



SERA TR 03-43-17-02c

Sulfometuron Methyl - Human Health and Ecological Risk Assessment - Final Report

Prepared for:

USDA, Forest Service

Forest Health Protection

GSA Contract No. GS-10F-0082F

USDA Forest Service BPA: WO-01-3187-0150

USDA Purchase Order No.: 43-1387-3-0716

Task No. 17



Submitted to:

Hank Appleton, COTR
Forest Health Protection Staff
USDA Forest Service

Rosslyn Plaza Building C, Room 7129C
1601 North Kent Street
Arlington, VA 22209

Prepared by Julie Klotzbach and Patrick Durkin

Submitted by:

Syracuse Environmental Research Associates, Inc.

5100 Highbridge St., 42C

Fayetteville, New York 13066-0950

Telephone: (315) 637-9560

Fax: (315) 637-0445

E-Mail: SERA_INC@msn.com

Home Page: www.sera-inc.com

December 14, 2004

TABLE OF CONTENTS

TABLE OF CONTENTS	ii
LIST OF TABLES	v
ACRONYMS, ABBREVIATIONS, AND SYMBOLS	vi
COMMON UNIT CONVERSIONS AND ABBREVIATIONS	viii
CONVERSION OF SCIENTIFIC NOTATION	ix
EXECUTIVE SUMMARY	x
1. INTRODUCTION	1-1
2. PROGRAM DESCRIPTION	2-1
2.1. Overview	2-1
2.2. Chemical Description and Commercial Formulations	2-1
2.3. Application Methods	2-2
2.4. Mixing and Application Rates	2-3
2.5. Use Statistics	2-4
3. HUMAN HEALTH RISK ASSESSMENT	3-1
3.1. HAZARD IDENTIFICATION	3-1
3.1.1. Overview.	3-1
3.1.2. Mechanism of Action.	3-2
3.1.3. Kinetics and Metabolism.	3-3
3.1.4. Acute Oral Toxicity.	3-4
3.1.5. Subchronic or Chronic Systemic Toxic Effects.	3-5
3.1.6. Effects on Nervous System.	3-6
3.1.7. Effects on Immune System.	3-6
3.1.8. Effects on Endocrine System.	3-7
3.1.9. Reproductive and Teratogenic Effects.	3-7
3.1.10. Carcinogenicity and Mutagenicity.	3-8
3.1.11. Irritation and Sensitization (Effects on the Skin and Eyes).	3-9
3.1.12. Systemic Toxic Effects from Dermal Exposure.	3-9
3.1.13. Inhalation Exposure.	3-10
3.1.14. Inerts and Adjuvants.	3-10
3.1.15. Impurities and Metabolites.	3-11

TABLE OF CONTENTS (continued)

3.2. EXPOSURE ASSESSMENT 3-12

- 3.2.1. Overview. 3-12
- 3.2.2. Workers. 3-12
 - 3.2.2.1. General Exposures 3-13
 - 3.2.2.2. Accidental Exposures 3-14
- 3.2.3. General Public. 3-16
 - 3.2.3.1. General Considerations 3-16
 - 3.2.3.2. Direct Spray 3-16
 - 3.2.3.3. Dermal Exposure from Contaminated Vegetation 3-17
 - 3.2.3.4. Contaminated Water 3-17
 - 3.2.3.4.1. Acute Exposure 3-17
 - 3.2.3.4.2. Longer-term Exposure 3-20
 - 3.2.3.5. Oral Exposure from Contaminated Fish 3-21
 - 3.2.3.6. Oral Exposure from Contaminated Vegetation 3-22

3.3. DOSE-RESPONSE ASSESSMENT 3-24

- 3.3.1. Overview 3-24
- 3.3.2. Existing Guidelines 3-24
- 3.3.2. Acute RfD 3-25

3.4. RISK CHARACTERIZATION 3-26

- 3.4.1. Overview 3-26
- 3.4.2. Workers 3-27
- 3.4.3. General Public. 3-28
- 3.4.4. Sensitive Subgroups. 3-29
- 3.4.5. Connected Actions. 3-29
- 3.4.6. Cumulative Effects. 3-29

TABLE OF CONTENTS (continued)

4. ECOLOGICAL RISK ASSESSMENT	4-1
4.1. HAZARD IDENTIFICATION	4-1
4.1.1. Overview.	4-1
4.1.2. Toxicity to Terrestrial Organisms.	4-2
4.1.2.1. Mammals	4-2
4.1.2.2. Birds	4-4
4.1.2.3. Terrestrial Invertebrates	4-4
4.1.2.4. Terrestrial Plants (Macrophytes)	4-5
4.1.2.5. Terrestrial Microorganisms	4-6
4.1.3. Aquatic Organisms.	4-6
4.1.3.1. Fish	4-6
4.1.3.2. Amphibians	4-7
4.1.3.3. Aquatic Invertebrates	4-8
4.1.3.4. Aquatic Plants	4-9
4.2. EXPOSURE ASSESSMENT	4-10
4.2.1. Overview.	4-10
4.2.2. Terrestrial Animals.	4-11
4.2.2.1. Direct Spray	4-12
4.2.2.2. Indirect Contact	4-13
4.2.2.3. Ingestion of Contaminated Vegetation or Prey	4-14
4.2.2.4. Ingestion of Contaminated Water	4-16
4.2.3. Terrestrial Plants.	4-16
4.2.3.1. Direct Spray	4-16
4.2.3.2. Off-Site Drift	4-16
4.2.3.3. Runoff	4-18
4.2.3.4. Contaminated Irrigation Water	4-19
4.2.3.5. Wind Erosion	4-20
4.2.4. Soil Organisms.	4-21
4.2.5. Aquatic Organisms.	4-21
4.3. DOSE-RESPONSE ASSESSMENT	4-23
4.3.1. Overview.	4-23
4.3.2. Toxicity to Terrestrial Organisms.	4-24
4.3.2.1. Mammals	4-24
4.3.2.2. Birds	4-25
4.3.2.3. Terrestrial Invertebrates	4-25
4.3.2.4. Terrestrial Plants (Macrophytes)	4-25
4.3.2.5. Terrestrial Microorganisms	4-26
4.3.3. Aquatic Organisms.	4-26
4.3.3.1. Fish	4-26
4.3.3.2. Amphibians	4-27

TABLE OF CONTENTS (continued)

4.3.3.3. Aquatic Invertebrates	4-27
4.3.3.4. Aquatic Plants	4-28
4.4. RISK CHARACTERIZATION	4-30
4.4.1. Overview.	4-30
4.4.2. Terrestrial Organisms.	4-31
4.4.2.1. Terrestrial Vertebrates	4-31
4.4.2.2. Terrestrial Invertebrates	4-32
4.4.2.3. Terrestrial Plants	4-32
4.4.2.4. Soil Microorganisms	4-33
4.4.3. Aquatic Organisms.	4-34
4.4.3.1. Aquatic Animals	4-34
4.4.3.2. Aquatic Plants	4-35
5. REFERENCES	5-1

NOTE: Tables and Figures follow the reference list.

LIST OF APPENDICES

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl

Appendix 3: Field Studies on the environmental fate of sulfometuron methyl

Appendix 4: Toxicity of sulfometuron methyl to experimental birds

Appendix 5: Bioassays of sulfometuron methyl toxicity in terrestrial plants

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants

Appendix 7: Toxicity of sulfometuron methyl to amphibians

Appendix 8: Effects of Sulfometuron methyl on microorganism and microbial populations in soil

LIST OF TABLES

Table 2-1: Identification and physical/chemical properties of sulfometuron methyl	Tables-1
Table 2-2: Use of sulfometuron methyl by USDA Forest Service in 2001 by Type of Use	Tables-2
Table 2-3: Use of sulfometuron methyl by USDA Forest Service in 2001 by Region . . .	Tables-3
Table 3-1: Chemical and site parameters used in GLEAMS Modeling for Sulfometuron methyl	Tables-4
Table 3-2: Summary of modeled concentrations of Sulfometuron methyl in streams	Tables-5
Table 3-3: Summary of modeled concentrations of Sulfometuron methyl in ponds	Tables-6
Table 4-1: Summary of Data on Short- and Long-term Exposure of African Clawed frog (<i>Xenopus laevis</i>) to sulfometuron methyl	Tables-7
Table 4-2: Summary of modeled concentrations of sulfometuron methyl in soil	Tables-8

LIST OF FIGURES

Figure 2-1: Use of sulfometuron methyl by the USDA Forest Service in various regions of the United States based on percentages of total use by the Forest Service	Figures-1
Figure 3-1: Proposed metabolic pathway of sulfometuron methyl in the goat	Figures-2

NOTE: Tables followed by figures are places after Section 5, References.

LIST OF WORKSHEETS

Supplement 1: Sulfometuron methyl – WordPerfect Worksheets for Human Health and Ecological Risk Assessments, SERA WPWS 04-43-17-02d, Version 2.04d, dated December 8, 2004.
Supplement 2: Sulfometuron Methyl – EXCEL Worksheets for Human Health and Ecological Risk Assessments, SERA EXWS 04-43-17-02d, Version 2.04d, dated December 8, 2004.

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
a.e.	acid equivalents
AEL	adverse-effect level
a.i.	active ingredient
ALS	acetolactate synthase
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
bw	body weight
CBI	confidential business information
CI	confidence interval
cm	centimeter
CNS	central nervous system
DAA	days after application
DAT	days after treatment
d.f.	degrees of freedom
EC _x	concentration causing X% inhibition of a process
EC ₂₅	concentration causing 25% inhibition of a process
EC ₅₀	concentration causing 50% inhibition of a process
ExToxNet	Extension Toxicology Network
F	female
FH	Forest Health
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FQPA	Food Quality Protection Act
g	gram
ha	hectare
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
k _a	absorption coefficient
k _e	elimination coefficient
kg	kilogram
K _{o/c}	organic carbon partition coefficient
K _{o/w}	octanol-water partition coefficient
K _p	skin permeability coefficient
L	liter
lb	pound
LC ₅₀	lethal concentration, 50% kill
LD ₅₀	lethal dose, 50% kill
LOAEL	lowest-observed-adverse-effect level
m	meter
M	male
MMAD	mass median aerodynamic diameter
MCS	multiple chemical sensitivity

ACRONYMS, ABBREVIATIONS, AND SYMBOLS (*continued*)

mg	milligram
mg/kg/day	milligrams of agent per kilogram of body weight per day
mL	milliliter
mM	millimole
MOS	margin of safety
MRID	Master Record Identification Number
MSDS	material safety data sheet
MW	molecular weight
NCI	National Cancer Institute
NIOSH	National Institute for Occupational Safety and Health
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NOS	not otherwise specified
NRC	National Research Council
NTP	National Toxicology Program
OM	organic matter
OPP	Office of Pesticide Programs
OPPTS	Office of Pesticide Planning and Toxic Substances
OSHA	Occupational Safety and Health Administration
ppm	parts per million
RBC	red blood cells
RED	re-registration eligibility decision
RfD	reference dose
SERA	Syracuse Environmental Research Associates
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SRC	Syracuse Research Corporation
UF	uncertainty factor
U.S.	United States
USDA	U.S. Department of Agriculture
U.S. EPA	U.S. Environmental Protection Agency
USGS	U.S. Geological Survey
WHO	World Health Organization
μ	micron
\blacktriangleright	greater than
\geq	greater than or equal to
$<$	less than
\leq	less than or equal to
$=$	equal to
\approx	approximately equal to
\sim	approximately

COMMON UNIT CONVERSIONS AND ABBREVIATIONS

To convert ...	Into ...	Multiply by ...
acres	hectares (ha)	0.4047
acres	square meters (m ²)	4,047
atmospheres	millimeters of mercury	760
centigrade	Fahrenheit	1.8 °C+32
centimeters	inches	0.3937
cubic meters (m ³)	liters (L)	1,000
Fahrenheit	centigrade	0.556 °F-17.8
feet per second (ft/sec)	miles/hour (mi/hr)	0.6818
gallons (gal)	liters (L)	3.785
gallons per acre (gal/acre)	liters per hectare (L/ha)	9.34
grams (g)	ounces, (oz)	0.03527
grams (g)	pounds, (oz)	0.002205
hectares (ha)	acres	2.471
inches (in)	centimeters (cm)	2.540
kilograms (kg)	ounces, (oz)	35.274
kilograms (kg)	pounds, (lb)	2.2046
kilograms per hectare (kg/ha)	pounds per acre (lb/acre)	0.892
kilometers (km)	miles (mi)	0.6214
liters (L)	cubic centimeters (cm ³)	1,000
liters (L)	gallons (gal)	0.2642
liters (L)	ounces, fluid (oz)	33.814
miles (mi)	kilometers (km)	1.609
miles per hour (mi/hr)	cm/sec	44.70
milligrams (mg)	ounces (oz)	0.000035
meters (m)	feet	3.281
ounces (oz)	grams (g)	28.3495
ounces per acre (oz/acre)	grams per hectare (g/ha)	70.1
ounces per acre (oz/acre)	kilograms per hectare (kg/ha)	0.0701
ounces fluid	cubic centimeters (cm ³)	29.5735
pounds (lb)	grams (g)	453.6
pounds (lb)	kilograms (kg)	0.4536
pounds per acre (lb/acre)	kilograms per hectare (kg/ha)	1.121
pounds per acre (lb/acre)	mg/square meter (mg/m ²)	112.1
pounds per acre (lb/acre)	µg/square centimeter (µg/cm ²)	11.21
pounds per gallon (lb/gal)	grams per liter (g/L)	119.8
square centimeters (cm ²)	square inches (in ²)	0.155
square centimeters (cm ²)	square meters (m ²)	0.0001
square meters (m ²)	square centimeters (cm ²)	10,000
yards	meters	0.9144

Note: All references to pounds and ounces refer to avoirdupois weights unless otherwise specified.

CONVERSION OF SCIENTIFIC NOTATION

Scientific Notation	Decimal Equivalent	Verbal Expression
$1 \cdot 10^{-10}$	0.0000000001	One in ten billion
$1 \cdot 10^{-9}$	0.000000001	One in one billion
$1 \cdot 10^{-8}$	0.00000001	One in one hundred million
$1 \cdot 10^{-7}$	0.0000001	One in ten million
$1 \cdot 10^{-6}$	0.000001	One in one million
$1 \cdot 10^{-5}$	0.00001	One in one hundred thousand
$1 \cdot 10^{-4}$	0.0001	One in ten thousand
$1 \cdot 10^{-3}$	0.001	One in one thousand
$1 \cdot 10^{-2}$	0.01	One in one hundred
$1 \cdot 10^{-1}$	0.1	One in ten
$1 \cdot 10^0$	1	One
$1 \cdot 10^1$	10	Ten
$1 \cdot 10^2$	100	One hundred
$1 \cdot 10^3$	1,000	One thousand
$1 \cdot 10^4$	10,000	Ten thousand
$1 \cdot 10^5$	100,000	One hundred thousand
$1 \cdot 10^6$	1,000,000	One million
$1 \cdot 10^7$	10,000,000	Ten million
$1 \cdot 10^8$	100,000,000	One hundred million
$1 \cdot 10^9$	1,000,000,000	One billion
$1 \cdot 10^{10}$	10,000,000,000	Ten billion

EXECUTIVE SUMMARY

OVERVIEW

Sulfometuron methyl is an effective and potent herbicide. Adverse effects on some nontarget terrestrial plant species and, to a lesser degree, some aquatic plant species are plausible under some conditions. For terrestrial plants, the dominant factor in the risk characterization is the potency of sulfometuron methyl relative to the application rate. The typical application rate considered in this risk assessment, 0.045 lb/acre, is about 1875 times higher than the NOEC in the vegetative vigor (direct spray) assay of the most sensitive non-target species – i.e., 0.000024 lb/acre – and almost 60 times higher than the NOEC for the most tolerant species in the same assay – i.e., 0.00078 lb/acre. The highest application rate that may be considered in Forest Service programs – i.e., 0.38 lb/acre – is over 15,000 times the NOEC in sensitive species and a factor of about 490 above the NOEC in tolerant species. Given these relationships, damage to sensitive nontarget species could be expected in ground broadcast applications at distances of about 900 feet from the application site in areas in which off-site drift is not reduced by foliar interception. This risk characterization applies only to ground broadcast applications. When used in directed foliar applications (i.e., backpack), offsite drift could be reduced substantially but the extent of this reduction cannot be quantified.

Damage to aquatic plants, particularly macrophytes, appears substantially less than for terrestrial plants. All hazard quotients for aquatic macrophytes were based on an NOEC of 0.00021 mg/L in duckweed for both acute and chronic exposures. Except for the hazard quotient of 4 associated with acute exposures based on the peak concentrations of sulfometuron methyl, all hazard quotients are below the level of concern, with a range of 0.01 to 0.4 for acute exposures and 0.002 to 0.01 for chronic exposures. Thus, if sulfometuron methyl is applied in areas where transport to water containing aquatic macrophytes is likely, it would be plausible that detectable but transient damage could be observed.

Aquatic algae do not appear to be as sensitive to sulfometuron methyl. The highest hazard quotient observed for acute exposure is 0.4 associated with the upper range for the most sensitive species. For chronic exposures, the highest hazard quotient is 0.001 associated with the upper range for the most sensitive species. Therefore, it is not anticipated that adverse effects in aquatic algae would result from exposure to sulfometuron methyl at application rates used by the Forest Service.

Just as there is little reason to doubt that adverse effects on some plant species are plausible, there is no clear basis for suggesting that effects on terrestrial or aquatic animals are likely or would be substantial.

PROGRAM DESCRIPTION

Sulfometuron methyl is a non-selective, sulfonyl urea herbicide used in the control the growth of broadleaf weeds and grasses. The only commercial formulations of sulfometuron methyl used by the Forest Service are Oust and Oust XP[®]. Oust and Oust XP are manufactured by Du Pont as a water dispersible granule. The composition of the product is 75% sulfometuron methyl and 25% inert ingredients.

Sulfometuron methyl is used in Forest Service programs primarily for the control of noxious weeds. Minor uses include conifer release and rights-of-way management. The most common methods of ground application for Oust and Oust XP involve backpack (selective foliar) and boom spray (broadcast foliar) operations. The Forest Service does not use aerial applications for Oust or Oust XP. Nonetheless, both formulations are registered for aerial applications and aerial applications are included in this risk assessment in the event the Forest Service may wish to consider this application method. For this risk assessment, the typical rate of 0.045 lbs/acre. A range of application rates will be taken as 0.03 lbs/acre to 0.38 lbs/acre to reflect plausible ranges that the Forest Service may use. An upper range of 0.38 lb/acre is used to assess the consequences of using the highest labeled rate should the Forest Service need to consider this option. The lower range is the lowest rate reported by the Forest Service.

HUMAN HEALTH RISK ASSESSMENT

Hazard Identification – In experimental mammals, the acute oral LD₅₀ for sulfometuron methyl is greater than 17,000 mg/kg, which indicates a low order of toxicity. The lowest dose reported to cause any apparent effects after single gavage administration to rats is 5000 mg/kg. Acute exposure studies of sulfometuron methyl and the sulfometuron methyl formulation Oust give similar results, indicating that formulations of sulfometuron methyl are not more toxic than sulfometuron methyl alone. The most common signs of toxicity involve changes in blood that are consistent with hemolytic anemia (i.e., a lysis or destruction of blood cells that results in a decreased number of red blood cells) and decreased body weight gain. It is plausible that the hemolytic anemia caused by sulfometuron methyl is attributable, at least partially, to sulfonamide and saccharin, which are metabolites of sulfometuron methyl. Appropriate tests have provided no evidence that sulfometuron methyl causes malformations or cancer. Sulfometuron methyl is irritating to the skin and eyes, but does not produce sensitizing effects following repeated dermal exposure.

There is some concern regarding potential reproductive and teratogenic effects from exposure to sulfometuron methyl. Gavage studies in rabbits suggest that sulfometuron methyl exposure may increase the number of fetuses with anomalies as well as the proportion of fetal anomalies per litter. In addition to the two teratogenicity studies in rabbits, there are three reproduction studies involving dietary exposure of rats to sulfometuron methyl, in which effects were observed in dams (decreases in maternal body weight gain associated with decreased food consumption) and offspring (decreased fetal weight, decreased numbers of pups, and decreases in brain weights). As detailed in the dose-response assessment, these effects were not consistently dose-related and do not appear to be the most sensitive effect for sulfometuron methyl.

Limited information is available on the toxicokinetics of sulfometuron methyl. The kinetics of absorption of sulfometuron methyl following dermal, oral or inhalation exposure are not documented in the available literature. In both mammals and bacteria, sulfometuron methyl is degraded by cleavage of the sulfonyl urea bridge to form sulfonamide and a dimethyl pyrimidine urea or pyrimidine amine. Sulfonamide may be further degraded by demethylation to the free benzoic acid which, in turn, may undergo a condensation reaction to form saccharin. Sulfometuron methyl does not appear to concentrate in tissues and is eliminated fairly rapidly, with a half-life in goats ranging from 28 to 40 hours. In goats, nearly all of the administered

sulfometuron methyl dose was excreted in urine. Studies on the toxicity of sulfometuron methyl metabolites have not been conducted, however, the toxicity of the metabolites of sulfometuron methyl is likely to be encompassed by the available mammalian toxicity studies.

As discussed in the exposure assessment, skin absorption is the primary route of exposure for workers. Data regarding the dermal absorption kinetics of sulfometuron methyl are not available in the published or unpublished literature. For this risk assessment, estimates of dermal absorption rates – both zero order and first order – are based on quantitative structure-activity relationships. These estimates of dermal absorption rates are used in turn to estimate the amounts of sulfometuron methyl that might be absorbed by workers, which then are used with the available dose-response data to characterize risk. The lack of experimental data regarding dermal absorption of sulfometuron methyl adds substantial uncertainties to this risk assessment. Uncertainties in the rates of dermal absorption, although they are substantial, can be estimated quantitatively and are incorporated in the human health exposure assessment.

The inhalation toxicity of sulfometuron methyl is not well documented in the literature. Available studies indicate that sulfometuron methyl induces irritant effects at very high exposure levels. Regardless, the potential inhalation toxicity of sulfometuron methyl is not of substantial concern to this risk assessment because of the implausibility of inhalation exposure involving high concentrations of this compound.

Exposure Assessment – Exposure assessments are conducted for both workers and members of the general public for the typical application rate of 0.045 lb/acre. The consequences of using the maximum application rate that might be used by the Forest Service, 0.38 lb/acre, are discussed in the risk characterization.

For workers, three types of application methods are generally modeled in Forest Service risk assessments: directed ground, broadcast ground, and aerial. Although Oust and Oust XP are registered for aerial applications (helicopter and sometimes fixed wing), the Forest Service does not currently use this method. Nonetheless, the aerial application method is included in this risk assessment in the event that the Forest Service considers it an option. Central estimates of exposure for ground workers are approximately 0.0006 mg/kg/day for directed ground spray and 0.001 mg/kg/day for broadcast ground spray. Upper range of exposures are approximately 0.004 mg/kg/day for directed ground spray and 0.007 mg/kg/day for broadcast ground spray. All of the accidental exposure scenarios for workers involve dermal exposures and all of these accidental exposures lead to estimates of dose that are either in the range of or substantially below the general exposure estimates for workers.

For the general public, the range of acute exposures is from approximately 1.2×10^{-7} mg/kg associated with the lower range for consumption of contaminated stream water by a child to 0.094 mg/kg/day associated with the upper range for consumption of contaminated water by a child following an accidental spill of sulfometuron methyl into a small pond. For chronic or longer term exposures, the modeled exposures are much lower than for acute exposures, ranging from approximately 2.3×10^{-11} mg/kg/day associated with the lower range for the normal consumption of fish by the general public to approximately 0.0016 mg/kg/day associated with the

upper range for consumption of contaminated fruit.

Dose-Response Assessment – According to a Federal Registry Notice (U.S. EPA 1997), the U.S. EPA has derived an RfD of 0.24 mg/kg/day. This RfD is based on a NOAEL for bladder toxicity of 500 ppm dietary sulfometuron methyl (equivalent to 24.4 mg/kg/day) and a 100-fold safety factor. Although an RfD has been derived by U.S. EPA, a more conservative provisional reference dose of 0.02 mg/kg/day, which was used in the previous Forest Service risk assessment on sulfometuron methyl (Durkin 1998), was derived from data reported in the 2-year feeding study in rats by Mullin (1984). The provisional reference dose is based on the 2 mg/kg/day (50 ppm) NOAEL for hematological effects in male rats and an uncertainty factor of 100:10 for species-to-species extrapolation and 10 for sensitive subgroups in the human population. The provisional RfD of 0.02 mg/kg/day is used in the current risk assessment for characterizing risks associated with chronic exposure to sulfometuron methyl. The U.S. EPA has not derived an acute/single dose RfD for sulfometuron methyl. A NOAEL of 86.6 mg/kg/day was reported for decreased maternal and fetal body weights in rats following 10-day gestational exposure of dams (Lu 1981). Using a NOAEL 86.6 mg/kg/day and margin of exposure of 100, a provisional acute RfD is calculated as 0.87 mg/kg/day and will be used for characterizing risks associated with acute exposure to sulfometuron methyl.

Risk Characterization – Typical exposures to sulfometuron methyl do not lead to estimated doses that exceed a level of concern. For workers, no exposure scenarios, acute or chronic, exceeds the RfD at the upper ranges of estimated dose associated with the typical application rate of 0.045 lb a.e./acre. For members of the general public, all upper limits for hazard quotients are below a level of concern for the typical application rate. Thus, based on the available information and under the foreseeable conditions of application, there is no route of exposure or scenario suggesting that workers or members of the general public will be at any substantial risk from acute or longer term exposures to sulfometuron methyl.

Irritation and damage to the skin and eyes can result from exposure to relatively high levels of sulfometuron methyl. From a practical perspective, eye or skin irritation is likely to be the only overt effect as a consequence of mishandling sulfometuron methyl. These effects can be minimized or avoided by prudent industrial hygiene practices during the handling of sulfometuron methyl.

ECOLOGICAL RISK ASSESSMENT

Hazard Identification – The mammalian toxicity of sulfometuron methyl is relatively well-characterized in experimental mammals; however, there is relatively little information regarding non-target wildlife species. In standard experimental toxicity studies, sulfometuron methyl has low acute and chronic oral toxicity. It seems reasonable to assume the most sensitive effects in wildlife mammalian species will be the same as those in experimental mammals (i.e., changes to blood and decreased body weight gain). Results of acute exposure studies in birds indicate that avian species appear no more sensitive than experimental mammals to the toxic effects of sulfometuron methyl. Chronic exposure studies in birds were not identified in the available literature. Results of two acute exposure studies in honey bees indicate that bees are no more sensitive than either mammals or birds to sulfometuron methyl. However, the available data are

not sufficient to determine whether this apparent low level of toxicity can be generalized to other species of terrestrial invertebrates.

The toxicity of sulfometuron methyl to terrestrial plants was studied extensively and is well characterized. Sulfometuron methyl inhibits acetolactate synthase (ALS), an enzyme that catalyzes the biosynthesis of three branched-chain amino acids, all of which are essential for plant growth. Bioassays have been conducted on pre-emergence and post-emergence toxicity to several species. Results of both pre-emergent and postemergent bioassays show that terrestrial plants are highly susceptible to the effects of sulfometuron methyl. Concern for the sensitivity of non-target plant species is further increased by field reports of substantial and prolonged damage to crops or ornamentals after the application of sulfometuron methyl in both an arid region, presumably due to the transport of soil contaminated with sulfometuron methyl by wind, and in a region with heavy rainfall, presumably due to the wash-off of sulfometuron methyl contaminated soil. Sulfometuron methyl exposure inhibited growth of several soil microorganisms and caused significant growth inhibition in *Salmonella typhimurium* after exposure periods of less than 3 hours.

As with potential effects on terrestrial species and as would be expected for a herbicide, the available data suggest that sulfometuron methyl is much more toxic to aquatic plants than to aquatic animals. The results of studies in fish suggest that frank toxic effects are not likely to be observed at concentrations less than or equal to 150 mg/L. Sulfometuron methyl also appears to be relatively non-toxic to aquatic invertebrates, based on acute bioassays in daphnids, crayfish, and field-collected species of other aquatic invertebrates. The most sensitive aquatic species tested appears to be the African clawed frog. In acute and chronic exposure studies, exposure to sulfometuron methyl produced alterations in limb development, organogenesis, and metamorphosis. Aquatic plants appear more sensitive than aquatic animals to the effects of sulfometuron methyl, although there appear to be substantial differences in sensitivity among species of macrophytes and unicellular algae. The macrophytes, however, appear to be generally more sensitive. There are no published or unpublished data regarding the toxicity of sulfometuron methyl to aquatic bacteria or fungi. By analogy to the effects on terrestrial bacteria and aquatic algae, it seems plausible that aquatic bacteria and fungi will be sensitive to the effects of sulfometuron methyl.

Exposure Assessment – Terrestrial animals might be exposed to any applied herbicide from direct spray, the ingestion of contaminated media (vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation. In acute exposure scenarios, the highest exposures for terrestrial vertebrates involves the consumption of contaminated insects by a small bird, which could reach up to about 5 mg/kg. There is a wide range of exposures anticipated from the consumption of contaminated vegetation by terrestrial animals: central estimates range from 0.06 mg/kg for a small mammal to 1.2 mg/kg for a large bird under typical exposure conditions, with upper ranges of about 0.1 mg/kg for a small mammal and 3.4 mg/kg for a large bird. The consumption of contaminated water will generally lead to much lower levels of exposure. A similar pattern is seen for chronic exposures. The central estimate for daily doses for a small mammal from the longer term consumption of contaminated vegetation at the application site is about 0.0009 mg/kg/day, with an upper estimate of about 0.004 mg/kg/day.

Longer term exposures from contaminated vegetation far exceed doses that are anticipated from the consumption of contaminated water, which has a central estimate of about 0.0000003 mg/kg/day and an upper range of about 0.0000005 for a small mammal. Based on general relationships of body size to body volume, larger vertebrates will be exposed to lower doses than small vertebrates under comparable exposure conditions. Because of the apparently low toxicity of sulfometuron methyl to animals, the rather substantial variations in the different exposure assessments have little impact on the assessment of risk to terrestrial animals.

For terrestrial plants, five exposure scenarios are considered quantitatively: direct spray, spray drift, runoff, wind erosion and the use of contaminated irrigation water. Unintended direct spray is expressed simply as the application rate considered in this risk assessment, 0.045 lb a.e./acre and should be regarded as an extreme/accidental form of exposure that is not likely to occur in most Forest Service applications. Estimated levels of exposure for the other scenarios are much less. All of these exposure scenarios are dominated by situational variability because the levels of exposure are highly dependent on site-specific conditions. Thus, the exposure estimates are intended to represent conservative but plausible ranges that could occur but these ranges may over-estimate or under-estimate actual exposures in some cases. Spray drift is based on estimates AGDRIFT. The proportion of the applied amount transported off-site from runoff is based on GLEAMS modeling of clay, loam, and sand. The amount of sulfometuron methyl that might be transported off-site from wind erosion is based on estimates of annual soil loss associated with wind erosion and the assumption that the herbicide is incorporated into the top 1 cm of soil. Exposure from the use of contaminated irrigation water is based on the same data used to estimate human exposure from the consumption of contaminated ambient water and involves both monitoring studies as well as GLEAMS modeling.

Exposures of aquatic plants and animals to sulfometuron methyl are based on essentially the same information used to assess the exposure to terrestrial species from contaminated water. The peak estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.001 (0.00006 to 0.02) mg a.e./L at an application rate of 1 lb a.e./acre. For longer-term exposures, average estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.00004 (0.00001 to 0.00007) mg a.e./L at an application rate of 1 lb a.e./acre. For the assessment of potential hazards, these contamination rates are adjusted based on the application rates considered in this risk assessment.

Dose-Response Assessment –For terrestrial mammals, the dose-response assessment for chronic exposure to sulfometuron methyl is based on the same data as the human health risk assessment (i.e., the chronic NOAEL of 2 mg/kg/day from a 2-year feeding study in rats is used to assess chronic risk). All of the potential longer-term exposures of terrestrial mammals to sulfometuron methyl are substantially below the NOAEL of 2 mg/kg/day. For acute exposure, the dose-response assessment is also based on the same data as the human health risk assessment (i.e. the chronic NOAEL in rats of 87 mg/kg/day from a 10-day gestational exposure study is used to assess acute risk). All of the potential acute exposures of terrestrial mammals to sulfometuron methyl are also substantially below the NOAEL of 87 mg/kg/day. Birds appear to exhibit the same low order of toxicity to sulfometuron methyl as mammals, with an acute NOAEL of 312 mg/kg based on changes in body weight observed following a single gavage administration to

mallard ducks. No chronic exposure studies of birds to sulfometuron methyl were identified in the available literature. Since results of acute exposure studies suggest that the sensitivity of birds to sulfometuron methyl is similar to that of mammals, in the absence of chronic exposure data in birds the chronic NOAEL of 2 mg/kg/day in rats is used for birds. For terrestrial invertebrates, based on direct spray studies in honey bees, no mortality would be expected following acute exposure to doses up to 1075 mg/kg. Although limited data are available, soil microorganisms appear sensitive to sulfometuron methyl at concentrations of about 70 µg/L.

The toxicity of sulfometuron methyl to terrestrial plants is relatively well characterized. Sulfometuron methyl is a potent herbicide that causes adverse effects in a variety of target and non-target plant species. Results of pre-emergent and post-emergent application studies in a variety of plant species yield NOELs ranging from 0.0000086 to 0.00078 lbs/acre. For assessing the potential consequences of exposure to nontarget plants via runoff, an LOEC for seedling emergence of 0.0000086 lb/acre is used for sensitive species and the corresponding value for tolerant species is 0.00025 lb/acre. For assessing the impact of drift, an LOEC for vegetative vigor of 0.000024 lb/acre is used for sensitive species and the corresponding value for tolerant species is 0.00078 lb/acre.

The data on toxicity to fish and aquatic invertebrates were obtained in several species. Fish do not appear to be highly sensitive to sulfometuron toxicity. However, investigations of acute toxicity have been hampered by the limited water solubility of sulfometuron methyl. For acute exposures in fish, the NOEC of 7.3 mg a.i./L in fathead minnow is used for the most sensitive species and the NOEC of 150 mg a.i./L in bluegill sunfish and rainbow trout is used for the most tolerant species. However, since both of these values were the highest concentration tested in both studies, identification of a most sensitive and a most tolerant species cannot be made with certainty. Toxicity values for chronic toxicity may be based on the available egg-and-fry/early life stage studies; only one study of chronic exposure in fish is available, a 30-day exposure of fathead minnow yielding an NOAEC of 1.17 mg a.i./L. This value is used for both the most sensitive and tolerant species for chronic exposure. For acute exposure of aquatic invertebrates, the most sensitive species appear to be *Alonella* sp. and *Cypria* sp., with LOAEC values of 75 mg a.i./L. *Daphnia* are the most tolerant species, with an NOEAC of 1800 mg a.i./L. Comparison of LOAEC values for *Daphnia* (2400 mg a.i./L) and *Alonella* and *Cypria* (75 mg a.i./L) show that *Daphnia* have a relative potency factor of 32 (i.e. *Daphnia* are 32 times more tolerant than *Alonella* and *Cypria* to acute exposure of sulfometuron methyl). For chronic exposure of aquatic invertebrates, data are only available from a single study in *Daphnia* with an NOAEC of 6.1 mg/L. This value is used for the most tolerant species for chronic exposure. Although no data are available to determine the most sensitive species for chronic exposures, parallels can be drawn to the acute exposure studies. As discussed above, the relative potency factor comparing *Daphnia* to *Alonella* and *Cypria* based on acute LOAEC values is 32. Using the relative potency factor for acute exposures of 32 and the chronic NOEC in *Daphnia* of 6.1 mg/L, an NOAEC for *Alonella* and *Cypria* is estimated to be 0.19 mg/L. This surrogate NOAEC for chronic exposure in *Alonella* and *Cypria* will be used to estimate the chronic NOAEC for the most sensitive species.

Aquatic plants appear to be much more sensitive to sulfometuron methyl than aquatic animals.

An NOAEC for growth inhibition of 0.00021 mg/L in duckweed is used to quantify effects for both acute and chronic exposure in aquatic macrophytes. Data are available also available in *Hydrilla* and yield a similar NOAEC. However, based on the limited data available as well as difference in experimental protocols, it is not possible to identify a most sensitive and most tolerant species for aquatic macrophytes. For algae, the most sensitive algal species appears to be *Selenastrum capricornutum*, with a 72-hour NOEC of 0.0025 mg/L and the most tolerant species appears to be *Navicula pelliculosa*, both with a 120-hour NOEC of 0.37 mg/L. The same data are used to quantify risk for both acute and chronic exposures.

Risk Characterization – Sulfometuron methyl is an effective and potent herbicide. Adverse effects on some nontarget terrestrial plant species and, to a lesser degree, some aquatic plant species are plausible under some conditions. For terrestrial plants, the dominant factor in the risk characterization is the potency of sulfometuron methyl relative to the application rate. The typical application rate considered in this risk assessment, 0.045 lb/acre, is about 1875 times higher than the NOEC in the vegetative vigor (direct spray) assay of the most sensitive non-target species – i.e., 0.000024 lb/acre – and almost 60 times higher than the NOEC for the most tolerant species in the same assay – i.e., 0.00078 lb/acre. The highest application rate that may be considered in Forest Service programs – i.e., 0.38 lb/acre – is over 15,000 times the NOEC in sensitive species and a factor of about 490 above the NOEC in tolerant species. Given these relationships, damage to sensitive nontarget species could be expected in ground broadcast applications at distances of about 900 feet from the application site in areas in which off-site drift is not reduced by foliar interception. This risk characterization applies only to ground broadcast applications. When used in directed foliar applications (i.e., backpack), offsite drift could be reduced substantially but the extent of this reduction cannot be quantified.

The NOEC values for soil exposures (assayed in the seedling emergence test) are 0.0000086 lb/acre for sensitive species and 0.00026 lb/acre for tolerant species. The offsite movement of sulfometuron methyl via runoff could be substantial under conditions that favor runoff – i.e., clay soils – and hazard quotients in the range of about 90 to nearly 2900 are estimated for sensitive species over a wide range of rainfall rates – i.e., 15 inches to 250 inches per year. In very arid regions in which runoff might not be substantial, wind erosion could result in damage to nontarget plant species. The plausibility of observing such damage would, however, be highly dependent on local conditions. This risk characterization for the potential effects of runoff would be applicable to either broadcast ground or directed foliar applications.

Damage to aquatic plants, particularly macrophytes, appears substantially less than for terrestrial plants. All hazard quotients for aquatic macrophytes were based on an NOEC of 0.00021 mg/L in duckweed for both acute and chronic exposures. No sensitive or tolerant species were identified. Except for the hazard quotient of 4 associated with acute exposures based on the peak concentrations of sulfometuron methyl, all hazard quotients are below the level of concern, with a range of 0.01 to 4 for acute exposures and 0.002 to 0.01 for chronic exposures. Thus, if sulfometuron methyl is applied in areas where transport to water containing aquatic macrophytes is likely, it would be plausible that detectable but transient damage could be observed.

Aquatic algae do not appear to be as sensitive to sulfometuron methyl. The highest hazard

quotient observed for acute exposure is 0.4 associated with the upper range for the most sensitive species, based on an NOEC for growth inhibition. For chronic exposures, the highest hazard quotient is 0.001 associated with the upper range for the most sensitive species. Both values were based on an acute NOEC. Therefore, it is not anticipated that adverse effects in aquatic algae would result from exposure to sulfometuron methyl at application rates used by the Forest Service.

There is no clear basis for suggesting that effects on terrestrial animals are likely or would be substantial. Adverse effects in mammals, birds, terrestrial insects, and microorganisms are not likely using typical or worst-case exposure assumptions at the typical application rate of 0.045 lb a.e./acre. The hazard quotients associated with the upper range for chronic consumption of vegetation by a large mammal (hazard quotient = 0.2) or large bird (hazard quotient = 0.3) feeding exclusively on treated vegetation slightly exceeds the level of concern of 0.1 associated with the maximum application rate of 0.38 lb a.e./acre. As with the human health risk assessment, this characterization of risk must be qualified. Sulfometuron methyl has been tested in only a limited number of species and under conditions that may not well-represent populations of free-ranging non-target species. Notwithstanding this limitation, the available data are sufficient to assert that no adverse effects are anticipated in terrestrial animals.

Similarly, the risk characterization for aquatic animals is relatively simple and unambiguous. Sulfometuron methyl appears to have a very low potential to cause any adverse effects in aquatic animals. All of the hazard quotients for aquatic animals are extremely low, with a range of 0.000000002 (lower range for acute exposures in tolerant aquatic invertebrates) to 0.004 (longer-term exposures to amphibians). It should be noted that confidence in this risk characterization is reduced by the lack of chronic toxicity studies in potentially tolerant fish and potentially sensitive aquatic invertebrates and lack of data in amphibians (data only available in a single species). Even with these uncertainties, there is no basis for asserting that adverse effects on aquatic animals are likely.

1. INTRODUCTION

The USDA Forest Service uses sulfometuron methyl in its vegetation management programs. This document is an update to a risk assessment prepared in 1998 (SERA 1998) and provides risk assessments for human-health effects and ecological effects to support an assessment of the environmental consequences of these uses.

This document has four chapters, including the introduction, program description, risk assessment for human health effects, and risk assessment for ecological effects or effects on wildlife species. Each of the two risk assessment chapters has four major sections, including an identification of the hazards associated with sulfometuron methyl and its commercial formulation, an assessment of potential exposure to the product, an assessment of the dose-response relationships, and a characterization of the risks associated with plausible levels of exposure. These are the basic steps recommended by the National Research Council of the National Academy of Sciences (NRC 1983) for conducting and organizing risk assessments.

Although this is a technical support document and addresses some specialized technical areas, an effort was made to ensure that the document can be understood by individuals who do not have specialized training in the chemical and biological sciences. Certain technical concepts, methods, and terms common to all parts of the risk assessment are described in plain language in a separate document (SERA 2001).

