMEDIA COTTONSEED FLOUR; VEGETABLE LIPID OIL	AW35550-50001
21	CUSTOM CHEMICIDES	DRIFTGARD	ANIONIC POLYACRYLAMIDE	AW39184-50003
22	CUSTOM CHEMICIDES	PARA SPRED	PARAFFINIC PETROLEUM OIL; POLYOL FATTY ACID ESTERS AND POLYETHOXLATED DERIVATIVES THEREOF	AW11656-70003
23	CUSTOM CHEMICIDES	FOAMGARD	SILICONE	AW1202-70007
24	CUSTOM CHEMICIDES	UNIFILM 707	ALKYLARYLPOLYOXYETHYLENE GLYCOLS-ISOPROPANOL	AW19713-70003
25	CUSTOM CHEMICIDES	BUFFERCIDE	PHOSPHORIC ACID	AW19713-70002
26	CUSTOM CHEMICIDES	STIK	ALKYLARYLPOLYOXYETHYLENE GLYCOL; BIS (2-ETHYLHEXYL) BENZENE DICARBOXYLATE; COMBINED FATTY ACIDS; GLYCOL ETHERS; ISOPROPANOL	AW19713-70001
27	CWC CHEMICAL INC.	HY-GRADE I (W) OIL	PETROLEUM OIL	AW99993-70001
28	DOWELANCO	IMPROVE	ETHOXYLATED NONYLPHENOL; FATTY AMINE ETHOXYLATE	AW61743-5000
29	DREXEL CHEMICAL COMPANY	SURFACTANT	ALKYLARYLPOLYETHOXYETHANOL; N-BUTANOL	AW99991-50001
30	DREXEL CHEMICAL COMPANY	SURF-AC 820	ALKYLARYLPOLYETHOXYETHANOL; N-BUTANOL	AW2935-50007
31	DREXEL CHEMICAL COMPANY	PEP OIL	PARAFFIN BASE PETROLEUM OIL; SURFACTANT BLEND	AW11656-50001
32	EXACTO CHEMICAL CO.	FORMULA 358	POLYVINYL POLYMER (POLYACRYLAMIDE)	AW39303-50005
33	EXACTO CHEMICAL CO.	EXACTROL	POLYAMIDE COPOLYMER	AW50241-50001
34	EXACTO CHEMICAL CO.	MORE	POLYACRYLAMIDE (POLYVINYL POLYMER)	AC11656-50051
35	EXACTO CHEMICAL CO.	GET-DOWN	POLYVINYL POLYMER (POLYACRYLAMIDE)	AC50775-50015
36	G. S. LONG CO., INC.	GSL 90 PLUS	ALKYLARYLPOLYETHOXYETHANOL; FREE FATTY ACICS; ISOPROPANOL; PROPYLENE GLYCOL; SILICONE ANTIFOAM AGENT	AW99972-70005
37	G. S. LONG CO., INC.	AMMONIUM SULFATE SPRAY GRADE	AMMONIUM SULATE (SPRAY GRADE)	AW11656-70001
38	GOLDEN WEST CHEMICAL DIST	PENOX	ALKYLARYL POLYOXYETHYLENE ETHER	AW52251-70005
39	HACO, INC.	HOPKINS UNITE	ACID POLYGLYCOLS; METHYL ALCOHOL	AC9319-50089
40	HACO, INC.	HOPKINS PLYAC	EMULSIFIABLE OXIDIZED POLYETHYLENE; ETHOXYLATED PHENOXY ALCOHOL	AW34704-50006
41	HELENA CHEMICAL COMPANY	KINETIC	POLYALKYLENEOXIDE MODIFIED POLYDIMETHYLSILOXANE; NONIONIC SURFACTANTS	AW2935-70009
42	HELENA CHEMICAL COMPANY	NALCOTROL II	POLYVINYL POLYMER	AC2935-50157
43	HELENA CHEMICAL COMPANY	NALCOTROL	POLYVINYL POLYMER	AW2935-70008
44	HELENA CHEMICAL COMPANY	STAY-PUT	POLYVINYL POLYMER	AW2935-70008
45	HELENA CHEMICAL COMPANY	COJET	AMMONIUM SALTS OF POLYACRYLIC; HYDROXY CARBOXYLIC; PHOSPHORIC ACIDS	AC2935-50098