The human health and ecological risk assessments presented in this document are not, and are not intended to be, comprehensive summaries of all of the available information. No published reviews regarding human health or ecological effects of sulfometuron methyl have been encountered. Moreover, almost all of the mammalian toxicology studies and most of the ecotoxicology studies are unpublished reports submitted to the U.S. EPA as part of the registration process for sulfometuron methyl.

Because of the lack of a detailed, recent review concerning sulfometuron methyl and the preponderance of unpublished relevant data in U.S. EPA files, a complete search of the U.S. EPA FIFRA/CBI files was conducted. Full text copies of relevant studies were kindly provided by the U.S. EPA Office of Pesticide Programs. These studies were reviewed and are discussed in Sections 3 and 4 as necessary. A synopsis of the most relevant studies are provided in the appendices to this document.

While this document discusses the studies required to support the risk assessments, it makes no attempt to summarize all of the information. The Forest Service will update this and other similar risk assessments on a periodic basis and welcomes input from the general public on the selection of studies included in the risk assessment. This input is helpful, however, only if recommendations for including additional studies specify why and/or how the new or not previously included information would be likely to alter the conclusions reached in the risk assessments.

For the most part, the risk assessment methods used in this document are similar to those used in

risk assessments previously conducted for the Forest Service as well as risk assessments conducted by other government agencies. Details regarding the specific methods used to prepare the human health risk assessment are provided in SERA (2001).

Variability and *uncertainty* may be dominant factors in any risk assessment, and these factors should be expressed. Within the context of a risk assessment, the terms *variability* and *uncertainty* signify different conditions.

Variability reflects the knowledge of how things may change. Variability may take several forms. For this risk assessment, three types of variability are distinguished: *statistical*, *situational*, and *arbitrary*. *Statistical variability* reflects, at least, apparently random patterns in data. For example, various types of estimates used in this risk assessment involve relationships of certain physical properties to certain biological properties. In such cases, best or maximum likelihood estimates can be calculated as well as upper and lower confidence intervals that reflect the statistical variability in the relationships. *Situational variability* describes variations depending on known circumstances. For example, the application rate or the applied concentration of a herbicide will vary according to local conditions and goals. As discussed in the following section, the limits on this variability are known and there is some information to indicate what the variations are. In other words, *situational variability* is not random. *Arbitrary variability*, as the name implies, represents an attempt to describe changes that cannot be characterized statistically or by a given set of conditions that cannot be well defined. This type of variability dominates some spill scenarios involving either a spill of a chemical on to the surface of the skin or a spill of a chemical into water. In either case, exposure depends on the amount of chemical spilled and the area of skin or volume of water that is contaminated.

Variability reflects a knowledge or at least an explicit assumption about how things may change, while *uncertainty* reflects a lack of knowledge. For example, the focus of the human health dose-response assessment is an estimation of an “acceptable” or “no adverse effect” dose that will not be associated with adverse human health effects. For sulfometuron methyl and for most other chemicals, however, this estimation regarding human health must be based on data from experimental animal studies, which cover only a limited number of effects. Generally, judgment is the basis for the methods used to make the assessment. Although the judgments may reflect a consensus (i.e., be used by many groups in a reasonably consistent manner), the resulting estimations of risk cannot be proven analytically. In other words, the estimates regarding risk involve uncertainty.

In considering different forms of variability, almost no risk estimate presented in this document is given as a single number. Usually, risk is expressed as a central estimate and a range, which is sometimes very large. Because of the need to encompass many different types of exposure as well as the need to express the uncertainties in the assessment, this risk assessment involves numerous calculations. Some of the calculations are relatively simple and are included in the body of the document. Some sets of the calculations, however, are cumbersome. For those calculations, worksheets are included with this risk assessment. The worksheets provide the detail for the estimates cited in the body of the document. As detailed in SERA (2003a), two versions of the worksheets are available: one in a word processing format (Supplement 1) and one in a

spreadsheet format (Supplement 2). The worksheets that are in the spreadsheet format are used only as a check of the worksheets that are in the word processing format. Both sets of worksheets are provided with the hard-text copy of this risk assessment as well as with the electronic version of the risk assessment.

2. PROGRAM DESCRIPTION

2.1. Overview

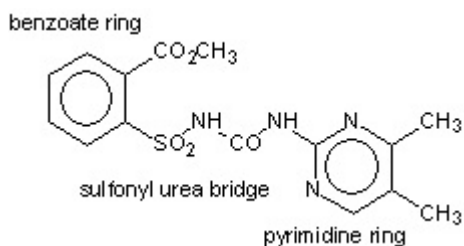
Sulfometuron methyl is a non-selective, sulfonyl urea herbicide used in the control the growth of broadleaf weeds and grasses. The only commercial formulations of sulfometuron methyl used by the Forest Service are Oust and Oust XP[®]. Oust and Oust XP are manufactured by Du Pont as a water dispersible granule. The composition of the product is 75% sulfometuron methyl and 25% inert ingredients.

Sulfometuron methyl is used in Forest Service programs primarily for the control of noxious weeds. Minor uses include conifer release and rights-of-way management. The most common methods of ground application for Oust and Oust XP involve backpack (selective foliar) and boom spray (broadcast foliar) operations. The Forest Service does not use aerial applications for Oust or Oust XP. Nonetheless, both formulations are registered for aerial applications and aerial applications are included in this risk assessment in the event the Forest Service may wish to consider this application method.

The labeled application rates for sulfometuron methyl range from 0.047 to 0.38 lb/acre. Typically, the Forest Service uses rates in the lower part of this range and some applications may be below the lower range of the labeled rate. For this risk assessment, the typical rate of 0.045 lbs/acre. A range of application rates will be taken as 0.03 lbs/acre to 0.38 lbs/acre to reflect plausible ranges that the Forest Service may use. An upper range of 0.38 lb/acre is used to assess the consequences of using the highest labeled rate should the Forest Service need to consider this option. The lower range is the lowest rate reported by the Forest Service.

2.2. Chemical Description and Commercial Formulations

Oust and Oust XP are the commercial formulations of sulfometuron methyl, a non-selective sulfonyl urea herbicide. Sulfometuron methyl is the common name for 2-[[[(4,6-dimethyl-2-pyrimidinyl)- amino] carbonyl] amino] sulfonyl] benzoic acid methyl ester and is essentially a methyl ester of a benzoate ring linked to a dimethyl substituted pyrimidine ring by a sulfonyl urea bridge:



Selected chemical and physical properties of sulfometuron methyl are summarized in Table 2-1. Additional information is presented in worksheet B03.

Oust[®] and Oust XP[®] are the only formulations of sulfometuron methyl used by the Forest Service. Both are formulated as a dry flowable water dispersible granule, which are mixed with

water and applied as a spray (section 2.4), containing 75% (w/w) sulfometuron methyl and 25% (w/w) inerts. The identity of all inerts in Oust XP has been disclosed to the U.S. EPA as part of the registration process and this information has been reviewed in the preparation of this risk assessment (DuPont Agricultural Products 1999). This information is classified as CBI (confidential business Information) under Section 7(d) and Section (10) of FIFRA. Except as noted below, this information cannot be specifically disclosed in this risk assessment. Since the inerts are not identified on the general product label (Du Pont 1999) or the general material safety data sheet (Du Pont 2002), the lack of disclosure indicates that none of the inerts are classified as hazardous. Nonetheless, as discussed by Levine (1996), the testing requirements for inerts are less rigorous than the testing requirements for active ingredients (i.e., sulfometuron methyl). The identity of inert ingredients for the sulfometuron methyl formulation Oust have been disclosed. The Northwest Coalition for Alternatives to Pesticides (NCAP) has obtained information on the identity of the inerts in Oust from U.S. EPA under the Freedom of Information Act and has listed this information on the NCAP web site (<http://www.pesticide.org/FOIA/clopyralid.html>). The inerts listed in this web site are sucrose, sodium salt of naphthalene-sulfonic acid formaldehyde condensate, polyvinyl pyrrolidone, sodium salt of sulfated alkyl carboxylated and sulfated alkyl naphthalene, and hydroxypropyl methylcellulose. However, the quantity of these inert compounds in the formulation is confidential and cannot be disclosed. The potential risks associated with the inerts in Oust formulations are discussed in Section 3.1.14.

Oust and Oust XP are used in forestry applications to control the growth of broadleaf weeds and grasses. Oust XP has no labeled uses for crops (Du Pont 1999a,b). According to the product label (Du Pont 1999a,b), when Oust XP is used in forest planting sites, periods of 60 days to 13 months after treatment are recommended before planting conifers. The use of a surfactant with Oust XP is not recommended. A cautionary note on the product label indicates that if a surfactant is used with Oust XP, contact with tree foliage may result in tree injury or death.

2.3. Application Methods

The most common methods of ground application for Oust and Oust XP involve backpack (selective foliar) and boom spray (broadcast foliar) operations. In selective foliar applications, the herbicide sprayer or container is carried by backpack and the herbicide is applied to selected target vegetation. Application crews may treat up to shoulder high brush, which means that chemical contact with the arms, hands, or face is plausible. To reduce the likelihood of significant exposure, application crews are directed not to walk through treated vegetation. Usually, a worker treats approximately 0.5 acre/hour with a plausible range of 0.25-1.0 acre/hour (USDA 1989a,b,c).

Boom spray is used primarily in rights-of-way management. Spray equipment mounted on tractors or trucks is used to apply the herbicide on either side of the roadway. Usually, about 8 acres are treated in a 45-minute period (approximately 11 acres/hour). Some special truck mounted spray systems may be used to treat up to 12 acres in a 35-minute period with approximately 300 gallons of herbicide mixture (approximately 21 acres/hour and 510 gallons/hour) (USDA 1989a, p. 2-9 to 2-10).

Oust XP is registered for aerial applications (Du Pont 1999a,b). Although this is not an application method that the Forest Service will typically employ for Oust or Oust XP, this method is covered by this risk assessment in the event that the Forest Service may need to consider aerial applications. Aerial applications may be made using helicopters. Oust and Oust XP are applied under pressure through specially designed spray nozzles and booms. The nozzles are designed to minimize turbulence and maintain a large droplet size, both of which contribute to a reduction in spray drift. In aerial applications, approximately 40–100 acres may be treated per hour (USDA 1989a,b,c).

2.4. Mixing and Application Rates

The specific application rates used in a ground application vary according to local conditions and the nature of the target vegetation. Application rates of Oust and Oust XP are expressed in ounces or pounds per acre. An application rate of 1 to 8 ounces of Oust XP per acre is recommended on the product labels (Du Pont 1999a,b). Given that both formulations contain 75% sulfometuron methyl by weight, these rates correspond to 0.75 to 6 ounces or 0.047 to 0.38 pounds of sulfometuron methyl per acre.

The use of sulfometuron methyl in Forest Service Programs for fiscal year 2001, the most recent year for which data are available, is summarized in Table 2-2. Sulfometuron methyl is used currently in Forest Service Programs primarily in conifer release (approximately 66% of the total number of pounds used). Smaller amounts are used for site preparation (19%), rights-of-way management (11%), and noxious weed control (4%). Based on the total amount used and number of acres treated, the application rates are approximately 0.030 lbs/acre for conifer release, 0.041 lbs/acre for noxious weed control, 0.150 lbs/acre for rights-of-way management, and 0.090 lbs/acre for site preparation. None of these application rates exceeds the maximum application rate recommended on the product labels (Du Pont 1999a,b).

For this risk assessment, the typical application rate for sulfometuron methyl will be taken as 0.045 lbs/acre. This is about the average value of all applications conducted by the Forest Service in 2001. The range of application rates will be taken as 0.03 lbs/acre to 0.38 lbs/acre to reflect plausible ranges that the Forest Service may use. The upper bound of the application rate will be taken as 0.38 lbs/acre, the maximum labeled rate for Oust XP. The lower bound of the application is the lowest rate reported by the Forest Service (Table 2-3). The worksheets that accompany this risk assessment are based on the typical application rate of 0.045 lb/acre rather than the full range of application rates. The consequences of varying application rates within the range of 0.030 to 0.38 lb/acre is considered in the risk characterization for human health (Section 3.4) and ecological effects (Section 4.4).

For forestry applications, mixing volumes of 5 to 40 gallons of water per acre are recommended, depending upon the application method. Recommended mixing volumes for ground broadcast applications range from 15 to 40 gallons of water per acre (Du Pont 1999a,b). For aerial applications, recommended mixing volumes are 5 to 15 gallons of water per acre (Du Pont 1999a,b). For this risk assessment, the extent to which the Oust or Oust XP formulation is diluted prior to application primarily influences dermal and direct spray scenarios, both of which are dependent on the ‘field dilution’ (i.e., the concentration of sulfometuron methyl in the applied

spray). The higher the concentration of sulfometuron methyl, the greater the risk. For this risk assessment, the lowest dilution will be taken at 5 gallons/acre, the minimum recommended for ground broadcast applications. The highest dilution (i.e., that which results in the lowest risk) will be based on 40 gallons of water per acre, the highest application volume recommended for both ground broadcast and aerial applications.

It should be noted that the selection of application rates and dilution volumes in this risk assessment is intended to simply reflect typical or central estimates as well as plausible lower and upper bounds. In the assessment of specific program activities, the Forest Service will use program specific application rates in the worksheets that are included with this report to assess any potential risks for a proposed application.

2.5. Use Statistics

The USDA Forest Service (USDA/FS 2002) tracks and reports use by geographical areas referred to as “*Regions*”. As illustrated in Figure 2-1, the Forest Service classification divides the U.S. into nine regions designated from Region 1 (Northern) to Region 10 (Alaska). [Note: There is no *Region 7* in the Forest Service system.] As illustrated in Figure 2-1 and detailed further in Table 2-3, the heaviest use of sulfometuron methyl occurs in Region 8 (Southern), followed by Region 9 (Eastern) and Region (Southwestern). Small quantities of sulfometuron methyl are used in the Region 1 (Northern), and Region 4 (Intermountain). Sulfometuron methyl is not used at all in Region 1 (Northern), Region 2 (Rocky Mountain), Region 3 (Southwestern), Region 5 (Pacific Southwest), or Region 6 (Pacific Northwest).

Sulfometuron methyl is not registered for use in agriculture. National production and use data on sulfometuron methyl have not been encountered in the open literature. In California, approximately 11,500 pounds of the sulfometuron methyl were applied in 2001 (California Department of Pesticide Regulation 2002). About 65% of the sulfometuron methyl was applied in rights-of-way management, about 25% was applied landscape maintenance and about 8% was applied in timberland. While sulfometuron methyl was not used by the Forest Service in California during 2001 (Table 2-3), it should be noted that the total use of sulfometuron methyl by the Forest Service in 2001 was about 57 lbs (Table 2-3), about 0.5 % of the total use of sulfometuron methyl in California during 2001. Thus, the Forest Service does not appear to use substantial amounts of sulfometuron methyl compared to the total use of sulfometuron methyl by other groups.

3. HUMAN HEALTH RISK ASSESSMENT

3.1 HAZARD IDENTIFICATION

3.1.1 Overview. There are several subchronic and chronic studies regarding exposure to sulfometuron methyl in the available literature. Although the mechanism of phytotoxic action of sulfonylurea herbicides including sulfometuron methyl is fairly well characterized, the mechanism of toxicity of sulfometuron methyl in mammals or other animal species is not well characterized. In experimental mammals, the acute oral LD₅₀ for sulfometuron methyl is greater than 17,000 mg/kg, which indicates a low order of toxicity. The lowest dose reported to cause any apparent effects after single gavage administration to rats is 5000 mg/kg. Acute exposure studies of sulfometuron methyl and the sulfometuron methyl formulation, Oust, give similar results, indicating that formulations of sulfometuron methyl are not more toxic than sulfometuron methyl alone. The most common signs of toxicity involve changes in blood that are consistent with hemolytic anemia (i.e., a lysis or destruction of blood cells that results in a decreased number of red blood cells) and decreased body weight gain. It is plausible that the hemolytic anemia caused by sulfometuron methyl is attributable, at least partially, to sulfonamide and saccharin, which are metabolites of sulfometuron methyl. In one study, the investigators observed several effects, in addition to changes in the blood, in dogs exposed to dietary concentrations of sulfometuron methyl for 1 year. These effects, which included increased alkaline phosphatase activity, increased serum cholesterol (females only), decreased serum albumin and creatinine, as well as changes in liver and thymus weights, were not, however, clearly attributable to sulfometuron methyl exposure. In chronic feeding studies with rats, mice and dogs and in several *in vitro* assays, sulfometuron methyl did not display carcinogenic or mutagenic activity.

There is some concern regarding potential reproductive and teratogenic effects from exposure to sulfometuron methyl. Gavage studies in rabbits suggest that sulfometuron methyl exposure may increase the number of fetuses with anomalies as well as the proportion of fetal anomalies per litter. In addition to the two teratogenicity studies in rabbits, there are three reproduction studies involving dietary exposure of rats to sulfometuron methyl, in which effects were observed in dams (decreases in maternal body weight gain associated with decreased food consumption) and offspring (decreased fetal weight, decreased numbers of pups, and decreases in brain weights). As detailed in the dose-response assessment, these effects were not consistently dose-related and do not appear to be the most sensitive effect for sulfometuron methyl.

Both sulfometuron methyl and the commercial formulations Oust and Oust XP, can cause skin and eye irritation. Although a direct comparison between the irritant effects of sulfometuron methyl and the irritant effects of Oust is precluded by the use of different exposure levels in the available studies, there appears to be no remarkable difference. Neither sulfometuron methyl nor Oust caused sensitization following repeated dermal exposure. The inhalation toxicity of sulfometuron methyl is not well documented in the literature. Sulfometuron methyl and Oust can induce irritant effects and possibly systemic toxic effects at very high exposure levels. The potential inhalation toxicity of sulfometuron methyl, however, is not of substantial concern to this risk assessment because of the implausibility of inhalation exposure involving high concentrations of this compound.

Limited information is available on the toxicokinetics of sulfometuron methyl. The kinetics of absorption of sulfometuron methyl following dermal, oral or inhalation exposure are not documented in the available literature. In both mammals and bacteria, sulfometuron methyl is degraded by cleavage of the sulfonyl urea bridge to form sulfonamide and a dimethyl pyrimidine urea or pyrimidine amine. Sulfonamide may be further degraded by demethylation to the free benzoic acid which, in turn, may undergo a condensation reaction to form saccharin. Sulfometuron methyl does not appear to concentrate in tissues and is eliminated fairly rapidly, with a half-life in goats ranging from 28 to 40 hours. In goats, nearly all of the administered sulfometuron methyl dose was excreted in urine. Studies on the toxicity of sulfometuron methyl metabolites have not been conducted, however, the toxicity of the metabolites of sulfometuron methyl is likely to be encompassed by the available mammalian toxicity studies.

As discussed in the exposure assessment, skin absorption is the primary route of exposure for workers. Data regarding the dermal absorption kinetics of sulfometuron methyl are not available in the published or unpublished literature. For this risk assessment, estimates of dermal absorption rates – both zero order and first order – are based on quantitative structure-activity relationships. These estimates of dermal absorption rates are used in turn to estimate the amounts of sulfometuron methyl that might be absorbed by workers, which then are used with the available dose-response data to characterize risk. The lack of experimental data regarding dermal absorption of sulfometuron methyl adds substantial uncertainties to this risk assessment. Uncertainties in the rates of dermal absorption, although they are substantial, can be estimated quantitatively and are incorporated in the human health exposure assessment.

3.1.2 Mechanism of Action

Although the mechanism of phytotoxic action of sulfonylurea herbicides including sulfometuron methyl is characterized in some detail (section 4.1.2.5), the mechanism of toxic action in mammals or other animal species is not well characterized.

As noted in the recent review on sulfometuron methyl by Cox (1993) and described in detail by Melander et al. (1989), several of the sulfonylureas are biologically active in humans and are used or were considered for use in the treatment of non-insulin-dependent diabetes mellitus (NIDDM or type 2 diabetes). A variety of sulfonylureas reduce blood glucose stimulating the release of insulin from pancreatic B cells, and some sulfonylureas may reduce the hepatic extraction of insulin. Secondarily, some sulfonylureas may affect levels of blood cholesterol and serum triglycerides. Sulfometuron methyl was not tested specifically for effects on glucose metabolism or cholesterol. With the exception of an increased level of serum cholesterol in female dogs (Wood and O'Neal 1983), there is no information indicating a relationship between this spectrum of effects and exposure to sulfometuron methyl.

It is plausible that some and perhaps most of the toxic effects observed in the studies on sulfometuron methyl are attributable to its metabolites. As summarized in section 3.1.3, hemolytic anemia is the most consistent systemic effect of exposure to sulfometuron methyl. As discussed further in section 3.3 (dose-response assessment), this effect is also the most sensitive (i.e., the adverse effect that occurs at the lowest dose). There is no information in the available literature suggesting that anemia is associated with the pyrimidine metabolites of sulfometuron

methyl. Recently, however, exposure to sulfonamides, was associated ($p=0.004$) with the development of hemolytic anemia in humans (Issaragrisil et al. 1997). This finding is supported by an earlier, more qualitative association of sulfonamide with anemia in humans (Dickerman 1981). Moreover, saccharin was shown to cause hematological effects in mice (Prasad and Rai 1987) that were similar to the hematological effects of sulfometuron methyl in rats (section 3.2.3). The doses of saccharin associated with the effects in mice—500, 1000, and 1500 mg/kg/day—are much higher than the doses of sulfometuron methyl that caused similar effects in rats and dogs (i.e., 20-30 mg/kg/day) (section 3.3). However, no mechanism of action for this effect has been identified.

3.1.3 Kinetics and Metabolism

Limited information is available on the toxicokinetics of sulfometuron methyl. The kinetics of absorption of chlorsulfuron following dermal, oral or inhalation exposure are not documented in the available literature. The lack of experimental data regarding the dermal absorption of sulfometuron methyl adds substantial uncertainties to this risk assessment. Nonetheless, the available laboratory data in rabbits and guinea pigs, albeit relatively sparse, do not suggest that sulfometuron methyl is likely to be absorbed through the skin in amounts that may cause systemic toxic effects (Appendix 1, Dermal Administration Studies). Uncertainties in the rates of dermal absorption, although they are substantial, can be estimated quantitatively and are incorporated in the human health exposure assessment (section 3.2).

Dermal exposure scenarios involving immersion or prolonged contact with chemical solutions use Fick's first law and require an estimate of the permeability coefficient, K_p , expressed in cm/hour. Using the method recommended by U.S. EPA (1992), the estimated dermal permeability coefficient for chlorsulfuron is 0.0000005 cm/hour with a 95% confidence interval of 0.0000001-0.000002 cm/hour. These estimates are used in all exposure assessments that are based on Fick's first law. The calculations for these estimates are presented in worksheet B05.

For exposure scenarios like direct sprays or accidental spills, which involve deposition of the compound on the skin's surface, dermal absorption rates (proportion of the deposited dose per unit time) rather than dermal permeability rates are used in the exposure assessment. The estimated first-order dermal absorption coefficient is 0.000087 hour⁻¹ with 95% confidence intervals of 0.000012-0.00063 hour⁻¹. The calculations for these estimates are presented in Worksheet B04.

An overview of the metabolism of sulfometuron methyl is presented in Figure 3-1. Because of the apparent similarities in metabolism of the compound by mammals and environmental media, information on both mammalian metabolism and environmental transformation are summarized in the figure.

In both mammals and bacteria, sulfometuron methyl is degraded by cleavage of the sulfonyl urea bridge to form sulfonamide and a dimethyl pyrimidine urea or pyrimidine amine. Sulfonamide may be further degraded by demethylation to the free benzoic acid which, in turn, may undergo a condensation reaction to form saccharin. At least in bacteria, the pyrimidine metabolites may be degraded further to hydroxypyrimidine amine and pyrimidine-ol. Although data regarding

mammalian metabolism of sulfometuron methyl are limited, there is an apparent qualitative difference between mammalian and microbial metabolism that involves changes to sulfometuron methyl prior to cleavage of the sulfonyl urea bridge. In mammals, the major metabolic route seems to involve hydroxylation of a methyl group on the pyrimidine ring (Keoppe and Mucha 1991); in bacteria, the major metabolic pathway seems to involve demethylation of the methyl ester group on the benzoate ring (Monson and Hoffman 1990).

There is only one detailed study regarding the metabolism of sulfometuron methyl by mammals. Keoppe and Mucha (1991) examined the metabolism of sulfometuron methyl in two lactating goats. The sulfometuron methyl used in the study was double labeled: pyrimidine-2-¹⁴C- and uniformly labeled phenyl ring. It was administered as capsules, 0.575 or 0.625 mg/kg, twice a day for 7 days. The authors give 'dietary' equivalents, apparently based on differences in food consumption, as 25 and 60 ppm; however, the actual dosing appears to have been by gavage. The animals were sacrificed 20 hours after the last dose. About 94-99% of dose was recovered in the urine, 60% in the form of hydroxysulfometuron methyl (i.e., no cleavage of the sulfonyl urea bridge). Most of the metabolites resulting from cleavage of the sulfonyl urea bridge were recovered in the liver and kidney and were tightly bound to protein. The only other information available on mammalian metabolism of sulfometuron methyl comes from an unpublished DuPont study, which reports half-times of 28 and 40 hours in rats after gavage doses of 16 and 3000 mg/kg, respectively (DuPont 1989). Thus, sulfometuron methyl is eliminated fairly rapidly and does not appear to accumulate in tissues.

3.1.4 Acute Oral Toxicity

Other than standard bioassays for acute toxicity that were conducted as part of the registration process, there is little information regarding the acute toxicity of chlorsulfuron. As summarized in Appendix 1, there are three acute oral studies in rats involving exposure to technical grade sulfometuron methyl (Dashiell and Hall 1980, Dashiell and Hinckle 1980c, Trivits 1979) and one acute oral study in rats involving exposure to the 75% sulfometuron methyl formulations Oust (Filliben 1995a) and Oust XP (Finlay 1999). Sulfometuron methyl doses in these studies ranged from 5,000 to 17,000 mg/kg. Results show that acute oral exposure to sulfometuron methyl has a low order of toxicity. As summarized in Appendix 1, neither mortality nor overt signs of toxicity were observed in rats given single oral doses of up to 17,000 mg/kg (Dashiell and Hall 1980, Dashiell and Hinckle 1980, Trivits 1979). Thus, the LD₅₀ value for sulfometuron methyl is > 17,000 mg/kg (Trivits 1979).

Qualitative assessments of toxicity were also made in all acute toxicity studies. The only effects commonly noted in the treated animals were weight loss and stained or wet perineal (genital) areas. Dashiell and Hall (1980) observed alopecia (hair loss) in male rats but not female rats, and the study by Dashiell and Hinckle (1980) reports an unspecified increase in lung weight in both male and female rats and 'pink thymus' in four of five female rats after a single gavage dose of 5000 mg/kg. It is not clear whether the changes in lung weight were relative to body weight or were absolute.

Comparison of acute toxicity studies of technical grade sulfometuron methyl and the formulations Oust and Oust XP show similar results. Oral administration of up to 5000 mg/kg

Oust (3750 mg a.i./kg) to rats did not result in a single mortality (Filliben 1995a). The only clinical sign of toxicity observed in this study was alopecia in one female rat. Acute oral administration of 5000 mg/kg Oust XP (3750 mg a.i./kg) did not result in any mortalities, clinical signs of toxicity or gross lesions in any animal (Finlay 1999). Thus, like technical grade sulfometuron methyl, acute exposure to the 75% formulations Oust and Oust XP does not appear to result in any significant toxicity.

3.1.5. Subchronic and Chronic Systemic Toxic Effects

Systemic toxicity encompasses virtually any effects that a chemical has after the chemical has been absorbed. Certain types of effects, however, are of particular concern and involve a specific subset of tests. Such special effects are considered in following subsections and include effects on the nervous system (Section 3.1.6), immune system (Section 3.1.7), endocrine function (3.1.8), development or reproduction (Section 3.1.9), and carcinogenicity or mutagenicity (Section 3.1.10). This section encompasses the remaining signs of general and non-specific toxicity.

The subchronic or chronic toxicity of sulfometuron methyl to humans or mammals is not documented in the published literature, and all of the available toxicological data comes from unpublished studies that were conducted to support the registration of sulfometuron methyl as a herbicide. As summarized in Appendix 1, there are two subchronic exposure studies in rats (Hinckle 1979, Wood et al. 1980), and chronic exposure studies in rats (Mullin 1984), mice (Summers 1990a), and dogs (Wood and O'Neal 1983). Subchronic and chronic exposure studies involving reproductive performance assays were also conducted in rats (Lu 1981, Mullin 1984) and rabbits (Hoberman et al. 1981, Serota et al. 1981) and are discussed further in section 3.1.9.

The most common signs of toxicity involve changes in blood (Wood and O'Neal 1983, Summers 1990a, Wood et al. 1980, Mullin 1984) and decreased body weight gain (Hoberman et al. 1981). The changes in the blood appear to be consistent with hemolytic anemia (i.e., a lysis or destruction of blood cells that results in a decreased number of red blood cells). Details of these studies are provided in Appendix 1. In rats, changes in red blood cell parameters were observed following subchronic dietary exposure to 1000 ppm sulfometuron methyl for male rats (NOAEL = 100 ppm) (Wood et al. 1980). In a 2-year feeding study, a NOAEL of 50 ppm for decreased erythrocyte count and hematocrit was observed in male rats (Mullins 1984). A NOAEL of 100 ppm was reported for anemia in female mice exposed to dietary sulfometuron methyl for 18 months (Summers 1990a) and a NOAEL of 200 ppm was reported for hemolytic anemia in dogs exposed to dietary sulfometuron methyl for 1 year (Wood and O'Neal).

No other specific signs of toxicity were noted consistently among the different subchronic or chronic bioassays summarized in Appendix 1. Following exposure of six rats to 3400 mg/kg bw/day sulfometuron methyl for 14 days, the investigators observed reduced testicular size in one rat and mild testicular lesions in another (Hinckle 1979). No such effects were observed in any of the six control rats. In a 1-year dog feeding study, several effects in addition to those on the blood were observed in various dose groups; however, the effects were not considered by the authors to be clearly dose-related (Wood and O'Neal 1983). The potentially significant effects reported in this study include increased alkaline phosphatase activity, increased serum cholesterol

(females only), and decreased serum albumin and creatinine. At dietary concentrations of 5000 ppm, the observed effects include increased absolute liver weights in females and increased relative liver weight in males and females, as well as increased absolute and relative thymus weights in females. Thymus weights were also increased in males at 200 and 1000 ppm but not at 5000 ppm. No pathological changes in the thymus were noted in either sex at any dose level.

3.1.6 Effects on Nervous System

As discussed in Durkin and Diamond (2002), a *neurotoxicant* is chemical that disrupts the function of nerves, either by interacting with nerves directly or by interacting with supporting cells in the nervous system. This definition of *neurotoxicant* is critical because it distinguishes agents that act directly on the nervous system (*direct neurotoxicants*) from those agents that might produce neurologic effects that are secondary to other forms of toxicity (*indirect neurotoxicants*). Virtually any chemical will cause signs of neurotoxicity in severely poisoned animals and thus can be classified as an indirect neurotoxicant. This is the case for sulfometuron methyl in that sulfometuron methyl was reported to cause signs of depression in rabbits exposed to up to 1000 mg/kg by gavage for 13 days (Hoberman et al. 1981). This report, however, does not implicate chlorsulfuron as a direct neurotoxicant.

3.1.7 Effects on Immune System

There is very little direct information on which to assess the immunotoxic potential of sulfometuron methyl. Dermal studies in rabbits show that chlorsulfuron does not produce sensitization (Section 3.1.11). Results of subchronic and chronic exposure studies show that sulfometuron methyl may produce changes to immune system function at high doses. In male rats exposed to 5000 ppm sulfometuron methyl in the diet for 90 days, elevated mean leukocyte and lymphocyte counts and decreased neutrophil count were reported (Wood et al. 1980). No effect on these parameters were observed at dietary concentrations of sulfometuron methyl of 100 and 1000 ppm. Increased thymus weights were observed in female dogs exposed to 5000 ppm and in male dogs exposed to 200 and 1000 ppm, but not 5000 ppm, dietary sulfometuron methyl for 1 year (Wood and O'Neal 1983). However, no pathological changes were observed in the thymus at any dose. While results of these studies suggest that exposure to sulfometuron methyl may produce changes in immune system parameters, the observations in these studies do not provide conclusive evidence supporting the immunotoxic potential of sulfometuron methyl.

3.1.8 Effects on Endocrine System

As noted in Section 3.1.1., a variety of sulfonylureas reduce blood glucose by stimulating the release of insulin from pancreatic B cells, and some sulfonylureas reduce the hepatic extraction of insulin. No studies investigating the effects of sulfometuron methyl on insulin release or metabolism were identified. As noted in Appendix 1 and Appendix 2, weight loss and decreased weight gain are observed in animals treated with sulfometuron methyl, implying a change in metabolic status. However, there is no evidence to suggest that changes in weight are due to effects of sulfometuron methyl on the endocrine system.

As noted in Section 3.1.5, following exposure of six rats to 3400 mg/kg/day sulfometuron methyl for 14 days, reduced testicular size in one rat and mild testicular lesions in another were reported (Hinckle 1979). In a 2-generation reproductive study, a decrease in reproductive performance

was observed in rats 5000 ppm dietary sulfometuron methyl for 90 days, but not at dietary concentrations of 50 and 500 ppm (Mullin 1984). While results of these studies suggest that exposure to sulfometuron methyl may produce changes in the function of the reproductive endocrine system, the observations in these studies do not provide conclusive evidence.

The administration of 2000 mg/kg sulfonamide over a 15-day period caused dose-related changes to the thyroid gland and changes in circulating levels of T3 and T4 in rats (Nishikawa 1983a,b). Elevated serum thyroxine levels have been observed in female rats exposed to 100 and 1000 ppm, but not 5000 ppm, dietary sulfometuron methyl for 90 days (Wood et al. 1980). As discussed in Section 4.1.3.2, a decrease in tail resorption rates, a morphological biomarker of thyroid disruption, was observed in African clawed frogs to 0.001 and 0.01 mg/L sulfometuron methyl for 14 days (Fort 1998). Effects were partially reversed by the administration of thyroxine. Based on results of these studies, it appears that sulfometuron methyl has the potential to produce changes in thyroid gland function. No mechanism has been identified for effects of sulfonamides on thyroid gland function.

3.1.9 Reproductive and Teratogenic Effects

Studies investigating the reproductive effects of sulfometuron methyl in humans or mammals are not documented in the published literature, and all of the available toxicological data comes from unpublished studies that were conducted to support the registration of sulfometuron methyl as a herbicide. As detailed in Appendix 1, studies assessing the reproductive and teratogenic effects of sulfometuron methyl have been conducted in rats (Lu 1981, Mullin 1984, Wood et al. 1980) and rabbits (Hoberman et al. 1981, Serota et al. 1981).

In the two teratogenicity studies in rabbits, sulfometuron methyl was administered by gavage, as shown in Appendix 1. The study by Hoberman et al. (1981) was a range finding study with daily doses of 100-1000 mg/kg, while the study by Serota et al. (1981) involved lower dose levels of 30-300 mg/kg. In the Hoberman et al. (1981) study, signs of maternal toxicity, including death in some dams, were apparent at all dose levels. In the study by Serota et al. (1981), there were no signs of toxicity in the dams or offspring at any exposure level. At the 30 and 100 mg/kg dose levels, an increase in the incidences of fetal anomalies was observed; however, at the 300 mg/kg dose level, there were actually fewer incidences of fetal anomalies than were observed at 100 mg/kg dose level. The authors state that statistical evaluation of all parameters, including fetal anomalies, revealed no statistical differences between the control and sulfometuron methyl treated groups. Given the clear lack of dose-response relationship, the NOAEL for this study for both maternal and fetal toxicity is 300 mg/kg/day.

The three studies in rats involve dietary exposure to sulfometuron methyl (Wood et al. 1980, Lu 1981, Mullin 1984). As Appendix 1 shows, decreases in maternal body weight gain associated with decreased food consumption (Lu 1981, Mullin 1984) and hematological changes (Mullin 1984, Wood et al. 1980) were the common effects observed in these studies. Gestational exposure of rats to 5000 ppm dietary sulfometuron methyl resulted in decreased maternal weight gain and decreased fetal weights, with NOAEL for the dams and fetuses of 1000 ppm (Lu 1981). Exposure of rats for 90 days to dietary levels of 5000 ppm was associated with a decreased number of pups in the F1 and F2 generations (Mullin 1984). In addition to these effects, mean

absolute brain weights were significantly decreased in male rats, with an NOAEL of 500 ppm (Mullin 1984). No adverse effects on reproductive parameters were observed in rats exposed to dietary sulfometuron methyl at dietary concentrations up to 5000 ppm (Wood et al. 1980).

3.1.10 Carcinogenicity and Mutagenicity

Sulfometuron methyl has been tested for mutagenicity in a number of different test systems and has been assayed for carcinogenic activity in rats, mice and dogs. Studies are summarized in Appendix 1. Rats were exposed to dietary sulfometuron methyl at concentrations up to 5000 ppm for one year (Mullin 1984), mice to concentrations up to 1000 ppm for 18 months (Summers), and dogs to concentrations up to 5000 ppm for 1 year. No evidence of carcinogenic activity was found in any sulfometuron methyl chronic exposure study. In all three studies, toxicity was indicated by hematological changes in the high dose groups (Appendix 1). Also, the study by Mullin (1984) reports bile duct hyperplasia and fibrosis in female rats exposed to the two higher dose levels and a significant decrease in mean absolute brain weight in male rats exposed to the highest dose level. Each of these studies can be viewed as involving doses that approximate the maximum tolerated dose based on alterations in body weight and clinical blood indices.

Sulfometuron methyl did not show mutagenic activity in assays in *Salmonella typhimurium* strains TA 1535, TA 1537, TA 98, and TA 100 (Taylor 1979, Taylor and Krahn 1990) and Chinese hamster ovary cells (Krahn and Fitzpatrick 1981). Moreover, sulfometuron methyl did not induce chromosomal damage in Chinese hamster ovary cells (Galloway 1981) or unscheduled DNA synthesis in rat hepatocytes (Ford 1982). These data provide no evidence that exposure to sulfometuron methyl poses a carcinogenic risk to humans.

3.1.11 Irritation and Sensitization (Effects on Skin and Eyes)

Both sulfometuron methyl and the commercial formulations, Oust and Oust XP, were tested for irritant effects on the skin and eyes as well as for sensitization resulting from dermal exposure. Details of all studies are summarized in Appendix 1.

Results of studies in rabbits and rats show that single and repeated dermal application of sulfometuron methyl (Dashiell and Henry 1980a, Dashiell and Hinckle 1983, Dashiell and Silber 1980c, 1981, Sarver 1990b) and single dermal applications of Oust (Filliben 1995b,c) and Oust XP (Finlay 1999b,c) induced skin irritation characterized by mild erythema and mild edema. A direct comparison between the irritant effects of sulfometuron methyl and those of Oust is difficult to make because of dissimilarities in study protocols. Nonetheless, there appears to be no remarkable difference between the irritant effects of sulfometuron methyl and the commercial formulations. Mild skin irritation was observed in guinea pigs exposed to 50% sulfometuron methyl in dimethyl phthalate (Dashiell and Silber 1980b; Edwards 1979a). Neither sulfometuron methyl nor Oust caused sensitization in guinea pigs (Edwards 1979a, Dashiell and Silber 1980a,b, Moore 1995).

Applications of technical grade sulfometuron methyl to the eyes of rabbits produced transient, mild irritant effects to the cornea and conjunctiva, including redness, transient corneal cloudiness, discharge, and chemosis. (Dashiell and Henry 1980a, Edwards, 1979b, Malek 1990).

Although sulfometuron methyl, Oust and Oust XP all cause mild eye irritation (Appendix 1), sulfometuron methyl caused transient corneal opacity in rabbits after ocular instillation of 61.8 mg a.i. (Dashiell and Henry 1980b), an effect not observed in rabbits exposed similarly to Oust at a dose of 46 mg or approximately 34.5 mg a.i. (Filliben 1995d) or Oust XP at a dose of 32 mg or approximately 24 mg a.i. (Finlay 1999b). In all studies, effects were resolved within 72 hours.

3.1.12 Systemic Toxic Effects from Dermal Exposure

Most of the occupational exposure scenarios and many of the exposure scenarios for the general public involve the dermal route of exposure. For these exposure scenarios, dermal absorption is estimated and compared to an estimated acceptable level of oral exposure based on subchronic or chronic toxicity studies. Thus, it is necessary to assess the consequences of dermal exposure relative to oral exposure and the extent to which sulfometuron methyl is likely to be absorbed from the surface of the skin.

Studies on the systemic toxicity of sulfometuron methyl following dermal exposure have been conducted in rabbits and rats (summarized in Appendix 1). Dermal exposure to doses up to 8000 mg/kg technical grade sulfometuron methyl were not associated with any signs of significant systemic toxicity in rabbits (Dashiell and Henry 1980a, Dashiell and Silber 1980c, 1981). Only 1 death, which was not considered to be treatment related, was reported (Dashiell and Silber 1980c). Thus, the LD₅₀ for dermal exposure of chlorsulfuron in rabbits is >8000 mg/kg (Dashiell and Silber 1980c). Dermal exposure to 2000 mg/kg sulfometuron methyl (Dashiell and Silber 1980c; Dashiell and Silber 1981) caused weight loss similar to that observed in rats after acute oral exposure to 5000 mg/kg sulfometuron methyl (Trivits 1979). This effect, however, was not reported in a subchronic dermal study in which doses of up to 2000 mg/kg/day were applied to the intact skin of rabbits for 21 days (Dashiell and Hinckle 1983). Furthermore, none of the dermal studies that examined hematological changes noted any effects. As discussed in sections 3.1.2. and 3.1.3, hematological effects are the most common effects observed after oral exposure to sulfometuron methyl. The results of the dermal studies with Oust in rabbits (Filliben 1995b,c) and Oust XP in rats (Finlay 1999c) suggest that there is no substantial difference between the dermal toxicity of the 75% sulfometuron formulations and technical grade sulfometuron methyl. The LC₅₀ value for dermal applications for both sulfometuron methyl formulations was greater than 5000 mg/kg (equivalent to 3750 mg a.i./kg).

3.1.13 Inhalation Exposure

As summarized in appendix 3, there is only one inhalation toxicity study on sulfometuron methyl (Kinney 1982), one inhalation toxicity study on Oust (Sarver 1995), and one on Oust XP (Bamberger 1999). All three studies involve acute (4-hour) exposure to relatively high concentration levels (>5 mg/L or >5000 mg/m³). Although no toxic effects were observed in rats after head-only exposure to 6.4 or 11 mg/L sulfometuron methyl (Kinney 1982), irritant effects (nasal and ocular discharge) were observed in male rats after head only exposure to 5.1 mg/L Oust (Sarver 1995). Transient weight loss and wet perineum were also observed in the Oust study, which is consistent with the signs of sulfometuron methyl toxicity after oral exposure. Similar transient effects were observed with following 4-hour exposure to Oust XP at a concentration of 5.3 mg/L formulation or about 4 mg a.i./L (Bamberger 1999). The extremely limited data suggest only that sulfometuron methyl can induce irritant effects as well as systemic

toxic effects at very high exposure levels. As discussed in Section 3.2, this finding is not directly relevant to this risk assessment because of the implausibility of exposure to such high concentrations of the compound.

3.1.14 Inerts and Adjuvants

The formulations of sulfometuron methyl used by the Forest Service contains materials other than sulfometuron methyl that are included as adjuvants to improve either efficacy or ease of handling and storage. As discussed in Section 2.2, the identity of these inert materials in Oust XP is confidential. The inerts were disclosed to the U.S. EPA (DuPont Agricultural Products 1999) and were reviewed in the preparation of this risk assessment. All that can be disclosed explicitly is that none of the additives in Oust XP are classified by the U.S. EPA as toxic. As also discussed in Section 2.2, the identity of inert ingredients for the sulfometuron methyl formulation Oust have been disclosed. The Northwest Coalition for Alternatives to Pesticides (NCAP) has obtained information on the identity of the inerts in Escort from U.S. EPA under the Freedom of Information Act and has listed this information on the NCAP web site (<http://www.pesticide.org/FOIA/clopyralid.html>). The inerts listed in this web site are sucrose, sodium salt of naphthalene-sulfonic acid formaldehyde condensate, polyvinyl pyrrolidone, sodium salt of sulfated alkyl carboxylated and sulfated alkyl naphthalene, and hydroxypropyl methylcellulose. Sucrose (CAS No. 57-50-1) is classified by the U.S. EPA as a List 4 inert and therefore, is generally recognized as a safe compound and is approved as a food additive (U.S. EPA 2003). Hydroxypropyl methylcellulose (Cas No. 009004-65-3) is classified as a List 4a inert, which is generally recognized as safe (U.S. EPA 2003). There is no evidence to assert that either sucrose or hydroxypropyl methylcellulose will materially impact the risks associated with the use of sulfometuron methyl. Polyvinyl pyrrolidone (CAS No. 88-12-0) is classified as a List 3 inert (U.S. EPA 2003). In other words, there is insufficient information to categorize this compound as either hazardous (Lists 1 or 2) or non-toxic (List 4). Sodium naphthalene sulfonate-formaldehyde condensate and the mixture of a sulfate of alkyl carboxylate and sulfonated alkyl naphthalene (sodium salt) were not identified in the EPA Inert List (U.S. EPA 2003). Other naphthalene derivatives identified on the EPA Inert List are classified as List 3 or List 4; no naphthalene derivatives are classified as List 1 or List 2 inerts (U.S. EPA 2003). Thus, there is insufficient information available to assess the impact of either polyvinyl pyrrolidone or the naphthalene derivatives on the risks associated with the use of sulfometuron methyl. However, as noted above, the toxicity of Oust and Oust XP appears to be comparable to that of technical grade sulfometuron methyl (Sections 3.1.4, 3.1.11, and 3.1.13). Therefore, there is no plausible basis for asserting that these inerts are present in Oust or Oust XP in toxicological amounts.

As noted in section 2.2, the manufacturers recommend that sulfometuron methyl formulations be mixed with a non-ionic surfactant. There is no published literature or information in the FIFRA files that would permit an assessment of toxicological effects of sulfometuron methyl mixed with surfactant.

3.1.15 Impurities and Metabolites

Virtually no chemical synthesis yields a totally pure product. Technical grade sulfometuron methyl, as with other technical grade products, undoubtedly contains some impurities. To some

extent, concern for impurities in technical grade sulfometuron methyl is reduced by the fact that the existing toxicity studies on sulfometuron methyl were conducted with the technical grade product. Thus, if toxic impurities are present in the technical grade product, they are likely to be encompassed by the available toxicity studies on the technical grade product.

No studies investigating the toxicity of the sulfometuron methyl metabolites produced by mammals were identified in the published literature or unpublished studies. The toxicity of the metabolites of sulfometuron methyl is likely to be encompassed by the available mammalian toxicity studies. As discussed in Section 3.1.3 (Kinetics and Metabolism), metabolites of sulfometuron methyl are rapidly excreted and do not appear to concentrate in any tissue.

3.2. EXPOSURE ASSESSMENT

3.2.1. Overview. Exposure assessments are conducted for both workers and members of the general public for the typical application rate of 0.045 lb/acre. The consequences of using the maximum application rate that might be used by the Forest Service, 0.38 lb/acre, are discussed in the risk characterization.

There are no occupational exposure studies in the available literature that are associated with the application of sulfometuron methyl. Consequently, worker exposure rates are estimated from an empirical relationship between absorbed dose per kilogram of body weight and the amount of chemical handled in worker exposure studies on nine different pesticides. For workers, three types of application methods are generally modeled in Forest Service risk assessments: directed ground, broadcast ground, and aerial. Although Oust and Oust XP are registered for aerial applications (helicopter and sometimes fixed wing), the Forest Service does not currently use this method. Nonetheless, the aerial application method is included in this risk assessment in the event that the Forest Service considers it an option. Central estimates of exposure for ground workers are approximately 0.0006 mg/kg/day for directed ground spray and 0.001 mg/kg/day for broadcast ground spray. Upper range of exposures are approximately 0.004 mg/kg/day for directed ground spray and 0.007 mg/kg/day for broadcast ground spray. All of the accidental exposure scenarios for workers involve dermal exposures and all of these accidental exposures lead to estimates of dose that are either in the range of or substantially below the general exposure estimates for workers.

For the general public, the range of acute exposures is from approximately 0.00000012 (1.2×10^{-7}) mg/kg associated with the lower bound for consumption of contaminated stream water by a child to 0.094 mg/kg/day associated with the upper bound for consumption of contaminated water by a child following an accidental spill of sulfometuron methyl into a small pond. For chronic or longer term exposures, the modeled exposures are much lower than for acute exposures, ranging from approximately 0.0000000023 (2.3×10^{-11}) mg/kg/day associated with the lower range for the normal consumption of fish by the general public to approximately 0.0016 mg/kg/day associated with the upper range for consumption of contaminated fruit.

3.2.2. Workers.

The Forest Service uses a standard set of exposure assessments in all risk assessment documents. While these exposure assessments vary depending on the characteristics of the specific chemical as well as the relevant data on the specific chemical, the organization and assumptions used in the exposure assessments are standard and consistent. All of the exposure assessments for workers as well as members of the general public are detailed in the worksheets on sulfometuron methyl that accompany this risk assessment (Supplement 1). This section on workers and the following section on the general public provides a plain verbal description of the worksheets and discuss sulfometuron methyl specific data that are used in the worksheets.

A summary of the exposure assessments for workers is presented in Worksheet E02 of the worksheets for sulfometuron methyl that accompany this risk assessment. Two types of exposure assessments are considered: general and accidental/incidental. The term *general* exposure assessment is used to designate those exposures that involve estimates of absorbed dose based on

the handling of a specified amount of a chemical during specific types of applications. The accidental/incidental exposure scenarios involve specific types of events that could occur during any type of application. The exposure assessments developed in this section as well as other similar assessments for the general public (Section 3.2.3) are based on the typical application rate of 0.045 lbs a.i./acre (Section 2). The consequences of using different application rates in the range considered by the Forest Service are discussed further in the risk characterization (Section 3.4).

3.2.2.1. General Exposures – As described in SERA (2001), worker exposure rates are expressed in units of mg of absorbed dose per kilogram of body weight per pound of chemical handled. Based on analyses of several different pesticides using a variety of application methods, default exposure rates are estimated for three different types of applications: directed foliar (backpack), boom spray (hydraulic ground spray), and aerial.

The specific assumptions used for each application method are detailed in worksheets C01a (directed foliar), C01b (broadcast foliar), and C01c (aerial). Although Escort is registered for aerial applications (Section 2), this is not an application method that the Forest Service will typically employ for Escort. However, aerial application is covered by this risk assessment in the event that the Forest Service may need to consider aerial applications. In the worksheets, the central estimate of the amount handled per day is calculated as the product of the central estimates of the acres treated per day and the application rate. The typical application rate is taken directly from the program description (see section 2.4). The central estimate of the amount handled per day (0.045 lbs sulfometuron methyl/acre) is calculated as the product of the central estimate of the acres treated per day and the application rate.

No worker exposure studies with sulfometuron methyl were found in the literature. As described in SERA (2001), worker exposure rates are expressed in units of mg of absorbed dose per kilogram of body weight per pound of chemical handled. These exposure rates are based on worker exposure studies on nine different pesticides with molecular weights ranging from 221 to 416 and log K_{ow} values at pH 7 ranging from -0.75 to 6.50. The estimated exposure rates are based on estimated absorbed doses in workers as well as the amounts of the chemical handled by the workers. As summarized in Table 2-1 of this risk assessment, the molecular weight of sulfometuron methyl is 364.38 and the log K_{ow} at pH 7 is approximately -0.46, with both parameters falling within the range defined above (SERA 2001). As described in SERA (2001), the ranges of estimated occupational exposure rates vary substantially among individuals and groups, (i.e., by a factor of 50 for backpack applicators and a factor of 100 for mechanical ground sprayers). It seems that much of the variability can be attributed to the hygienic measures taken by individual workers (i.e., how careful the workers are to avoid unnecessary exposure); however, pharmacokinetic differences among individuals (i.e., how individuals absorb and excrete the compound) also may be important.

An estimate of the number of acres treated per hour is needed to apply these worker exposure rates. These values are taken from previous USDA risk assessments (USDA 1989a,b,c). The number of hours worked per day is expressed as a range, the lower end of which, 6 hours per day, is based on an 8-hour work day with 1 hour at each end of the work day spent in activities that do

not involve herbicide exposure. The upper end of the range, 8 hours per day, is based on an extended (10-hour) work day, allowing for 1 hour at each end of the work day to be spent in activities that do not involve herbicide exposure.

It is recognized that the use of 6 hours as the lower range of time spent per day applying herbicides is not a true lower limit. It is conceivable and perhaps common for workers to spend much less time in the actual application of a herbicide if they are engaged in other activities. Thus, using 6 hours may overestimate exposure. In the absence of any published or otherwise documented work practice statistics to support the use of a lower limit, this approach is used as a protective assumption.

The range of acres treated per hour and hours worked per day is used to calculate a range for the number of acres treated per day. For this calculation as well as others in this section involving the multiplication of ranges, the lower end of the resulting range is the product of the lower end of one range and the lower end of the other range. Similarly, the upper end of the resulting range is the product of the upper end of one range and the upper end of the other range. This approach is taken to encompass as broadly as possible the range of potential exposures.

The central estimate of the acres treated per day is taken as the arithmetic average of the range. Because of the relatively narrow limits of the ranges for backpack and boom spray workers, the use of the arithmetic mean rather than some other measure of central tendency, like the geometric mean, has no marked effect on the risk assessment.

3.2.2.2. Accidental Exposures – Typical occupational exposures may involve multiple routes of exposure (i.e., oral, dermal, and inhalation); nonetheless, dermal exposure is generally the predominant route for herbicide applicators (Ecobichon 1998; van Hemmen 1992). Typical multi-route exposures are encompassed by the methods used in Section 3.2.2.1 on general exposures. Accidental exposures, on the other hand, are most likely to involve splashing a solution of herbicides into the eyes or to involve various dermal exposure scenarios.

Sulfometuron methyl can cause irritant effects to the skin and eyes (see Section 3.1.11). The available literature does not include quantitative methods for characterizing exposure or responses associated with splashing a solution of a chemical into the eyes; furthermore, there appear to be no reasonable approaches to modeling this type of exposure scenario quantitatively. Consequently, accidental exposure scenarios of this type are considered qualitatively in the risk characterization (section 3.4).

There are various methods for estimating absorbed doses associated with accidental dermal exposure (U.S. EPA/ORD 1992; SERA 2001). Two general types of exposure are modeled: those involving direct contact with a solution of the herbicide and those associated with accidental spills of the herbicide onto the surface of the skin. Any number of specific exposure scenarios could be developed for direct contact or accidental spills by varying the amount or concentration of the chemical on or in contact with the surface of the skin and by varying the surface area of the skin that is contaminated.

For this risk assessment, two exposure scenarios are developed for each of the two types of dermal exposure, and the estimated absorbed dose for each scenario is expressed in units of mg chemical/kg body weight. Both sets of exposure scenarios are summarized in Worksheet E01, which references other worksheets in which the specific calculations are detailed.

Exposure scenarios involving direct contact with solutions of the chemical are characterized by immersion of the hands for 1 minute or wearing contaminated gloves for 1 hour. Generally, it is not reasonable to assume or postulate that the hands or any other part of a worker will be immersed in a solution of a herbicide for any period of time. On the other hand, contamination of gloves or other clothing is quite plausible. For these exposure scenarios, the key element is the assumption that wearing gloves grossly contaminated with a chemical solution is equivalent to immersing the hands in a solution. In either case, the concentration of the chemical in solution that is in contact with the surface of the skin and the resulting dermal absorption rate are essentially constant.

For both scenarios (the hand immersion and the contaminated glove), the assumption of zero-order absorption kinetics is appropriate. Following the general recommendations of U.S. EPA/ORD (1992), Fick's first law is used to estimate dermal exposure. As discussed in Section 3.1.3, an experimental dermal permeability coefficient (K_p) for sulfometuron methyl is not available. Thus, the K_p for sulfometuron methyl is estimated using the algorithm from U.S. EPA/ORD (1992), which is detailed in Worksheet A07b. The application of this algorithm to sulfometuron methyl, based on molecular weight and the $k_{o/w}$, is given in Worksheet B04.

Exposure scenarios involving chemical spills onto the skin are characterized by a spill on to the lower legs as well as a spill on to the hands. In these scenarios, it is assumed that a solution of the chemical is spilled on to a given surface area of skin and that a certain amount of the chemical adheres to the skin. The absorbed dose is then calculated as the product of the amount of the chemical on the surface of the skin (i.e., the amount of liquid per unit surface area multiplied by the surface area of the skin over which the spill occurs and the concentration of the chemical in the liquid) the first-order absorption rate, and the duration of exposure.

For both scenarios, it is assumed that the contaminated skin is effectively cleaned after 1 hour. As with the exposure assessments based on Fick's first law, this product (mg of absorbed dose) is divided by body weight (kg) to yield an estimated dose in units of mg chemical/kg body weight. The specific equation used in these exposure assessments is specified in Worksheet B03.

Confidence in these exposure assessments is diminished by the lack of experimental data on the dermal absorption of sulfometuron methyl. Nonetheless, as summarized in Worksheet E01, there is a noteworthy similarity between the exposure scenario in which contaminated gloves are worn for 1 hour (Worksheet C02b) and the exposure scenario in which a chemical solution is spilled on to the skin surface of the hands and cleaned after 1 hour (Worksheet C03a). Confidence in these assessments is enhanced somewhat by the fact that two similar scenarios based on different empirical relationships yield similar estimates of exposure.

3.2.3. General Public.

3.2.3.1. General Considerations – Under normal conditions, members of the general public should not be exposed to substantial levels of sulfometuron methyl. Nonetheless, any number of exposure scenarios can be constructed for the general public, depending on various assumptions regarding application rates, dispersion, canopy interception, and human activity. Several scenarios are developed for this risk assessment which should tend to over-estimate exposures in general.

The two types of exposure scenarios developed for the general public include acute exposure and longer-term or chronic exposure. All of the acute exposure scenarios are primarily accidental. They assume that an individual is exposed to the compound either during or shortly after its application. Specific scenarios are developed for direct spray, dermal contact with contaminated vegetation, as well as the consumption of contaminated fruit, water, and fish. Most of these scenarios should be regarded as extreme, some to the point of limited plausibility. The longer-term or chronic exposure scenarios parallel the acute exposure scenarios for the consumption of contaminated fruit, water, and fish but are based on estimated levels of exposure for longer periods after application.

The exposure scenarios developed for the general public are summarized in Worksheet E03. As with the worker exposure scenarios, details of the assumptions and calculations involved in these exposure assessments are given in the worksheets that accompany this risk assessment (Worksheets D01–D09). The remainder of this section focuses on a qualitative description of the rationale for and quality of the data supporting each of the assessments.

3.2.3.2. Direct Spray – Direct sprays involving ground applications are modeled in a manner similar to accidental spills for workers (Section 3.2.2.2). In other words, it is assumed that the individual is sprayed with a solution containing the compound and that an amount of the compound remains on the skin and is absorbed by first-order kinetics. For these exposure scenarios, it is assumed that during a ground application, a naked child is sprayed directly with sulfometuron methyl. These scenarios also assume that the child is completely covered (that is, 100% of the surface area of the body is exposed). These exposure scenarios are likely to represent upper limits of plausible exposure. An additional set of scenarios are included involving a young woman who is accidentally sprayed over the feet and legs. For each of these scenarios, some assumptions are made regarding the surface area of the skin and body weight, as detailed in Worksheet A03.

3.2.3.3. Dermal Exposure from Contaminated Vegetation – In this exposure scenario, it is assumed that the herbicide is sprayed at a given application rate and that an individual comes in contact with sprayed vegetation or other contaminated surfaces at some period after the spray operation. For these exposure scenarios, some estimates of dislodgeable residue and the rate of transfer from the contaminated vegetation to the surface of the skin must be available. No such data are available on dermal transfer rates for sulfometuron methyl and the estimation methods of Durkin et al. (1995) are used as defined in Worksheet D02. The exposure scenario assumes a contact period of one hour and assumes that the chemical is not effectively removed by washing for 24 hours. Other estimates used in this exposure scenario involve estimates of body weight, skin surface area, and first-order dermal absorption rates, as discussed in the previous section.

3.2.3.4. Contaminated Water – Water can be contaminated from runoff, as a result of leaching from contaminated soil, from a direct spill, or from unintentional contamination from aerial applications. For this risk assessment, the two types of estimates made for the concentration of sulfometuron methyl in ambient water are acute/accidental exposure from an accidental spill and longer-term exposure to sulfometuron methyl in ambient water that could be associated with the application of this compound to a 10 acre block that is adjacent to and drains into a small stream or pond.

3.2.3.4.1. ACUTE EXPOSURE – Two exposure scenarios are presented for the acute consumption of contaminated water: an accidental spill into a small pond (0.25 acres in surface area and 1 meter deep) and the contamination of a small stream by runoff or percolation.

The accidental spill scenario assumes that a young child consumes contaminated water shortly after an accidental spill into a small pond. The specifics of this scenario are given in Worksheet D05. Because this scenario is based on the assumption that exposure occurs shortly after the spill, no dissipation or degradation of sulfometuron methyl is considered. This scenario is dominated by arbitrary variability and the specific assumptions used will generally overestimate exposure. The actual concentrations in the water would depend heavily on the amount of compound spilled, the size of the water body into which it is spilled, the time at which water consumption occurs relative to the time of the spill, and the amount of contaminated water that is consumed. Based on the spill scenario used in this risk assessment, the concentration of sulfometuron methyl in a small pond is estimated to range from about 0.098 mg/L to 0.83 mg/L with a central estimate of about 0.30 mg/L (Worksheet D05).

The other acute exposure scenario for the consumption of contaminated water involves runoff into a small stream. Monitoring data on sulfometuron methyl are available from Battaglin et al. (1999) and Neary and Michael (1989, 1993, 1996). A single random sampling study reports monitoring data on sulfometuron methyl concentrations obtained from midwestern streams, rivers, and ground-water in 1998 (Battaglin et al. 1999). Of the 130 samples collected from streams and rivers, sulfometuron methyl was detected in only two samples, with a maximum concentration of 0.018 µg/L, just slightly above the method reporting limit of 0.01 µg/L. In ground-water, sulfometuron methyl was below the method reporting limit in the 25 samples tested. Unfortunately, these monitoring data are of limited use in the exposure assessment because sampling was random and not associated with a specific application of sulfometuron methyl.

Monitoring data are associated with a known application rate of sulfometuron methyl are available from the study by Neary and Michael (1989). Additional details are taken from Michael and Neary (1993) and Neary and Michael (1996). In the Neary and Michael (1989) study at a site in Florida, sulfometuron methyl was applied at a rate of 0.4 kg a.i./ha—equivalent to 0.3568 lbs/acre—as either dispersible granules (DG) or an experimental pellet formulation (P). Application was made by broadcast ground applications with predominantly sandy soil. Sulfometuron methyl was monitored at maximum concentrations of 5 µg/L(P) and 7 µg/L (DG). Sulfometuron methyl was detected in only 10 of 185 stream samples and only from day 3 to day

7 after treatment. Monitoring was conducted up to 203 days after treatment. In most instances, the sulfometuron methyl was detected in the surface water after storm events. In each of these applications, rainfall began 24 hours after treatment and a total of 54 mm of rain fell over the first 3 days after treatment. Monitoring data are also available from broadcast aerial applications in an area of Mississippi with predominantly clay soil (Michael and Neary 1993, Neary and Michael 1996); fewer details are available from this application site. Aerial application was made at a rate of 0.4 kg a.i./ha (equivalent to 0.3568 lbs/acre). At this site, the maximum reported levels of sulfometuron methyl in surface water were 23 µg/L(P) and 44 µg/L (DG).

For this risk assessment, the maximum concentration reported by Michael and Neary (1989), 7 µg/L, is used as the basis for the upper estimate of sulfometuron methyl in surface water. Normalized for an application rate of 1 lb a.e./acre, this value is converted to approximately 20 µg/L per lbs/acre [$7 \mu\text{g/L} \div 0.3568 \text{ lbs/acre} = 19.57$]. In some respects, this approach may be viewed as extremely conservative. The higher concentrations from the Michael and Neary (1993) are both associated with aerial application and are approximately 3- to 9-fold higher than the concentrations based on ground applications. Since the Forest Service does not anticipate using aerial applications for sulfometuron methyl, a case could be made for using only the lower values for estimating potential human exposure from ground applications. On the other hand, this risk assessment is intended to encompass the broad use of sulfometuron methyl in several different regions of the country. The relatively sparse monitoring data from only two locations are not likely to reflect the diversity of meteorological or hydrogeological conditions under which sulfometuron methyl may be applied. Since data obtained following aerial applications may not be representative of ground applications, they are not used in this risk assessment.

Although these values are used for the longer-term exposure scenario for humans, it is implausible to suggest that these concentrations would be maintained for prolonged periods of time. For the characterization of potential human health effects (section 3.4), this extremely conservative approach makes no difference because the exposure levels are far below those of toxicological concern. A fuller use of these monitoring studies, however, is required for the assessment of toxicological effects on aquatic vegetation, as discussed in Section 4.2.3.

While monitoring data provide practical and documented instances of water contamination, monitoring studies may not encompass a broad range of conditions which may occur during program applications – e.g., extremely heavy rainfall – or they may reflect atypical applications that do not reflect program practices. Consequently, for this component of the exposure assessment, modeled estimates are made based on GLEAMS (Groundwater Loading Effects of Agricultural Management Systems).

GLEAMS is a root zone model that can be used to examine the fate of chemicals in various types of soils under different meteorological and hydrogeological conditions (Knisel and Davis 2000). As with many environmental fate and transport models, the input and output files for GLEAMS can be complex. The general application of the GLEAMS model and the use of the output from this model to estimate concentrations in ambient water are detailed in SERA (2003b).

For the current risk assessment, the application site was assumed to consist of a 10 acre square area that drained directly into a small pond or stream. The chemical specific values as well as the details of the pond and stream scenarios used in the GLEAMS modeling are summarized in Table 3-1. The GLEAMS model yields estimates runoff, sediment loss and percolation that were used to estimate concentrations in the stream adjacent to a treated plot, as detailed in Section 6.4 of SERA (2003b). The results of the GLEAMS modeling for the small stream are summarized in Table 3-2 and the corresponding values for the small pond are summarized in Table 3-3. These estimates are expressed as both average and maximum water contamination rates (WCR) - i.e., the concentration of the compound in water in units of mg/L normalized for an application rate of 1 lb a.e./acre.

As indicated in Table 3-2, no stream contamination is estimated in very arid regions – i.e., annual rainfall of 10 to 15 inches or less depending on soil type. The modeled maximum concentrations in the stream range from about 0.08 µg/L (less in loam or sand) to somewhat over 2 µg/L (clay) at annual rainfall rates from 15 to 250 inches per year, with the highest concentrations associated with clay at annual rainfall rates of 150 inches or more. While not detailed in Table 3-2, the losses from clay and loam are associated primarily with runoff (about 86 to 90%), with the remaining amount due to sediment loss. For sand, the pesticide loss is associated almost exclusively with percolation. For both clay and loam, the maximum losses occur with the first rainfall after application. For sand, time to maximum loss is delayed due to the time required to percolate through the soil column.

Modeled peak concentrations in ponds (Table 3-3) are generally similar to those in streams, ranging from about 0.06 to 1.6 µg/L in clay soil with much lower concentrations for other soil types – i.e., up to about 0.4 µg/L in sand and 0.03 µg/L in loam. Modeled average concentrations in ponds, however, are substantially higher than those in streams. The highest average concentration is estimated at about 0.07 µg/L – i.e., clay or sandy soil at a rainfall rates of 50 to 200 inches per year. Over all soil types, typical concentrations are in the range of 0.02 to 0.07 µg/L. As with the stream modeling, virtually no contamination is modeled in very arid regions.

Comparisons of the modeled maximum concentrations in streams to the maximum concentration of 19.57 µg/L reported by Neary and Michael (1989) show that modeled maximum concentrations are approximately 10-fold less than the observed maximum concentration of 2.1 in clay associated with 150 inches of rainfall (Table 3-2). While the reasons for this discrepancy cannot be determined, it is possible that the concentrations noted in the Neary and Michael (1989) study could have been due to drift during application rather than offsite transfer of sulfometuron methyl after deposition on to soil.

The GLEAMS scenarios do not specifically consider the effects of accidental direct spray. For example, the stream modeled using GLEAMS is about 6 feet wide and it is assumed that the herbicide is applied along a 660 foot length of the stream with a flow rate of 4,420,000 L/day. At an application rate of 1 lb/acre, accidental direct spray onto the surface of the stream would deposit about 41,252,800 µg [1 lb/acre = 112,100 µg/m², 6'x660' = 3960 ft² = 368 m², 112,100 µg/m² × 368 m² = 41,252,800 µg]. This would result in a downstream concentration of about 10

µg/L [41,252,800 µg/day ÷ 4,420,000 L/day], similar to the concentrations reported by Neary and Michael (1989). As indicated in Table 3-2, the expected peak concentrations from runoff or percolation are below this value by a factor of about 5 or more.

For the current risk assessment, the upper bound for the short-term water contamination rate will be taken as 20 µg/L per lb/acre, the maximum value observed in the monitoring study by Neary and Michael (1989). This value, converted to 0.02 mg/L per lb/acre, is entered into Worksheet B06. The central estimate will be taken as 1 µg/L (0.001 mg/L), about the concentration for clay at annual rainfall rate of 100 inches. The lower range will be taken as 0.06 µg/L (0.00006 mg/L), concentrations that might be expected in relatively arid regions with clay soil – i.e., annual rainfall of 15 inches. Note that much lesser concentrations are modeled for loam and sand and this may need to be considered in any site-specific application of GLEAMS.

3.2.3.4.2. LONGER-TERM EXPOSURE – The scenario for chronic exposure from contaminated water is detailed in worksheet D07. This scenario assumes that an adult (70 kg male) consumes contaminated ambient water from a contaminated pond for a lifetime. The estimated concentrations in pond water are based on the modeled estimates from GLEAMS, discussed in the previous section.

For this risk assessment, the typical longer term WCR is taken as 0.04 µg/L or 0.00004 mg/L per lb/acre. This is about the average concentration that modeled in a pond using GLEAMS at a rainfall rate of 20 to about 50 inches per year in clay soil (Table 3-3). The upper bound of the WCR is taken as 0.07 µg/L or 0.00007 mg/L per lb/acre. This is the highest average concentration modeled from sandy soil at an rainfall rate of 100 inches per year or clay soil at annual rainfall rates of 150 inches per year. The lower bound is taken as 0.01 µg/L or 0.00001 mg/L per lb/acre. This selection is somewhat arbitrary but would tend to encompass concentrations that might be found in relatively arid areas.

The WCR values discussed in this section summarized in Worksheet B06 and used for all longer term exposure assessments involving contaminated water. As with the corresponding values for a small stream, these estimates are expressed as the water contamination rates (WCR) in units of mg/L per lb/acre.

3.2.3.5. Oral Exposure from Contaminated Fish – Many chemicals may be concentrated or partitioned from water into the tissues of animals or plants in the water. This process is referred to as bioconcentration. Generally, bioconcentration is measured as the ratio of the concentration in the organism to the concentration in the water. For example, if the concentration in the organism is 5 mg/kg and the concentration in the water is 1 mg/L, the bioconcentration factor (BCF) is 5 L/kg [5 mg/kg ÷ 1 mg/L]. As with most absorption processes, bioconcentration depends initially on the duration of exposure but eventually reaches steady state. Details regarding the relationship of bioconcentration factor to standard pharmacokinetic principles are provided in Calabrese and Baldwin (1993).

The potential for accumulation of sulfometuron methyl in fish was studied in bluegill fish exposed to 0.01 and 1.0 mg/L and in channel catfish exposed to 0.01-0.02 mg/L

¹⁴C-sulfometuron methyl (Harvey 1981a) . The bioconcentration of ¹⁴C-sulfometuron methyl in muscle (edible tissue), liver and viscera was examined during a 28-day exposure period. Details of these studies are provided in Appendix 2. For this risk assessment, concentrations in viscera are considered to reflect the concentration in whole fish. No bioaccumulation of sulfometuron methyl in either muscle or viscera occurred in bluegill sunfish. In catfish, bioaccumulation occurred in both muscle and viscera. Following 1 day of exposure, the bioconcentration factor (BCF) in muscle was 3, which is used for acute exposure to edible tissue, and the BCF observed in viscera was 3.5, which is used for acute exposure to whole fish. Over the 28-day exposure period, the highest BCF in edible tissue was 7, which was observed following 21 days of exposure, and the highest BCF in viscera 6, observed after 28 days of exposure. For this risk assessment, a bioconcentration factor for edible tissue for chronic exposure in fish for edible tissue will be taken as 7 and for whole fish will be taken as 6.

For both the acute and longer-term exposure scenarios involving the consumption of contaminated fish, the water concentrations of sulfometuron methyl used are identical to the concentrations used in the contaminated water scenarios (see Section 3.2.3.4). The acute exposure scenario is based on the assumption that an adult angler consumes fish taken from contaminated water shortly after an accidental spill of 200 gallons of a field solution into a pond that has an average depth of 1 m and a surface area of 1000 m² or about one-quarter acre. No dissipation or degradation is considered. Because of the available and well-documented information and substantial differences in the amount of caught fish consumed by the general public and native American subsistence populations, separate exposure estimates are made for these two groups, as illustrated in worksheet D08. The chronic exposure scenario is constructed in a similar way, as detailed in worksheet D09, except that estimates of sulfometuron methyl concentrations in ambient water are based on GLEAMS modeling as discussed in Section 3.2.3.4.

3.2.3.6. Oral Exposure from Contaminated Vegetation – None of the Forest Service applications of sulfometuron methyl will involve the treatment of crops. Thus, under normal circumstances and in most types of applications conducted as part of Forest Service programs, the consumption by humans of vegetation contaminated with sulfometuron methyl is unlikely. Nonetheless, any number of scenarios could be developed involving either accidental spraying of crops or the spraying of edible wild vegetation, like berries. In most instances, and particularly for longer-term scenarios, treated vegetation would probably show signs of damage from exposure to sulfometuron methyl (Section 4.3.2.4), thereby reducing the likelihood of consumption that would lead to significant levels of human exposure. Notwithstanding that assertion, it is conceivable that individuals could consume contaminated vegetation. One of the more plausible scenarios involves the consumption of contaminated berries after treatment of a right-of-way or some other area in which wild berries grow.

The two accidental exposure scenarios developed for this exposure assessment include one scenario for acute exposure, as defined in Worksheet D03 and one scenario for longer-term exposure, as defined in Worksheet D04. In both scenarios, the concentration of sulfometuron methyl on contaminated vegetation is estimated using the empirical relationships between application rate and concentration on vegetation developed by Fletcher et al. (1994) which is in

turn based on a re-analysis of data from Hoerger and Kenaga (1972). These relationships are defined in worksheet A04. For the acute exposure scenario, the estimated residue level is taken as the product of the application rate and the residue rate (Worksheet D03).

For the longer-term exposure scenario (D04), a duration of 90 days is used. The rate of decrease in the residues over time is taken from the vegetation half-time of 10 days reported by Knisel and Davis (2000). Although the duration of exposure of 90 days is somewhat arbitrarily chosen, this duration is intended to represent the consumption of contaminated fruit that might be available over one season. Longer durations could be used for certain kinds of vegetation but would lower the estimated dose (i.e., would reduce the estimate of risk).

For the longer-term exposure scenarios, the time-weighted average concentration on fruit is calculated from the equation for first-order dissipation. Assuming a first-order decrease in concentrations in contaminated vegetation, the concentration in the vegetation at time t after spray, C_t , can be calculated based on the initial concentration, C_0 , as:

$$C_t = C_0 \times e^{-kt}$$

where k is the first-order decay coefficient [$k = \ln(2) \div t_{50}$]. Time-weighted average concentration (C_{TWA}) over time t can be calculated as the integral of C_t (De Sapiro 1976, p. p. 97 ff) divided by the duration (t):

$$C_{TWA} = C_0 (1 - e^{-k t}) \div (k t).$$

A separate scenario involving the consumption of contaminated vegetation by drift rather than direct spray is not developed in this risk assessment. As detailed further in Section 3.4, this elaboration is not necessary because the direct spray scenario leads to estimates of risk that are below a level of concern. Thus, considering spray drift and a buffer zone quantitatively would have no impact on the characterization of risk.

3.3. DOSE-RESPONSE ASSESSMENT

3.3.1. Overview.

The U.S. EPA has derived an RfD of 0.24 mg/kg/day. This RfD is based on a NOAEL for bladder toxicity of 500 ppm dietary sulfometuron methyl (equivalent to 24.4 mg/kg/day) and a 100-fold safety factor. Although an RfD has been derived by U.S. EPA, a more conservative provisional reference dose of 0.02 mg/kg/day, which was used in the previous Forest Service risk assessment on sulfometuron methyl (Durkin 1998), was derived from data reported in the 2-year feeding study in rats by Mullin (1984). The provisional reference dose is based on the 2 mg/kg/day (50 ppm) NOAEL for hematological effects in male rats and an uncertainty factor of 100: a factor of 10 for species-to-species extrapolation and a factor of 10 for sensitive subgroups in the human population. The provisional RfD of 0.02 mg/kg/day is used in the current risk assessment for characterizing risks associated with chronic exposure to sulfometuron methyl. The U.S. EPA has not derived an acute/single dose RfD for sulfometuron methyl. A NOAEL of 86.6 mg/kg/day was reported for decreased maternal and fetal body weights in rats following 10-day gestational exposure of dams (Lu 1981). Using a NOAEL 86.6 mg/kg/day and margin of exposure of 100, a provisional acute RfD is calculated as 0.87 mg/kg/day and will be used for characterizing risks associated with acute exposure to sulfometuron methyl.

3.3.2. Existing Guidelines.

According to a Federal Registry Notice (U.S. EPA 1997), the U.S. EPA has derived an RfD of 0.24 mg/kg/day. This RfD is based on a NOAEL of 500 ppm dietary sulfometuron methyl (equivalent to 24.4 mg/kg/day, according to U.S. EPA 1997) and a 100-fold safety factor. As reported in the Federal Registry Notice, the NOAEL of 24.4 mg/kg/day is reported for the development of chronic toxicity, primarily of the urinary bladder, in male rats. In female rats, a NOAEL of 30 mg/kg/day for bladder toxicity is cited by U.S. EPA (1997).

The RfD of 0.24 mg/kg/day derived by U.S. EPA was not cited in a previous Forest Service risk assessment on sulfometuron methyl (Durkin 1998). Instead, a more conservative provisional reference dose of 0.02 mg/kg/day, based on data reported in the 2-year feeding study by Mullin (1984), was used. The provisional reference dose is based on the 2 mg/kg/day (50 ppm) NOAEL for hematological effects in male rats and an uncertainty factor of 100:10 for species-to-species extrapolation and 10 for sensitive subgroups in the human population. Because the study by Mullin (1984) entailed a 2-year exposure period which approximates the life span of rats, there is no need for an additional uncertainty factor to account for subchronic to chronic exposure. At 20 mg/kg/day, hematological effects were observed in male rats. Thus, at a dose that is 10-fold higher than the provisional reference dose, 0.2 mg/kg/day, there would be concern for hematologic effects in humans. At intermediate levels of exposure (i.e., those between 0.02 and 0.2 mg/kg/day) the concern for potential adverse effects cannot be defined well. As reported by Mullin (1984) and summarized in Appendix 1, the dietary concentration of 50 ppm is equivalent to a daily dose of 2 mg/kg/day and was determined using time-weighted averages for daily food consumption. In female rats, a NOAEL of 3 mg/kg/day for hematological, equivalent to a dietary concentration of 50 ppm, was reported (Mullin 1984). Thus, in terms of dietary concentration of sulfometuron methyl, NOAELs reported for male and female rats for hematological effects are the same (50 ppm), although the NOAEL reported in terms of the daily dose is higher in females

than in males. For this risk assessment, the provisional RfD of 0.02 mg/kg/day is used for characterizing risks associated with chronic exposure to sulfometuron methyl.

With regard to species sensitivity for hematological effects, rats appear to be most sensitive with reported NOAELs of 2-3 mg/kg/day and an AEL of 20-26 mg/kg/day (Appendix 1). Dogs appear to have a sensitivity similar to that of rats, with a reported NOAEL of 5 mg/kg/day and a LOAEL of 28 mg/kg/day (Wood and O'Neal 1983). Mice appear to be much less sensitive than either rats or dogs to the hematological effects of sulfometuron methyl with a NOAEL of about 18 mg/kg/day and a LOAEL of 180 mg/kg/day (Summers 1990a; dose conversions provided in Appendix 1). Although these data are not amenable to formal statistical analysis, they lend qualitative support to the use of an uncertainty factor for species-to-species extrapolation for the human health risk assessment (i.e., the larger animals appear to be more sensitive than smaller animals to sulfometuron methyl).

3.3.3. Acute RfD.

The U.S. EPA has not derived an acute/single dose RfD for sulfometuron methyl. However, a provisional acute RfD can be calculated based on the short-term exposure NOAEL for decreased body weight of 86.6 mg/kg/day reported in the teratology study in rats by Lu (1981). As discussed in Section 3.1.9, exposure of rats to 5000 ppm dietary for 10 days during gestation resulted in decreased maternal, with a NOAEL of 1000 ppm. As reported by the author, the dietary concentration of 5000 ppm is equivalent to a daily dose of 433 mg/kg/day and was determined using mean daily food consumption and body weight. Thus, assuming that food consumption and body weights were similar between the 1000 and 5000 ppm exposure groups, the concentration of 1000 ppm is estimated as equivalent to 86.6 mg/kg/day [433 mg/kg/day ÷ 5]. Using a margin of exposure of 100, a provisional acute RfD can be calculated as 0.87 mg/kg/day [86.6 mg/kg/day ÷ 100]. For this risk assessment, the provisional RfD of 0.87 mg/kg/day is used for characterizing risks associated with acute exposure to sulfometuron methyl.

3.4. RISK CHARACTERIZATION

3.4.1. Overview.

Typical exposures to sulfometuron methyl do not lead to estimated doses that exceed a level of concern. For workers, no exposure scenarios, acute or chronic, exceeds the RfD at the upper bound of the estimated dose associated with the typical application rate of 0.045 lb a.e./acre. Hazard quotients for the upper ranges for directed ground spray, broadcast ground spray, and aerial applications slightly exceeded the level of concern of 0.1 associated with the maximum application rate of 0.38 lb a.e./acre. These upper limits of exposure are constructed using the highest anticipated application rate, the highest anticipated number of acres treated per day, and the upper limit of the occupational exposure rate. If any of these conservative assumptions are modified (e.g., the compound is applied at the typical rather than the maximum application rate) the hazard indices would most likely be below the level of concern. Given the conservative nature of the RfD itself, it is unlikely that there would be any signs of toxicity in workers applying sulfometuron methyl. The simple verbal interpretation of the quantitative characterization of risk is that under the most conservative set of exposure assumptions, workers could be exposed to levels of sulfometuron methyl that are regarded as unacceptable. If sulfometuron methyl is not applied at the highest application rate or if appropriate steps are taken to ensure that workers are not exposed at the maximum plausible rates (i.e., worker hygiene practices and/or reduced areas of treatment per day) there is no indication that the workers would be at risk of incurring systemic toxic effects.

Irritation and damage to the skin and eyes can result from exposure to relatively high levels of sulfometuron methyl. From a practical perspective, eye or skin irritation is likely to be the only overt effect as a consequence of mishandling sulfometuron methyl. These effects can be minimized or avoided by prudent industrial hygiene practices during the handling of sulfometuron methyl.