46	HELENA CHEMICAL COMPANY	BEN DEX	ALKYL ARYL POLYETHOXY ETHANOL; PHOSPHATE ESTERS	AC30573-50002
47	HELENA CHEMICAL COMPANY	FOAMER	SODIUM ALPHA-SOEFIN SULFONATE	AW39184-50001
48	HELENA CHEMICAL COMPANY	HELENA AGRI-DEX	PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AW39184-50002
49	HELENA CHEMICAL COMPANY	INDUCE	ALKYLARYL POLYOXYLKANE ETHER; FREE FATTY ACIDS; IPA	AW17545-50002
50	HELENA CHEMICAL COMPANY	SURFIX	BETA PINENE POLYMER	AW17545-50001
51	HELENA CHEMICAL COMPANY	BUFFER P.S.	ALKYL ARYL POLYETHOXY ETHANOL PHOSPHATES; ORGANIC PHOSPHATIC ACIDS	AW17545-50004
52	HELENA CHEMICAL COMPANY	PENETRATOR	PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AW17545-50003
53	HELENA CHEMICAL COMPANY	BAYFOLAN PLUS	AMMONIACAL NITRATE; AMMONIUM MOLYBDATE; COBALT SULFATE; DIAMMONIUM PHOSPATE; EDTA (Fe, Cu, Mn, and Zn); PHOSPHORIC ACID; POTASSIUM NITRA	AW2935-50008
54	HELENA CHEMICAL COMPANY	DYNAMIC	POLYALKYLENEOXIDE; POLYDIMETHYLSILOXIDE; NONIONIC EMULSIFIERS; METHYLATED VEGETABLE OILS	AW17545-50005
55	JLB INTERNATIONAL	JLB OIL	LIMONENE PLUS SELECTED EMULSIFIERS	AC2792-50102
56	JLB INTERNATIONAL	JLB OIL PLUS - SPRAY ADJUVANT	ALIPHATIC HYDROCARBON OIL; LIMONENE PENETRANTS	AW2839-70002
57	JLB INTERNATIONAL	CIDE-KICK II	D, L-LIMONENE; RELATED ISOMERS PLUS SELECTED EMULSIFIERS	AW39303-50004
58	JONES-HAMILTON CO.	SODIUM BISULFATE	SODIUM BISULFATE; SULFUR	AW39303-50003
59	KALO AGRICULTURAL CHEMICA	REGULAID	POLYOXYETHYLENEPOLYPROPOXYPROPANOL; DIHYDROXYPROPANE; 2-BUTOXYETHANOL	AW39303-50002
60	KALO AGRICULTURAL CHEMICA	COMPEX	ALCOHOL SULFATES	AW47528-50001
61	KALO AGRICULTURAL CHEMICA	BUFFER-X	ALKYLARYLPOLYETHOXYETHANOL; DI-ALKYL BENZENEDICARBOXYLATE; FREE AND COMBINED FATTY ACIDS; GLYCOL ETHERS; ISOPROPANOL;	AC9319-50085
62	KALO AGRICULTURAL CHEMICA	BIO 38	ALKYLARYLPOLYETHOXYETHANOL; DIMETHYLPOLYSILOXANE; FREE FATTY ACIDS; ISOPROPANOL	AW2935-70011

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#	REGISTRANT	NAME	ACTIVE INGREDIENTS	EPA
63	KALO AGRICULTURAL CHEMICA	BIO FILM	ALKYLARYLPOLYETHOXYETHANOL; FREE AND COMBINED FATTY AND PHOSPHATIC ACIDS; ISOPROPANOL	AW2935-70007
64	KALO AGRICULTURAL CHEMICAL	HYDRO WET	GLYCOL BUTYL ETHER; POLYOXYETHYLENEPOLYPROPOXYPROPANOL	AW34704-50004
65	LANDO LAKES/CENEX	PRECISION SPRAY CONTROL	ACRYLAMIDE ACRYLIC ACID COPOLYMER	AC59639-50001
66	LEFFINGWELL/BUSINESS OF U CO	SOFBA-SPRAY ZNP 10-12-0	ALKYLBENZENESULFONATES; AMMONIUM PHOSPHATE; UREA; ZINC SULFATES;	AC2935-50152
67	LEFFINGWELL/BUSINESS OF U CO	ACTIVATE 9-0	ALKYLPOLYETHOXY-ETHANOL; DIMETHYLPOLY-SILOXANE; DIMETHYLPOLY-SILOXANE; ISOPROPANOL; N-BUTANOL	AC1148-50036
68	LEFFINGWELL/BUSINESS OF U CO	SOFBA-SPRAY ZIP	ALKYLBENZENESULFONATES; CITRIC ACID; IRON; PHOSPHORIC AID; SULFUR; ZINC	AW39509-50004
69	LEFFINGWELL/BUSINESS OF U CO	SOFBA-SPRAY MN	ALKYLBENZENESULFONATES; CITRIC ACID; MANGANESE; PHOSPHORIC ACID; SULFUR; ZINC	AW39509-50003
70	LEFFINGWELL/BUSINESS OF U CO	SOFBA-SPRAY MG	ALKYLBENZENESULFONATES; MANGANESE; PHOSPHORIC ACID; SULFUR; ZINC	AW34704-50003
71	LEFFINGWELL/BUSINESS OF U CO	SOFBA-SPRAY ZKP	ALKYLBENZENESULFONATES; PHOSPHORIC ACID; SULFUR; SOLUBLE POTASH; ZINC	AW7969-50001
72	LEFFINGWELL/BUSINESS OF U CO.	SOFBA-SPRAY ZBK	ALKYLBENZENESULFONATES; BORON; NITROGEN; SOLUBLE POTASH; ZINC;	AC36208-50015
73	LEFFINGWELL/BUSINESS OF U CO.	ACTIVATE 3	ALKYL OXY POLYETHOXY ETHANOL; DIMETHYLPOLYSILOXANE; FORMULATION AID; PROPYLENE GLYCOL	AW52251-70003
74	LOVELAND INDUSTRIES, INC.	CHEM-TROL	POLYVINYL POLYMER (POLYACRYLAMIDE)	AW52251-70001
75	LOVELAND INDUSTRIES, INC.	LI 700 PENETRATING SURFACTANT	PHOSPHATIDYLCHOLINE; METHYLACETIC ACID	AC17545-50004
76	LOVELAND INDUSTRIES, INC.	ACTIVATOR 90	ALKYL POLYOXYETHYLENE ETHER; FREE FATTY ACIDS; ISOPROPANOL	AC17545-50013
77	LOVELAND INDUSTRIES, INC.	WIDESPREAD	ALKYL ARYL POLYOXYETHYLENE ETHER	AC17545-50012
78	LOVELAND INDUSTRIES, INC.	TAKE-DOWN	ANIONIC SURFACTANTS; ETHOXYLATED NONIONIC SURFACTANTS; LOW MOLECULAR WT. ORGANIC ACIDS	AW99999-4
79	LOVELAND INDUSTRIES, INC.	FLOZINE	POLYSACCHARIDE GUMS	AW2935-70003
80	LOVELAND INDUSTRIES, INC.	ACTIVATOR N.F.	PRIMARY ALIPHATIC OXYALKYLATED ALCOHOL; DIMETHYLPOLYSILOXANE	AW62719-70001
81	LOVELAND INDUSTRIES, INC.	HELIMAX	PETROLEUM HYDROCARBONS (PARAFFINIC DISTILLATE; ALIPHATIC PETROLEUM SOLVENT); SURFACTANT (MONO AND DIESTERS OF OMEGA HYDROXYPOLY OX	AW1381-50003
82	LOVELAND INDUSTRIES, INC.	FIGHTER F	DIMETHYLPOLYSILOXANE	5887-37
83	LOVELAND INDUSTRIES, INC.	BOND	SYNTHETIC LATEX; PRIMARY ALIPHATIC OLYALKYLATED ALCOHOL	AW17545-50009
84	LOVELAND INDUSTRIES, INC.	E-Z MIX	ESTERS OF ALKYL POLYOXYETHYLENE ETHERS	AW17545-50005