For members of the general public, all upper limits for hazard quotients are below a level of concern for the typical application rate. The upper end of the range of exposure resulting from the consumption by a child of contaminated water from a small pond immediately after an accidental spill is at the level of concern for the maximum application rate of 0.38 lbs/acre— i.e., a hazard quotient of 0.1 and a level of concern of 0.1. For chronic exposure, all upper limits are well below the level of concern for the maximum application rate. Thus, based on the available information and under the foreseeable conditions of application, there is no route of exposure or scenario suggesting that workers or members of the general public will be at any substantial risk from acute or longer term exposures to sulfometuron methyl.

Irritation to the skin and eyes can result from exposure to relatively high levels of sulfometuron methyl. From a practical perspective, eye or skin irritation is likely to be the only overt effect as a consequence of mishandling sulfometuron methyl. These effects can be minimized or avoided by prudent industrial hygiene practices during the handling of the compound.

3.4.2. Workers.

A quantitative summary of the risk characterization for workers associated with exposure to sulfometuron methyl is presented in Worksheet E02 (Supplement 1). The quantitative risk characterization is expressed as the hazard quotient, the ratio of the estimated doses from Worksheet E01 to the RfD. For acute exposures – i.e., accidental or incidental exposures – a provisional acute RfD of 0.87 mg/kg/day is used (Section 3.3.3). For general exposures – i.e., daily exposures that might occur over the course of an application season – a provisional chronic RfD of 0.02 mg/kg/day is used (Section 3.3.2).

As indicated in Section 2, the exposures in Worksheet E01 and the subsequent hazard quotients in Worksheet E02 are based on the typical application rate of 0.045 lb a.e./acre and the “level of concern” is one – i.e., if the hazard quotient is below 1.0, the exposure is less than the RfD. For all exposure scenarios, the estimated dose scales linearly with application rate. Thus, at an application rate of 0.38 lb a.e./acre, the highest application rate contemplated by the Forest Service, the level of concern would be 0.1 – i.e., $0.045 \text{ lb/acre} \div 0.38 \text{ lb/acre}$. All hazard quotients are below the level of concern for the typical application rate of 0.045 lb a.e./acre. It should be noted that confidence in these assessments is diminished by the lack of a worker exposure study and the lack of experimental data on the dermal absorption kinetics of sulfometuron methyl (Section 3.1). Nonetheless, the statistical uncertainties in the estimated dermal absorption rates, both zero-order and first-order, are incorporated into the exposure assessment and risk characterization. As shown in Worksheet E02, hazard quotients for the upper end of the ranges for directed ground spray (0.2), broadcast spray (0.3) and aerial applications (0.2) are slightly above the level of concern of 0.1 associated with the highest application rate of 0.38 lb a.e./acre. As discussed in Section 3.2 and detailed in Worksheets C01a and C01b, these upper limits of exposure are constructed using the highest anticipated application rate, the highest anticipated number of acres treated per day, and the upper limit of the occupational exposure rate. If any of these conservative assumptions are modified (e.g., the compound is applied at a rate below at the typical application rate) the hazard indices would most likely be below the level of concern. The simple verbal interpretation of this quantitative characterization of risk is that under the most conservative set of exposure assumptions, workers could be exposed to levels of sulfometuron methyl that are regarded as unacceptable. If sulfometuron methyl is not applied at the highest application rate or if appropriate steps are taken to ensure that workers are not exposed at the maximum plausible rates (i.e., worker hygiene practices and/or reduced areas of treatment per day) there is no indication that the workers would be at risk of incurring systemic toxic effects.

While the accidental exposure scenarios are not the most severe one might imagine (e.g., complete immersion of the worker or contamination of the entire body surface for a prolonged period of time) they are representative of reasonable accidental exposures. None of these hazard quotients approach a level of concern at the upper ranges, even when considering the level of concern associated with an application rate of 0.38 lbs a.e./acre – i.e., a level of concern of 0.1. The simple verbal interpretation of this quantitative characterization of risk is that under the most protective set of exposure assumptions, workers would not be exposed to levels of sulfometuron methyl that are regarded as unacceptable so long as reasonable and prudent handling practices are followed.

As discussed in Section 3.1.11, sulfometuron methyl can cause irritation to eyes and skin. Quantitative risk assessments for irritation are not derived; however, from a practical perspective, effects on the eyes or skin are likely to be the only overt effects as a consequence of mishandling sulfometuron methyl. These effects can be minimized or avoided by prudent industrial hygiene practices during the handling of sulfometuron methyl.

3.4.3. General Public.

The quantitative hazard characterization for the general public associated with exposure to sulfometuron methyl is summarized in Worksheet E04 (Supplement 1). Like the quantitative risk characterization for workers, the quantitative risk characterization for the general public is expressed as the hazard quotient using the acute RfD of 0.87 mg/kg/day for acute/short term exposure scenarios and the provisional chronic RfD of 0.02 mg/kg/day chronic or longer term exposures.

Although there are several uncertainties in the longer-term exposure assessments for the general public, as discussed in Section 3.2.3, the upper limits for hazard quotients associated with the longer-term exposures at an application rate of 0.045 lbs/acre are sufficiently below a level of concern. Thus, the risk characterization is relatively unambiguous: based on the available information and under the foreseeable conditions of application, there is no route of exposure or scenario suggesting that the general public will be at any substantial risk from longer-term exposure to sulfometuron methyl even if the level of concern is set to 0.1 – i.e., that associated with the maximum application rate of 0.38 lbs/acre that will be used in Forest Service programs.

For the acute/accidental scenarios, none of the upper estimates representing typical exposures exceed the level of concern. Exposure resulting from the consumption of contaminated water is of greatest concern. The estimate of the upper bound of exposure resulting from the consumption by a child of contaminated water from a small pond immediately after an accidental spill (Section 3.2.3.4.1) is at the level of concern for the maximum application rate of 0.38 lbs/acre– i.e., a hazard quotient of 0.1 and a level of concern of 0.1. This is an extremely conservative scenario that typically results in an excursion above the RfD. This is not the case with sulfometuron methyl.

Each of the hazard quotients summarized in Worksheet E04 involves a single exposure scenario. In some cases, individuals could be exposed by more than one route and in such cases risk can be quantitatively characterized by simply adding the hazard quotients for each exposure scenario. For sulfometuron methyl, considerations of multiple exposure scenarios has little impact on the risk assessment. For example, based on the upper ranges for typical levels of acute/accidental exposure for being directly sprayed on the lower legs, staying in contact with contaminated vegetation, eating contaminated fruit, drinking contaminated water from a stream, and consuming contaminated fish at rates characteristic of subsistence populations leads to a combined hazard quotient of 0.05 (0.0005 + 0.0001 + 0.02 + 0.00006 + 0.03). Similarly, for all of the chronic exposure scenarios, the addition of all possible pathways lead to hazard quotients that are below the level of concern of 0.1 – i.e., the level of concern at the maximum application rate.

3.4.4. Sensitive Subgroups. There is no information to suggest that specific groups or individuals may be especially sensitive to the systemic effects of sulfometuron methyl. Due to the lack of data in humans, the likely critical effect of sulfometuron methyl in humans cannot be identified clearly. As indicated in Section 3.1.5, the most sensitive effect reported in animals for chronic sulfometuron methyl exposure appears to involve changes in blood that are consistent with hemolytic anemia. Thus, individuals with pre-existing anemia could potentially be at an increased risk. As discussed in Section 3.1.8, it appears that sulfometuron methyl has the potential to alter thyroid gland function. Individuals with pre-existing thyroid dysfunction may, therefore, be at increased risk. However, there are no data to directly support these speculations.

3.4.5. Connected Actions. As noted in section 2.2, the manufacturers recommend that sulfometuron methyl formulations be mixed with a surfactant. There is no published literature or information in the FIFRA files that would permit an assessment of toxicological effects or risk assessment of sulfometuron methyl mixed with a surfactant. According to the product label, Escort may be applied in combination with other herbicides. However, there are no animal data to suggest that sulfometuron methyl will interact, either synergistically or antagonistically with any other herbicide.

3.4.6. Cumulative Effects. As noted above, this risk assessment specifically considers the effect of both single and repeated exposures. Based on the hazard quotients summarized in Worksheet E04, as discussed above, there is no indication that repeated exposures will exceed the threshold for toxicity.

4. ECOLOGICAL RISK ASSESSMENT

4.1. HAZARD IDENTIFICATION

4.1.1. Overview.

The mammalian toxicity of sulfometuron methyl is relatively well-characterized in experimental mammals; however, there is relatively little information regarding non-target wildlife species. In standard experimental toxicity studies, sulfometuron methyl has low acute and chronic oral toxicity. It seems reasonable to assume the most sensitive effects in wildlife mammalian species will be the same as those in experimental mammals (i.e., changes to blood and decreased body weight gain). Results of acute exposure studies in birds indicate that avian species appear no more sensitive than experimental mammals to the toxic effects of sulfometuron methyl. Chronic exposure studies in birds were not identified in the available literature. Because the studies on birds are different in design from those on experimental mammals, it is difficult to assess the sensitivity of birds, relative to mammals. Nonetheless, on the basis of the limited comparisons that can be made, birds appear to be somewhat less sensitive than experimental mammals to the toxic effects of sulfometuron methyl. Results of two acute exposure studies in honey bees indicate that bees are no more sensitive than either mammals or birds to sulfometuron methyl. However, the available data are not sufficient to determine whether this apparent low level of toxicity can be generalized to other species of terrestrial invertebrates.

The toxicity of sulfometuron methyl to terrestrial plants was studied extensively and is well characterized. Sulfometuron methyl inhibits acetolactate synthase (ALS), an enzyme that catalyzes the biosynthesis of three branched-chain amino acids, all of which are essential for plant growth. Bioassays have been conducted on pre-emergence and post-emergence toxicity to several species. In the pre-emergence assay, the most sensitive species based on the NOEC were rape, tomato, sorghum, wheat, and corn with an NOEC of 0.0000086 lb ai/acre. The most tolerant species based on the NOEC were onion, pea, cucumber, and soybean with an NOEC of 0.00026 lb/acre. In the post-emergence assay, the most sensitive species based on the NOEC is corn with an NOEC of 0.000024 lb ai/acre. The most tolerant species in the post-emergence assay was pea with an NOEC of 0.00078 lb ai/acre. Assays using an application rate of 0.01 kg/ha [0.00892 lbs a.i./acre] sulfometuron methyl show high toxicity to seedlings of several broadleaves and grasses, either preemergence or postemergence. Moreover, adverse effects were observed in most plants tested at application rates of 0.001 kg/ha [0.000892 lbs a.i./acre]. This application rate is a factor of about 50-fold less than the application rate that the Forest Service would typically use. Concern for the sensitivity of non-target plant species is further increased by field reports of substantial and prolonged damage to crops or ornamentals after the application of sulfometuron methyl in both an arid region, presumably due to the transport of soil contaminated with sulfometuron methyl by wind, and in a region with heavy rainfall, presumably due to the wash-off of sulfometuron methyl contaminated soil. Sulfometuron methyl exposure inhibited growth of several soil microorganisms and, at concentrations as low as 0.2 μM [~ 73 $\mu\text{g/L}$] caused significant growth inhibition in *Salmonella typhimurium* after exposure periods of less than 3 hours.

As with potential effects on terrestrial species and as would be expected for a herbicide, the available data suggest that sulfometuron methyl is much more toxic to aquatic plants than to

aquatic animals. The effects of acute and chronic exposure to sulfometuron methyl has been studied in several types of aquatic animals, including fish, amphibians and several species of aquatic invertebrates. The results of studies in fish suggest that frank toxic effects are not likely to be observed at concentrations less than or equal to 150 mg/L. Based on assays of fathead minnow embryo hatch, larval survival, or larval growth over 30-day exposure periods, no adverse effects would be expected at concentrations up to 1.17 mg sulfometuron methyl/L. Sulfometuron methyl also appears to be relatively non-toxic to aquatic invertebrates, based on acute bioassays in daphnids, crayfish, and field-collected species of other aquatic invertebrates. One daphnid reproduction study noted a decrease in the number of neonates at 24 mg/L but not at 97 mg/L or any of the lower concentrations tested. The authors report the NOEL as 6.1 mg a.i./L. Although the effect observed at 24 mg/L may have been a random variation, it is treated as an LOAEL for the purpose of this risk assessment. While this approach may be regarded as conservative, in the absence of additional studies regarding reproductive effects in aquatic invertebrates, the approach seems prudent. The most sensitive aquatic species tested appears to be the African clawed frog. In acute and chronic exposure studies, exposure to sulfometuron methyl produced alterations in limb development, organogenesis, and metamorphosis, with the lowest NOEL of 0.001 mg/L for metamorphosis.

Aquatic plants are far more sensitive than aquatic animals to the effects of sulfometuron methyl, although there appear to be substantial differences in sensitivity among species of macrophytes and unicellular algae. The macrophytes, however, appear to be generally more sensitive. Although the effect on aquatic plants have not been extensively studied, there is a wide variation in species sensitivity, with EC₅₀ values for growth inhibition ranging from 0.462 µg/L in duckweed to 10 µg/L in hydrilla. In algae, EC₅₀ values for growth inhibition range from 4.6 µg/L in *Selenastrum capricornutum* to > 370 µg/L (the NOEC value) in *Navicula pelliculosa*. There are no published or unpublished data regarding the toxicity of sulfometuron methyl to aquatic bacteria or fungi. By analogy to the effects on terrestrial bacteria and aquatic algae, it seems plausible that aquatic bacteria and fungi will be sensitive to the effects of sulfometuron methyl.

4.1.2. Toxicity to Terrestrial Organisms.

4.1.2.1. Mammals— As summarized in the human health risk assessment (see Section 3.1), the mode of action of sulfometuron methyl in mammals is not well understood. There are several standard toxicity studies in experimental mammals that were conducted as part of the registration process. The most common signs of toxicity involve changes in blood that are consistent with hemolytic anemia (i.e., a lysis or destruction of blood cells that results in a decreased number of red blood cells) and decreased body weight gain. It is plausible that the hemolytic anemia caused by sulfometuron methyl is attributable, at least partially, to sulfonamide and saccharin, which are metabolites of sulfometuron methyl. Other than these effects, sulfometuron methyl does not appear to cause specific target organ toxicity in mammals.

No field studies are available in which the impact of sulfometuron methyl applications were assessed on mammalian wildlife communities. In standard experimental toxicity studies, sulfometuron methyl has low acute oral toxicity. A common measure of acute oral toxicity is the LD₅₀, the estimate of the dose that may be lethal to 50% of the exposed animals. As summarized in Section 3.1.4, in rats the acute oral LD₅₀ for technical grade sulfometuron methyl is greater

than 17,000 mg/kg (Dashiell and Hall 1982a) and for a 75% formulation of sulfometuron methyl is greater than 5000 mg/kg (equivalent to 3750 mg a.i./kg/day) (Filliben 1995a), indicating a low order of toxicity. In both of these studies, no mortalities occurred at any dose. Thus, the acute LD₅₀ values derived from experimental mammals are several orders of magnitude higher than any plausible exposures and have no practical impact on the risk assessment. As summarized in Appendix 1, clinical signs of toxicity, including weight loss and stained or wet perineal (genital) areas, were observed at doses of 5000 mg a.i./kg (Dashiell and Hinckle 1980c). The toxicological significance of the latter effect is unclear.

The U.S. EPA has not derived an acute/single dose RfD for sulfometuron methyl. However, a provisional acute RfD can be calculated based on the short-term NOAEL for decreased body weight of 86.6 mg/kg/day reported in the teratology study in rats by Lu (1981). As discussed in Section 3.1.9, exposure of rats to 5000 ppm dietary for 10 days during gestation resulted in decreased maternal body weight, with a NOAEL of 1000 ppm. According to the authors, the dietary concentration of 5000 ppm is equivalent to a daily dose of 433 mg/kg/day and was determined using mean daily food consumption and body weight. Thus, assuming that food consumption and body weights were similar between the 1000 and 5000 ppm exposure groups, the concentration of 1000 ppm is estimated as equivalent to 86.6 mg/kg/day [433 mg/kg/day ÷ 5]. Using a margin of exposure of 100, a provisional acute RfD can be calculated as 0.87 mg/kg/day [86.6 mg/kg/day ÷ 100].

The subchronic and chronic toxicity studies on sulfometuron methyl were conducted in rats, mice, rabbits and dogs. The most sensitive effects involve changes to blood that are consistent with hemolytic anemia, and decreased body weight gain. Study details are provided in Section 3.1.5 and Appendix 1. As discussed in Section 3.3, according to a Federal Registry Notice (U.S. EPA 1997), the U.S. EPA has derived an RfD of 0.24 mg/kg/day. This RfD is based on a NOAEL for bladder toxicity of 500 ppm dietary sulfometuron methyl (equivalent to 24.4 mg/kg/day) and a 100-fold safety factor. Although an RfD has been derived by U.S. EPA, a more conservative provisional reference dose of 0.02 mg/kg/day, which was used in the previous Forest Service risk assessment on sulfometuron methyl (Durkin 1998), was derived from data reported in the 2-year feeding study in rats by Mullin (1984). In the Mullin study, a NOAEL of 50 ppm and a LOAEL of 500 ppm for decreased erythrocyte count and hematocrit was observed in male rats. According to Mullin (1984), the dietary concentration of 50 ppm is equivalent to 2 mg a.i./kg/day. The provisional reference dose is based on the 2 mg/kg/day NOAEL for hematological effects in male rats and an uncertainty factor of 100:10 for species-to-species extrapolation and 10 for sensitive subgroups in the human population.

4.1.2.2. Birds— As summarized in Appendix 4, there are only four available studies regarding the toxicity of sulfometuron methyl to birds. In one of these studies, Dudeck and Bristol (1981b), considerably high and unexplained mortality was observed in the control group (5 of 10 animals died). Consequently, the study is not used in this risk assessment. Apparently, the other three studies, one single dose gavage study in mallard ducks (Dudeck and Bristol 1981a), one 9-day dietary study in mallard ducks (Dudeck and Twigg 1980), and one 5-day dietary study in young bobwhite quail (Fink et al. 1981) assayed only for relatively gross effects (i.e., overt signs of toxicity, changes in body weight and food consumption). No longer-term or chronic exposure

studies or standard bioassays on reproductive effects in bird were identified in the available literature. No studies investigating the acute or chronic toxicity of formulations of sulfometuron methyl to birds were identified in the available literature.

In adult mallard ducks administered single doses of technical grade sulfometuron methyl ranging from 312 to 5000 mg/kg, no mortality was observed, placing the LD₅₀ value > 5000 mg a.i./kg (Dudeck and Bristol 1981a). Compared to control birds, males, but not females had decreased weight gain at doses of 625 mg/kg and higher. Although the decrease in weight gain did not exhibit dose-dependence with respect to the magnitude of effect, the NOAEL for changes in body weight is 312 mg/kg. In the dietary exposure studies with technical grade sulfometuron methyl, no mortality or signs of toxicity were reported at concentrations up to 5000 ppm in ducks (equivalent to 332.5 mg/kg/day) (Dudeck and Twigg 1980) or up to 5620 ppm quail (equivalent to 1068 mg/kg/day) (Fink et al. 1981). All dose-conversions are described in Appendix 4. Thus, the NOAEL for short-term dietary exposure in mallard ducks is 5000 ppm (332.5 mg/kg/day) and in bobwhite quail is 5620 ppm (1068 mg/kg/day).

4.1.2.3. Terrestrial Invertebrates— There are only two available studies regarding the toxicity of sulfometuron methyl to a terrestrial invertebrate (Hoxter and Smith 1990, DuPont de Nemours 1983). Both studies used the standard contact toxicity test in bees that is required by the U.S. EPA for pesticide registration. No mortality was noted in bees exposed to up to 100 µg/bee (Hoxter and Smith 1990). In this study, nominal doses of 13, 22, 36, 60, or 100 µg a.i./bee in an ethanol vehicle were applied to 1- to 4-day post-emergence bees, with two replicates per dose level and 25 bees per replicate. The bees were observed twice a day on days 1 and 2. Similar results were reported by DuPont de Nemours 1983, with no mortality or signs of toxicity at doses of 6.25 and 12.5 µg a.i./bee. Using a body weight of 0.093 g for the honey bee (USDA 1993), these values correspond to doses ranging from about 65 mg/kg [0.00625 mg/0.000093 kg] to 1075 mg/kg [0.1 mg/0.000093 kg], with an LD₅₀ value >1075 mg/kg and the NOAEL for mortality and toxicity of 1075 mg/kg. No longer-term exposure studies for terrestrial invertebrate were identified in the available literature. No studies investigating the acute or chronic toxicity of formulations of sulfometuron methyl to terrestrial invertebrates were identified in the available literature.

4.1.2.4. Terrestrial Plants (Macrophytes)—The toxicity of sulfometuron methyl to terrestrial plants was studied extensively and is well characterized (e.g., Aulgur 1996, Gaeddert et al. 1997, Landstein et al. 1995, Schloss et al. 1988, Shaner et al. 1990, Stidham 1991). Sulfometuron methyl inhibits acetolactate synthase (ALS), an enzyme that catalyzes the biosynthesis of three branched-chain amino acids (valine, leucine, and isoleucine), all of which are essential for plant growth. This target enzyme (ALS) is also referred to as acetohydroxy acid synthase or AHAS (e.g., Epelbaum et al. 1996). Other ALS inhibiting herbicides include other sulfonylureas as well as imidazolinones, triazolopyrimidines, and pyrimidinylthiobenzoates.

The most relevant laboratory bioassay regarding the toxicity of sulfometuron methyl to terrestrial plants is summarized in appendix 5. The quantitative use of these studies for this risk assessment is discussed in Section 4.3. In a recent study submitted to the U.S. EPA (McKelvey 1995), bioassays were conducted on pre-emergence and post-emergence toxicity to corn, cucumber,

onion, pea, rape, sugar beet, sorghum, soybean, tomato, wheat. In the pre-emergence assay, the most sensitive species based on the NOEC were rape, tomato, sorghum, wheat, and corn with an NOEC of 0.0000086 lb ai/acre. The most tolerant species based on the NOEC were onion, pea, cucumber, and soybean with an NOEC of 0.00026 lb/acre. In the post-emergence assay, most sensitive species based on the NOEC is corn with an NOEC of 0.000024 lb ai/acre. The most tolerant species in the post-emergence assay was pea with an NOEC of 0.00078 lb ai/acre.

In terms of a hazard identification, however, it is noteworthy that some target species, like the leafy spurge (Beck et al. 1993) and certain species of pine (Barnes et al. 1990) are much less sensitive than a number of non-target dicots and monocots (Drake 1990) to the effects of sulfometuron methyl. Drake (1990) reports that at an application rate of 0.01 kg/ha [0.00892 lbs a.i./acre] sulfometuron methyl is highly toxic to seedlings of several broadleaves and grasses, either preemergence or postemergence. Moreover, adverse effects were observed in most plants tested at application rates of 0.001 kg/ha [0.000892 lbs a.i./acre]. This application rate is about 100-fold less than the application rate that the Forest Service would typically use.

The species differences in sensitivity may be attributable to differences in metabolism. For example, centipede grass, compared with bahiagrass, is more resistant to the effects of sulfometuron methyl because of the higher rate at which it metabolizes the compound. Another factor regarding sensitivity differences among plant species may relate to genetic differences in the form of the ALS enzyme, as appears to be the case with the dicotyledonous weed, *Sonchus oleraceus* (Boutsalis and Powles 1995).

As reviewed by Cox (1993), concern for the sensitivity of non-target species is further increased by a report of non-target plant damage after the application of sulfometuron methyl in rights-of-way maintenance. Extensive and prolonged damage to crops and ornamentals was observed after the application of sulfometuron methyl in an arid region, presumably due to wind transport of soil contaminated with sulfometuron methyl (Turner 1987), and in a region with heavy rainfall, presumably due to the wash-off of sulfometuron methyl contaminated soil (Bridges 1992).

4.1.2.5. Terrestrial Microorganisms–

Terrestrial microorganisms have an enzyme that is involved in the synthesis of branched chain amino acids, which is functionally equivalent to the target enzyme in terrestrial macrophytes. Sulfometuron methyl, at concentrations as low as 0.2 μM [$\sim 73 \mu\text{g/L}$] in a liquid glucose medium, caused significant growth inhibition in *Salmonella typhimurium* after exposure periods of less than 3 hours (Epelbaum et al. 1996). In plate cultures using solid growth media, Burnet and Hodgson (1991) found that sulfometuron methyl also inhibited the growth of several soil microorganisms. At concentrations up to 1000 $\mu\text{g/g}$ soil, no effects were observed on the microbial populations (Hadley 19??) and at soil concentrations ranging from 0.098 to 0.98 ppm no significant effects were observed on nitrate production, or cellulose, protein or starch metabolism (Anderson and Berg 1980). Study details are provided in Appendix 8.

Burnet and Hodgson (1991) suggest that soil residues of sulfometuron methyl may alter the composition of soil microorganisms and speculate further that such changes to the microbial populations in soil may lead to the proliferation of plant pathogens. This speculation is not

supported by any experimental evidence or field observations. At least one terrestrial microorganism, *Streptomyces griseolus*, metabolizes sulfometuron methyl by an inducible cytochrome P-450 (O'Keefe et al. 1988). The extent to which the induction may alter the toxicity of sulfometuron methyl to microorganisms with an inducible cytochrome P-450 was not determined. Like plants, at least some forms of bacteria may develop resistance to sulfometuron methyl (Xie and Jimenez 1996).

4.1.3. Aquatic Organisms.

4.1.3.1. Fish – Standard toxicity bioassays to assess the effects of exposure of fish to sulfometuron methyl are summarized in Appendix 5. Acute toxicity studies have been conducted in fathead minnow (Muska and Driscoll 1982), rainbow trout (Brown 1994b, Muska and Trivits 1980b) and bluegill sunfish (Brown 1994a, Muska and Hall 1980) and chronic exposure was studied in fathead minnow (Muska and Driscoll). No field studies on the effect of sulfometuron methyl in fish were identified in the published literature or the U.S. EPA files. Due to the poor solubility of sulfometuron methyl, the maximum concentration tested was 150 mg/L in the studies by Brown (1994 a,b); the maximum concentrations tested in all other studies were lower than 150 mg/L. In some studies dimethyl formamide (DMF) was added (Muska and Driscoll 1982, Muska and Hall 1980, Muska and Trivits 1980b) or the pH of the water was adjusted (Brown 1994a, b) to increase solubility. In each case, appropriate control groups were included. No studies investigating the acute or chronic toxicity of formulations of sulfometuron methyl in fish were identified in the available literature.

Investigations of the acute LC₅₀ have been hampered by the limited water solubility of sulfometuron methyl. For acute toxicity studies, the LC₅₀ values range from >7.3 mg/L in fathead minnow (Muska and Driscoll 1982) to > 150 mg/L in bluegill sunfish (Brown 1994a) and rainbow trout (Brown 1994b). The lowest concentration at which any mortality was observed in any species of fish is 1.25 mg/L. At this level, mortality was observed in 1/10 bluegill sunfish. No mortality, however, was observed in 10 bluegills exposed to 12.5 mg/L (Muska and Hall 1980). Thus, mortality does not appear to be treatment related. Since no signs of toxicity were observed in any study, NOEC values are placed at the highest concentration of sulfometuron methyl tested in each study – 7.3 mg/L for fathead minnows (Muska and Driscoll 1982), 12.5 mg/L (Muska and Hall 1980) and 150 mg/L (Brown 1994a) in bluegill sunfish, and 12.5 mg/L (Muska and Trivits 1980b) and 150 mg/L (Brown 1994b) in rainbow trout.

Muska and Driscoll (1982) is the only study available regarding the toxicity of sulfometuron methyl to fish eggs or fry. These investigators observed no effects on fathead minnow embryo hatch, larval survival, or larval growth over 30-day exposure periods in which the measured average concentrations were 0.06, 0.14, 0.32, 0.65, and 1.17 mg a.i./L.

4.1.3.2. Amphibians– The toxicity of sulfometuron methyl to amphibians has been evaluated in a single study in African Clawed frogs (*Xenopus laevis*) (Fort 1998). Results show that short -term (96 hours) and longer-term (14 and 30 days) exposure to the sulfometuron methyl resulted in alterations in limb development, organogenesis, and metamorphosis. All LC₅₀, EC₅₀, NOAEC and LOAEC values are summarized in Table 4-1. The most sensitive endpoint examined in this study was metamorphosis. To enhance solubility of the test material, DMSO (1% v/v) was

added; the authors state that this concentration of DMSO did not adversely alter normal larval development. The author did not state whether data were reported in terms of mg sulfometuron methyl/L or mg Oust/L. Taking the most conservative approach, values are assumed to be expressed in terms of mg Oust/L.

The effects of short-term exposures to sulfometuron methyl on lethality and organogenesis was studied in blastulae stage embryos of the African clawed frog (*Xenopus laevis*) (Fort 1998). The embryos were exposed to concentrations of sulfometuron methyl ranging from 0.001 to 10 mg Oust/L (the limit of solubility) for 4 days. No mortality was observed at concentrations up to 0.1 mg Oust/L (0.00075 to 7.5 mg a.i./L). Although 2.5% mortality was observed at the 0.38 mg a.i./L concentration, this was not statistically different from the control group. Malformations observed in this study include miscoiling of the gut, incomplete lens formation of the eye, and abnormal craniofacial development. The severity of these malformations was graded as moderate. At the highest concentration tested, malformations were observed in 100% of animals. As shown in Table 4-1, NOAEL and LOAEC values are the same for both mortality and malformations.

The effects of longer-term exposure of organogenesis was studied by exposing blastula stage embryos to concentrations of sulfometuron methyl ranging from 0.001 to 10 mg Oust/L for 30 days. Malformations were observed in 100% of animals at concentrations of 1.0 mg/L and 10.0 mg/L (0.75 and 7.5 mg a.i./L). A decrease in tail resorption rates, a morphological indicator of thyroid disruption, was observed in blastula stage embryos exposed to sulfometuron methyl for 14 days. In animals exposed for 14 days to concentration ranging from 0.0001 to 1 mg/L (0.000075 to 0.75 mg a.i./L), resorption rates were increased in the 0.001 and 0.01 mg/L (0.00075 and 0.0075 mg/L) treatment groups. Effects were partially reversed by the addition of thyroxine. Results indicate that exposure to sulfometuron methyl adversely affects metamorphosis in frogs.

4.1.3.3. Aquatic Invertebrates – Standard toxicity bioassays to assess the effects of exposure of aquatic invertebrates to sulfometuron methyl are summarized in Appendix 6. Acute toxicity studies on technical grade sulfometuron methyl have been conducted in *Daphnia magna* (Brown 1994b, Muska and Trivits 1980a), and on Oust have been conducted in *Daphnia magna* (Wetzel 1984), crayfish (Naqvi et al. 1987), and four other species of fresh water aquatic invertebrates (Naqvi and Hawkins 1989). Chronic exposure to technical grade sulfometuron methyl was studied in *Daphnia magna* (Bear 1990). No field studies on the effect of sulfometuron methyl in aquatic invertebrates were identified in the published literature or the U.S. EPA files.

Acute exposure to sulfometuron methyl appears to be relatively non-toxic to aquatic invertebrates, based on acute bioassays in *Daphnia* (Muska and Trivits 1980a, Brown 1994b, Wetzel 1984), crayfish (Naqvi et al. 1987), and field-collected species of *Diatomus*, *Eucyclops*, *Alonella*, and *Cypria* (Naqvi and Hawkins 1989). For studies on technical grade sulfometuron methyl in *Daphnia*, 48-hour LC₅₀ values range from > 12.5 mg/L (Muska and Trivits 1980a) to >150 mg/L (Brown 1994b), the maximum concentrations tested. In the Muska and Trivits (1980a) study, mortality was observed in 1 of 10 daphnids in the lowest exposure group (0.125 ppm) (Muska and Trivits 1980a) and in the DMF control group, but not in groups exposed to

1.25 and 12.5 ppm. Thus, mortality does not appear to be related to sulfometuron methyl exposure.

The LC₅₀ value reported in *Daphnia* for Oust is considerably higher than for technical grade sulfometuron methyl (8500 mg Oust/L or 6375 mg a.i./L), with a NOEC of 2400 mg Oust/L (1800 mg a.i./L) and an LOEC of 3200 mg Oust/L (2400 mg a.i./L) (Wetzel 1984). High LC₅₀ values ranging from 802 mg Oust/L (602 mg a.i./L) in *Alonella* sp. to 2241 mg Oust/L (1681 mg a.i./L) in *Cypria* sp. were reported by Naqvi and Hawkins 1989, with LOEC values for both species of 100 mg Oust/L (75 mg a.i./L), the lowest dose tested. The highest LC₅₀ value of 12,174 mg/L was reported in crayfish (Naqvi et al. 1987). Neither of the Naqvi studies specifically state whether data are reported in term of mg Oust/L or mg a.i./L, although it is implied that data are reported in terms of the formulation; taking the most conservative approach, it is inferred that data are reported in terms of mg Oust/L. However, regardless of how these data are expressed, crayfish appear to be far more tolerant to the effects of sulfometuron methyl than other aquatic invertebrate species tested based on LC₅₀ values. Due to the higher water solubility of Oust, acute toxicity studies could test much higher concentrations than those evaluated in studies on technical grade sulfometuron methyl. Unfortunately, since the full dose-response relationship could not be defined in studies with technical grade sulfometuron methyl, comparisons of LC₅₀ values of Oust and technical grade sulfometuron methyl are of limited value.

One daphnid reproduction study on technical grade sulfometuron methyl was conducted (Baer 1990). As indicated in Appendix 6, the number of neonates per surviving adult was significantly reduced at 24 mg/L but not at 97 mg/L or any of the lower concentrations. The authors report the NOEC as 6.1 mg a.i./L. Although the effect observed at 24 mg/L may have been a random variation, it is treated as an LOEC for the purpose of this risk assessment. While this approach may be regarded as conservative, in the absence of additional studies regarding reproductive effects in aquatic invertebrates, the approach seems prudent. Studies investigating effects of chronic exposure to sulfometuron methyl formulations were not identified in the available literature.

4.1.3.4. Aquatic Plants– The toxicity of sulfometuron methyl has been examined in both algae and aquatic macrophytes. Study results are summarized in Appendix 6. Studies on the mechanism of action of sulfometuron methyl in aquatic plants were not identified. However, sulfometuron methyl is assumed have the same mechanism in aquatic plants as in terrestrial plants (i.e., the inhibition of ALS as described in Section 4.1.2.4). As might be expected for a herbicide, aquatic plants are far more sensitive than aquatic animals to the effects of sulfometuron methyl.

Little information is available on the effects of sulfometuron methyl on aquatic macrophytes – one 7-day exposure study in *Hydrilla verticillata* (Byl et al. 1994), an aquatic angiosperm commonly called *Hydrilla* or water thyme, and one 14-day exposure study in *Lemna gibba*, a species of duckweed (Kannuck and Sloman 1995). For the Byl et al. (1994) study, the authors did not state whether data were reported in terms of mg sulfometuron methyl/L or mg Oust/L. Taking the most conservative approach, values are assumed to be expressed in terms of mg

Oust/L. In hydrilla the 7-day EC_{50} value for growth inhibition was reported as 10 μg Oust/L (7.5 μg a.i./L), with an NOEC value for growth inhibition of 1 μg Oust/L (0.75 μg a.i./L). In duckweed exposed to technical grade sulfometuron methyl for 14 days, the most sensitive effect was on frond count, with an EC_{50} value of 0.462 μg a.i./L and an NOEC of 0.207 μg a.i./L. No field studies on the effects of sulfometuron methyl in aquatic macrophytes were identified in the available literature.

Three studies have investigated the effects of sulfometuron methyl on algae, as detailed in Appendix 6 (Hoberg 1990, Landstein et al. 1993, Thompson 1994), with dose-response data available in *Selenastrum capricornutum* (Hoberg 1990) and *Anabaen flosaquae* (Thompson 1994). Based on growth inhibition as measured by cell density, *Selenastrum capricornutum*, with a 120-hour EC_{50} value of 4.6 $\mu\text{g}/\text{L}$ (Hoberg 1990), appears to be more sensitive to the effects of sulfometuron methyl than *Anabaen flosaquae*, with a 120-hour EC_{50} value of 65 $\mu\text{g}/\text{L}$ (Thompson 1994). In the Hoberg study, growth stimulation was observed at concentrations up to 2.5 $\mu\text{g}/\text{L}$ following 72 hours of exposure; thus, the NOEC could be taken as 2.5 $\mu\text{g}/\text{L}$. In *Navicula pelliculosa* exposed to 370 $\mu\text{g}/\text{L}$, no growth inhibition was observed (Thompson 1994); thus, the NOEC for *Navicula pelliculosa* is 370 $\mu\text{g}/\text{L}$. No field studies on the effects of sulfometuron methyl in aquatic macrophytes were identified in the available literature.

4.2. EXPOSURE ASSESSMENT

4.2.1. Overview.

Terrestrial animals might be exposed to any applied herbicide from direct spray, the ingestion of contaminated media (vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation. In acute exposure scenarios, the highest exposure for terrestrial vertebrates involves the consumption of contaminated insects by a small bird, which could reach up to about 5 mg/kg. There is a wide range of exposures anticipated from the consumption of contaminated vegetation by terrestrial animals: central estimates range from 0.06 mg/kg for a small mammal to 1.2 mg/kg for a large bird under typical exposure conditions, with upper ranges of about 0.1 mg/kg for a small mammal and 3.4 mg/kg for a large bird. The consumption of contaminated water will generally lead to much lower levels of exposure. A similar pattern is seen for chronic exposures. The central estimate for daily doses for a small mammal from the longer term consumption of contaminated vegetation at the application site is about 0.0009 mg/kg/day, with an upper estimate of about 0.004 mg/kg/day. Longer term exposures from contaminated vegetation far exceed doses that are anticipated from the consumption of contaminated water, which has a central estimate of about 0.0000003 mg/kg/day and an upper range of about 0.0000005 for a small mammal. Based on general relationships of body size to body volume, larger vertebrates will be exposed to lower doses than small vertebrates under comparable exposure conditions. Because of the apparently low toxicity of sulfometuron methyl to animals, the rather substantial variations in the different exposure assessments have little impact on the assessment of risk to terrestrial animals.

For terrestrial plants, five exposure scenarios are considered quantitatively: direct spray, spray drift, runoff, wind erosion and the use of contaminated irrigation water. Unintended direct spray is expressed simply as the application rate considered in this risk assessment, 0.045 lb a.e./acre and should be regarded as an extreme/accidental form of exposure that is not likely to occur in most Forest Service applications. Estimated levels of exposure for the other scenarios are much less. All of these exposure scenarios are dominated by situational variability because the levels of exposure are highly dependent on site-specific conditions. Thus, the exposure estimates are intended to represent conservative but plausible ranges that could occur but these ranges may over-estimate or under-estimate actual exposures in some cases. Spray drift is based on estimates AGDRIFT. The proportion of the applied amount transported off-site from runoff is based on GLEAMS modeling of clay, loam, and sand. The amount of sulfometuron methyl that might be transported off-site from wind erosion is based on estimates of annual soil loss associated with wind erosion and the assumption that the herbicide is incorporated into the top 1 cm of soil. Exposure from the use of contaminated irrigation water is based on the same data used to estimate human exposure from the consumption of contaminated ambient water and involves both monitoring studies as well as GLEAMS modeling.

Exposures of aquatic plants and animals to sulfometuron methyl are based on essentially the same information used to assess the exposure to terrestrial species from contaminated water. The peak estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.001 (0.00006 to 0.02) mg a.e./L at an application rate of 1 lb a.e./acre. For longer-term exposures, average estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.00004 (0.00001 to 0.00007) mg a.e./L at

an application rate of 1 lb a.e./acre. For the assessment of potential hazards, these contamination rates are adjusted based on the application rates considered in this risk assessment.

4.2.2. Terrestrial Animals. Terrestrial animals might be exposed to any applied herbicide from direct spray, the ingestion of contaminated media (vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation.

In this exposure assessment, estimates of oral exposure are expressed in the same units as the available toxicity data. As in the human health risk assessment, these units are usually expressed as mg of agent per kg of body weight and abbreviated as mg/kg. For dermal exposure, the units of measure usually are expressed in mg of agent per cm of surface area of the organism and abbreviated as mg/cm². In estimating dose, however, a distinction is made between the exposure dose and the absorbed dose. The *exposure dose* is the amount of material on the organism (i.e., the product of the residue level in mg/cm² and the amount of surface area exposed), which can be expressed either as mg/organism or mg/kg body weight. The *absorbed dose* is the proportion of the exposure dose that is actually taken in or absorbed by the animal.

The exposure assessments for terrestrial animals are summarized in Worksheet G01. As with the human health exposure assessment, the computational details for each exposure assessment presented in this section are provided scenario specific worksheets (Worksheets F01 through F16b). Given the large number of species that could be exposed to herbicides and the varied diets in each of these species, a very large number of different exposure scenarios could be generated. For this generic – i.e., not site- or species-specific – risk assessment, an attempt is made to limit the number of exposure scenarios.

Because of the relationship of body weight to surface area as well as the consumption of food and water, small animals will generally receive a higher dose, in terms of mg/kg body weight, than large animals will receive for a given type of exposure. Consequently, most general exposure scenarios for mammals and birds are based on a small mammal or bird. For small mammals, the body weight is taken as 20 grams, typical of mice, and exposure assessments are conducted for direct spray (F01 and F02a), consumption of contaminated fruit (F03, F04a, F04b), and the consumption of contaminated water (F05, F06, F07). Grasses will generally have higher concentrations of herbicides than fruits and other types of vegetation (Fletcher et al. 1994; Hoerger and Kenaga 1972). Because small mammals do not generally consume large amounts of grass, the scenario for the assessment of contaminated grass is based on a large mammal – a deer (Worksheets F10, F11a, and F11b). Other exposure scenarios for a mammals involve the consumption of contaminated insects by a small mammal (Worksheet F14a) and the consumption of small mammals by a large mammalian carnivore (Worksheet F16a). Exposure scenarios for birds involve the consumption of contaminated insects by a small bird (Worksheet F14b), the consumption of contaminated fish by a predatory bird (Worksheets F08 and F09), the consumption of small mammals by a predatory bird (F16b), and the consumption of contaminated grasses by a large bird (F12, F13a, and F13b).

While a very large number of other exposure scenarios could be generated, the specific exposure scenarios developed in this section are designed as conservative screening scenarios that may serve as guides for more detailed site-specific assessments.

4.2.2.1. Direct Spray – In the broadcast application of any herbicide, wildlife species may be sprayed directly. This scenario is similar to the accidental exposure scenarios for the general public discussed in Section 3.2.3.2. In a scenario involving exposure to direct spray, the amount absorbed depends on the application rate, the surface area of the organism, and the rate of absorption.

For this risk assessment, three groups of direct spray exposure assessments are conducted. The first, which is defined in Worksheet F01, involves a 20 g mammal that is sprayed directly over one half of the body surface as the chemical is being applied. The range of application rates as well as the typical application rate is used to define the amount deposited on the organism. The absorbed dose over the first day (i.e., a 24-hour period) is estimated using the assumption of first-order dermal absorption. In the absence of any data regarding dermal absorption in a small mammal, the estimated absorption rate for humans is used (see Section 3.1.3). An empirical relationship between body weight and surface area (Boxenbaum and D'Souza 1990) is used to estimate the surface area of the animal. The estimates of absorbed doses in this scenario may bracket plausible levels of exposure for small mammals based on uncertainties in the dermal absorption rate of sulfometuron methyl.

Other, perhaps more substantial, uncertainties affect the estimates for absorbed dose. For example, the estimate based on first-order dermal absorption does not consider fugitive losses from the surface of the animal and may overestimate the absorbed dose. Conversely, some animals, particularly birds and mammals, groom frequently, and grooming may contribute to the total absorbed dose by direct ingestion of the compound residing on fur or feathers. Furthermore, other vertebrates, particularly amphibians, may have skin that is far more permeable than the skin of most mammals. Quantitative methods for considering the effects of grooming or increased dermal permeability are not available. As a conservative upper limit, the second exposure scenario, detailed in Worksheet F02, is developed in which complete absorption over day 1 of exposure is assumed.

Because of the relationship of body size to surface area, very small organisms, like bees and other terrestrial insects, might be exposed to much greater amounts of sulfometuron methyl per unit body weight, compared with small mammals. Consequently, a third exposure assessment is developed using a body weight of 0.093 g for the honey bee (USDA/APHIS 1993) and the equation above for body surface area proposed by Boxenbaum and D'Souza (1990). Because there is no information regarding the dermal absorption rate of sulfometuron methyl by bees or other invertebrates, this exposure scenario, detailed in Worksheet F02b, also assumes complete absorption over the first day of exposure.

Direct spray scenarios are not given for large mammals. As noted above, allometric relationships dictate that large mammals will be exposed to lesser amounts of a compound in any direct spray scenario than smaller mammals. As detailed further in Section 4.4, the direct spray scenarios for the small mammal are substantially below a level of concern. Consequently, elaborating direct spray scenarios for a large mammal would have no impact on the characterization of risk.

4.2.2.2. Indirect Contact – As in the human health risk assessment (see Section 3.2.3.3), the only approach for estimating the potential significance of indirect dermal contact is to assume a relationship between the application rate and dislodgeable foliar residue. The study by Harris and Solomon (1992) (Worksheet A04) is used to estimate that the dislodgeable residue will be approximately 10 times less than the nominal application rate.

Unlike the human health risk assessment in which transfer rates for humans are available, there are no transfer rates available for wildlife species. As discussed in Durkin et al. (1995), the transfer rates for humans are based on brief (e.g., 0.5 to 1-hour) exposures that measure the transfer from contaminated soil to uncontaminated skin. Wildlife, compared with humans, are likely to spend longer periods of time in contact with contaminated vegetation.

It is reasonable to assume that for prolonged exposures an equilibrium may be reached between levels on the skin, rates of absorption, and levels on contaminated vegetation, although there are no data regarding the kinetics of such a process. The bioconcentration data on sulfometuron methyl suggest that sulfometuron methyl may accumulate to a small degree in muscle and viscera of fish (section 3.2.3.5). However, the high water solubility and low octanol/water partition coefficient for sulfometuron methyl suggest that sulfometuron methyl is not likely to partition from the surface of contaminated vegetation to the surface of skin, feathers, or fur. Thus, a plausible partition coefficient is unity (i.e., the concentration of the chemical on the surface of the animal will be equal to the dislodgeable residue on the vegetation).

Under these assumptions, the absorbed dose resulting from contact with contaminated vegetation will be one-tenth that associated with comparable direct spray scenarios. As discussed in the risk characterization for ecological effects (Section 4.4), the direct spray scenarios result in exposure levels below the estimated NOAEL (i.e., hazard quotients below one). Consequently, details of the indirect exposure scenarios for contaminated vegetation are not further elaborated in this document.

4.2.2.3. Ingestion of Contaminated Vegetation or Prey – Since sulfometuron methyl will be applied to vegetation, the consumption of contaminated vegetation is an obvious concern and separate exposure scenarios are developed for acute and chronic exposure scenarios for a small mammal (Worksheets F04a and F04b) and large mammal (Worksheets F10, F11a, and F11b) as well as large birds (Worksheets F12, F13a, and F13b).

For the consumption of contaminated vegetation, a small mammal is used because allometric relationships indicate that small mammals will ingest greater amounts of food per unit body

weight, compared with large mammals. The amount of food consumed per day by a small mammal (i.e., an animal weighing approximately 20 g) is equal to about 15% of the mammal's total body weight (U.S. EPA/ORD 1989). When applied generally, this value may overestimate or underestimate exposure in some circumstances. For example, a 20 g herbivore has a caloric requirement of about 13.5 kcal/day. If the diet of the herbivore consists largely of seeds (4.92 kcal/g), the animal would have to consume a daily amount of food equivalent to approximately 14% of its body weight $[(13.5 \text{ kcal/day} \div 4.92 \text{ kcal/g}) \div 20 \text{ g} = 0.137]$. Conversely, if the diet of the herbivore consists largely of vegetation (2.46 kcal/g), the animal would have to consume a daily amount of food equivalent to approximately 27% of its body weight $[(13.5 \text{ kcal/day} \div 2.46 \text{ kcal/g}) \div 20 \text{ g} = 0.274]$ (U.S. EPA/ORD 1993, pp.3-5 to 3-6). For this exposure assessment (Worksheet F03), the amount of food consumed per day by a small mammal weighing 20 g is estimated at about 3.6 g/day or about 18% of body weight per day from the general allometric relationship for food consumption in rodents (U.S. EPA/ORD 1993, p. 3-6).

A large herbivorous mammal is included because empirical relationships of concentrations of pesticides in vegetation, discussed below, indicate that grasses may have substantially higher pesticide residues than other types of vegetation such as forage crops or fruits (Worksheet A04). Grasses are an important part of the diet for some large herbivores, but most small mammals do not consume grasses as a substantial proportion of their diet. Thus, even though using residues from grass to model exposure for a small mammal is the most conservative approach, it is not generally applicable to the assessment of potential adverse effects. Hence, in the exposure scenarios for large mammals, the consumption of contaminated range grass is modeled for a 70 kg herbivore, such as a deer. Caloric requirements for herbivores and the caloric content of vegetation are used to estimate food consumption based on data from U.S. EPA/ORD (1993). Details of these exposure scenarios are given in worksheets F10 for acute exposures as well as Worksheets F11a and F11b for longer-term exposures.

For the acute exposures, the assumption is made that the vegetation is sprayed directly – i.e., the animal grazes on site – and that 100% of the animal's diet is contaminated. While appropriately conservative for acute exposures, neither of these assumptions are plausible for longer-term exposures. Thus, for the longer-term exposure scenarios for the large mammal, two sub-scenarios are given. The first is an on-site scenario that assumes that a 70 kg herbivore consumes short grass for a 90 day period after application of the chemical. In the worksheets, the contaminated vegetation is assumed to account for 30% of the diet with a range of 10% to 100% of the diet. These are essentially arbitrary assumptions reflecting grazing time at the application site by the animal. Because the animal is assumed to be feeding at the application site, drift is set to unity - i.e., direct spray. This scenario is detailed in Worksheet 11a. The second sub-scenario is similar except the assumption is made that the animal is grazing at distances of 25 to 100 feet from the application site (lowering risk) but that the animal consumes 100% of the diet from the contaminated area (increasing risk). For this scenario, detailed in Worksheet F12b, AgDRIFT is used to estimate deposition on the off-site vegetation. Drift estimates from AgDrift are summarized in Worksheet A06 and this model is discussed further in Section 4.2.3.2.

The consumption of contaminated vegetation is also modeled for a large bird. For these exposure scenarios, the consumption of range grass by a 4 kg herbivorous bird, like a Canada Goose, is modeled for both acute (Worksheet F12) and chronic exposures (Worksheets F13a and F13b). As with the large mammal, the two chronic exposure scenarios involve sub-scenarios for on-site as well as off-site exposure.

For this component of the exposure assessment, the estimated amounts of pesticide residue in vegetation are based on the relationship between application rate and residue rates on different types of vegetation. As summarized in Worksheet A04, these residue rates are based on estimated residue rates from Fletcher et al. (1994).

Similarly, the consumption of contaminated insects is modeled for a small (10g) bird and a small (20g) mammal. No monitoring data have been encountered on the concentrations of sulfometuron methyl in insects after applications of sulfometuron methyl. The empirical relationships recommended by Fletcher et al. (1994) are used as surrogates as detailed in Worksheets F14a and F14b. To be conservative, the residue rates from small insects are used – i.e., 45 to 135 ppm per lb/ac – rather than the residue rates from large insects – i.e., 7 to 15 ppm per lb/ac.

A similar set of scenarios are provided for the consumption of small mammals by either a predatory mammal (Worksheet 16a) or a predatory bird (Worksheet 16a). Each of these scenarios assume that the small mammal is directly sprayed at the specified application and the concentration of the compound in the small mammal is taken from the worksheet for direct spray of a small mammal under the assumption of 100% absorption (Worksheet F02a).

In addition to the consumption of contaminated vegetation and insects, sulfometuron methyl may reach ambient water and fish. Thus, a separate exposure scenario is developed for the consumption of contaminated fish by a predatory bird in both acute (Worksheet F08) and chronic (Worksheet F09) exposures. Because predatory birds usually consume more food per unit body weight than do predatory mammals (U.S. EPA 1993, pp. 3-4 to 3-6), separate exposure scenarios for the consumption of contaminated fish by predatory mammals are not developed.

4.2.2.4. Ingestion of Contaminated Water – Estimated concentrations of sulfometuron methyl in water are identical to those used in the human health risk assessment (Worksheet B06). The only major differences involve the weight of the animal and the amount of water consumed. There are well-established relationships between body weight and water consumption across a wide range of mammalian species (e.g., U.S. EPA 1989). Mice, weighing about 0.02 kg, consume approximately 0.005 L of water/day (i.e., 0.25 L/kg body weight/day). These values are used in the exposure assessment for the small (20 g) mammal. Unlike the human health risk assessment, estimates of the variability of water consumption are not available. Thus, for the acute scenario, the only factors affecting the variability of the ingested dose estimates include the field dilution rates (i.e., the concentration of the chemical in the solution that is spilled) and the amount of solution that is spilled. As in the acute exposure scenario for the human health risk

assessment, the amount of the spilled solution is taken as 200 gallons. In the exposure scenario involving contaminated ponds or streams due to contamination by runoff or percolation, the factors that affect the variability are the water contamination rate, (see Section 3.2.3.4.2) and the application rate. Details regarding these calculations are summarized in Worksheets F06 and Worksheet F07.

4.2.3. Terrestrial Plants. In general, the primary hazard to non-target terrestrial plants associated with the application of most herbicides is unintended direct deposition or spray drift. In addition, herbicides may be transported off-site by percolation or runoff or by wind erosion of soil.

4.2.3.1. Direct Spray – Unintended direct spray will result in an exposure level equivalent to the application rate. For many types of herbicide applications – e.g., rights-of-way management – it is plausible that some non-target plants immediately adjacent to the application site could be sprayed directly. This type of scenario is modeled in the human health risk assessment for the consumption of contaminated vegetation.

4.2.3.2. Off-Site Drift – Because off-site drift is more or less a physical process that depends on droplet size and meteorological conditions rather than the specific properties of the herbicide, estimates of off-site drift can be modeled using AgDRIFT (Teske et al. 2001). AGDRIFT is a model developed as a joint effort by the EPA Office of Research and Development and the Spray Drift Task Force, a coalition of pesticide registrants. AGDRIFT is based on the algorithms in FSCBG (Teske and Curbishley, 1990), a drift model previously used by USDA.

For aerial applications, AGDRIFT permits very detailed modeling of drift based on the chemical and physical properties of the applied product, the configuration of the aircraft, as well as wind speed and temperature. For ground applications, AGDRIFT provides estimates of drift based solely on distance downwind as well as the types of ground application: low boom spray, high boom spray, and orchard airblast. Representative estimates based on AGDRIFT (Version 1.16) are given in Worksheet A06. For the current risk assessment, the AGDRIFT estimates are used for consistency with comparable exposure assessments conducted by the U.S. EPA. In addition, AGDRIFT represents a detailed evaluation of a very large number of field studies and is likely to provide more reliable estimates of drift. Further details of AGDRIFT are available at <http://www.agdrift.com/>.

Estimates of drift for ground and aerial applications is given in Worksheet A06. In ground broadcast applications, sulfometuron methyl will typically be applied by low boom ground spray and thus these estimates are used in the current risk assessment.

Drift associated with backpack (directed foliar applications) are likely to be much less although studies quantitatively assessing drift after backpack applications have not been encountered. Drift distance can be estimated using Stoke's law, which describes the viscous drag on a moving sphere. According to Stoke's law:

$$v = \frac{D^2 \cdot g}{18n}$$

or

$$v = 2.87 \cdot 10^5 \cdot D^2$$

where v is the velocity of fall (cm sec^{-1}), D is the diameter of the sphere (cm), g is the force of gravity (980 cm sec^{-2}), and n is the viscosity of air ($1.9 \cdot 10^{-4} \text{ g sec}^{-1} \text{ cm}^{-1}$ at 20°C) (Goldstein et al. 1974).

In typical backpack ground sprays, droplet sizes are greater than 100μ , and the distance from the spray nozzle to the ground is 3 feet or less. In mechanical sprays, raindrop nozzles might be used. These nozzles generate droplets that are usually greater than 400μ , and the maximum distance above the ground is about 6 feet. In both cases, the sprays are directed downward.

Thus, the amount of time required for a 100μ droplet to fall 3 feet (91.4 cm) is approximately 3.2 seconds,

$$91.4 \div (2.87 \cdot 10^5 (0.01)^2).$$

The comparable time for a 400μ droplet to fall 6 feet (182.8 cm) is approximately 0.4 seconds,

$$182.8 \div (2.87 \cdot 10^5 (0.04)^2).$$

For most applications, the wind velocity will be no more than 5 miles/hour, which is equivalent to approximately 7.5 feet/second (1 mile/hour = 1.467 feet/second). Assuming a wind direction perpendicular to the line of application, 100μ particles falling from 3 feet above the surface could drift as far as 23 feet (3 seconds \cdot 7.5 feet/second). A raindrop or 400μ particle applied at 6 feet above the surface could drift about 3 feet (0.4 seconds \cdot 7.5 feet/second).

For backpack applications, wind speeds of up to 15 miles/hour are allowed in Forest Service programs. At this wind speed, a 100μ droplet can drift as far as 68 feet (3 seconds \cdot 15 \cdot 1.5 feet/second). Smaller droplets will of course drift further, and the proportion of these particles in the spray as well as the wind speed and turbulence will affect the proportion of the applied herbicide that drifts off-site.

4.2.3.3. Runoff – Sulfometuron methyl or any other herbicide may be transported to off-site soil by runoff or percolation. Both runoff and percolation are considered in estimating contamination of ambient water. For assessing off-site soil contamination, however, only runoff is considered. This approach is reasonable because off-site runoff will contaminate the off-site soil surface and could impact non-target plants. Percolation, on the other hand, represents the amount of the herbicide that is transported below the root zone and thus may impact water quality but should not affect off-site vegetation.

Based on the results of the GLEAMS modeling (Section 3.2.3.4.2), the proportion of the applied sulfometuron methyl lost by runoff was estimated for clay, loam, and sand at rainfall rates ranging from 5 inches to 250 inches per year. These results are summarized in Worksheet G04 and indicate that runoff will be negligible in relatively arid environments as well as sandy or loam soils. In clay soils, which have the highest runoff potential, off-site loss may reach up to about 60% of the applied amount in regions with very high rainfall rates.

Two detailed studies (Hubbard et al. 1989, Wauchope et al. 1990) that investigate the fate and transport of sulfometuron methyl in soil are useful for assessing the potential for off-site vegetation to be exposed to sulfometuron methyl.

In the Hubbard et al. (1989) study, 0.6-4.48 kg/ha sulfometuron methyl was applied to three types of soil: sandy clay loam, loamy sand, and sand. The soil surfaces were free of vegetation, and the soil slope was 2%. One day before application, the soils were saturated with water by backflushing, which maximized the potential for runoff. Rainfall rates of 125, 75, and 43 mm/hour were then simulated for 2 hours, and runoff and percolation were measured. Concentrations of sulfometuron methyl in runoff were less than 2.4 $\mu\text{g/mL}$ (2.4 ppm), and the concentrations in percolate were less than 0.1 $\mu\text{g/mL}$ (0.1 ppm). Under low rainfall conditions (43 mm/hour), relatively little sulfometuron methyl was removed by runoff: 0-4.2% of the applied amount with the greatest proportion found in sandy clay loam. Under moderate or high levels of rainfall, however, up to 34.7% of the applied amount was lost by runoff. Again, the greatest losses were noted in the sandy clay loam soil, and losses were not as great in loamy sand or sand. As would be expected, percolation was generally greater in the sandier soils. As part of the study, Hubbard et al. (1989) compared the results of GLEAMS modeling with the monitoring runoff. In all instances, GLEAMS under-predicted runoff, in some cases by a factor of more than 30, with the greatest discrepancies apparent under heavy rainfall. According to the investigators, these discrepancies are probably attributable to the 1-day time step used by GLEAMS, which fails to account for rapid water and herbicide movement during short-term but intense rainfall events.

In the Wauchope et al. (1990), study, sulfometuron methyl was applied at a rate of 0.4 kg/ha to a sandy loam soil with an average slope of 2.5%. Bare soil as well as soil covered with Bermudagrass and Bahiagrass were used. Beginning 5 days before application, simulated rainfall was applied until runoff occurred. Thus, although the soil was moist at the time of application, like it was in the Hubbard et al. (1989) study, the soil was probably not as moist because of the longer period of time between effective soil saturation and herbicide application. After application, simulated rainfall was applied until 10 to 12 500 mL runoff samples were collected. Although rainfall rates are not specified, total rainfall ranged from about 12 to 30 mm at each site. Thus, the amount of rainfall in this study was substantially less than that in the Hubbard et al. (1989) study, in which the lowest rate used was 43 mm/hour for 2 hours. In all cases, the fractional loss in runoff ranged from 0.7 to 1.4% of the applied sulfometuron methyl and did not differ substantially on bare and covered plots. For this study, unlike the study by Hubbard et al. (1989), the GLEAMS model did a good job of predicting the amount of sulfometuron methyl

runoff. The difference may be due to the lesser amounts of rainfall in the Wauchope et al. (1990), which would tend to diminish the importance of brief intense rainfall events.

These studies by Hubbard et al. (1989) and Wauchope et al. (1990) are fairly consistent with one another. The runoff losses of 0.7-1.4% from sandy loam soil after 12-30 mm of rain, observed by Wauchope et al. (1990), are comparable to the 0-4.2% runoff losses after a total rainfall of 84 mm (43 mm/hour for 2 hours), reported by Hubbard et al. (1989).

For this exposure assessment, these studies generally support the supposition that at least 1% of the applied sulfometuron methyl could run off from the application site to adjoining areas after a moderate rain. In the case of a heavy rain, losses could be much greater and might approach 50% in cases of extremely heavy rain and a steep soil slope.

4.2.3.4. Contaminated Irrigation Water – Unintended direct exposures of nontarget plant species may occur through the use of contaminated ambient water for irrigation. Although there are no studies in the literature addressing the impact of sulfometuron methyl in contaminated irrigation water, the effects of such exposure scenarios on non-target vegetation have been observed with other herbicides (e.g., Bhandary et al. 1991). Furthermore, given the mobility of sulfometuron methyl, the contamination of irrigation water is a plausible scenario.

The levels of exposure associated with this scenario will depend on the concentration of sulfometuron methyl in the ambient water used for irrigation and the amount of irrigation water that is applied. As detailed in Section 3.2.3.4, sulfometuron methyl is relatively mobile and contamination of ambient water may be anticipated and can be quantified (i.e., 0.001 [0.00006 to 0.02] mg a.e./L at an application rate of 1 lb a.e./acre [Worksheet B06]).

The amount of irrigation water that may be applied will be highly dependent on the climate, soil type, topography, and plant species under cultivation. Thus, the selection of an irrigation rate is somewhat arbitrary. Typically, plants require 0.1 to 0.3 inch of water per day (Delaware Cooperative Extension Service 1999). In the absence of any general approach of determining and expressing the variability of irrigation rates, the application of one inch of irrigation water will be used in this risk assessment. This is somewhat higher than the maximum daily irrigation rate for sandy soil (0.75 inches/day) and substantially higher than the maximum daily irrigation rate for clay (0.15 inches/day) (Delaware Cooperative Extension Service 1999). This variability is addressed further in the risk characterization (Section 4.4.2.2).

Based on the estimated concentrations of sulfometuron methyl in ambient water and an irrigation rate of 1 inch per day, the estimated functional application rate of sulfometuron methyl to the irrigated area is 1.02×10^{-6} ($6.11 \times 10^{-8} - 2.04 \times 10^{-5}$) lb a.e./acre (see Worksheet F15 for details of these calculations). This level of exposure is inconsequential relative to off-site drift and runoff. Specifically, off-site movement from runoff can result in functional offsite application rates of 2.46×10^{-2} lb a.e./acre (Worksheet G04) and offsite movement from drift can result in functional

offsite application rates of about 8×10^{-4} lb a.e. after ground broadcast applications (Worksheet G05a).

4.2.3.5. Wind Erosion – Wind erosion is a major transport mechanism for soil (e.g., Winegardner 1996). Although no specific incidents of nontarget damage from wind erosion have been encountered in the literature for sulfometuron methyl, this mechanism has been associated with the environmental transport of other herbicides (Buser 1990). Numerous models have been developed for wind erosion (e.g., Streck and Spaan 1997; Streck and Stein 1997) and the quantitative aspects of soil erosion by wind are extremely complex and site specific. Field studies conducted on agricultural sites found that wind erosion may account for annual soil losses ranging from 2 to 6.5 metric tons/ha (Allen and Fryrear 1977). The upper range reported by Allen and Fryrear (1977) is nearly the same as the rate of 2.2 tons/acre (5.4 tons/ha) recently reported by the USDA (1998). The temporal sequence of soil loss (i.e., the amount lost after a specific storm event involving high winds) depends heavily on soil characteristics as well as meteorological and topographical conditions.

To estimate the potential transport of sulfometuron methyl by wind erosion, this risk assessment uses average soil losses ranging from 1 to 10 tons/ha·year, with a typical value of 5 tons/ha·year. The value of 5 tons/ha·year is equivalent to 500 g/m^2 (1 ton=1000 kg and 1 ha = 10,000 m^2) or 0.05 g/cm^2 ($1 \text{ m}^2=10,000 \text{ cm}^2$). Using a soil density of 2 g/cm^3 , the depth of soil removed from the surface per year would be 0.025 cm [$(0.05 \text{ g/cm}^2) \div (2 \text{ g/cm}^3)$]. The average amount per day would be about 0.00007 cm/day ($0.025 \text{ cm per year} \div 365 \text{ days/year}$). This central estimate is based on a typical soil loss rate of 5 tons/ha·year. Since the range of plausible rates of annual soil loss is 1 to 10 tons/ha·year, the range of soil loss per day may be calculated as 0.00001 cm/day ($0.00007 \div 5 = 0.000014$) to 0.0001 cm/day ($0.00007 \times 2 = 0.00014$).

The amount of sulfometuron methyl that might be transported by wind erosion depends on several factors, including the application, the depth of incorporation into the soil, the persistence in the soil, the wind speed, and the topographical and surface conditions of the soil. Under desirable conditions, like relatively deep (10 cm) soil incorporation, low wind speed, and surface conditions that inhibit wind erosion, it is likely that wind transport of sulfometuron methyl would be neither substantial nor significant. For this risk assessment, it will be assumed that sulfometuron methyl is incorporated into the top 1 cm of soil. Thus, daily soil losses expressed as a proportion of applied amount would be 0.00007 with a range of 0.00001 to 0.001 .

As with the deposition of sulfometuron methyl in runoff, the deposition of the sulfometuron methyl contaminated soil from wind erosion will vary substantially with local conditions and, for this risk assessment, neither concentration nor dispersion is considered quantitatively. Nonetheless, these factors together with the general and substantial uncertainties in the exposure assessment are considered in the risk characterization (see Section 4.4).

4.2.4. Soil Organisms. Limited data are available on the toxicity of sulfometuron methyl to microorganisms (Section 4.1.2.5). The toxicity data are expressed in units of soil concentration –

i.e., mg sulfometuron methyl/kg soil which is equivalent to parts per million (ppm) concentrations in soil. The GLEAMS modeling discussed in Section 3.2.3.4 provides estimates of concentration in soil as well as estimates of off-site movement (runoff, sediment, and percolation). Based on the GLEAMS modeling, concentrations in clay, loam, and sand over a wide range of rainfall rates are summarized in Table 4-2. As indicated in this table, peak soil concentrations in the range of about 6 ppm are likely in relatively arid soils at an application rate of 1 lb a.e./acre. As rainfall rate increases, maximum soil concentrations are substantially reduced in sand and, to a lesser extent, in loam because of losses from soil through percolation. The potential consequences of such exposures are discussed in Section 4.4 (Risk Characterization).

4.2.5. Aquatic Organisms. The potential for effects on aquatic species are based on estimated concentrations of sulfometuron methyl in water that are identical to those used in the human health risk assessment (Worksheet B06). As summarized in Worksheet B06, the peak estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.001 (0.00006 to 0.02) mg a.e./L at an application rate of 1 lb a.e./acre. For longer-term exposures, average estimated rate of contamination of ambient water associated with the normal application of sulfometuron methyl is 0.00004 (0.00001 to 0.00007 mg a.e./L at an application rate of 1 lb a.e./acre. For the assessment of potential hazards, these contamination rates are adjusted based on the application considered in this risk assessment – i.e., 0.045 lb a.e./acre. The consequences of using higher application rates is discussed in the risk characterization (Section 4.4).

4.3. DOSE-RESPONSE ASSESSMENT

4.3.1. Overview. For terrestrial mammals, the dose-response assessment for chronic exposure to sulfometuron methyl is based on the same data as the human health risk assessment (i.e., the chronic NOAEL of 2 mg/kg/day from a 2-year feeding study in rats is used to assess chronic risk). All of the potential longer-term exposures of terrestrial mammals to sulfometuron methyl are substantially below the NOAEL of 2 mg/kg/day. For acute exposure, the dose-response assessment is also based on the same data as the human health risk assessment (i.e. the chronic NOAEL in rats of 87 mg/kg/day from a 10-day gestational exposure study is used to assess acute risk). All of the potential acute exposures of terrestrial mammals to sulfometuron methyl are also substantially below the NOAEL of 87 mg/kg/day. Birds appear to exhibit the same low order of toxicity to sulfometuron methyl as mammals, with an acute NOAEL of 312 mg/kg based on changes in body weight observed following a single gavage administration to mallard ducks. No chronic exposure studies of birds to sulfometuron methyl were identified in the available literature. Since results of acute exposure studies suggest that the sensitivity of birds to sulfometuron methyl is similar to that of mammals, in the absence of chronic exposure data in birds the chronic NOAEL of 2 mg/kg/day in rats is used for birds. For terrestrial invertebrates, based on direct spray studies in honey bees, no mortality would be expected following acute exposure to doses up to 1075 mg/kg. Although limited data are available, soil microorganisms appear sensitive to sulfometuron methyl at concentrations of about 70 µg/L.

The toxicity of sulfometuron methyl to terrestrial plants is relatively well characterized. Sulfometuron methyl is a potent herbicide that causes adverse effects in a variety of target and non-target plant species. Results of pre-emergent and post-emergent application studies in a variety of plant species yield NOELs ranging from 0.0000086 to 0.00078 lbs/acre. For assessing the potential consequences of exposure to nontarget plants via runoff, an LOEC for seedling emergence of 0.0000086 lb/acre is used for sensitive species and the corresponding value for tolerant species is 0.00025 lb/acre. For assessing the impact of drift, an LOEC for vegetative vigor of 0.000024 lb/acre is used for sensitive species and the corresponding value for tolerant species is 0.00078 lb/acre.

The data on toxicity to fish and aquatic invertebrates were obtained in several species. Fish do not appear to be highly sensitive to sulfometuron toxicity. However, investigations of acute toxicity have been hampered by the limited water solubility of sulfometuron methyl. For acute exposures in fish, the NOEC of 7.3 mg a.i./L in fathead minnow is used for the most sensitive species and the NOEC of 150 mg a.i./L in bluegill sunfish and rainbow trout is used for the most tolerant species. However, since both of these values were the highest concentration tested in both studies, identification of a most sensitive and a most tolerant species cannot be made with certainty. Toxicity values for chronic toxicity may be based on the available egg-and-fry/early life stage studies; only one study of chronic exposure in fish is available, a 30-day exposure of fathead minnow yielding an NOAEC of 1.17 mg a.i./L. This value is used for both the most sensitive and tolerant species for chronic exposure. For acute exposure of aquatic invertebrates, the most sensitive species appear to be *Alonella* sp. and *Cypria* sp., with LOAEC values of 75 mg a.i./L. *Daphnia* are the most tolerant species, with an NOEC of 1800 mg a.i./L. Comparison

of LOAEC values for *Daphnia* (2400 mg a.i./L) and *Alonella* and *Cypria* (75 mg a.i./L) show that *Daphnia* have a relative potency factor of 32 (i.e. *Daphnia* are 32 times more tolerant than *Alonella* and *Cypria* to acute exposure of sulfometuron methyl). For chronic exposure of aquatic invertebrates, data are only available from a single study in *Daphnia* with an NOAEC of 6.1 mg/L. This value is used for the most tolerant species for chronic exposure. Although no data are available to determine the most sensitive species for chronic exposures, parallels can be drawn to the acute exposure studies. As discussed above, the relative potency factor comparing *Daphnia* to *Alonella* and *Cypria* based on acute LOAEC values is 32. Using the relative potency factor for acute exposures of 32 and the chronic NOEC in *Daphnia* of 6.1 mg/L, an NOAEC for *Alonella* and *Cypria* is estimated to be 0.19 mg/L. This surrogate NOAEC for chronic exposure in *Alonella* and *Cypria* will be used to estimate the chronic NOAEC for the most sensitive species.

Aquatic plants appear to be much more sensitive to sulfometuron methyl than aquatic animals. An NOAEC for growth inhibition of 0.00021 mg/L in duckweed is used to quantify effects for both acute and chronic exposure in aquatic macrophytes. Data are also available in *Hydrilla* and yield a similar NOAEC. However, based on the limited data available as well as difference in experimental protocols, it is not possible to identify a most sensitive and most tolerant species for aquatic macrophytes. For algae, the most sensitive algal species appears to be *Selenastrum capricornutum*, with a 72-hour NOEC of 0.0025 mg/L and the most tolerant species appears to be *Navicula pelliculosa*, both with a 120-hour NOEC of 0.37 mg/L. The same data are used to quantify risk for both acute and chronic exposures.

4.3.2. Toxicity to Terrestrial Organisms.

4.3.2.1. Mammals – As summarized in the dose-response assessment for the human health risk assessment (see section 3.3.3.), according to a Federal Registry Notice, the U.S. EPA has derived a chronic RfD of 0.24 mg/kg/day based on a NOAEL for bladder toxicity of 500 ppm dietary sulfometuron methyl (equivalent to 24.4 mg/kg/day) and a 100-fold safety factor (U.S. EPA 1997). Although an RfD has been derived by U.S. EPA, a more conservative provisional reference dose of 0.02 mg/kg/day was derived from data reported in the 2-year feeding study in rats by Mullin (1984). The provisional reference dose is based on the 2 mg/kg/day (50 ppm, dose conversions described in Appendix 1) NOAEL for hematological effects in male rats and an uncertainty factor of 100:10 for species-to-species extrapolation and 10 for sensitive subgroups in the human population. All of the potential longer-term exposures of terrestrial mammals to sulfometuron methyl are substantially below the NOAEL of 2 mg/kg/day (see Worksheet G01); thus, it is not necessary to elaborate much more on the dose-response assessment for longer-term exposures. A dose of 2 mg/kg/day is used to assess the consequences of all longer-term exposures.

As discussed in Section 3.3.3, since the U.S. EPA has not derived an acute/single dose RfD for sulfometuron methyl, a provisional acute RfD is calculated as 0.87 mg/kg/day. The provisional acute RfD is based on a NOAEL for dietary exposure of 86.6 mg/kg/day (1000 ppm, dose conversions described in Appendix 1) reported for decreased maternal and fetal body weights in

rats following 10-day gestational exposure of dams (Lu 1981) and margin of exposure of 100. All of the potential acute exposures of terrestrial mammals to sulfometuron methyl are substantially below the NOAEL of 87 mg/kg/day (see Worksheet G01); thus, it is not necessary to elaborate much more on the dose-response assessment for acute exposures. A dose of 87 mg/kg/day is used to assess the consequences of all acute exposures.

4.3.2.2. Birds – As discussed in Section 4.1.2.2, results of all acute exposure studies in birds show that sulfometuron methyl has very low toxicity, with LD₅₀ values exceeding 5000 mg/kg by gavage (Dudeck and Bristol 1981a) and exceeding 5620 ppm in the diet (equivalent to 1068 mg/kg/day, dose conversions described in Appendix 4) (Fink et al. 1981). Only one study reported signs of toxicity following acute sulfometuron methyl exposure - a gavage study in mallard ducks using single doses of technical grade sulfometuron methyl ranging from 312 to 5000 mg/kg (Dudeck and Bristol 1981a). Results show that compared to control birds, male birds, but not females, had decreased weight gain at doses of 625 mg/kg and higher. Although the decrease in weight gain did not exhibit dose-dependence with respect to the magnitude of effect, the NOAEL for changes in body weight is 312 mg/kg. For this risk assessment, the NOAEL for acute exposure to birds is taken as 312 mg/kg/day. No chronic exposure studies in birds were identified in the available literature. However, LD₅₀ values obtained from acute exposure studies in both mammals and birds are of similar magnitude and show that both birds and mammal appear to have a very low order of toxicity to sulfometuron methyl. In the absence of chronic exposure data in birds, the NOAEL of 2 mg/kg/day in obtained in rats is used for chronic exposure of birds to sulfometuron methyl. Since acute and chronic NOAELs for birds greatly exceed all exposure scenarios, it is not necessary to elaborate this dose-response assessment.

4.3.2.3. Terrestrial Invertebrates – Two standard bioassays were conducted on the toxicity of sulfometuron methyl to bees, as detailed in Section 4.1.2.3. Results of these studies are unremarkable, yielding LD₅₀ values of sulfometuron methyl greater than the highest dose tested in each study – 12.5 µg/bee (DuPont de Nemours 1983) and 100 µg/bee (Hoxter and Smith 1990,). Using a body weight of 0.093 g for the honey bee (USDA/APHIS 1993), these values correspond to doses of about 134 to 1075 mg/kg [0.0125 mg/0.000093 kg to 0.1 mg/0.000093 kg]. For the purposes of this risk assessment, the NOAEL 1075 mg/kg will be used for risk characterization. Since this NOAEL greatly exceeds all exposure scenarios, it is not necessary to elaborate this dose-response assessment.

4.3.2.4. Terrestrial Plants (Macrophytes) –Sulfometuron methyl is a herbicide and causes adverse effects in a variety of non-target plant species (Section 3.1.2.4 and Appendix 4). The most relevant studies for assessing the effects of direct spray or drift are the series of bioassays conducted by Drake (1990) and McKelvey (1995). As detailed in Appendix 4 and discussed in Section 3.1.2.4, the more recent study by McKelvey (1995) clearly defines NOEC's for growth inhibition whereas the earlier study by Drake (1990) did not.

For assessing the potential consequences of exposure to nontarget plants via runoff, results of pre-emergence studies are used from the study by McKelvey (1995). In this assay, the most sensitive species based on the NOEC rape, tomato, sorghum, wheat, and corn with an NOEC of 0.0000086 lb ai/acre. Onion, pea, cucumber, and soybean are most tolerant species based on the NOEC, 0.00026 lb/acre. These values are used in Worksheet G04 to assess the risks to non-target plant species from soil contamination associated with the runoff of sulfometuron methyl from the application site.

For assessing the impact of drift, bioassays on vegetative vigor from the study by McKelvey (1995) will be used. In this assay, the most sensitive species based on the NOEC is corn with an NOEC of 0.000024 lb ai/acre. The most tolerant species based on the NOEC is pea with an NOEC of 0.00078 lb ai/acre. These NOEC values are used in Worksheets G05a and G05b for characterizing risks associated with off-site drift.

4.3.2.5. Terrestrial Microorganisms – As discussed in section 4.1.2.5, the sensitivity of terrestrial microorganisms appears to operate and be governed by the same mechanism involved in plant toxicity. The lowest reported effect level is about 70 µg/L. At this concentration, exposure periods of less than 3 hours inhibited the growth of terrestrial/soil microorganisms in a liquid glucose medium (Epelbaum et al. 1996). The extent to which these findings can be applied to soil levels of sulfometuron methyl is uncertain.

4.3.3. Aquatic Organisms.

The toxicity values used in this risk assessment are summarized in Worksheet G03 based on the information presented in Section 4.1.3.

4.3.3.1. Fish – As discussed in Section 4.1.3.1 and summarized in Appendix 6, fish do not appear to be highly sensitive to sulfometuron toxicity, although investigations of the acute LC₅₀ have been hampered by the limited water solubility of sulfometuron methyl. Results of all acute exposure studies yield LC₅₀ values at the highest concentration tested in each study - a range of >7.3 mg/L in fathead minnow (Muska and Driscoll 1982) to >150 mg/L in bluegill sunfish (Brown 1994a) and rainbow trout (Brown 1994b). For this risk assessment, the NOEC of 7.3 mg/L in fathead minnow is used for the most sensitive species and the NOEC of 150 mg/L in bluegill sunfish and rainbow trout is used for the most tolerant species. Due to the limited water solubility of sulfometuron methyl, the most sensitive and tolerant species cannot truly be identified and it is quite possible that no species would respond at concentrations of 150 mg/L or substantially higher. However, for this risk assessment, fathead minnows are considered to be approximately 20 times more sensitive to sulfometuron methyl toxicity than bluegill sunfish and rainbow trout.

Toxicity values for chronic toxicity may be based on the available egg-and-fry/early life stage studies; only one study of chronic exposure in fish, a 90-day exposure of fathead minnow, was identified in the available literature (Muska and Driscoll 1982). No effects on fathead minnow embryo hatch, larval survival, or larval growth was observed over 30-day exposure periods at

measured average concentrations up to 1.17 mg a.i./L. As discussed above for acute exposure studies in fish, it is not possible to identify with certainty the most sensitive and tolerant species based on the available data. Thus, for this risk assessment, the NOEC of 1.17 mg/L is used for both the most sensitive and tolerant species for chronic exposure.

4.3.3.2. Amphibians – As discussed in Section 4.1.3.2 and summarized in Appendix 7, the toxicity of acute and chronic exposure to sulfometuron methyl to amphibians has been evaluated in a single study in African Clawed frogs (*Xenopus laevis*) (Fort 1998). In this report, the author did not state whether data were reported in terms of mg sulfometuron methyl/L or mg Oust/L. Taking the most conservative approach, values are assumed to be expressed in terms of mg a.i./L. For this risk assessment, the acute NOEC of 0.38 mg a.i./L for both lethality and malformations and the chronic NOEC of 0.00075 mg a.i./L for decreased tail resorption rate will be used. Since no studies on other amphibian species were identified in the available literature, it is not possible to identify a most tolerant and most sensitive amphibian species.

4.3.3.3. Aquatic Invertebrates – Studies assessing the toxicity of sulfometuron methyl have been conducted in several species of aquatic invertebrates, as detailed in Section 4.1.3.3 and Appendix 6. As reported for studies in fish, studies on the toxicity of technical grade sulfometuron methyl have been somewhat hampered by the limited water solubility of sulfometuron methyl. Although studies have been conducted using both technical grade sulfometuron methyl and Oust, studies using Oust are considered to be most relevant to this risk assessment. Based on the results of acute exposure studies, the most sensitive aquatic invertebrates appear to be *Alonella* sp. and *Cypria* sp., with LOAEC values of 100 mg Oust/L (75 mg a.i./L) (Naqvi and Hawkins 1989). Since 100 mg Oust/L was the lowest concentration tested, NOEC values could not be determined. Although Naqvi and Hawkins do not specifically state whether data are reported as mg a.i./L or mg Oust/L, it is implied that data are reported in terms of the formulation; taking the most conservative approach, values are assumed to be expressed as mg Oust/L. Based on reported NOAEC values, *Daphnia* appear to be the most tolerant species, with an LC₅₀ of 6375 mg a.i./L, an NOAEC of 1800 mg a.i./L and an LOAEC of 2400 mg a.i./L (Wetzel 1984). A higher LC₅₀ value of 12,175 mg/L was reported in crayfish, however an NOAEC and LOAEC values were not reported (Naqvi et al. 1987). For this risk assessment for acute exposure of aquatic invertebrates, the LOAEC of 75 mg a.i./L in *Alonella* sp. and *Cypria* sp. will be used for the most sensitive species and the NOAEC of 2400 mg a.i./L will be used for the most tolerant species. Comparison of LOAEC values for *Daphnia* (2400 mg a.i./L) and *Alonella* and *Cypria* (75 mg a.i./L) show that *Daphnia* have a relative potency factor of 32 (i.e. *Daphnia* are 32 times more tolerant than *Alonella* and *Cypria* to acute exposure of sulfometuron methyl).