85	MANDOPS INC.	SPRODNAM	POLYMER OF CYCLOHEXANE; 1-METHYL-4-(1-METHYLETHYL)	AW17545-50007
86	McGREGOR CO.	M-SUL-45	AMMONIUM SULFATE	AW17545-50008
87	McGREGOR CO.	CROP OIL M	PARAFFINIC PETROLEUM OIL; SURFACTANT BLEND	AW39303-50001
88	McGREGOR CO.	BUFFER-M	ALKYLARYLPOLYETHOXYETHOXYETHANOL; FREE AND COMBINED FATTY AND PHOSPHORIC ACIDS	AW36208-70004
89	McGREGOR CO.	MIX M	POLYESTERS OF SODIUM THIOBUTANEDIOATE AND ALCOHOL SULFATES; SODIUM ALKYLBUTANEDIAMATE	AC36208-50014
90	McGREGOR CO.	MCGREGOR M90 BIODEGRADABLE	ALKYLARYLPOLYETHOXYETHANOL; DIMETHYLPOLYSILOXANE; FREE FATTY ACIDS; ISOPROPANOL	59639-20
91	MILLER CHEM & FERT. CO.	EXIT	POLY (ETHYLENE-P-NONYL PHENOXY) POLY (OXYPROPYLENE) PROPANOL; MODIFIED RESIN; PETROLEUM DISTILLATE	5905-NONE
92	MILLER CHEM & FERT. CO.	SPRAY-AIDE	ALKYLARYL POLYOXYETHYLENE GLYCOL PHOSPHATE ESTER	AC36208-50020
93	MILLER CHEM & FERT. CO.	FOAM FIGHTER	DIMETHYL SILICONE FLUID EMULSION	AC72-50006
94	MILLER CHEM & FERT. CO.	NU-FILM-17	DI-1-P-METHENE	AC72-50005
95	MILLER CHEM & FERT. CO.	NU-FILM-P	POLY-1-P-METHENE	AC36208-16
96	MONTEREY CHEMICAL CO.	MONTEREY ZPK 0-16-9	IRON SULFATE; MANGANESE SULFATE; NONYLPHENOXY POLYETHOXY ETHANOL; PHOSPHORIC ACID; POLYDIMETHYLSILOXANE; POTASSIUM CHLORIDE; ZINC S	AW99999-3
97	MONTEREY CHEMICAL CO.	MONTEREY NPK 6-8-2	IRON SULFATE; MANGANESE SULFATE; NONYLPHENOXY POLYETHOXY ETHANOL; PHOSPHORIC ACID; POLYDIMETHYLSILOXANE; POTASSIUM CHLORIDE; UREA;	AW99999-2
98	MONTEREY CHEMICAL CO.	MONTEREY ZNP 10-12-0	NONYLPHENOXY POLYETHOXY ETHANOL; PHOSPHORIC ACID; POLYDIMETHYLSILOXANE; SULFUR; UREA; ZINC SULFATE	AW99999-1
99	MONTEREY CHEMICAL CO.	MONTEREY ZIP 0-8-0	CITRIC ACID; FERROUS SULFATES; MALIC ACID; NONYLPHENOXY POLYETHOXY ETHANOL; PHOSPHORIC ACID; POLYDIMETHYLSILOXANE; ZINC SULFATES	AW99987-50001
100	MONTEREY CHEMICAL CO.	NUTRIENT-BUFFER 11-4-6	ALKYL ARYLOXY POLYOXYETHYLENE PHOSPHATE; EDTA; HEDTA; PHOSPHORIC ACID; POTASSIUM NITRATE; POTASSIUM PHOSPHATE; UREA	AW99972-70004
101	MONTEREY CHEMICAL CO.	NALCOTROL II	POLYAMIDE COPOLYEMER	AW99963-7001
102	MONTEREY CHEMICAL CO.	NALCOTROL	POLYVINYL POLYMER	AW99963-50002
103	MONTEREY CHEMICAL CO.	EXCEL 90	ISOPROPANOL; NONYLPHENOXY POLYETHOXY ETHANOLS; POLYDIMETHYL-SILOXANE	AW99963-50001
104	MONTEREY CHEMICAL CO.	STIMULATOR PLUS	POLYETHOXYLATED ALKANOLS	AW707-50040