Data regarding chronic exposure to aquatic invertebrates are only available from a single reproductive study with technical grade in *Daphnia* (Baer 1990). As discussed in Section 4.1.3.3, the number of neonates per surviving adult was significantly reduced at 24 mg a.i./L but not at 97 mg a.i./L or any of the lower concentrations. The authors report the NOEC as 6.1 mg a.i./L. Although the effect observed at 24 mg/L may have been a random variation, it is treated

as an LOEC for the purpose of this risk assessment. While this approach may be regarded as conservative, in the absence of additional studies regarding reproductive effects in aquatic invertebrates, the approach seems prudent. Thus, the NOEC of 6.1 mg a.i./L will be used to assess the risk of chronic exposure to the most tolerant species. Although no data are available to determine the most sensitive species for chronic exposures, parallels can be drawn to the acute exposure studies. As discussed above, the relative potency factor comparing *Daphnia* to *Alonella* and *Cypria* based on acute LOAEC values is 32, (i.e., *Daphnia* are 32 times more tolerant to sulfometuron methyl toxicity than *Alonella* and *Cypria* in acute exposures). Using the relative potency factor for acute exposures of 32 and the chronic NOEC in *Daphnia* of 6.1 mg/L, an NOEC for *Alonella* and *Cypria* is estimated to be 0.19 mg/L [$6.1 \text{ mg/L} \div 32 = 0.19 \text{ mg/L}$]. This surrogate NOEC for chronic exposure in *Alonella* and *Cypria* will be used to estimate the chronic NOEC for the most sensitive species.

4.3.3.4. Aquatic Plants – The relevant data on the toxicity of sulfometuron methyl to aquatic plants is summarized in Appendix 6. The most sensitive algal species appears to be *Selenastrum capricornutum*, with a 72-hour NOAEC value for growth inhibition based on cell density of 2.5 µg/L (0.0025 mg/L) (Hoberg 1990). The 120-hour NOAEC in *Navicula pelliculosa* for growth inhibition is reported as 370 µg/L (0.37 mg/L) and is the value used for the most tolerant species (Thompson 1994). The maximum exposure period in all studies was 120 hours; no long-term exposure studies were identified in the available literature. Therefore, for risk characterization for both acute and chronic exposure, the NOAEC 0.0025 mg/L will be used as the most sensitive species and the NOAEC of 0.37 for the most tolerant species.

As reviewed in Section 4.1.3.4 and Appendix 6, only two studies in aquatic plants were identified in the available literature – a 7-day exposure study in *Hydrilla* using Oust (Byl et al. 1994) and a 14-day exposure study in *Lemna* (duckweed) using technical grade sulfometuron methyl (Kannuck and Sloman 1995). For the Byl et al. (1994) study, the authors did not state whether data were reported in terms of mg sulfometuron methyl/L or mg Oust/L. Taking the most conservative approach, values are assumed to be expressed in terms of mg Oust/L. In *Hydrilla*, the 7-day EC₅₀ value for growth inhibition was reported as 10 µg Oust/L (7.5 µg a.i./L), with an NOAEC of 1 µg Oust/L (0.75 µg a.i./L). In duckweed exposed to technical grade sulfometuron methyl for 14 days, the most sensitive effect was on frond count, with an EC₅₀ of 0.46 µg a.i./L (0.00046 mg a.i./L) and an NOAEC of 0.21 µg a.i./L (0.00021 mg/L). Comparison of NOAEC value for the two species are similar, although the duckweed appears to be slightly more sensitive than *Hydrilla*. However, due to differences in the exposure period in these two studies, it is not possible to determine which, if either, of these two species is more sensitive to sulfometuron toxicity. Taking the most conservative approach, the lower NOAEC value of 0.00021 mg a.i./L in duckweed will be used for both the sensitive and tolerant species. Since data are only available for 14 days, this value will also be used for both acute and chronic exposures.

4.4. RISK CHARACTERIZATION

4.4.1. Overview. Sulfometuron methyl is an effective and potent herbicide. Adverse effects on some nontarget terrestrial plant species and, to a lesser degree, some aquatic plant species are plausible under some conditions. For terrestrial plants, the dominant factor in the risk characterization is the potency of sulfometuron methyl relative to the application rate. The typical application rate considered in this risk assessment, 0.045 lb/acre, is about 1875 times higher than the NOEC in the vegetative vigor (direct spray) assay of the most sensitive non-target species – i.e., 0.000024 lb/acre – and almost 60 times higher than the NOEC for the most tolerant species in the same assay – i.e., 0.00078 lb/acre. The highest application rate that may be considered in Forest Service programs – i.e., 0.38 lb/acre – is over 15,000 times the NOEC in sensitive species and a factor of about 490 above the NOEC in tolerant species. Given these relationships, damage to sensitive nontarget species could be expected in ground broadcast applications at distances of up to about 900 feet from the application site in areas in which off-site drift is not reduced by foliar interception. This risk characterization applies only to ground broadcast applications. When used in directed foliar applications (i.e., backpack), offsite drift could be reduced substantially but the extent of this reduction cannot be quantified.

The NOEC values for soil exposures (assayed in the seedling emergence test) are 0.0000086 lb/acre for sensitive species and 0.00026 lb/acre for tolerant species. The offsite movement of sulfometuron methyl via runoff could be substantial under conditions that favor runoff – i.e., clay soils – and hazard quotients in the range of about 90 to nearly 2900 are estimated for sensitive species over a wide range of rainfall rates – i.e., 15 inches to 250 inches per year. In very arid regions in which runoff might not be substantial, wind erosion could result in damage to nontarget plant species. The plausibility of observing such damage would, however, be highly dependent on local conditions. This risk characterization for the potential effects of runoff would be applicable to either broadcast ground or directed foliar applications.

Damage to aquatic plants, particularly macrophytes, appears substantially less than for terrestrial plants. All hazard quotients for aquatic macrophytes were based on an NOEC of 0.00021 mg/L in duckweed for both acute and chronic exposures. No sensitive or tolerant species were identified. Except for the hazard quotient of 4 associated with acute exposures based on the peak concentrations of sulfometuron methyl, all hazard quotients are below the level of concern, with a range of 0.01 to 0.4 for acute exposures and 0.002 to 0.01 for chronic exposures. Thus, if sulfometuron methyl is applied in areas where transport to water containing aquatic macrophytes is likely, it would be plausible that detectable but transient damage could be observed.

Aquatic algae do not appear to be as sensitive to sulfometuron methyl. The highest hazard quotient observed for acute exposure is 0.4 associated with the upper range for the most sensitive species, based on an NOEC for growth inhibition. For chronic exposures, the highest hazard quotient is 0.001 associated with the upper range for the most sensitive species. Both values were based on an acute NOEC. Therefore, it is not anticipated that adverse effects in aquatic algae would result from exposure to sulfometuron methyl at application rates used by the Forest Service.

There is no clear basis for suggesting that effects on terrestrial animals are likely or would be substantial. Adverse effects in mammals, birds, terrestrial insects, and microorganisms are not likely using typical or worst-case exposure assumptions at the typical application rate of 0.045 lb a.e./acre. The hazard quotients associated with the upper bound for chronic consumption of vegetation by a large mammal (hazard quotient = 0.2) or large bird (hazard quotient = 0.3) feeding exclusively on treated vegetation slightly exceeds the level of concern of 0.1 associated with the maximum application rate of 0.38 lb a.e./acre. As with the human health risk assessment, this characterization of risk must be qualified. Sulfometuron methyl has been tested in only a limited number of species and under conditions that may not well-represent populations of free-ranging non-target species. Notwithstanding this limitation, the available data are sufficient to assert that no adverse effects are anticipated in terrestrial animals.

Similarly, the risk characterization for aquatic animals is relatively simple and unambiguous. Sulfometuron methyl appears to have a very low potential to cause any adverse effects in aquatic animals. All of the hazard quotients for aquatic animals are extremely low, with a range of 0.000000002 (lower range for acute exposures in tolerant aquatic invertebrates) to 0.004 (longer-term exposures to amphibians). It should be noted that confidence in this risk characterization is reduced by the lack of chronic toxicity studies in potentially tolerant fish and potentially sensitive aquatic invertebrates and lack of data in amphibians (data only available in a single species). Even with these uncertainties, there is no basis for asserting that adverse effects on aquatic animals are likely.

4.4.2. Terrestrial Organisms.

4.4.2.1. Terrestrial Vertebrates – The quantitative risk characterization for mammals and birds is summarized in Worksheet G02. The toxicity values used for each group of animals is summarized at the bottom of Worksheet G02 and refer to values derived in the dose-response assessment (Sections 4.3.2.1 and 4.3.2.2). In this worksheet, risk is characterized as the estimated dose, taken from Worksheet G01, divided by toxicity value. This ratio is referred to as the hazard quotient (HQ). All exposures summarized in Worksheet G01 are based on the typical application rate of 0.045 lb a.e./acre. At this application rate, an HQ of one or less indicates that the estimated exposure is less than the toxicity value. When this is the case, there is no basis for asserting that adverse effects are plausible.

As discussed in Section 2 (Program Description), the maximum application rate that might be used in Forest Service programs is 0.38 lb a.e./acre. Because exposure is directly related to application rate, the level of concern for the hazard quotients given in Worksheet G02 for an application rate of 0.38 lb a.e./acre is 0.1 [$0.045 \text{ lb a.e./acre} \div 0.38 \text{ lb a.e./acre} = 0.1$].

As indicated in Worksheet G02, the highest hazard quotient for any acute exposure is 0.04 [4e-02], the upper range of the hazard quotient for the consumption of contaminated insects by a small mammal. Thus, there is no basis for asserting that adverse effects are likely from the application of sulfometuron methyl at any application rate, even the maximum application rate of

0.38 lb a.e./acre, that might be used in Forest Service programs. Thus, at the typical application rate of 0.045 lb a.e./acre, there is also no basis for asserting that adverse effects are likely.

For chronic exposures, all hazard quotients are well below one. The highest hazard quotients, which slightly exceed the level of concern for the maximum application rate of 0.38 lb a.e./acre are 0.2 [2e-01] associated with the upper end of the range for chronic consumption of vegetation by a large mammal feeding exclusively on treated vegetation (i.e., labeled “on-site” in Worksheet G02) and 0.3 [3e-01] associated with the upper bound for chronic consumption of vegetation by a large bird feeding exclusively on treated vegetation (i.e., labeled “on-site” in Worksheet G02). These scenarios are essentially used in these risk assessments as a very conservative/extreme screening scenario and assume that the vegetation is treated and that the animal stays in the treated area consuming nothing but the contaminated vegetation. Given that most forms of vegetation treated at an effective (i.e., herbicidal) application rate would likely die or at least be substantially damaged, this exposure scenario is implausible. It is, however, routinely used in Forest Service risk assessments as a very conservative upper estimate of potential exposures and risks. Thus, it is unlikely that adverse effects are likely to results, even at the highest application rate.

The simple verbal interpretation of this quantitative risk characterization is similar to that of the human health risk assessment: the weight of evidence suggests that no adverse effects in mammals or birds are plausible using typical or worst-case exposure assumptions at the typical application rate of 0.045 lb a.e./acre or the maximum application rate of 0.38 lb a.e./acre. As with the human health risk assessment, this characterization of risk must be qualified. Sulfometuron methyl has been tested in only a limited number of species and under conditions that may not well-represent populations of free-ranging non-target terrestrial mammals or birds. Notwithstanding this limitation, the available data are sufficient to assert that no adverse effects are anticipated in terrestrial mammals or birds.

4.4.2.2. Terrestrial Invertebrates – As shown in Worksheet G02, for the honey bee, the hazard quotient of 0.007 [7e-03] is well below the level of concern of one associated with the typical application rate of 0.045 lb a.e./acre and the level of concern of 0.1 associated with the maximum application rate of 0.38 lb a.e./acre. Thus, there is no basis for anticipating the occurrence of adverse effects in bees exposed to sulfometuron methyl at application rates that might be used in Forest Service programs.

4.4.2.3. Terrestrial Plants – A quantitative summary of the risk characterization for terrestrial plants is presented in Worksheet G04 for runoff and Worksheets G05a and G05b for drift. Analogous to the approach taken for terrestrial animals, risk in these worksheets is characterized as a ratio of the estimated exposure to a benchmark exposure (i.e., exposure associated with a defined response). For both worksheets, the benchmark exposure is a NOEC, as derived in Section 4.3.2.2, for both sensitive and tolerant species.

Sulfometuron methyl is an effective and potent herbicide and adverse effects on some nontarget plant species due to drift are likely under certain application conditions and circumstances. As indicated in Worksheets G05a, off-site drift of sulfometuron methyl associated with ground broadcast applications may cause damage to sensitive plant species at distances of greater than 900 feet from the application site. The closer that the non-target species is to the application site, the greater is the likelihood of damage. Whether or not damage due to drift would actually be observed after the application of sulfometuron methyl would depend on a several site-specific conditions, including wind speed and foliar interception by the target vegetation. In other words, in some right-of-way applications conducted at low wind speeds and under conditions in which vegetation at or immediately adjacent to the application site would limit off-site drift, damage due to drift would probably be inconsequential or limited to the area immediately adjacent to the application site. Tolerant plant species would probably not be impacted by the drift of sulfometuron methyl and might show relatively little damage unless they were directly sprayed.

As summarized in Worksheet G04, runoff could pose a substantial risk to sensitive non-target plant species under conditions in which runoff is favored – i.e., clay soil over a very wide range of rainfall rates. Some tolerant plants species could also be adversely affected under conditions which favor runoff and in regions with high rainfall rates.

The situational variability in the exposure assessments for runoff, wind erosion, and irrigation water does have a substantial impact on the characterization of risk for sensitive nontarget plant species. All of these scenarios may overestimate or underestimate risk under certain conditions.

As summarized in Section 4.2.3.5, daily soil losses due to wind erosion, expressed as a proportion of an application rate, could be in the range of 0.00001 to 0.001. This is substantially less than off-site losses associated with runoff from clay (Worksheet G04) and similar to off-site losses associated with drift at a distance of 500 feet or more from the application site (Worksheet G05a). As with the drift scenarios, wind erosion could lead to adverse effects in sensitive plant species. Wind erosion of soil contaminated with sulfometuron methyl is most plausible in relatively arid environments and if local soil surface and topographic conditions favor wind erosion.

The simple verbal interpretation for this quantitative risk characterization is that sensitive and tolerant plant species could be adversely affected by the off-site drift or runoff of sulfometuron methyl under a variety of different scenarios depending on local site-specific conditions. If sulfometuron methyl is applied in the proximity of sensitive crops or other desirable plant species, site-specific conditions and anticipated weather patterns will need to be considered if unintended damage is to be avoided.

4.4.2.4. Soil Microorganisms – Based on the study by Hubbard et al. (1989), sulfometuron methyl concentrations after light to heavy rainfalls were less than 2.4 µg/mL (2400 µg/L or 2.4 ppm) in runoff and 0.1 µg/mL (100 µg/L) in percolate at applications rates within the range used by the Forest Service. Data regarding the toxicity of soil-incorporated sulfometuron methyl to

soil microorganisms is not available. Based on the study by Epelbaum et al. (1996), sulfometuron methyl concentrations of $\approx 73 \mu\text{g/L}$ in a liquid glucose medium inhibited the growth of soil microorganisms after exposure periods of less than 3 hours (see section 4.3.2.5). Although the level of sulfometuron methyl in runoff may be substantially greater than levels that might inhibit microbial growth, the compound would be diluted substantially in the soil column. Concentrations of sulfometuron methyl in the percolate are more directly relevant to soil bacteria. If the level used by Epelbaum et al. (1996) in glucose medium is relevant to soil exposure, microbial inhibition is likely to occur and could be substantial. There is no certainty, however, that the finding is relevant.

From a practical perspective, this uncertainty has relatively little impact on this risk assessment. As discussed in the previous section, sulfometuron methyl applied to vegetation at rates that control undesirable vegetation will cause substantial damage to the vegetation, target or non-target. This damage would probably be accompanied by secondary changes in the local environment affecting the soil microbial community to a greater extent or at least more certainly than any direct toxic action by sulfometuron methyl on the microorganisms.

4.4.3. Aquatic Organisms.

4.4.3.1. Aquatic Animals – The risk characterization for aquatic animals is relatively simple and unambiguous. Sulfometuron methyl appears to have a very low potential to cause any adverse effects in aquatic animals. As detailed in Section 4.2.3 and summarized in Worksheet G03, concentrations of sulfometuron methyl in ambient water over prolonged periods of time are estimated to be no greater than 0.0000032 mg/L and peak concentration of sulfometuron methyl associated with runoff or percolation are estimated to be no more than 0.0009 mg/L . As summarized in Worksheet G03, all of the hazard quotients for aquatic animals are extremely low, ranging from $0.000000002 [2\text{e-}9]$ (lower range for acute exposures in tolerant aquatic invertebrates) to $0.004 [4\text{e-}03]$ (longer-term exposures to amphibian). Thus, there is no basis for asserting that effects on nontarget aquatic species are likely. As detailed in Section 4.3.3.1, since chronic exposure data in fish are only available in one species (fathead minnow), confidence in this risk characterization is reduced by the lack of chronic toxicity studies in potentially sensitive fish. Similarly, confidence in the chronic exposure data is reduced for the sensitive species of invertebrates because data are only available in a single species (*Daphnia*) (Section 4.3.3.3) and tolerant and sensitive species of amphibians could not be identified due to insufficient data (Section 4.3.3.2).

As with other risk characterization worksheets, Worksheet G03 is based on the typical application rate of 0.045 lbs/acre . At the maximum application rate of 0.38 lbs/acre , all of the hazard quotients would be increased by a factor of about 8 [$0.38 \text{ lbs/acre} \div 0.045 \text{ lbs/acre} = 8.4$]. This difference would have no impact on the risk characterization for aquatic animals – i.e., the highest hazard quotient 0.0004 (upper range for chronic exposure of amphibians) in Worksheet G03 would be increased to 0.03 , below the level of concern by a factor of over 30.

The simple verbal interpretation of this quantitative risk characterization is similar to that of the human health risk assessment and the assessment for terrestrial animals: the weight of evidence suggests that no adverse effects in aquatic animals are plausible using typical or worst-case exposure assumptions at the typical application rate of 0.045 lb a.e./acre or the maximum application rate of 0.38 lb a.e./acre. As with the human health risk assessment, this characterization of risk must be qualified. Sulfometuron methyl has been tested in only a limited number of species and under conditions that may not well-represent populations of free-ranging aquatic animals. Notwithstanding this limitation, the available data are sufficient to assert that no adverse effects are anticipated in fish, amphibians or aquatic invertebrates.

4.4.3.2. Aquatic Plants – The risk assessment for aquatic plants differs substantially from that of aquatic animals, particularly for acute exposure of macrophytes. For acute exposures based on the peak concentrations of sulfometuron methyl, aquatic macrophytes appear to be at risk at the upper range of plausible exposures, with a hazard quotient of 4. Hazard quotients for the central estimate is 0.2 and for the lower bound is 0.01, both below the level of concern of one. For chronic exposures, all hazard quotients are below the level of concern, ranging from 0.002 for the lower range to 0.01 for the upper bound. It should be noted that the risk characterization is based on NOEC values in a single species and that a most sensitive and most tolerant species could not be identified due to a lack of data. Thus, as with terrestrial plants, aquatic macrophytes appear to be at some risk if sulfometuron methyl is applied near bodies of water containing aquatic macrophytes. In such applications, it would be desirable to take measures that would substantially reduce the anticipated levels of exposure.

Algae appear to be much less sensitive to sulfometuron methyl than macrophytes and neither sensitive or tolerant species of algae would be at risk in either chronic or acute exposures scenarios. Based on the upper range of exposure, the highest hazard quotient for sensitive algae for acute exposure is 0.4 and for chronic exposure is 0.001, both well below the level of concern associated with the typical application rate of 0.045 lb a.e./acre. Thus, algal species do not appear to be at risk based on estimated longer term concentrations of sulfometuron methyl in water. However, effects may be evident in sensitive species at the higher application rates.

5. REFERENCES

- Allen RR; Fryrear DW. 1977. Limited tillage saves soil, water, and energy. ASAE Annual Meeting, NC State Univ., Raleigh, NC. June 26-29, 1977. 14 pp.
- Anderson JJ. 1980. Terrestrial Field Dissipation Study of 14 C-DPX-5648 in Delaware, North Carolina, and Mississippi Soils. Document No. AMR-35-81-B. Interim rept. (Unpublished study received Jun 17, 1981 under 352-EX-108; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:245375-F) MRID 00078705
- Anderson J. 1990a. Phase 3 summary of MRID 00143540: The fate of ¹⁴C-DPX-T5648 in an anaerobic environment. du Pont Report No. 65-82, AMR-1848-90. Prepared by D.I. du Pont de Nemours and Co., Wilmington, DE. 10 p. MRID 93206025.
- Anderson J. 1990b. Phase 3 summary of study MRID No. 78701: Microbial metabolism of ¹⁴C-phenyl-labeled DPX-T5648 in a viable soil ecosystem. du Pont Report No. AMR 34-81. Guideline Reference: 162-1: Aerobic soil. (Unpublished study prepared by E.I. du Pont de Nemours and Co., DuPont Agricultural Products, Wilmington, DE.) 7 p. MRID 93206024.
- Anderson J. 1994. Microbial Metabolism of (carbon 14)-Phenyl-Labeled DPX-T5648 in a Viable Soil Ecosystem. Supplement: Lab Project Number: AMR 34-81. Unpublished study prepared by DuPont Agricultural Products. 7 p. MRID 43174102
- Anderson JJ; Berg DS. 1980?. Studies To Determine the Effect of DPX-5648 on Metabolic Processes of Soil Microbes. Document No. AMR-01-81. (Unpublished study received Jan 23, 1981 under 352-EX-107; prepared in cooperation with Univ. of Delaware, College of Agricultural Sciences, submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:244195-W) MRID 0007
- Anderson JJ; Dulka JJ. 1985. Environmental fate of sulfometuron methyl in aerobic soils. J. Agric. Food Chem. 33(4): 596-602.
- Arnold GW; Weeldenberg JW; Leone J. 1998. Herbicide control of exotic annual plant species in *Acacia acuminata*-*Eucalyptus loxophleba* woodland in south-western Australia and effects on native ground flora. Plant Prot Q. 13 (1):39-43
- Arthur MA; Wang Y. 1999. Soil nutrients and microbial biomass following weed-control treatments in a Christmas tree plantation. Soil Sci Soc Am J. 63 (3):629-637
- Ashby WC. 1997. Soil ripping and herbicides enhance tree and shrub restoration on stripmines. Restoration Ecol. 5 (2):169-177

Aulgur LF. 1996. Du Pont OUST herbicide update. Proc. Ann. For. Veg. Manag. Conf. 17: 32-36.

Baer K. 1990. Chronic Toxicity of IN T5648-40 to *Daphnia magna*. Lab Project Number: 614-90: MR-4581-804. Unpublished study prepared by Haskell Laboratory. 260 p. MRID 41672806

Bamberger, J. (1999) Paste Extruded 75WG Formulation of Sulfometuron Methyl: Inhalation Median Lethal Concentration (LC50) Study in Rats: Lab Project Number: 721: 12479: 2658. Unpublished study prepared by E.I. du Pont de Nemours and Co. 45 p. {OPPTS 870.1300} MRID 44874105

Barefoot AC; Anderson JJ; Naidu MV; Ryan DL. 2001. Evaluation of cell cultures as an in vitro tool to study metabolism of crop protection chemicals. Abstr Papers Am Chem Soc. 222 (1-2)

Barnes AD; Zedaker SM; Feret PP; Seiler JR. 1990. The effects of sulfometuron on the root growth of loblolly pine. New For. 3(4): 289-295.

Battaglin WA; Furlong ET; Burkhardt MR; Peter CJ. 1999. Proceedings of the Technical Meeting Charleston South Carolina March 8-12,1999--Volume 2 of 3--Contamination of Hydrologic Systems and Related Ecosystems, Water-Resources Investigation Report 99-4018B. Available at: http://toxics.usgs.gov/pubs/wri99-4018/Volume2/sectionC/2402_Battaglin/index.html.

Beck KG; Lym RG; Becker RL; Ferrel MA; Finnerty DW; Frank RJ; Henson MA; Peterson MA. 1993. Leaf spurge (*Euphorbia esula*) control and grass injury with sulfometuron. Weed Technol. 7(1): 212-215.

Bloemer D. 1999. Physical and Chemical Characteristics of End-Use Product Sulfometuron Methyl 75% Paste Extruded Formulation: Lab Project Number: 2494. Unpublished study prepared by E.I. du Pont de Nemours and Co. 10 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.7000, 830.. MRID 44874102

Boutsalis P; Powles SB. 1995. Inheritance and mechanism of resistance to herbicides inhibiting acetolactate synthase in *Sonchus oleraceus* L. Theoret. Appl. Gen. 91(2): 242-247.

Boxenbaum J; D'Souza R. 1990. Interspecies pharmacokinetic scaling, biological design and neoteny. Adv. Drug Res. 19: 139-195.

Brattsten LB. 1987. Hydrolysis of [pyrimidine-2-¹⁴C] sulfometuron methyl. Laboratory Project ID. AMR-541-86. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Inc., Wilmington, DE). 10 p. MRID 41672811.

Brennan D. 1990. Sulfometuron Methyl: Product Identity and Composition, Analysis and Certification of Product Ingredients: Lab Project Number: AMR-1674-90. Unpublished study prepared by E.I. du Pont de Nemours and Co. 129 p.. MRID 41672801

Brennan D. 1990. Sulfometron Methyl: Physical and Chemical Characteristics: Lab Project Number: AMR-1746-90. Unpublished study prepared by E.I. du Pont de Nemours & Co. 111 p.. MRID 41672802

Bridges A. 1992. Roadside ousst vs. a flower farm: It kills plants, but can't be detected in a lab. *J. Pest. Reform.* 12(2): 17.

Brown MR. 1994a. Static, acute, 96-hour LC50 of DPX-T5648-42 (sulfometuron methyl) to Bluegill Sunfish, *Lepomis macrochirus* (Limit test). Haskell Laboratory Report No. 590-94. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE). 27 p. MRID 43501801.

Brown MR. 1994b. Static, acute, 48-hour EC50 of DPX-T5648-42 (sulfometuron methyl) to *Daphnia magna*. Haskell Laboratory Report No. 588-94. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE). 27 p. MRID 43501803.

Brown MR. 1994c. Static, acute, 96-hour LC50 of DPX-T5648-42 (sulfometuron methyl) to Rainbow trout, *Oncorhynchus mykiss*: (Limit test). Haskell Laboratory Report No. 589-94. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE). 28 p. MRID 43501802.

Budavari S. (Ed). 1989. *The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals*, 11th ed. Merck & Co., Inc., Rahway, New Jersey.

Burnet M; Hodgson B. 1991. Differential effects of the sulfonylurea herbicides chlorsulfuron and sulfometuron methyl on microorganisms. *Arch. Microbiol.* 155 (6): 521-525.

Burnmaster DE. 1998. Lognormal distributions for total water intake and tap water intake by pregnant and lactating women in the United States. *Risk Analysis.* 18(2): 215-219.

Buser HR. 1990. Atrazine and other s-triazine herbicides in lakes and in rain in Switzerland. *Environ. Sci. Technol.* 24(7): 1049-1058.

Byl TD; Sutton HD; Klaine S. 1994. Evaluation of peroxidase as a biochemical indicator of toxic chemical exposure in the aquatic plant *Hydrilla verticillata*, Royle. *Environ. Toxicol. Chem.* 13(3): 509-515.

Cadwgan GE. 1990a. Phase 3 summary study of MRID No. 41273602: n-Octanol/water partition coefficient of sulfometuron methyl at pH 5, 7, and 9. du Pont Report No. AMR-1203-88. Guideline Reference: Series 63-11: Octanol/Water Partition Coefficient. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Wilmington, DE). 7 p. MRID 93206001.

Cadwgan GE. 1990b. Phase 3 summary study of MRID Nos. 71423 and 78702: Soil mobility of sulfometuron methyl. du Pont Report Nos. AMR-25-80 and AMR -11-81. Guideline Reference: Series 163-1: Leaching/Adsorption/Desorption. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Wilmington, DE). 12 p. MRID 93206026.

Calabrese EJ; Baldwin LA. 1993. Performing Ecological Risk Assessments. Lewis Publishers, Boca Raton, LA, pp. 12-24.

California Department of Pesticide Regulation. 2002. Summary of Pesticide Use Report Data 2001, Indexed by Chemical. Available at: <http://www.cdpr.ca.gov/docs/pur/pur01rep/chmrpt01.pdf>.

Cambon JP; Zheng SQ; Bastide J. 1992. Chemical or microbial degradation of sulfonylurea herbicides in soil. I. Sulfometuron methyl. Weed Res. 32(1): 1-7.

Cantrell RL; Hyland JR. 1985. Application techniques. In: A Guide to Silvicultural Herbicide Use in the Southern United States. Auburn University School of Forestry, Alabama Agricultural Experiment Station. November 1985. 612 pp.

Cox C. 1993. Sulfometuron methyl (Oust). J. Pest. Reform. 13 (4): 30-35.

CPR (Crop Protection Reference). 1997. Crop Protection Reference. 13th ed. C&P Press, Inc., New York, NY.

Dashiell OL; Hall JA. 1980. Oral LD50 test in rats--EPA proposed guidelines. Haskell Laboratory Report No. 965-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL: 244195-K) MRID 00071409.

Dashiell OL; Henry JE. 1980a. Skin Irritation test on rabbits for EPA pesticide registration. Haskell Laboratory Report No. 964-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-M). MRID 00071411.

Dashiell OL; Hinckle L. 1980a. Oral LD50 test in rats--EPA proposed guidelines. Haskell Laboratory Report No. 870-80. Rev. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-H). MRID 00071406.

Dashiell OL; Henry JE. 1980b. Eye irritation in rabbits--EPA pesticide registration. Haskell Laboratory Report No. 963-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours, Wilmington, DE; CDL: 244195-N). MRID 00071412.

Dashiell OL; Hinckle L. 1980c. Oral LD50 Test in Rats--EPA Proposed Guidelines: Haskell Laboratory Report No. 870-80. Rev.. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:244195-H) MRID 00071406

Dashiell OL; Hinckle L. 1983. Subacute dermal toxicity study (21-days) in rabbits. Haskell Laboratory Report No. 792-82. (Unpublished study received Apr 5, 1983 under 352-401; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL: 249865-A). MRID 00126714.

Dashiell OL; Silber LS. 1980a. Primary skin irritation and sensitization test on guinea pigs. Haskell Laboratory Report No. 966-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-O). MRID 00071413.

Dashiell OL; Silber L. 1980b. Acute skin absorption LD50 test on rabbits--EPA pesticide registration. Haskell Laboratory Report No. 1068-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-L). MRID 00071410.

Dashiell OL; Silber LS. 1980c. Primary skin irritation and sensitization test on guinea pigs. Haskell Laboratory Report No. 966-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-O). MRID 00071413.

Dashiell OL; Silber LS. 1981. Acute skin absorption LD50 test on rabbits--EPA pesticide registration. Haskell Laboratory Report No. 1078-80. (Unpublished study received Jul 1, 1981 under 352-401; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245515-J). MRID 00078791.

De Sapio R. 1976. Calculus for the Life Sciences. W.H. Freeman and Company, San Francisco, California. 740 pp.

Dickens R; Wehtje G. 1986. Mobility and soil solution characteristics of imazapyr (Arsenal) and sulfometuron methyl (Oust). Proc. South. Weed Sci. Soc. 39th Ann. Meet. p. 368.

Dickerman JD. 1981. A familial hemolytic anemia associated with sulfa administration. Hosp. Pract. (Off Ed). 16(9): 41, 44, 47.

Dourson ML; Knauf LA; Durkin PR; Stiteler WM. 1997. Categorical regression of toxicity data: A case study using aldicarb. *Reg. Toxicol. Pharmacol.* 25:121-129.

Drake D. 1990. Tier testing evaluation of sulfometuron methyl on plants grown under greenhouse. Lab Project No. DCD-90-2. (Unpublished study prepared by E. I. Du Pont de Nemours and Co., Wilmington DE). 13 p. MRID 41672809.

Driscoll R. 1984. Early life stage toxicity of INT-5648-18 to fathead minnow. Haskell Laboratory Report No. 765-82. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE). 29 p. MRID 00143539.

Dudeck SH; Bristol KL. 1981a. Final report: Single-dose oral toxicity in mallard ducks. Project No. 201-552. (Unpublished study received Jun 17, 1981 under 352-EX-108; prepared by Hazleton Laboratories America, Inc.; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245375-A). MRID 00078700.

Dudeck SH; Bristol KL. 1981b. Final report: Avian dietary toxicity study in Bobwhite quail. Project No. 201-549; HLO-17-81. (Unpublished study received Jan 23, 1981 under 352-EX-107; prepared by Hazleton Laboratories America, Inc.; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-Q). MRID 00071415.

Dudeck SH; Twigg CJ. 1980. Final report: Avian dietary toxicity (LC50) study in Mallard ducks. Project No. 201-548; HLO-788- 80. (Unpublished study received Jan 23, 1981 under 352-EX-107; prepared by Hazleton Laboratories America, Inc.; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-P). MRID 00071414.

Dulka J; Anderson J. 1982. The fate of [¹⁴C-]-DPX-T5648 in an anaerobic environment. (Unpublished study prepared by E. I. du Pont de Nemours and Co., Inc., Wilmington, DE). 28 p. MRID 00143540.

Du Pont de Nemours. 1981. Typical Impurity Profile for Technical DPX-5648.. (Unpublished study received Jan 23, 1981 under 352-EX-107; CDL:244195-C) MRID 00071401

Du Pont de Nemours. 1981. Du Pont OustTM Weed Killer: Analysis for Nitrosamines. (Unpublished study received Jul 1, 1981 under 352-401; CDL:245515-H). (Unpublished study received Jul 1, 1981 under 352-401; CDL:245515-H) MRID 00078789

Du Pont de Nemours. 1981. Purity of Starting Materials for Technical DPX 5648. (Unpublished study received Jul 1, 1981 under 352-401; CDL:245515-I). (Unpublished study received Jul 1, 1981 under 352-401; CDL:245515-I) MRID 00078790

Du Pont de Nemours. 1983. Honey Bee Toxicity Test. (Unpublished study received Apr 12, 1984 under 352-401; CDL: 252941-A). (Unpublished study received Apr 12, 1984 under 352-401; CDL: 252941-A) MRID 00137862

DuPont. 1989. Metabolism of sulfometuron methyl in rats. Unpublished study by DuPont Chemical Company, February 3, 1989. Not submitted to the EPA. (Cited in Extoxnet 1994).

Du Pont de Nemours. 1990. Submission of Product Chemistry, Environmental Fate and Phytotoxicity Data to Support the Reregistration of Sulfometuron methyl. Transmittal of 3 Studies.. MRID 41680100

DuPont. 1996. DuPont Material Safety Data Sheet for "Oust" Herbicide. M0000028. Revised 19-NOV-1994.

DuPont. 1997a. Oust herbicide: Product label for states other than California. EPA Reg. 352-401. H-63401. E.I. du Pont de Nemours and Company, Agricultural Products, Wilmington, DE. 9 pp.

DuPont. 1997b. Oust herbicide: Product label for California. EPA Reg. 352-401. H-63246. E.I. du Pont de Nemours and Company, Agricultural Products, Wilmington, DE. 9 pp.

Dupont. 1999a. Product Label for Oust XP. Available at www.greenbook.net.

Dupont 1999b. California label for Oust XP. Available at www.greenbook.net.

Dupont 2002a. MSDS for Oust XP. Available at www.greenbook.net.

Dupont 2002b. California MSDS for Oust XP. Available at www.greenbook.net.

Dulka JJ. 1980. Dissipation of ¹⁴C-DPX-5648 in the Field. Document No. AMR-35-81-C. Interim rept. (Unpublished study received Jun 17, 1981 under 352-EX-108; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:245375-G) MRID 00078706

Dulka JJ. 1980. The Fate of ¹⁴C -DPX-5648 in an Anaerobic Environment. Document No. AMR-35-81-D. Interim rept. (Un published study received Jun 17, 1981 under 352-EX-108; submit ted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL: 245375-H) MRID 00078707

Dulka JJ. 1981. Mobility of ¹⁴C-Phenyl Labeled DPX-5648 in Soil - Soil Column Leaching Studies. AMR-11-81. (Unpublished study received Jun 17, 1981 under 352-EX-108; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:245375-C) MRID 00078702

DuPont Agricultural Products (1999) Submission of Product Chemistry and Toxicity Data in Support of the Application for Registration of Dupont Oust XP Herbicide. Transmittal of 8 Studies. MRID 44874100

Durkin PR; Diamond G. 2002. Neurotoxicity, Immunotoxicity, and Endocrine Disruption with Specific Commentary on Glyphosate, Triclopyr, and Hexazinone: Final Report. SERA TR 01-43-08-04a dated January 30, 2002. Available at www.fs.fed.us/foresthealth/pesticide/risk.htm.

Durkin PR; Rubin L; Withey J; Meylan W. 1995. Methods of assessing dermal absorption with emphasis on uptake from contaminated vegetation. *Toxicol. Indust. Health.* 11(1): 63-79.

Edwards DF. 1979a. Primary skin irritation and sensitization tests on guinea pigs. Haskell Laboratory Report No. 232-79. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-I). MRID 00071407.

Edwards DF. 1979b. Eye irritation test in rabbits. Haskell Laboratory Report No. 230-79. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-J). MRID 00071408.

Epelbaum S; Chipman DM; Barak Z. 1996. Metabolic effects of inhibitors of two enzymes of the branched-chain amino acid pathway in *Salmonella typhimurium*. *J. Bacteriol.* 178(4): 1187-96.

Extoxnet. 1994. Sulfometuron methyl. Pesticide Information Profile. Extension Toxicology Network, University of Oregon. <http://ace.orst.edu/info/extoxnet/pips/sulfomet.htm>

Fallon R;. 1989. Anaerobic Aquatic Metabolism of [Pyrimidine-2 Carbon 14!Sulfometuron Methyl in Landenberg, Pennsylvania and Bradenton, Florida Pond Water and Sediment Systems. Lab Project Number: AMR 1084-88: 587-87. Unpublished study prepared by E.I. du Pont de Nemours MRID 42091402

Fedtke C; Schmalfluss J; Couderchet M. 1999. Chloroacetanilides, oxyacetamides, tetrazolinones: Mode of action. 1. Cross resistance and oleic acid incorporation in algal model systems. *Pestic Sci.* 55 (5):580-582

Filliben T. 1995a. Acute oral toxicity study with DPX-T5648-71 (Oust) in male and female rats. Laboratory Project No. HLR 721-95: 10360-001. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I du Pont de Nemours and Co, Inc., Newark, DE). 26 p. MRID 43848401.

Filliben T. 1995b. Primary dermal irritation study with DPX-T5648-71 (Oust) in rabbits. Laboratory Project No. HLR 709-95: 10360-001. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I du Pont de Nemours and Co, Inc., Newark, DE). 22 p. MRID 43848405.

Filliben T. 1995c. Acute dermal toxicity study with DPX-T5648-71 (Oust) in rabbits: Revision No.1. Laboratory Project No. HLR 720-95: 10360-001. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I du Pont de Nemours and Co, Inc., Newark, DE). 28 p. MRID 43848402

Filliben T. 1995d. Primary eye irritation study with DPX-T5648-71 (Oust) in rabbits. Laboratory Project No. HLR 710-95: 10360-001. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I du Pont de Nemours and Co, Inc., Newark, DE). 23 p. MRID 43848404.

Fink R; Beavers JB; Joiner G; et al. 1981. Final report: Eight-day dietary LC₅₀--Bobwhite Quail. Project No. 112-122. (Unpublished study received Dec 12, 1981 under 352-401; prepared by Wildlife International, Ltd., I.D. No. WI-734; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL: 246409-A). MRID 00088813.

Finlay, C. (1999a) Paste Extruded 75WG Formulation of Sulfometuron Methyl Acute Oral Toxicity Study in Male and Females Rats: Lab Project Number: 12479: 2692: 828. Unpublished study prepared by E.I. du Pont de Nemours and Co. 28 p. {OPPTS 870.1100} MRID 44874103

Finlay, C. (1999b) Paste Extruded 75WG Formulation of Sulfometuron Methyl: Primary Dermal Irritation Study in Rabbits: Lab Project Number: 631: 12479: 2807. Unpublished study prepared by E.I. du Pont de Nemours and Co. 25 p. {OPPTS 870.2500} MRID 44874107

Finlay, C. (1999c) Paste Extruded 75WG Formulation of Sulfometuron Methyl: Acute Dermal Toxicity Study in Rats: Lab Project Number: 673: 12479: 2690. Unpublished study prepared by E.I. du Pont de Nemours and Co. 31 p. {OPPTS 870.1200} MRID 44874104

Finlay, C. (1999d) Paste Extruded 75WG Formulation of Sulfometuron Methyl: Primary Eye Irritation Study in Rabbits: Lab Project Number: 601: 12479: 2944. Unpublished study prepared by E.I. du Pont de Nemours and Co. 25 p. {OPPTS 870.2400} MRID 44874106

Fort D. 1998. Effects of Sulfonyl Urea Herbicides in *Xenopus laevis*: An Evaluation of Developmental Toxicity and Impact on Metamorphosis. (Accent and Sulfometuron Methyl): Lab Project Number: 1-1. Unpublished study prepared by The Stover Group. 26 p. MRID 44682601

Ford LS. 1982. Unscheduled DNA synthesis/rat hepatocytes in vitro. Haskell Laboratory Report No. 769-82. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 00146847.

Fort DJ; Rogers R; Copley H; Bruning L; Stover EL; Rapaport D. 1999. Effect of sulfometuron methyl and nicosulfuron on development and metamorphosis in *Xenopus laevis*: Impact of purity. *Environ Toxicol Chem.* 18 (12):2934-2940

Gaeddert JW; Peterson DE; Horak MJ. 1997. Control and cross-resistance of an acetolactate synthase inhibitor-resistant Palmer amaranth (*Amaranthus palmeri*) biotype. *Weed Technol.* 11(1): 132-137.