105	MONTEREY CHEMICAL CO.	MONTEREY X-100	ALKYLARYL POLYOXYETHYLENE GLYCOLS; PROPYL CARBINOL	AW5905-7003
106	MONTEREY CHEMICAL CO.	CHCP OIL CONC. NON-IONIC SURFACTANT	PARAFFIN-BASED PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AW5905-7002
107	NALCO CHEMICAL COMPANY	STA PUT DEPOSITION AID	POLYVINYL POLYMER	AW5905-70001
108	NALCO CHEMICAL COMPANY	NALQUATIC	POLYCARBOXYLATE POLYMER	AC36208-50001
109	NALCO CHEMICAL COMPANY	NALCO-TROL II	POLYAMIDE COPOLYMER	AW34704-50001
110	NALCO CHEMICAL COMPANY	NALCO-TROL	POLYVINYL POLYMER	AW2935-70006
111	OR-CAL	FYNOL-90 SPRAY ADJUVANT	MINERAL OIL (UR 90 MIN.)	AW2935-70001
112	OR-CAL	HEAT	PARAFFIN BASE PETROLEUM OIL	AW2839-70001
113	OR-CAL	FYNOL-4	OCTYLPHENOXY POLYETHOXY ETHANOL	AC2393-50007
114	PLATTE CHEMICAL COMPANY	ACTIMASTER	AMMONIUM SULFATE	AC1706-50005
115	PLATTE CHEMICAL COMPANY	HY-STOP SPRAY BUFFER	PHOSPHORIC ACID	AW1202-70005
116	PLATTE CHEMICAL COMPANY	OIL CONCENTRATE	PARAFFIN BASE PETROLEUM OIL	AW1202-70001
117	PLATTE CHEMICAL COMPANY	ACTIMASTER SPRAY ADJUVANT	AMMONIUM SULFATE	AW11600-70003
118	PUREGRO COMPANY	LEAF ACT PG-21 PLUS	AMMONIUM SULFATE; ALKYL GLYCOL; AMMONIACAL NITROGEN; NITROGEN; PHOSPHORIC ACID; SOLUBLE POTASH; SULFUR	AW11600-70002
119	PUREGRO COMPANY	PURE GRO CROP OIL (CONCENTRATE)	ALKANOIC AND ALKENOIC ACIDS AND MONO AND DIHYDROGEN PHOSPHATE ESTERS; ISOPROPYL ALCOHOL; PARAFFIN BASE PETROLEUM OIL	AW45989-70001
120	PUREGRO COMPANY	WET-SPREAD IRRIGATION AID	ALKYLPHENOXY POLYETHOXYETHANOL; PROPANOL	AW1148-70001
121	PUREGRO COMPANY	LEAF ACT 80 HE HERBICIDE ENHANCER	ALKYL POLYETHOXYETHYLENE GLYCOLS; ALKYD RESIN; ISOPROPANOL; MODIFIED PTHALIC GLYCEROL	[illegible]9-50084
122	PUREGRO COMPANY	LEAF LIFE FOLIAR NUTRIENT N-P-K-S 8-8-2	PHOSPHORIC ACID; POTASSIUM PHOSPHATE; S[illegible] MANGENESE; ZINC; UREA	9-50081
123	PUREGRO COMPAN	LEAF ACT 80S SPREADER STICKER	ALKYD RESIN; ALKYLPHENOXY POLYETHOXYE[illegible], ISOPROPANOL; MODIFIED PHTHALIC GLYCEROL; PROPYLENE GLYCOL	[illegible]9-50063
124	PUREGRO COMPANY	LEAF ACT 80A SPREADER ACTIVATOR	ALKYLPHENOXY POLYETHOXYETHANOL; ISOPROPANOL	AC9319-50048

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125	PURECHO COMPANY	LEAF LIFE FOLIAR NUTRIENT 3 10-12-0	NITROGEN; PHOSPHORIC ACID; MANGANESE; ZINC; PHOSPHORIC ACID; ZINC SULFATE	AC9319-50062