Galloway SM. 1981. Mutagenicity evaluation of H#13,647-03: In an *in vitro* cytogenetic assay measuring chromosome aberration frequencies in Chinese hamster ovary (CHO) cells. (Unpublished study prepared by Litton Bionetics, Inc., Kensington, MD; LBI Project No. 20990; submitted to E.I. du Pont de Nemours Co., Inc., Wilmington, DE). MRID 00146846.

Goldstein A; Aronow L; Kaman SM. 1974. Principles of Drug Action: The Basis of Pharmacology. 2nd ed. John Wiley and Sons, New York, NY. 854 p.

Gunther P; Rahman A; Pestemer W. 1989. Quantitative bioassays for determining residues and availability to plants of sulphonylurea herbicides. *Weed Res. UK.* 29(2): 141-146.

Hadley BA; Bembry RP; Larsen JM; et al. 19???. The Effect of DPX-5648 on Soil Microorganism Populations. . (Unpublished study received Jan 23, 1981 under 352-EX-107; prepared in co operation with Univ. of Delaware, Soil Testing Laboratory, sub mitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; (Unpublished study received Jan 23, 1981 under 352-EX-1

Halasz-Laky V. 2000. Physico-Chemical Properties of Sulfometuron Methy. Lab Project Number: 00507-349AN. Unpublished study prepared by Toxicological Research Centre Ltd. 51 p. MRID 45415001

Halasz-Laky V. 2001. Physico-chemical Properties: Color, Physical State, Odor, Density, pH, Storage Stability and Corrosion Characteristics. Lab Project Number: 01/574-314AN. Unpublished study prepared by Toxicological Research Centre Ltd. 16 p. MRID 45451902

Halasz-Lasky V. 2000. Five-batch Analysis of Sulfometuron Methyl. Lab Project Number: 00/504-350AN. Unpublished study prepared by Toxicological Research Centre Ltd. 49 p. MRID 45339502

Hardesty P. 1983. Soil Photolysis of Sulfometuron Methyl. Document No. AMR-123-83.(Unpublished study received Apr 12, 1984 under 352-401; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL:252941-C) MRID 00137864

Harris SA; Solomon KR. 1992. Human exposure to 2,4-D following controlled activities on recently sprayed turf. J. Environ. Sci. Health. B27(1): 9-22.

Harvey J Jr. 1981a. Bioaccumulation studies of [¹⁴C]-phenyl-DPX-5648 in fish. (Unpublished study prepared by E.I. du Pont de Nemours Co., Inc., Wilmington, DE). MRID 00146279.

Harvey J Jr. 1981b. Hydrolysis of ¹⁴C-DPX-5648. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-U). MRID 00071419.

Harvey J Jr. 1990a. Phase 3 summary of MRID No. 146279: Bioaccumulation of sulfometuron methyl in fish. AMR-41-81. Guideline Reference 165-4: Bioaccumulation in fish. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Newark DE and E.I. du Pont de Nemours and Co. Wilmington, DE). MRID 93206028.

Harvey J Jr. 1990b. Phase 3 summary of MRID No. 71429: Hydrolysis of sulfometuron methyl. Guideline Reference 161-1: Hydrolysis. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Wilmington, DE). MRID 93206022.

Harvey J Jr; Bennett CT; Reiser RW. 1980. Photolysis of ¹⁴C-DPX-5648: Document No. AMR-01-81. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:244195-V). MRID 00071420.

Harvey J Jr; Dulka JJ; Anderson JJ. 1985. Properties of sulfometuron methyl affecting its environmental fate: Aqueous hydrolysis and photolysis, mobility and adsorption on soils, and bioaccumulation potential. J. Agric. Food Chem. 33(4): 590-596.

Harvey J Jr; Bennett CT; Reiser RW. 19??. Hydrolysis of ¹⁴C-DPX-5648. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:244195-U). MRID No. 00071419.

Hershman R. 1999. Paste Extruded 75WG Formulation of Sulfometuron Methyl: Evaluation of the Potential Dermal Sensitization in the Guinea Pig (Modified Buehler Method). Lab Project Number: 12479: 99897: 2420. Unpublished study prepared by White Eagle Toxicology Labs. 76 p. MRID 44874108

Hinckle L. 1979. Ten-dose oral subacute test. Haskell Laboratory No. 523-79. (Unpublished study received Jul 1, 1981 under 352- 401; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245515-M). MRID 00078794.

Hoberg J. 1990. Sulfometuron methyl-toxicity to the freshwater green alga *Selenastrum capricornutum*. Lab Project No. AMR- 1893-90. (Unpublished study prepared by Springborn Laboratories, Inc.). 24 p. MRID 41680102.

Hoberman AM; Bristol KL; Wolfe GW. 1981. Pilot oral teratology study in rabbits. Project No. 201-554; HLO-234-81. Final report. (Unpublished study received Jul 1, 1981 under 352-401; prepared by Hazleton Laboratories America, Inc., submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245515-P) MRID 00078797.

Hoerger F; Kenaga EE. 1972. Pesticide residues on plants: Correlation of representative data as a basis for estimation of their magnitude in the environment. In: Environmental Quality and Safety, Volume I: Global Aspects of Toxicology and Technology as Applied to the Environment. F. Coulston and F. Kerte (eds.). Academic Press, New York, NY. pp. 9-28.

Hoxter K; Smith G. 1990. An Acute contact toxicity study with the honey bee. Lab Project No. 112-243; 583-90. (Unpublished study prepared by Wildlife International, Ltd., Easton, MD). 14 p. MRID 41672810.

Hubbard RK; Williams RG; Erdman MD; Marti LR 1989. Chemical transport from Coastal Plain soils under simulated rainfall. II. Movement of cyanazine, sulfometuron-methyl, and bromide. Am. Soc. Agric. Eng. 32(4): 1250-1257.

ICRP (International Commission on Radiologic Protection). 1975. Report of the Task Group on Reference Man. Recommendations of the International Commission on Radiological Protection (ICRP) Publ. No. 23. Pergamon Press, New York, NY.

Issaragrisil S; Kaufman DW; Anderson T; Chansung K; Thamprasit T; Sirijirachai J; Piankijagum A; Porapakham Y; Vannasaeng S; Leaverton PE; Shapiro S; Young NS. 1997. Low drug attributability of aplastic anemia in Thailand. The Aplastic Anemia Study Group. Blood. 89(11): 4034-4039.

Jia MH; Larossa RA; Lee JM; Rafalski A; Derosé E; Gonye G; Xue Z. 2000. Global expression profiling of yeast treated with an inhibitor of amino acid biosynthesis, sulfometuron methyl. Physiol Genomics. 9;3 (2):83-92

James TK; Rahman A. 1992. Effect of simulated rainfall and adjuvants on the phytotoxicity of sulfonyleurea herbicides. Proc 1st Internat. Weed Cont. Congr. 2: 229-232.

Jokela EJ; Wilson DS; Allen JE. 2000. Early growth responses of slash and loblolly pine following fertilization and herbaceous weed control treatments at establishment. South J Appl Forestry. 24 (1):23-30

Kaniga K; Compton MS; Curtiss R 3rd; Sundaram P. 1998. Molecular and functional characterization of *Salmonella enterica* serovar typhimurium *poxA* gene: Effect on attenuation of virulence and protection. Infect Immun. 66 (12):5599-5606

Kannuck R; Sloman T. 1995. Sulfometuron methyl (DPX-T5648): Influence on growth and reproduction of *Lemna gibba* G3. Lab Project No. AMR 2902-94. (Unpublished study prepared by Stine-Haskell Research Center, E.I. du Pont de Nemours and Co, Newark, DE). 41p. MRID 43538503.

Kaplun A; Chipman DM; Barak Z. 2002. Isoleucine starvation caused by sulfometuron methyl in *Salmonella typhimurium* measured by translational frameshifting. Microbiology. 148 (3):713-717

Kinney L. 1982. Inhalation median lethal concentration (LC50) of INT-5648-18 by EPA protocol: [Benzoic acid, 2-[[[(4,6-dimethyl-2-pyrimidinyl)amino]carbonyl]amino]-sulfonyl]-, methyl ester]. Haskell Laboratory Report No. 657-82; MR No. 0581-944. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 00146848.

Koeppel MK; Mucha CF. 1991. Metabolism of sulfometuron methyl in lactating goats. J. Agric. Food Chem. 39(12): 2304-2309.

Koskinen WC; Stone DM; Harris AR. 1996. Sorption of hexazinone, sulfometuron methyl, and tebuthiuron on acid, low base saturated sands. Chemosphere. 32(9): 1681-1689.

Krahn DF; Fitzpatrick K. 1981. Chinese hamster ovary cell assay for mutagenicity. Haskell Laboratory Report No. 1074-80. (Unpublished study received Jul 1, 1981 under 352-401; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL: 245515-L) MRID 00078793.

Kraut G. 1993. Batch Equilibrium (Adsorption/Desorption) Study of (Phenyl(U)-- -(carbon 14))Sulfometuron Methyl, IN-X993, IN-D5803, and Saccharin. Lab Project Number: AMR 2607-93. Unpublished study prepared by E.I. duPont de Nemours and Co. 84 p. MRID 42789301

Kuhn JO. 1989a. Primary eye irritation study in rats: Knockout HC. Guideline No. 81-4. Laboratory Study No. 6418-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366606.

Kuhn JO. 1989b. Primary dermal irritation study in rabbits: Knockout HC. Guideline No. 81-5. Laboratory Study No. 6419-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366607.

Kuhn JO. 1989c. Primary eye irritation study in rats: Knockout HC. Guideline No. 81-4. Laboratory Study No. 6418-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366707.

Kuhn JO. 1989d. Acute dermal toxicity study in rabbits: Knockout HC. Guideline No. 81-2. Laboratory Study No. 6417-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366604.

Kuhn JO. 1989e. Dermal sensitization study in guinea pigs: Knockout HC. Guideline No. 81-6. Laboratory Study No. 6420-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366608.

Kuhn JO. 1989f. Primary dermal irritation study in rabbits: Knockout HC. Guideline No. 81-5. Laboratory Study No. 6419-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366607.

Kuhn JO. 1989g. Acute oral toxicity study in rats: Knockout. Guideline No. 81-1. Laboratory Study No. 6416-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366704.

Kuhn JO. 1989h. Dermal sensitization study in guinea pigs: Knockout. Guideline No. 81-6. Laboratory Study No. 6420-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366709.

Kuhn JO. 1989i. Acute oral toxicity study in rats: Knockout. Guideline No. 81-1. Laboratory Study No. 6416-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366603.

Kuhn JO. 1989j. Acute dermal toxicity study in rabbits: Knockout Guideline No. 81-2. Laboratory Study No. 6417-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366705.

Kuhn JO. 1989k. Acute pulmonary (intratracheal) toxicity study in rats: Knockout. Guideline No. 81-3. Laboratory Study No. 6421-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366706.

Kuhn JO. 1989l. Acute pulmonary (intratracheal) toxicity study in rats: Knockout H.C. Guideline No. 81-3. Laboratory Study No. 6421-89. (Unpublished study prepared by Stillmeadow, Inc., Houston, TX; submitted December 29, 1989 by Landis Associates, Inc., Valdosta, GA). MRID 41366605.

Landstein D; Arad S; Barak Z; Chipman DM. 1993. Relationships among the herbicide and functional sites of acetohydroxy acid synthase from *Chlorella emersonii*. *Planta*. 191(1): 1-6.

Landstein D; Epelbaum S; Arad S; Barak Z; Chipman DM. 1995. Metabolic response of *Chlorella emersonii* to the herbicide sulfometuron methyl. *Planta*. 197 (2):219-224.

Levine TE. 1996. The regulation of inert ingredients in the United States. In: *Pesticide Formulation and Adjuvant Technology*. CL Foy; DW Pritchard (Eds). CRC Press, New York, NY.

Lovell J. 2001. Nations AG II Sulfometuron Methyl 75. Product Identity and Composition; Description of Materials Used to Produce the Product; Description of Formulation Process and Discussion of the Formation of Impurities: Lab Project Number: NA II#0101. Unpublished st MRID 45451901

Lu M.H. 1981. Teratogenicity study by diet in the rat with benzoic acid: 2-[[[[[4,6-dimethyl-2-pyrimidinyl)-amino]-carbonyl]-amino]-sulfonyl]-methyl ester. Haskell Laboratory Report No. 316-81. (Unpublished study received Jul 1, 1981 under 352-401; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245515-O). MRID 00078796.

Lu M. 1990. E.I. du Pont De Nemours Co. Phase 3 Reformat of MRID 00078796. Teratogenicity Study by Diet in the Rat with Benzoic Acid, 2-[[[[[4,6-Dimethyl-2-Pyrimidinuy)Amino] Carbonyl] Amino] Sulfonyl]-,Methyl Ester: HLR 316-81, HLR 344-90. Prepared by E.I. DU PONT DE NEMOURS CO. INC. 174 p. MRID 93206029

Lym RG. 1992. Absorption and translocation of foliar-applied sulfometuron in leafy spurge (*Euphorbia esula*). *Weed Sci.* 40(3): 477-481.

Lym RG; Swenson OR. 1991. Sulfometuron persistence and movement in soil and water in North Dakota. *J. Environ. Qual.* 20(1): 209-215.

Malek DE. 1990. Primary eye irritation study with IN T5648-40 in rabbits. Medical Research No. 4581-804. Haskell Laboratory Report No. 126-90. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 41672807.

Malek D. 1994. Combined chronic toxicity/oncogenicity study with INT-5648: Long-term feeding and two-generation, four-litter reproduction study in rats: Supplement No. 2. Laboratory Project No. 4052-001: 367-84. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Newark, DE). 145 p. MRID 43154101.

Mason RW; Johnson BL. 1987. Ergonomic factors in chemical hazard control. In: *Handbook of Human Factors*. Salvendy, G; ed. John Wiley and Sons, New York, NY. pp. 772-741.

Matchett WH; Winnik W; Betowski LO; Brumley WC. 1997. A kinetic study of the methanolysis of the sulfonylureas bensulfuron methyl and sulfometuron methyl using capillary electrophoresis. *Electrophoresis.* 18(2): 205-13.

McCord E. 1994. Anaerobic Aquatic Metabolism of (Pyrimidine-2--(carbon 14))Sulfometuron Methyl in Landenberg, Pennsylvania and Bradenton, Florida Pond Water and Sediment Systems. Supplement: Lab Project Number: AMR 1084-88: 587-87: 7831-001. Unpublished study prepared by Haskell Laboratory for Toxicology and Indust

McCullagh P. 1980. Regression models for ordinal data. *J. Roy. Statist. Soc. B.* 42: 109-142.

McKelvey R. 1995. Influence of Sulfometuron Methyl (DPX-T5648) on Seedling Emergence and Vegetative Vigor of Several Terrestrial Plants. Lab Project Number: AMR 2871-93. Unpublished study prepared by DuPont Agricultural Products. 415 p. MRID 43538501

McGill DW; Brenneman BB. 2002. Six-year development of regenerating natural hardwood stands with herbaceous weed control. *North J Appl Forestry.* 19 (1):14-21

Melander A; Bitzen P-O; Faber O; Groop L. 1989. Sulphonylurea antidiabetic drugs: An update of their clinical pharmacology and rational therapeutic use. *Drugs*. 37: 58-72.

Meylan WM; Howard PH. 1993. Computer estimation of the atmospheric gas-phase reaction rate of organic compounds with hydroxyl radicals and ozone. *Chemosphere*. 26: 2293-2299. (Computer estimation program available from Syracuse Research Corporation, Syracuse, NY.).

Michael JL; Neary DG. 1993. Herbicide dissipation studies in southern forest ecosystems. *Environ. Toxicol. Chem.* 12(3): 405-410.

Mitchell RJ; Runion GB; Kelley WD; Gjerstad DH; Brewer CH. 1991. Factors associated with loblolly pine mortality on former agricultural sites in the Conservation Reserve Program. *J. Soil Water Conserv.* 46(4): 306-311.

Monson K; Hoffman D. 1990. Aerobic soil metabolism of [pyrimidine-2-¹⁴C]sulfometuron methyl in keyport silt loam. Laboratory Project ID AMR-1113-88. (Unpublished study prepared by E.I. du Pont de Nemours and Company, Wilmington, DE). MRID 42091401.

Moore JA. 1964. *Physiology of the Amphibia*. Academic Press, New York, NY. (Cited in USDA 1989b).

Moore GS. 1995. Delayed contact hypersensitivity test (Buehler method) with DPX-T5648-71 (Oust) (H-21296) in guinea pigs. Haskell Laboratory Project ID 481-95. (Unpublished study prepared by Biosearch, Inc., Philadelphia, PA; prepared for Haskell Laboratory for Toxicology and Industrial Medicine, I.E. du Pont de Nemours and Co, Newark, DE). MRID 43848406.

Moyer J. 2001. Sulfometuron-Methyl Technical: Summary of the OPPTS 830.1000 Product Properties Test Guidelines. Lab Project Number: 6603-01. Unpublished study prepared by Micro Flo Company. 8 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.6313, 830.6314, 830.6315, 830.631. MRID 45579901

Mullin LS. 1984. Long-term feeding study in rats with benzoic acid, 2-[[[(4,6-dimethyl-2-pyrimidinyl)amino]carbonyl]-amino]sulfonyl]-, methyl ester (INT-5648). Medical Research Project - 4052-001; Haskell Laboratory Report 367-84. Final report on the feeding and two-generation reproduction study conducted 1/27/81-2/24/83. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 00146849.

Muska C; Driscoll R. 1982. Early life stage toxicity of INT-5648-18 to fathead minnow. Haskell Laboratory Report No. 765-82. (Unpublished study received March 28, 1983, under 352-401; submitted by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 00126600.

Muska CF; Hall CL. 1980. 96-hour LC50 to Bluegill sunfish. Haskell Laboratory Report No. 629-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours and Co, Wilmington, DE; CDL:244195-S). MRID 00071417.

Muska CF; Trivits RL. 1980a. 48-hour LC50 to *Daphnia magna*. Haskell Laboratory Report No. 631-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours and Co, Wilmington, DE; CDL:244195-T). MRID 00071418.

Muska CF; Trivits RL. 1980b. 96-hour LC50 to Rainbow trout. Haskell Laboratory Report No. 630-80. (Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours and Co, Wilmington, DE; CDL:244195-R). MRID 00071416.

Naidu M. 1990. Aqueous Photolysis of Pyrimidine-2-C14 Sulfometuron Methyl. Lab Project Number: AMR-564-86. Unpublished study prepared by E. I. Du Pont de Nemours and Co. 43 p. MRID 41672812

Naqvi SM; Hawkins R; Naqvi NH. 1987. Mortality response and LC50 values for juvenile and adult crayfish, *Procambarus clarkii* exposed to Thiodan (insecticide), Treflan, MSMA, Oust (herbicides) and Cutrine-plus (algicide). Environ. Pollut. 48(4): 275-283.

Naqvi SM; Hawkins RH. 1989. Responses and LC sub(50) values for selected microcrustaceans exposed to Spartan), Malathion, Sonar), Weedtrine-D) and Oust) pesticides. Bull. Environ. Contam. Toxicol. 43(3): 386-393.

NCAP (Northwest Coalition for Alternatives to Pesticides). 2003. Lists of Inert Ingredients in Pesticides. <http://www.pesticide.org/FOIA/clopyralid.html>.

Neary DG; Michael JL. 1989. Effect of sulfometuron methyl on ground water and stream quality in coastal plain forest watersheds. Wat. Res. Bull. 25(3): 617-623.

Neary DG; Michael JL. 1996. Herbicides - protecting long-term sustainability and water quality in forest ecosystems. New Zeal. J. For. Sci. 26(½): 241-264.

Nishikawa S. 1983a. Effects of sulfonamide on the pituitary-thyroid gland: 1. Morphological changes of thyroid gland and variation in plasma thyroxine and triiodothyronine. J. Toxicol. Sci. 8: 47-59.

Nishikawa S. 1983b. Effects of sulfonamide on the pituitary-thyroid gland: 2. Morphological changes of thyrotrophs in anterior pituitary gland. J. Toxicol. Sci. 8: 61-70.

NRC (National Research Council). 1983. Risk assessment in the Federal government: managing the process. Washington, DC: National Academy Press; 176 p. + app.

O'Keefe DP; Romesser JA; Leto KJ. 1988. Identification of constitutive and herbicide-inducible cytochromes P-450 in *Streptomyces griseolus*. *Arch. Microbiol.* 149(5): 406-412.

Oldham JD Jr. 1984. Combined chronic toxicity/oncogenicity study with INT-5648: Long-term feeding and two-generation, four litter reproduction study in rats. Medical Research Project No. 4052-001; Supplemental data to Haskell Laboratory Report No. 367-84. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 42385706.

Oliveira RS Jr; Koskinen WC; Ferreira FA. 2001. Sorption and leaching potential of herbicides on Brazilian soils. *Weed Res.* 41 (2):97-110

O'Neill AJ. 1990. Acute inhalation toxicity study with INT-5648-18 in rats. Medical Research No. 0581-944; Laboratory Project ID 657-82. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 43089203.

Patton A. 1999. Physical and Chemical Characteristics of End-Use Product DPX-GH427 Paste Extruded Granule Blend Formulation. Lab Project Number: 2661. Unpublished study prepared by E.I. du Pont de Nemours and Company. 10 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.6316, MRID 44882903

Prasad O; Rai G. 1987. Hematological abnormalities induced by feeding a common artificial sweetener, saccharin, in ICR Swiss mice. *Toxicol. Lett.* 36(1): 81-8.

Rhodes B. 1991. Aerobic Aquatic Metabolism of Carbon 14-Sulfometuron Methyl. Lab Project Number: AMR-1896-90. Unpublished study prepared by E.I. du Pont de Nemours and Co. 40 p. MRID 42091403

Rhodes B. 1994. Aerobic Aquatic Metabolism of (carbon 14)-Sulfometuron Methyl: Supplement. Lab Project Number: AMR 1896-90. Unpublished study prepared by DuPont Agricultural Products. 7 p. MRID 43174103

Rhodes B; Cooke L. 1990. Solubility of Sulfometuron methyl in Various Organic Solvents at 25 degree C. Lab Project Number: AMR/1746/90/63/8. Unpublished study prepared by E. I. du Pont de Nemours and Co. 15 p. MRID 41680101

Rickard RW. 1992. Combined chronic toxicity/oncogenicity study with INT-5648: Long-term feeding and two-generation, four litter reproduction study in rats. Revision No.1. Medical Research Project No. 4052-001; Supplemental data to Haskell Laboratory Report No. 367-84. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 1437 p. MRID 42385705.

Roche R. 1999. Product Identity and Composition of End-Use Product Sulfometuron Methyl 75% Paste Extruded Formulation. Lab Project Number: 3109. Unpublished study prepared by DuPont Agricultural Products. 40 p. MRID 44876401

Roche R. 1999. Product Identity and Composition of End-Use Product Hexazinone/Sulfometuron Methyl (63.2:11.8) 75% (DPX-GH427) Blended Formulation. Lab Project Number: 3110. Unpublished study prepared by E.I. du Pont de Nemours and Company. 36 p. {OPPTS 830.1550, 830.1600, 830.1650, 830.1670, 830.1750} MRID 44882901

Roshon RD; McCann JH; Thompson DG; Stephenson GR. 1999. Effects of seven forestry management herbicides on *Myriophyllum sibiricum*, as compared with other nontarget aquatic organisms. *Can J Forest Res.* 29 (7):1158-1169

Rowland J. 1998. Personal communication between J. Rowland, U.S. EPA, Office of Pesticide Programs, Health Effects Division, Phone: (703) 308-2719 and Patrick R. Durkin, SERA, Inc., Fayetteville, NY. January 15, 1998.

Rudinsky JA; Kline LN; Diekman JD. 1975. Response-inhibition by four analogues of MCH, an antiaggregative pheromone of the Douglas-fir beetle. *Journal of Economic Entomology.* 68(4): 527-528.

Ruffle B; Burmaster DE; Anderson PD; Gordon HD. 1994. Lognormal distributions for fish consumption by the general U.S. population. *Risk Analy.* 14(4): 395-404.

Ryan D; Atkins B. 1986. Photodegradation of Phenyl(U)-Carbon 14 Sulfometuron Methyl and Pyrimidine-2-Carbon 14 Sulfometuron Methyl on Soil. Lab Project Number: AMR-573-86. Unpublished study prepared by E. I. du Pont de Nemours and Co. 35 p. MRID 41420601

Ryan D; Atkins B. 1986. Aged Soil Column Leaching Study with Phenyl l(U)-carbon 14 Sulfometuron Methyl and Pyrimidine-2- carbon 14 Sulfometuron methyl. Lab Project Number: AMR/643/86. Un published study prepared by E. I. du Pont de Nemours & Co. 35 p. MRID 41680103

Ryan D; Atkins B. 1987. Photodegradation of [Phenyl(U)-[carbon 14!!-Sulfometuron Methyl and [Pyrimidine-2-[carbon 14!!-Sulfometuron Methyl on Soil: Supplement No.. Lab Project Number: AMR 573-86. Unpub lished study prepared by E. I. du Pont de Nemours and Co. 15 p. MRID 42385707

Sarver JW. 1990a. Primary skin irritation and sensitization test with INT-5648-03 on guinea pigs. Medical Research No. 0581-790; Haskell Laboratory Report No. 232-79. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 69 pp. MRID 43089204.

Sarver JW. 1990b. Primary dermal irritation and sensitization test with IN T5648-40 rabbits. Medical Research No. 0581-804; Haskell Laboratory Report No. 137-90. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 13 pp. MRID 41672808.

Sarver JW. 1990c. Acute skin absorption LD50 test with INT-5648-13. Medical Research No. 0581-907; Haskell Laboratory Report No. 1078-80. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 12 pp. MRID 43089202.

Sarver J. 1990d. Oral LD50 test with INT-5648-11 in male and female rats. Lab Project No. 0581-886; HLR 870-80. (Unpublished study prepared by E. I. du Pont de Nemours and Co., Wilmington, DE). 15 p. MRID 43089201.

Sarver J. 1995. Inhalation median lethal concentration (LC50) study with DPX-T5648-72 (Oust) in rats. Medical Research Project No. 10361-001; Haskell Laboratory Report No. 589-95. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Newark, DE). 42 p. MRID 43848403.

Sauers RF; Levitt G. 1984. Sulfonylureas: new high potency herbicides (for control of weeds and grasses, chlorsulfuron, sulfometuron methyl, chemical structures). ACS Symposium series. Am. Chem. Soc. 255: 21-28.

Scheel D; Casida JE. 1985. Sulfonylurea herbicides: growth inhibition in soybean cell suspension cultures and in bacteria correlated with block in biosynthesis of valine, leucine and isoleucine. Pest. Biochem. Physiol. 23(3): 398-412.

Schloss JV; Ciskanik LM; Dyk DEvan. 1988. Origin of the herbicide binding site of acetolactate synthase. Nature, UK. 331(6154): 360-362.

Schneiders GE. 1993. Hydrolysis of [phenyl-¹⁴C(U)]- and [pyrimidine-2-¹⁴C]sulfometuron methyl in buffer solutions of pH 5, 7, and 9. DuPont Project ID AMR 2426-92. (Unpublished study prepared by E.I. du Pont de Nemours and Co., Wilmington, DE). 48 p. MRID 42715201.

Schneiders G. 1994. Photodegradation of (Phenyl-(carbon 14)(U)) and (Pyrimidine-2- -(carbon 14)) Sulfometuron Methyl in Water Conducted in Simulated Sunlight: Supplement. Lab Project Number: 2179-91. Unpublished study prepared by DuPont Agricultural Products. 18 p. MRID 43174101

Schneiders G; Bachmura S. 1992. Photodegradation of Phenyl-Carbon 14(U) and Pyrimidine-2-carbon 14 Sulfometuron Methyl in Water--Conducted in Simulated Sunlight. Lab Project Number: AMR 2179-91. Unpublished study prepared by E.I. du Pont de Nemours and Co. 48 p. MRID 42182401

SERA (Syracuse Environmental Research Associates, Inc.). 1998. Sulfometuron Methyl. Final Report, SERA TR98-21-09-02d, Report dated September 11, 1998. Prepared under USDA/FS Contract 53-3187-5-12, Order No. 43-3187-8-0222. Syracuse Environmental Research Associates, Inc., Fayetteville, NY.

SERA (Syracuse Environmental Research Associates, Inc.). 2001. Preparation of Environmental Documentation and Risk Assessments, SERA MD 2001-01a, draft dated July 2001. Syracuse Environmental Research Associates, Inc., Fayetteville, NY. Available at www.sera-inc.com.

SERA (Syracuse Environmental Research Associates, Inc.). 2003a. Documentation for Worksheets Version 2.04 - Human Health and Ecological Risk Assessments, SERA WSD 01-2.04, report dated February 25, 2003. Available at www.sera-inc.com.

SERA (Syracuse Environmental Research Associates, Inc.). 2003b. Documentation for the Use of GLEAMS (Version 3) and Auxiliary Programs in Forest Service Risk Assessments (Version 2.01), SERA TD 2003-02e. Syracuse Environmental Research Associates, Inc., Fayetteville, NY. Available at www.sera-inc.com.

Serota DG.; Fieser S; Durloo RS; et al. 1981. Teratology study in rabbits: H-13647-02: Benzoic acid, 2-[[[(4,6-dimethyl-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-, methyl ester. Project No. 201-555; HLO-331-81. Final report. (Unpublished study received Jul 1, 1981 under 352-401; prepared by Hazleton Laboratories America, Inc., submitted by E.I. du Pont de Nemours and Co., Wilmington, DE; CDL:245515-Q). MRID 00078798.

Serota DG. 1990. Phase 3 reformat of MRID 00078798 and related MRID 00078797: Teratology study in rabbits H-13647-02 benzoic acid, 2-[[[(4,6-dimethyl-2-pyrimidinyl)-amino]carbonyl]amino]sulfonyl]- methyl ester. HLO 331-81; Project No. 201-555. (Unpublished study prepared by Hazleton Laboratories America, Inc., Vienna, VA; reformatted by E.I. du Pont de Nemours and Co., Wilimington, DE). 169 p. MRID 932060630.

Shaner DL; Singh BK; Stidham MA. 1990. Interaction of imidazolinones with plant acetohydroxy acid synthase: evidence for in vivo binding and competition with sulfometuron. J. Agric. Food Chem. 38(5): 1279-1282.

Stevenson I. 1988. n-Octanol/Water Partition Coefficient Determination of Sulfometuron Methyl at pH 5, pH 7, and pH 9. Project ID: AMR-1203-88. Unpublished study prepared by E.I. du Pont de Nemours & Co. 26 p. MRID 41273601

Stidham MA. 1991. Herbicides that inhibit acetohydroxyacid synthase. Weed Sci. 39(3): 428-434.

Stone DM; Harris AR; Koskinen WC. 1993. Leaching of soil-active herbicides in acid, low base saturated sands: Worst-case conditions. Environ. Toxicol. Chem. 12(3): 399-404.

Strek G; Spaan WP. 1997. Wind erosion control with crop residues in the Sahel. *Soil Sci. Soc. Am. J.* 61(3): 911-917.

Strek G; Stein A. 1997. Mapping wind-blown mass transport by modeling variability in space and time. *Soil Sci. Soc. Am. J.* 61(1): 232-239

Strek HJ; Dulka JJ; Parsells AJ. 1990. Humic matter content vs. organic matter content for making herbicide recommendations. *Comm. Soil Sci. Plant Anal.* 21(13-16): 1985-1995.

Summers JC. 1990a. Phase 3 summary of MRID 41273602: Oncogenicity study with INT-5648: Long-term feeding study in mice. Haskell Laboratory Report No. 355-87. Guideline Reference 83-2(b) Oncogenicity - Mouse. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 26 p. MRID 93206015.

Summers JC. 1990b. Phase 3 summary of MRID 00126714: Subacute dermal toxicity study (21-days) in rabbits. Haskell Laboratory Report No. 792-82. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 31 p. MRID 93206014.

Summers JC. 1990c. Phase 3 summary of MRID 00078798 and related MRID 0078797: Teratology study in rabbits H-13,647-02 for 78798 and pilot oral teratology study in rabbits H-13,647-02 for 78797. Guideline Reference 83-3(b) Teratogenicity - Rabbit. Original study prepared by Hazleton Laboratories America, Inc., Vienna, VA; HLO 331-81, Project No. 210-55 for '78/'79 and HLO 234-81, Project No. 210-554 for 78797. (Unpublished study submitted by E.I. du Pont de Nemours and Co., Wilmington, DE). 21 p. MRID 93206017.

Summers JC. 1990d. Phase 3 summary of MRID 00071406: Oral LD₅₀ test in rats - EPA Proposed Guidelines: HLR 870-80. Guideline Reference: 81-1 Acute oral toxicity rat. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 93206009.

Summers JC. 1990e. Phase 3 summary of MRID 00146848: Inhalation median lethal concentration (LC₅₀) of INT-5648-18 by EPA protocol: HLR 657-82. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 93206011.

Summers J. 1990f. Phase 3 summary of MRID 00078796: Teratogenicity study by diet in the rat with benzoic acid, 2[[[(4,6-dimethyl-2-pyrimidinyl) amino]carbonyl]amino]sulfonyl]-, methyl ester. Haskell Laboratory Report No. 316-81. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 14 p. MRID 93206016.

Summers J. 1990g. Phase 3 Summary of MRID 00071407: Primary skin irritation and sensitization test on guinea pigs. Haskell Laboratory Report No. 232-79. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 9 p. MRID 93206012.

Summers J. 1990h. Phase 3 Summary of MRID 00078793: Chinese hamster ovary cell assay for mutagenicity. Haskell Laboratory Report No. 1074-80. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 10 p. MRID 93206019.

Summers JC. 1990i. Phase 3 summary of MRID 00071414: Avian dietary toxicity (LC₅₀) study in Mallard ducks H-13,647: HLO 788-80, Hazleton Project No. 210-548. Guideline Reference 71-1(a) Acute avian oral quail/duck. (Original unpublished study prepared by Hazleton Laboratories America, Inc., Vienna, VA; submitted by E.I. du Pont de Nemours and Co., Wilmington, DE). 12 p. MRID 93206004.

Summers JC. 1990j. Phase 3 summary of MRID 00078700: Single-dose oral toxicity study in Mallard ducks H-13,647: HLO 90-81, Hazleton Project No. 210-552. Guideline Reference 71-1(a) Acute avian oral quail/duck. (Original unpublished study prepared by Hazleton Laboratories America, Inc., Vienna, VA; submitted by E.I. du Pont de Nemours and Co., Wilmington, DE). 13 p. MRID 63206002.

Summers JC. 1990k. Phase 3 summary of MRID 00088813. Eight-day dietary LC50-Bobwhite quail H-13,647-03:HLO 464-81, Wildlife Project No. 112-122. (Original unpublished study prepared by Wildlife International Ltd., Easton, MD; submitted by E.I. du Pont de Nemours and Co., Wilmington, DE). 13 p. MRID 93206003

Summers JC. 1990l. Phase 3 summary of MRID 00143539: Early life stage toxicity of INT-5648-18 to fathead minnows: HLR 765-82: Supplement 1. Guideline Reference: 72-4(a) Early life stage fish. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 76 p. MRID 42385704.

Summers JC. 1990m. Phase 3 summary of MRID 00143539: Early life stage toxicity of INT-5648-18 to fathead minnows: HLR 765-82: Supplement 1. Guideline Reference: 72-4(a) Early life stage fish. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 16 p. MRID 93206008.

Summers JC. 1990n. Phase 3 summary of MRID 00071417: 96-Hour LC50 to bluegill sunfish: HLR 629-80: Supplement 1. Guideline Reference: 72-1(a) Fish toxicity bluegill. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 5 p. MRID 42385701.

Summers JC. 1990o. Phase 3 summary of MRID 00071417: 96-Hour LC50 to bluegill sunfish: HLR 629-80: Supplement 1. Guideline Reference: 72-1(a) Fish toxicity bluegill. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 11 p. MRID 93206005.

Summers JC. 1990p. Phase 3 summary of MRID 00071416: 96-Hour LC50 to rainbow trout: HLR 630-80: Supplement 1. Guideline Reference 72-1(c): Fish toxicity rainbow trout. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). MRID 93206006.

Summers JC. 1990q. Phase 3 summary of MRID 00071416: 96-Hour LC50 to rainbow trout: HLR 630-80: Supplement 1. Guideline Reference 72-1(c): Fish toxicity rainbow trout. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 11 p. MRID 93206006.

Summers J. 1992r. Phase 3 Summary of MRID 00071418: 48-Hour LD50 to Daphnia magna. Haskell Laboratory Report No. 631-80. Supplement No. 1. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 5 p. MRID 42385703.

Summers J. 1990s. Phase 3 Summary of MRID 00071418: 48-Hour LC50 to Daphnia magna. Haskell Laboratory Report No. 631-80. Guideline Reference 72-2(a) Invertebrate Toxicity. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 11 p. MRID 93206007.

Summers J. 1990t. Phase 3 Summary of MRID 00146847: Unscheduled DNA synthesis/rat hepatocytes in vitro. Haskell Laboratory Report No. 769-82. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 9 p. MRID 93206021.

Summers J. 1990u. Phase 3 Summary of MRID 00078792: Mutagenic activity in the Salmonella/microsome assay. Haskell Laboratory Report No. 271-79. (Unpublished study prepared by E.I. Haskell Laboratory for Toxicology and Industrial Medicine, E.I. de Pont de Nemours and Co., Inc., Newark, DE). 10 p. MRID 93206018.

Summers J. 1990v. Phase 3 summary of MRID 00078795: Ninety-day feeding and one-generation reproduction study with INT- 5648 in rats. Haskell Laboratory Report No. 928-80. (Unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). 28 p. MRID 93206013

Summers JC. 1990w. Phase 3 summary of MRID 00146846: Mutagenicity evaluation of H 13, 647-03 in an *in vitro* cytogenetic assay measuring chromosome aberration frequencies in chinese hamster ovary (CHO) cells: HLO 792-81. (Original unpublished study prepared by Litton

Bionetics, Inc., Kensington, MD; submitted by E.I. du Pont de Nemours and Co., Wilmington, DE). MRID 93206020.

Sun JH; Kulhavy DL; Roques A. 2000. Effects of fertilizer and herbicide application on Nantucket pine tip moth infestation (Lep. , Tortricidae). J Appl Entomol. 124 (3-4):191-195

Taylor JD. 1979. Mutagenic activity in the Salmonella/microsome assay: Haskell Laboratory Report No. 271-79. (Unpublished study received Jul 1, 1981 under 352-401; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:245515-K) MRID 00078792.

Taylor J; Krahn D. 1990. E.I. du Pont De Nemours Co. Phase 3 Reformat of MRID 00078792. Mutagenic Activity in the Salmonella/Microsome Assay. HLR 271-79. Prepared by E.I. DU PONT DE NEMOURS CO. INC. 14 p. MRID 93206031

Thompson S. 1994. Sulfometuron methyl (DPX-T5648): Influence on growth and reproduction of three select algal species: Lab Project Number: AMR 2892-93: 112A-121. (Unpublished study prepared by Wildlife International Ltd, Easton, MD and Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co, Inc., Newark, DE). 58 p. MRID 43538502.

Tomlin C. 1997. The Pesticide Manual. 11th Ed. Crop Protection Publications, UK. p. 1128-1130.

Triangle Labs of Columbus Inc. 1993. Occupational Health Consultants Analysis for Selected Sulfonyl Urea Herbicides from Filter Wipe Samples. Lab Project Number: 1093004. Unpublished study. 71 p. MRID 43356901

Trivits RL. 1979. Acute Oral Test. Haskell Laboratory Report No. 210-79.(Unpublished study received Jan 23, 1981 under 352-EX-107; submitted by E.I. du Pont de Nemours & Co., Wilmington, Del.; CDL:244195-G) MRID 00071405

Trubey R. 1994. Movement of Sulfometuron Methyl in Forest Watersheds After Aerial Application of Oust for Herbaceous Weed Control (75% Dispersible Granule). Lab Project Number: AMR 1641-90. Unpublished study prepared by Southern Forest Experiment Station, Auburn University. 38 p. MRID 43174104

Trubey R. 1994. Field Soil Dissipation on Oust Herbicide. Sulfometuron Methyl: Lab Project Number: AMR/1922/91: DUPDE01B 2. Unpublished study prepared by E.I. du Pont de Nemours and Co. and Alta Analytical Lab., Inc. 1151 p. MRID 43212101

Trubey R. 1994. Movement of Sulfometuron Methyl in Forest Watersheds After Aerial Application of Oust for Herbaceous Weed Control (75 (percent) Dispersible Granule). Supplement No. 2: Lab Project Numbers: FS-SO-4105-1.19: AMR 1641-90. Unpublished study prepared by Auburn University. 143 p. MRID 43492501

Trubey RK; Bethem RA; Peterson B. 1998. Degradation and mobility of sulfometuron-methyl (Oust herbicide) in field soil. *J Agric Food Chem.* 46 (6):2360-2367

Turner SA. 1987. Post-application movement of sulfometuron methyl from treated rights-of-way areas via wind (soil) erosion. Proc. 4th Symp. Environ. Concerns Rights-of-Way Manag., Indianapolis, IN, October 25-28, 1987. Purdue University, Depart. For. Nat. Res., West Lafayette, IN.

Turner S. 1992. Compilation of Incident Reports on Oust. Unpublished study prepared by US EPA. 86 p. MRID 42276601

USDA (U.S. Department of Agriculture). 1998. Cropland acreage, soil erosion, and installation of conservation buffer strips: preliminary estimates of the 1997 National Resource Inventory. [Http://www.nhq.nrcs.usda.gov/land/pubs/buffer1.html](http://www.nhq.nrcs.usda.gov/land/pubs/buffer1.html).

USDA/FS (U.S. Department of Agriculture/Forest Service). 1989a. Final Environmental Impact Statement: Vegetation Management in the Coastal Plain/Piedmont, Management Bulletin R8-MB-23, dated January, 1989. 1213 pp.