126	PURECHO COMPANY	LEAF ACT 80B BUFFER SPREADER	ALKYL POLYETHOXYETHYLENE GLYCOLS; ISOPROPANOL; PHOSPHORIC ACID	AC707-50033
127	QUINCY FARM CHEMICALS	QFC TACK-90	FATTY ACIDS; ISOPROPANOL; NONYLPHENOXY POLYETHOXY ETHANOLS	AC707-50032
128	RIVERSIDE/TERRA CORP.	SURF AID	ALKYL POLYETHOXY ETHERS; OTHER ETHOXYLATED DERIVATIVES	AC6718-50002
129	RIVERSIDE/TERRA CORP.	ACTIVATE PLUS	ALKYLARYLPOLYOXYETHYLENE GLYCOLS; FREE FATTY ACIDS; IPA	AC6718-50001
130	RIVERSIDE/TERRA CORP.	ASSIST	SILICON DIOXIDE; ALUMINUM OXIDE	AC5905-50066
131	RIVERSIDE/TERRA CORP.	INVADE PLUS	PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; ALKYL ARYL POLYOXYETHYLENE	AC5905-50063
132	RIVERSIDE/TERRA CORP.	COMPLEX	ALKYL POLYOXYETHYLENE ETHERS; POLYMERIZED RESINS; FATTY ACIDS; REACTED AMINES; AROMATIC PETROLEUM SOLVENT	AC5905-50062
133	RIVERSIDE/TERRA CORP.	COMBINE	PHOSPHATE ESTERS OF ALKYL ARYL POLYOXYETHANOL; ALCOHOL	AC5905-50058
134	RIVERSIDE/TERRA CORP.	AD 100	NONIONIC EMULSIFIERS; OIL OF LIMONENE	AC5905-50057
135	RIVERSIDE/TERRA CORP.	PLEX	ALPHA PHELLANDRENE; ALPHA TERPINENE; CAMPHENE	AC707-50036
136	ROHM AND HAAS COMPANY	LATRON B-1956	MODIFIED PHTALIC GLYCEROL ALKYD RESIN	AC50975-50001
137	ROHM AND HAAS COMPANY	LATRON CS-7	ALKYLARYL POLYETHOXYLATE; SODIUM SALT OF ALKYLSULFONATEDALKYLATE	AC50903-50001
138	ROHM AND HAAS COMPANY	LATRON AG-44M	ALKYLARYL POLYOXYETHYLENE GLYCOL PHOSPHATE ESTER SURFACTANTS; SOLUBILIZERS	AC50775-50021
139	ROHM AND HAAS COMPANY	LATRON AG-98	ALKYLARYL POLYOXYETHYLENE GLYCOLS	AC50775-50010
140	ROHM AND HAAS COMPANY	TRITON AG-44M	ALKYLARYL POLYOXYETHYLENE GLYCOL PHOSPHATE ESTER SURFACTANTS; SOLUBILIZERS	AC50775-50008
141	ROHM AND HAAS COMPANY	TRITON CS-7	ALKYLARYL POLYETHOXYLATE; SODIUM SALT OF ALKYLSULFONATEDALKYLATE	AC45989-50012
142	ROHM AND HAAS COMPANY	TRITON B-1956	PHTHALIC GLYCEROL ALKYD RESIN	AC45989-50007
143	ROHM AND HAAS COMPANY	TRITON AG-98	ALKYLARYL POLYOXYETHYLENE GLYCOLS	AC45989-50005
144	S & A CHEMICALS /SMITH &	SPREED-IT	ALKYLPHENOXY POLYETHOXYETHANOL ISOPROPANOL	AC45989-50004
145	S & A CHEMICALS /SMITH &	BUFF-IT	ALKYLPOLYETHOXYETHYLENE GLYCOLS; ISOPROPANOL; PHOSPHORIC ACID	AC45989-50003
146	S-K-H INC	S-K-H AGRICULTURAL ADHESIVE	BENTONITE; CASEIN; LACTOSE	AC36208-50008
147	SANAG DIV. OF SANITEK PRO	41-A	POLYACRYLAMIDE POLYMER	AC36208-50011
148	SANAG DIV. OF SANITEK PRO	18-F DRIFT RETARDENT ADDITIVE	POLYACRYLAMIDE POLYMER	AC36208-50003
149	SEACROP CO	CROCKERS FISH OIL STICKER-SPREADER	FISH OIL	AC2935-50144
150	TRI RIVER CHEMICAL	FLOTHRU SOIL PENETRANT 24	ALKYLARYL POLYETHYLENE ETHANOLS; ALKYL POLYOXYETHYLENE; MIXED ISOALKYL	AC2935-50142
151	TRI RIVER CHEMICAL	DEPO CONCENTRATE	POLYVINYL POLYMER (POLYACRYLAMIDE)	AC2935-50137
152	TRI RIVER CHEMICAL	DEPO RTU	POLYVINYL POLYMER (POLYACRYLAMIDE)	AC2935-50098
153	VALENT U.S.A. CORPORATION	X-77 SPREADER	ALKYLARYLPOLYOXYETHYLENE; FREE FATTY ACIDS; GLYCOLS; ISOPROPANOL	AC2935-50098
154	VALENT U.S.A. CORPORATION	/CLICK SUPREME SPRAY(ALFALFA)	PETROLEUM OIL; PETROLEUM OIL CLASSIFICATION-UNCLASSIFIED; PETROLEUM OIL MIN UNSULFONATED RESIDUE	AC2393-50002
155	WALLA WALLA FARMERS CO-OP	CO-OP 90	ISOPROPANOL; NONYLPHENOXY POLYETHOXY ETHANOLS; POLYDIMETHYLSILOXANE	AC11160-50001
156	WESTERN FARM SERVICE, IN	FIRST CHOICE SURPHTAC ADJUVANT		AC1050987-5000
157	WESTERN FARM SERVICE, IN	FIRST CHOICE NEUTRALIZER	ALKYLARYLPOLYOXYETHYLENE GLYCOLS; ISOPROPANOL; PHOSPHORIC ACID	AC50775-50020
158	WESTERN FARM SERVICE, IN	FIRST CHOICE CROP OIL CONCENTRATE	PARAFFIN-BASED PETROLEUM OIL; POLYOL FATTY ACID ESTERS AND POLYETHOXYLATED DERIVATIVES THEREOF	AC50775-50019
159	WESTERN FARM SERVICE, IN	FIRST CHOICE EXCEL 90	ISOPROPANOL; NONYLPHENOXY POLYETHAOXY ETHANOLS; POLYDIMETHYLSILOXANE	AC1706-50004
160	WESTERN FARM SERVICE, IN	WFSI CROP OIL CONCENTRATE	PARAFFIN-BASED PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATEVES THEREOF	AC1706-50002
161	WESTERN FARM SERVICE, IN	WFSI NEUTRILIZER	ALKYLARYLPOLYOXYETHYLENE GLYCOLS; ISOPROPANOL	AC1148-50034
162	WESTERN FARM SERVICE, IN	EXCEL 90	ISOPROPANOL; NONYLPHENOXY POLYETHOXY ETHANOLS; POLYDIMETHYLSILOXANE	AC1148-50033
163	WILBUR ELLIS COMPANY	NALCOTROL	POLYVINYL POLYMER	AC1148-50032
164	WILBUR ELLIS COMPANY	CAYUSE	AMMONIATED SALTS; PHOSPHATE ESTER OF POLYGLYCOLETHERS	AC1148-50035
165	WILBUR ELLIS COMPANY	PH	FATTY ACIDS; FATTY AMINE SALTS; PETROLEUM DISTALLATE	AC1202-50310
166	WILBUR ELLIS COMPANY	BIVERT	AMINE SALTS OF ORGANIC ACIDS; AROMATIC ACID; AROMATIC AND ALIPHATIC PETROLEUM DISTILLATE	AC1148-50062
167	WILBUR ELLIS COMPANY	TME (FOREST)	AMINE SALTS OF VEGETABLE FATTY ACIDS; ORGANIC AROMATIC ACID; AROMATIC AND ALIPHATIC PETROLEUM DISTILLATE	AC1202-50306
168	WILBUR ELLIS COMPANY	TME	AMINE SALTS OF VEGETABLE FATTY ACIDS; ORGANIC AROMATIC ACID; AROMATIC AND ALIPHATIC PETROLEUM DISTILLATE	AC1202-50307
169	WILBUR ELLIS COMPANY	RED-TOP MOR-ACT ADJUVANT (INDUSTRIAL)	NON-PHYTOTOXYC PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AC1148-50065
170	WILBUR ELLIS COMPANY	X-TA-PUT DEPOSITION AID	POLYVINYL POLMER	AC1202-50308
171	WILBUR ELLIS COMPANY	X-151 PESTICIDE ANTI-FOAM	SILICONE-BASED EMULSION	AC1202-50311
172	WILBUR ELLIS COMPANY	MOR-ACT ADJUVANT	NON-PHYTOTOXIC PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS AND POLYETHOXYLATED DERIVATIVES THEREOF	AC1706-50001
173	WILBUR ELLIS COMPANY	TRI-FOL	ALPHATIC POLYCARBOXYLATE; CALCIUM	AC1202-50309
174	WILBUR ELLIS COMPANY	NUTRIENT BUFFER SPRAY	AMMONIA NITROGEN; DIALKYL PHENOXYBENZENE DISULFONATE MIXTURE; ETHYLENEDIAMINETETRAACETE (EDTA); PHOSPHORIC ACID; SODIUM MONALKYL; U	AC36208-50005
175	WILBUR ELLIS COMPANY	X-900	ETHOXYLATED ALCOHOLS; LINEAR ALCOHOLS; COMPOUNDED SILICONE	AC72-50001
176	WILBUR ELLIS COMPANY	PENN-CLEAN	DIALKYL SODIUM SULFO DICARBOXYLATE	AC72-50003
177	WILBUR ELLIS COMPANY	X-56 SPREADER-STICKER	ALPHA-(P-NONYLPHENYL)-2-HYDROXYPOLY (OXYLETHYLENE); POLY (METHYLENE P-NONYLPHENOXY); POLYOXYPROPYLENE PROPANOL	AC36208-50006
178	WILBUR ELLIS COMPANY	X-77 SPREADER-ACTIVATOR	OCTYL PHENOXY POLYETHOXY ETHANOL; ISOPROPANOL; COMPOUNDED SILICONE	AW9779-50008
179	WILBUR ELLIS COMPANY	NO-FOAM	NON-IONIC EMULSIFIERS; SILICONE	AW9779-50007
180	WILBUR ELLIS COMPANY	MOR-ACT ADJUVANT (FOREST SITE PREP.)	NON-PHYTOTOXYC PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AW9779-50006
181	WILBUR ELLIS COMPANY	MOR-ACT ADJUVANT	NON-PHYTOTOXYC PARAFFIN BASE PETROLEUM OIL; POLYOL FATTY ACID ESTERS; POLYETHOXYLATED DERIVATIVES THEREOF	AW9779-50005
182	WITCO CHEMICAL CORPORATIO	SPONTO 168-D	ALKYLPHENOXYPOLY-(ETHYLENEOXY) ETHYL PHOSPHATE; (ALKYLPHENYL)-HYDROXYPOLY (OXYLETHYLENE); PHOSPHORIC ACID; METHANOL; MONOPOTSSIU	AW9779-50004
183	WITCO CHEMICAL CORPORATIO	ADSEE 100-80	ISOPROPANOL; (NONYLPHENYL)-HYDROXYPOLY (OXYLETHYLENE); SEE LABEL	AW9779-50003
184	WITCO CHEMICAL CORPORATIO	ADSEE 775	(NONYLPHENYL)-HYDROXYPOLY (OXYETHYLENE); POLY (METHYLENE NONYLPHENOXY POLYOXYPROPYLENE PROPANOL: ALKYL FATTY ACIDS; SEE LABEL	AW9779-50002
185	WITCO CHEMICAL CORPORATIO	ADSEE 801	ALKYL POLYOXYETHYLENE ETHER; ISOPROPANOL	AW9779-50001