USDA/FS (U.S. Department of Agriculture/Forest Service). 1989b. Draft Environmental Impact Statement: Vegetation Management in the Ozark/Ouachita Mountains, Management Bulletin R8-MB-23, dated June, 1989. 499 pp.

USDA/FS (U.S. Department of Agriculture/Forest Service). 1989c. Final Environmental Impact Statement: Vegetation Management in the Appalachian Mountains, Management Bulletin R8-MB-38, dated July, 1989. 1104 pp.

USDA/APHIS (United States Department of Agriculture/Animal and Plant Health Inspection Service). 1993. Nontarget Risk Assessment for the MEDFLY Cooperative Eradication Program. APHIS, USDA, Riverdale, MD. [Pagination not continuous.]

USDA/ARS (U.S. Department of Agriculture). 1996. ARS Pesticides Property Database. U.S. Department of Agriculture. Internet Website access (<http://ncsr.arsusda.gov/ppdb3/>)

U.S. EPA (U.S. Environmental Protection Agency). 1989a. Recommendations for and Documentation of Biological Values for use in Risk Assessment. U.S. EPA, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. ECAO-CIN-554. [pagination not continuous].

U.S. EPA (U.S. Environmental Protection Agency). 1989b. Exposure Factors Handbook. U.S. EPA, Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC. EPA/600/8-89/043. [pagination not continuous].

U.S. EPA (U.S. Environmental Protection Agency). 1992. Dermal Exposure Assessment: Principles and Applications. EPA/600/8-91/011B. Interim Report. Exposure Assessment Group, Office of Health and Environmental Assessment, Washington, DC.

U.S. EPA (U.S. Environmental Protection Agency). 1996. Exposure Factors Handbook: Volumes I-III. EPA/600/P-95/002Ba, Bb, and Bc. NTIS PB97-117683, 117691, and 117709. [pagination not continuous].

U.S. EPA 1997. Sulfometuron Methyl (Oust) Pesticide Petition Filing 12/97. Federal Register 62 (242): 66083-66091.

U.S. EPA/OPP (United States EPA/Office of Pesticide Programs. 2003. List of Inert Pesticide Ingredients. Updated August 14, 2003. Available at: <http://ww.epa.opprd001/inerts/>.

Van Dyk TK; Ayers BL; Morgan RW; Larossa RA. 1998. Constricted flux through the branched-chain amino acid biosynthetic enzyme acetolactate synthase triggers elevated expression of genes regulated by rpoS and internal acidification. J Bacteriol. 180 (4):785-792

van Hemmen JJ. 1992. Agricultural pesticide exposure data bases for risk assessment. Rev. Environ. Contam. Toxicol. 126: 1-85.

Ward TJ; Boeri RL. 1990a. Static acute toxicity of IN T5648-40 to the sheepshead minnow, *Cyprinodon variegatus*. (Unpublished study prepared by EnviroSystems Division, Resource Analysts, Inc., Hampton, NH). 32 p. MRID 41672803.

Ward TJ; Boeri RL. 1990b. Static acute toxicity of IN T5648-40 to the bivalve mollusc embryos and larvae. (Unpublished study prepared by EnviroSystems Division, Resource Analysts, Inc., Hampton, NH). 32 p. MRID 41672805.

Ward TJ; Boeri RL. 1990c. Static acute toxicity of IN T5648-40 to the mysid, *Mysidopsis bahia*. (Unpublished study prepared by EnviroSystems Division, Resource Analysts, Inc., Hampton, NH). 32 p. MRID 41672804.

Washburn K. 2001. Sulfometuron-Methyl: Product Chemistry: Technical Grade Product. Final Report: Lab Project Number: 6604-01. Unpublished study prepared by Stillmeadow, Inc. 30 p. {OPPTS 830.6302, 830.6303, 830.6304, 830.6313, 830.6314, 830.7000, 830.7050, 830.7200, 830.7 MRID 45579902

Wauchope RD; Williams RG; Marti LR. 1990. Runoff of sulfometuron-methyl and cyanazine from small plots: Effects of formulation and grass cover. *J. Environ. Qual.* 19(1): 119-125.

Wehtje G; Dickens R; Wilcut JW; Hajek BF. 1987. Sorption and mobility of sulfometuron and imazapyr in five Alabama soils. *Weed Sci.* 35(6): 858-864.

Wetzel J. 1984. 48-Hour EC50 to *Daphnia magna*. Haskell Laboratory Report No. 147-84. (Revised unpublished study prepared by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Inc., Newark, DE). MRID 00145514.

Whitcomb C. 1991. Compilation Concerning the Use of Sulfonylurea-type Herbicides and the Fate and Effects in plants and the Environment. Unpublished study. 264 p. MRID 43039601

Willard R. 1996. Preliminary Analysis of Sulfometuron Methyl (T5648) in Oust Herbicide. Lab Project Number: AMR 3447-95. Unpublished study prepared by DuPont Agrichemicals Caribe, Inc. 20 p. MRID 44035901

Winegardner, DL. 1996. An Introduction to Soils for Environmental Professionals. Lewis Publishers, New York, NY.

Wood CK; McAlack JW; Schneider PW; Barba CM. 1980. Ninety-day feeding and one-generation reproduction study with benzoic acid, 2-[[[(4-methyl-2-pyrimidinyl)aminocarbonyl]-amonsulfonyl], methyl ester, INT-5648 in rats. Medical Research Project No. 3492; Haskell Laboratory Report No. 928-80. Final Report. (Unpublished study received Jul 1, 1981 under 352-401; submitted by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Newark, DE; CDL:2455155N). MRID 00078795.

Wood C; O'Neal F. 1983. One-year feeding study in dogs with benzoic acid, 2-[[[(4,6-dimethyl-2-pyrimidinyl)-amino]carbonyl]amino]sulfonyl]-, methyl ester, INT 5648. Haskell Laboratory Report No. 482-82. Final Report. (Unpublished study received Jul 1, 1981 under 352-401; submitted by Haskell Laboratory for Toxicology and Industrial Medicine, E.I. du Pont de Nemours and Co., Newark, DE; CDL:250950A). MRID 00129051.

WSSA (Weed Science Society of America). 1989. *Herbicide Handbook of the Weed Science Society of America*. Sixth edition. Champaign, IL: Weed Science Society of America. p. 239-240.

Xie Q; Jimenez A. 1996. Molecular cloning of a novel allele of SMR1 which determines sulfometuron methyl resistance in *Saccharomyces cerevisiae*. *FEMS Microbiol. Lett.* 137(2-3): 165-8.

Table 2-1. Identification and Physical/Chemical Properties of Sulfometuron Methyl.

Property	Value	Reference
Synonyms	Formulations: Oust XP, Oust Synonyms: T-5648, DPX-5648, Haskell #13,647, NB 8415-21, INT-5648-1, DPX- T5648-71 (Oust), DPX-T5648-72 (Oust),	
CAS Number	74222-97-2	Budavari 1989
U.S. EPA Registration Number	352-601	Du Pont 1999a,b
MW $C_6H_3Cl_3N_2O_2$	364.38	Budavari 1989
Henry's Law Constant (atm m ³ /mole)	$< 5 \times 10^{-17}$ (atm-m ³ /mole), calculated from vapor pressure	
pK _a	5.2 5.7	Washburn 2001 Budavari 1989
Vapor pressure	5.5×10^{-16}	Tomlin 1997, WSSA 1989
Water solubility	0.897 mg/L at 25°C, pH not specified 10 mg/L at 25°C, pH 5 300 mg/L at 25°C, pH 7	Washburn 2001 Budavari 1989 Budavari 1989
K _{o/w} (acid)	pH 5, log K _{o/w} = 1.01 pH 5, log K _{o/w} = 1.04 pH 5, log K _{o/w} = 1.01 to 1.07 pH 7, log K _{o/w} = -0.46 pH 9, log K _{o/w} = -1.87 pH 9, log K _{o/w} = -1.86	USDA/ARS 2003 Cadwgan 1990a Stevenson 1988 USDA/ARS 2003, Cadwgan 1990a, Stevenson 1988 USDA/ARS 2003, Cadwgan 1990a Stevenson 1988
K _{o/c} (acid, ml/g)	61-122 16-50	USDA/ARS 2003 Oliviera et al. 2001

Table 2-2: Use of sulfometuron methyl by USDA Forest Service in 2001 by Type of Use (USDA/FS 2002)

Use Classification	Total Pounds	Total Acres	Pounds per acre average	Proportion of Use	
				by Pounds	by Acres
Conifer Release	37.20	1,043.00	0.030	0.6564	0.8242
Noxious Weed Control	2.50	60.50	0.041	0.0441	0.0478
Rights-of-Way	6.00	40.00	0.150	0.1059	0.0316
Site Preparation	10.97	122.00	0.090	0.1936	0.0964
Grand Total	56.67	1,265.50	0.045	1	1

Table 2-3: Use of sulfometuron methyl by USDA Forest Service in 2001 by Region (USDA/FS 2002)

Region	Pounds	Acres	lbs/acre	Proportion of Total Pounds	Proportion of Total Acres
Northern (R1)	0.00	0.00	0.000	0.000	0.00
Rocky Mountain (R2)	0.00	0.00	0.000	0.000	0.00
Southwestern (R3)	0.00	0.00	0.000	0.000	0.00
Intermountain (R4)	1.50	50.00	0.030	0.026	0.04
Pacific Southwest (R5)	0.00	0.00	0.000	0.000	0.00
Pacific Northwest (R6)	0.00	0.00	0.000	0.000	0.00
Southern (R8)	38.20	1053.50	0.036	0.674	0.832
Eastern (R9)	16.97	162.00	0.105	0.299	0.128
Total	56.67	1265.50	0.045	1	1

Table 3-1: Chemical and site parameters used in GLEAMS Modeling for Sulfometuron methyl.

Chemical Specific Parameters				
Parameter	Clay	Loam	Sand	Comment/ Reference
Halftimes (days)				
Aquatic Sediment		60		Note 1
Foliar		10		Knisel and Davis 2000
Soil	10	30	100	Note 2
Water		113		Note 3
Ko/c, mL/g		78		Note 4
K _d , mL/g	0.15	0.6	1	Note 5
Water Solubility, mg/L		300		Budavari 1989 (pH 7, 25°C)
Foliar wash-off fraction		0.65		Knisel and Davis 2000
Note 1	Upper limit of range (3-weeks to 2 months) reported by (Dulka 1980b) and (Anderson 1990a) in in fresh anaerobic aquatic system and flooded soil.			
Note 2	Value for loam is the approximate value for silt-loam soil reported by Anderson (1990) and Anderson and Dulka (1985). Longer halftimes of about 50 to 170 days are reported in sterile soil (Monson and Hoffman 1990; Anderson and Dulka 1985). The shorter soil halftime of 20 days recommended by Knisel and Davis (2000) may reflect a more active or abundant soil microorganisms. The wider range of field dissipation halftimes of 10 to 120 days (USDA/ARS 1995) probably reflect combination of arid conditions with low microbial populations (100 to 120 days) and moist field conditions with richer microbial populations (10 to 20 days). For GLEAMS models, the upper range of 100 is used for sand and lower range of 10 is used for clay. These may vary substantially with site conditions.			
Note 3	Based on pH 7 hydrolysis half-time reported by Schneiders (1993). A somewhat shorter pH 7 hydrolysis rate of 43.6 days has been reported by Naidu (1990). For acidic waters (pH of about 5), halftimes of about 14 days are more representative (Brattsten 1987; Harvey 1990b; Naidu 1990; Schneiders 1993).			
Note 4	Value recommended by Knisel and Davis (2000). A wide range of Ko/c values have been reported – i.e, 61-122 in USDA/ARS (1995) and 16-50 Oliviera et al.(2001). Differences in humic acid content of various soils may account for some of this variability (Strek et al. 1990) and could need to be considered in site-specific assessments.			
Note 5	Value for clay taken as average of 0.12 and 0.17 reported by Wehtje et al. (1987). Value of 1.0 for sand taken from Cadwgan (1990b). Value for loam taken as approximate average of values used for sand and clay.			
Site Parameters (see SERA 2003, SERA AT 2003-02d dated for details)				
Pond	1 acre pond, 2 meters deep, with a 0.01 sediment fraction. 10 acre square field (660' by 660') with a root zone of 60 inches and four soil layers.			
Stream	Base flow rate of 4,420,000 L/day with a flow velocity of 0.08 m/second or 6912 meters/day. Stream width of 2 meters (about 6.6 feet') and depth of about 1 foot. 10 acre square field (660' by 660') with a root zone of 60 inches and four soil layers.			

Table 3-2: Summary of modeled concentrations of sulfometuron methyl in streams (all units are $\mu\text{g/L}$ or ppb per lb/acre applied)

Annual Rainfall (inches)	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
15	0.00044	0.07473	0.00000	0.00000	0.00000	0.00004
20	0.00093	0.16841	0.00000	0.00000	0.00056	0.01324
25	0.00144	0.27655	0.00000	0.00000	0.00174	0.02907
50	0.00358	0.81902	0.00000	0.00102	0.00677	0.12316
100	0.00576	1.63619	0.00030	0.04322	0.00962	0.26951
150	0.00639	2.10070	0.00049	0.04651	0.00913	0.34413
200	0.00605	1.98950	0.00060	0.03894	0.00822	0.37910
250	0.00563	1.85116	0.00065	0.03135	0.00736	0.39285

Table 3-3: Summary of modeled concentrations of Sulfometuron methyl in ponds (all units are $\mu\text{g/L}$ or ppb per lb/acre applied)

Annual Rainfall (inches)	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
10	0.00000	0.00000	0.00000	0.00000	0.00000	0.00000
15	0.02102	0.06455	0.00000	0.00000	0.00001	0.00009
20	0.02916	0.13608	0.00000	0.00000	0.01022	0.01795
25	0.03589	0.21133	0.00000	0.00000	0.02576	0.04038
50	0.05446	0.56238	0.00006	0.00066	0.06528	0.16301
100	0.06556	1.15455	0.00246	0.02971	0.06851	0.29436
150	0.06629	1.61771	0.00310	0.03412	0.06113	0.34969
200	0.05952	1.63274	0.00322	0.03024	0.05427	0.37395
250	0.05370	1.59417	0.00317	0.02556	0.04859	0.38694

Table 4-1: Summary of Data on Short- and Long-term Exposure of African Clawed frog (*Xenopus laevis*) to sulfometuron methyl¹

Exposure Duration	Effect	LC₅₀ or EC₅₀ (mg a.i./L)²	NOAEL (mg a.i./L)²	LOAEL (mg a.i./L)²
4 hours	lethality	> 7.5	0.38	0.75
4 hours	malformations	0.71	0.38	0.75
14 days	tail resorption rate	–	0.00075	0.0075
30 days	malformations	between 0.0756 and 0.38	0.0075	0.038

¹ All data from Fort 1998

² The author did not state whether data were reported in terms of mg sulfometuron methyl/L or mg Oust/L. Taking the most conservative approach, values reported by Fort 1998 are assumed to be expressed as mg Oust/L. To calculate all values in terms of mg a.i./L, reported values were multiplied by 0.75.

Table 4-2: Summary of modeled concentrations of Sulfometuron methyl in soil (all units are mg/kg soil or ppm per lb/acre applied)

Annual Rainfall (inches)	Clay		Loam		Sand	
	Average	Maximum	Average	Maximum	Average	Maximum
5	0.29710	5.44359	0.52422	4.85915	0.79146	3.94014
10	0.27002	4.97065	0.38725	4.17880	0.28655	3.55616
15	0.23510	4.62861	0.26479	3.55233	0.12702	3.52830
20	0.20562	4.29300	0.19391	3.52516	0.07456	3.52545
25	0.18320	4.00415	0.14874	3.52516	0.05038	3.52516
50	0.11725	3.99075	0.05973	3.52516	0.02003	3.52516
100	0.04983	3.99075	0.02269	3.52516	0.01411	3.52516
150	0.01248	3.99075	0.01535	3.52516	0.01289	3.52516
200	0.01240	3.99075	0.01304	3.52516	0.01231	3.52516
250	0.01235	3.99075	0.01261	3.52516	0.01199	3.52516

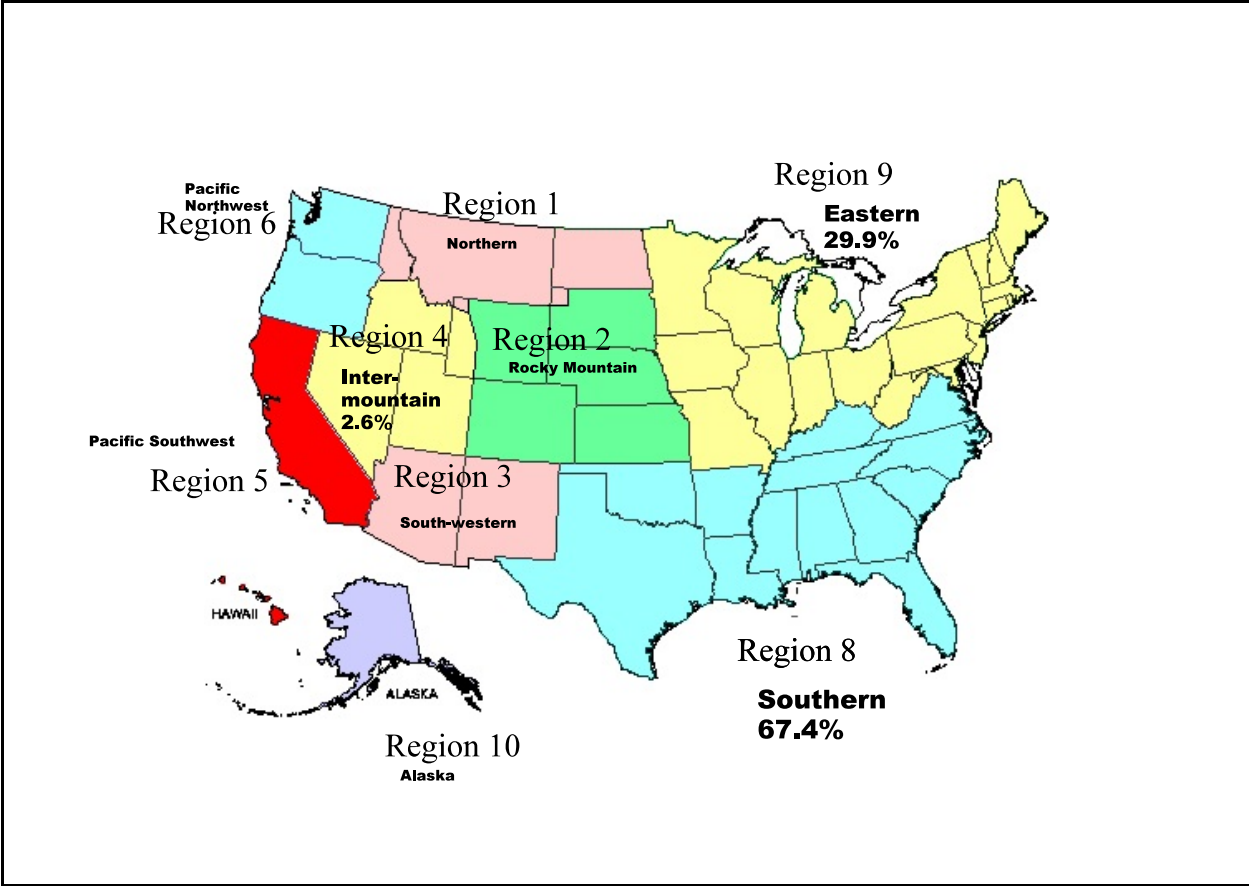


Figure 2-1. Use of sulfometuron methyl by the USDA Forest Service in various regions of the United States based on percentages of total use by the F S.

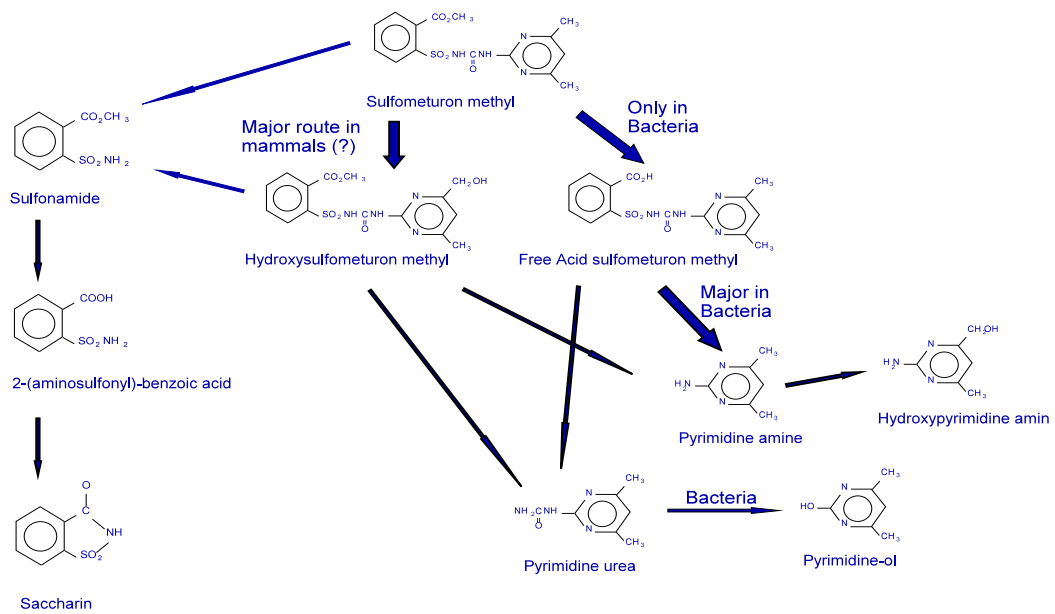


Figure 3-1: Proposed metabolic pathway of sulfometuron methyl in the goat (adapted and modified from Cambon et al. 1992; Koeppe and Mucha 1991; Monson and Hoffman 1990).

APPENDICES

- Appendix 1:** Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals
- Appendix 2:** Laboratory and simulation studies on environmental sulfometuron methyl
- Appendix 3:** Field Studies on the environmental fate of sulfometuron methyl
- Appendix 4:** Toxicity of sulfometuron methyl to experimental birds
- Appendix 5:** Bioassays of sulfometuron methyl toxicity in terrestrial plants
- Appendix 6:** Toxicity to fish, aquatic invertebrates, and aquatic plants
- Appendix 7:** Toxicity of sulfometuron methyl to amphibians
- Appendix 8:** Effects of Sulfometuron methyl on microorganism and microbial populations in soil

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
ORAL - ACUTE			
Rats, CrI:CD, male and female, 7-8 weeks old, 15 per sex	5000 mg/kg by gavage. Test substance appears to be Oust (75% formulation).	No mortality. Alopecia on left hind quarters of 1 female rat. No gross lesions on necropsy. LD ₅₀ > 5,000 mg/kg (= 3750 mg a.i./kg)	Filliben 1995a MRID 43848401 Summarized by Summers 1990e MRID 93206011
Rats, CrI:CD; 5 males and 5 females	5000 mg/kg by gavage. Animals observed for 15 days after dosing Sulfometuron methyl past extruded 75WG Formulation. According to DuPont Agricultural Products (1999) (MRID 44874100), this formulation is Oust XP	No deaths, signs of toxicity, weight loss or gross lesions in any rat LD ₅₀ > 5000 mg/kg (> 3750 mg a.i./kg)	Finlay 1999 MRID 44874103
Rats, young adult males, 1/dose group	Single gavage dose of 0, 5000, 7000, 11,000, or 17,000 mg/kg of body weight “Benzoic acid, 2[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] Amino-sulfonyl]-, Methyl ester” suspended in corn oil, followed by 14-day observation period.	Weight loss for 1 day at 5000, 7000, or 17,000 mg/kg; wet and or stained perineal area for 1-2 days at 11,000 or 17,000 mg/kg; stained underside for 1 day at 17,000 mg/kg. No mortality at any dose. LD ₅₀ > 17,000 mg a.i./kg	Trivits 1979 MRID 00071405 Summarized by Summers 1990b MRID 93206014
Rats, ChR-DC, young adults, 5 males (fasted avg wt =224g) and 5 females (fasted avg wt = 149 g)	Single gavage dose of 5000 mg/kg body weight “Benzoic acid, 2[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] Amino-sulfonyl]-, Methyl ester” (other codes: Haskell #13,647; NB 8415-21; and INT-5648-11) suspended in corn oil, followed by 14-day observation period.	No mortality. No clinical signs in males; females had wet perineal area and slight weight loss. Increase (NOS) in lung weight in males and females with histological changes [apparent inflammation]. 'pink thymus' in 4/5 females. LD ₅₀ >5000 mg a.i./kg (males and females)	Dashiell and Hinckle 1980c MRID 00071406 Summarized by Summers 1990d MRID 93206009

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rats, CrI:CD, young male and female, 5 per dose level per sex.	5000 mg a.i./kg. Gavage in corn oil.	Alopecia in males only. No mortality. LD ₅₀ >5000 mg a.i./kg (males and females)	Dashiell and Hall 1980 MRID 00071409
ORAL - SUBCHRONIC			
Rats, ChR-DC, young adult male, 6 rats per dose level.	0 and 3400 mg/kg bw, 5 times per week for 2 weeks followed by 14 day recovery period. Gavage in corn oil.	Testis of 1 test rat weighted only 0.97 g, expected is 3 grams and another exhibited mild testicular lesions involving later stages of germ cell maturation. No other gross or microscopic pathology. No mortality.	Hinckle 1979 MRID 00078794
Rats, CD, 16 animals per dose group per sex.	Dietary levels of 0, 100, 1000, or 5000 ppm for 90 days. [Average doses for males from Table IX, p. 30: 0, 9, 74, 370 mg/kg/day. Average doses for females from Table X, p. 31: 0, 9, 91, 432 mg/kg/day.] Partial sacrifice (10 per group) after 90 day. Other animals allowed to mate. Includes reproductive parameters	Elevated mean leukocyte and lymphocyte counts and decreased neutrophils in males at 5000 ppm. No effects on reproductive parameters. Other hematologic changes - not considered by the study authors to be treatment related - included reduced mean corpuscular hemoglobin concentrations in males at 1000 and 5000 ppm and decreased hemoglobin in females at 5000 ppm. Also elevated serum thyroxine content in female rats at 100 and 1000 ppm.	Wood et al. 1980 MRID 00078795

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rats, female, ChR-CD	0, 50, 1000, and 5000 ppm in the diet on days 6-15 of gestation. At 5000 ppm, the average daily dose was 433 mg/kg. Based similar values for food consumption [Summers 1990, Item 10, p. 9; Lu 1990, Table 3, p. 22], diets containing 50 and 1000 ppm are estimated to correspond to doses of 4.33 mg/kg/day and 86.6 mg/kg/day. Teratology study	Decreased maternal weight gain associated with decrease food consumption at 5000 ppm. Also, decreased fetal weight at 5000 ppm.	Lu 1981 MRID 00078796 Summarized by Summers 1990f MRID 93206016 Also summarized by Lu 1990 MRID 93206029

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rabbits, New Zealand White, 5 per dose at all doses except 300 mg/kg. At this dose level, 6 animals were used. This occurred because of an injury (NOS) in one of five animals originally assigned to this group. The injured animal was anticipated to die but survived the duration of the study.	0, 100, 300, 750, 1000 mg/kg bw on days 6-18 of gestation by gavage in 0.5% methylcellulose in distilled water. A range finding teratology study	2/5 animals at 1000 mg/kg and 1/5 animals at each of the 100, 300, and 750 mg/kg dose levels died or were sacrificed after evidence of abortion. One rabbit in the 300 mg/kg group was found dead due to possible tracheal intubation. One rabbit in the 750 mg/kg group was found dead during the study for no apparent reason. Five of the animals - one at 100 mg/kg, two at 750 mg/kg, and two at 1000 mg/kg were sacrificed upon evidence of abortion. Signs of toxicity included anorexia, depression, and thinness as well as decreased weight. In the post-treatment period, animals at 1000 mg/kg continued to loose weight. Animals at 300 and 750 mg/kg evidenced decreased weight gain. No clear association of pathology with dose levels. Possible spontaneous abortions in 1/5 at 300 mg/kg/day and 2/5 at 750 and 1000 mg/kg. Increased resorptions and no fetuses at 1000 mg/kg.	Hoberman et al. 1981 MRID 00078797

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rabbits, New Zealand White, 17 per dose level	0, 30, 100, and 300 mg/kg on days 6-18 of gestation by intubation in 0.5% methylcellulose in distilled water. Teratology study	Statistical evaluation of all parameters (maternal body weights changes, clinical observations, survival, gross pathology, and pregnancy rates; numbers and percentages of copus lutea, implantations, and resorptions in each maternal animal; and fetal sex, viability and development) revealed no significant differences between the control and treatment groups. The total number of fetuses with anomalies was increased [1/100, 2/87, 5/90, 3/96] as was the mean percent of fetal anomalies per litter [0.7, 3.3, 7.2, 3.3]. Treatment groups were not statistically different from the control group.	Serota et al. 1981 MRID 00078798 Summarized by Summers 1990c MRID 93206017 Reformatted by Serota 1990 MRID 93206030

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
ORAL - CHRONIC			
Dogs, Beagles, 1 to 2 years old, six per dose level	0, 200, 1000, and 5000 ppm in the diet for 1 year. Dose levels correspond to 5, 28, and 150 mg/kg respectively based on measured food consumption.	No changes in food consumption or body weight gain. At 1000 and 5000 ppm, mild hemolytic anemia - i.e. dose related decreases in erythrocyte counts, hematocrit, and hemoglobin. Potentially significant effects include increased alkaline phosphatase activity, increased serum cholesterol [females only], decreased serum albumin and creatinine. At 5000 ppm, increased liver weights in females [absolute] and males and females [relative] and increased absolute and relative thymus weights in females. Thymus weights (absolute) were increased in males at 200 and 1000 ppm but not at 5000 ppm. No pathological changes in the thymus at any dose level in either sex.	Wood and O'Neal 1983 MRID 00129051
Mice, Crl:CD-1 (ICR) BR, 80 per sex per dose level	Dietary exposure to 0, 5, 20, 100, and 1000 ppm for 18 months. Mean food consumption in all groups of about 5.5 grams/day and mean body weight for all groups of about 30.5g. The fractional food consumption is calculated as 0.18 (5.5 g/day ÷ 30.5g). Thus, daily doses are calculated as 0.9, 3.6, 18 and 180 mg/kg/day [the concentration in food × the fractional food consumption]	Decreased body weight gain (6%) in females at 1000 ppm. Mild anemia and hypoproteinemia and a statistically significant increase in incidence of amyloidosis at 1000 ppm in females. No significant effects in males. For hematological effects in females – NOAEL = 100 ppm LOAEL = 1000 ppm	Summers 1990a MRID 93206015 This is a summary of Cadwgan 1990a MRID 41273602. This was not identified by EPA in U.S. EPA's search of its files.

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rats, CrI:CD, 80 male and 80 female animals per group.	0, 50, 500, and 5000 ppm in the diet for 24 months. Partial sacrifice [10/group] at 1 year. Based on food consumption and body weights, doses were 0, 2, 20, and 199 mg/kg bw for males and 0, 3, 26, and 260 mg/kg bw for females. Includes reproductive study. After 90 days on study, two-generation, four litter reproduction substudy was conducted using 20 animals from each group.	At 5000 ppm, females evidenced decreased weight gain and decreased food consumption. No gross signs of toxicity. Decreased erythrocyte count and hematocrit in males at 500 and 5000 ppm. Mean absolute brain weights in males at 5000 ppm were significantly lower than controls. Dose dependent increase in bile duct hyperplasia and fibrosis in females at 500 and 5000 ppm. At 5000 ppm, number of pups was decreased in the F1 and F2 generations.	Mullin 1984 MRID 00146849 Appears to be identical to Rickard 1992 MRID 42385705 Individual animal pathology given in Oldham 1984 MRID 42385706
DERMAL			
Guinea pigs, male, albino, 10 animals	50% w/v in dimethyl phthalate on day 1 with challenge on day 13.	Mild skin irritation in one challenged animal.	Edwards 1979a MRID 00071407 Individual animal data in Sarver 1990a, MRID 43089204 Summarized by Summers 1990g MRID 93206012
Guinea pigs, Duncan-Hartley, albino, male	0.05 ml of 5% and 50% in dimethyl phthalate on shaved and intact shoulder skin..	No irritation with 5% solution and no to mild irritation with 50% solution. No sensitization on challenge after 13 days.	Dashiell and Silber 1980b MRID 00071413

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Guinea pigs, Hartly, male, n=20 in treatment group, n=10 in saline control group and n=5 in dinitrochlorobenzene positive control group. <i>OUST: 75% ai</i>	0.5 g moistened in saline on to clipped skin covered. Removed after 6 hours and scored for irritation at 24 and 48 hours. Procedure performed once per week for 3 weeks.	No delayed contact hypersensitivity. Positive results found with positive control.	Moore 1995 MRID 43848406
Guinea pigs, Hartley, 30 males, weight between 339 and 452 g <i>Paste Extruded 75WG Formulation (75% DPX-T5648 a.i.).</i>	occluded application of 0.5 g aliquot test material moistened with 0.5 mL normal saline solution to clipped intact skin for 6 hours. Test sites were wiped with saline and then deionized water.	No redness observed 24 or 48 hours after induction phase; no redness at test sites 24 or 48 hours after challenge phase; incidence of sensitization was 0%.	Hershman 1999 MRID 44874108
Rabbits, New Zealand, male, 5 per dose group.	1500, 2000, 5000, and 8000 mg/kg	moderate and mild redness, slight swelling, sporadic weight loss. One animal died in the 2000 mg/kg group. No compound related pathology.	Dashiell and Silber 1980c MRID 00071410
Rabbits, New Zealand, female, 5 per dose group.	2000 mg/kg	Severe to mild redness, severe to slight swelling, sporadic weight loss. No compound related pathology.	
Rabbits, New Zealand, male, 6 per dose group.	0.5 g applied to 2 areas each of intact and abraded skin.	No primary skin irritation.	Dashiell and Henry 1980a MRID 00071411
Rabbits, New Zealand, male and female, 5 per group	2000 mg/kg in physiological saline to the abraded back for 24 hours. Observed for 14-15 days.	Diarrhea, sporadic weight loss, slight erythema and edema.	Dashiell and Silber 1981 MRID 00078791 Summarized by Summers 1990h MRID 93206010
Rabbits, New Zealand, male and female, 5 per sex per dose group	0, 125, 500, 500, and 2000 mg/kg, 6 hours per day for 21 consecutive days.	No signs of toxicity, pathological changes, or changes in clinical chemistry attributed to treatment.	Dashiell and Hinckle 1983 MRID 00126714

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rabbits, New Zealand, male, young, n=6	0.5 g moistened with distilled applied to gauze on the shaved back. Observations at 30-60 minutes and 24, 48, and 72 hours.	No dermal irritation. During the study, one animal died. This was attributed to handling procedure rather than the test compound.	Sarver 1990b MRID 41672808 Individual animal data in Sarver 1990c MRID 43089202
Rabbits, New Zealand White, 5 per sex <i>OUST: 75.57% a.i.</i>	5000 mg/kg to the shaved intact skin occluded for 24 hours then removed. 14 day observation period.	No mortality or clinical signs of toxicity. Mild to severe erythema and slight to moderate edema after 2 hours post-removal. Most erythema and all edema resolved by 5 days. Slight to mild erythema and epidermal scaling, sloughing, or desquamation from day 5 to end of study. No gross lesions. Minimal and mild skin discoloration in 1 male and 1 female attributed to shaving prior to necropsy.	Filliben 1995c MDIR 43848402
Rabbits, New Zealand White, female, n=6 <i>OUST: 75.57% a.i.</i>	0.5 g, occluded for 4 hours then removed. Observations at 1, 24, 48, and 72 hours.	Mild to slight primary irritation based on erythema in 1 of 6 animals at 1 hour after application. No effects at 24 hour or later. No signs of systemic toxicity. Weight loss of about 3% in one animal by end of study {this was <i>not</i> the animal that evidenced skin irritation.}.	Filliben 1995b MRID 43848405

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rabbits, New Zealand White, 6 males	0.5 g test substance (0.376 g a.i.) applied to shaved, intact skin, occluded for 4 hours, then removed. Rats observed for 72 hours. Paste extruded 75 WG Formulation of sulfometuron methyl. According to DuPont Agricultural Products (1999) (MRID 44874100), this formulation is Oust XP	Primary dermal irritation study. Erythema observed in all animals. 3/6 rabbits had mild edema. At 72 hours after application, no skin irritation was observed. No clinical signs of toxicity or changes in body weight were observed. Test substance classified as a “non-irritant”	Finlay 1999 MRID 44874107
rats, Crl:CS; 5 males and 5 females	Single dose of 5000 mg/kg applied to shaved, intact skin, occluded for 24 hours, then removed. Rats observed for 15 days Paste extruded 75 WG Formulation of sulfometuron methyl. According to DuPont Agricultural Products (1999) (MRID 44874100), this formulation is Oust XP	No deaths, no substance-related clinical signs of toxicity. No gross lesions on necropsy. Mild erythema observed in 5 rats which resolved within 7 days. No edema in any rats. LD ₅₀ > 5000 mg 75 WG/kg (> 3750 mg a.i./kg)	Finlay 1999 MRID 44874104
EYES			
Rabbits, albino, 2 each group	1 mg a.i. in right conjunctival sac with or without washing after 20 seconds	Without washing, mild redness at 1 hour to 1 day and slight swelling at 1-4 hours. With washing, only mild redness at 1 hour.	Edwards 1979b [MRID 00071408]
Rabbits, albino, male, 9	61.8 mg a.i. in right eye with (n=3) or without washing (n=6) after 20 seconds. Observations at 1, 2, 3, and 4 days.	Without washing, slight transient corneal cloudiness in 2/6 animals. With washing, similar effects in 2/3 animals. All eyes were normal within 2-3 days.	Dashiell and Henry 1980a [MRID 00071411]

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
Rabbits, New Zealand White, 6 females	32 mg 75 WG Formulation (24 mg a.i.) in 0.1 mL administered to 1 eye of each animal; eye were unwashed. Animals observed for 72 hours.	Test substance produced conjunctival redness and conjunctival chemosis in all rabbits. Effects were resolved between 24 and 72 hours after treatment.	Finlay 1999 MRID 44874106
	Paste extruded 75 WG Formulation of sulfometuron methyl. According to DuPont Agricultural Products (1999) (MRID 44874100), this formulation is Oust XP	No corneal opacity, iritis or conjunctiva discharge was observed. Test substance classified as a “non-irritant”	
Rabbits, New Zealand White, male, young, n=6	0.079 g a.i. (0.1 mL) into the lower conjunctival sac of the right eye. No washing. Observations at 1, 24, 48, and 72 hours.	After 1 hour, redness and discharge from the conjunctiva of 3/6 animals. After 24 hours, conjunctival discharge in 1/6 animals. No effects at 48 or 72 hours.	Malek 1990 MRID 41672807
Rabbits, New Zealand, White, young adult, n=6 <i>Oust</i>	46 mg (~0.1 mL, ~34.5 mg a.i.) in one eye. Evaluations at 1, 24, 48, and 72 hours.	At 24 to 48 hours, conjunctival redness, chemosis, and discharge. No corneal opacity or iritis. No effects after 48 hours.	Filliben 1995d MRID 43848404

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
INHALATION			
rats, CrI:CD; 5 males and 5 females	nose-only inhalation for 4 hours to 5.3 mg 75 WG formulation /L; rats observed to 15 days	No deaths. Immediately after exposure, clinical signs of toxicity included nasal and/or oral discharge and stained fur.	Bamberger 1999 MRID 44874105
	Paste extruded 75 WG Formulation of sulfometuron methyl. According to DuPont Agricultural Products (1999) (MRID 44874100), this formulation is Oust XP	All rats had slight to moderate body weight loss the day after exposure, but losses were not maintained during the 15-day observation period. No gross pathology findings or evidence of organ-specific toxicity. LC50 > 5.3 mg 75 MG formulation/L (> 4.0 mg a.i./L)	
Rats, CrI:CD, 7-8 weeks old, male and female, 5 per group	Mean air concentrations of 6.4 or 11 mg/L air for 4 hours, head only.	No apparent signs of toxicity or pathology.	Kinney 1982 MRID 00146848 Individual animal data in O'Neill 1990 MRID 43089203
Rats, CrI:CD, male and female, 8 weeks old, 15 per sex OUST: 75.25% a.i.	Mean air concentrations of 5.1 mg/L air for 4 hours, head only. 14 day recovery period.	Nasal and ocular discharge in male rats. Nasal discharge and wet perineum in female rats. Slight and generally transient weight loss. No gross pathology.	Sarver 1995 MDIR 43848403

Appendix 1: Toxicity of sulfometuron methyl, Oust and Oust XP to experimental mammals [a.i. unless specified as *Oust* or *Oust XP*, in which case the commercial formulation was used].

Animal	Dose/Exposure	Response	Reference
KINETICS			
Rats	16 mg/kg and 3000 mg/kg	$t_{1/2}$ s of 28 and 40 hours respectively	DuPont 1989. Metabolism of Sulfometuron Methyl in Rats. Unpublished, Feb. 3, 1989, not submitted to EPA, summarized in EXTOXNET, 1994, ref. 10
Lactating goats, n=2, 40 kg bw	sufometuron methyl with double label: pyrimidine-2- ¹⁴ C- and uniformly labelled phenyl ring, capsules, 0.575 mg/kg or 0.625 mg/kg, twice per day for 7 days. [Author give 'dietary' equivalent, apparently based on differences in food consumption of 25 ppm and 60 ppm but the dosing seems to have been by gavage.] Animals sacrificed 20 h after last dose.	94-99% of dose recovered in the urine.	Keoppe and Mucha 1991

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
Aquatic Sediments	
Fate in sediment/pond water systems and flooded soil. $t_{1/2}$ of 3 weeks to 2 months in fresh anaerobic sediment systems. Soil types in the sediment varied ($t_{1/2}$ range only given, not specific for sediment type) $t_{1/2}$ of 4 months in sterile soils. Major metabolites saccharin and 2-(aminosulfonyl)-benzoic acid. A.I. has no effect on catabolism of cellulose to CO_2 by anaerobic organisms.	Dulka and Anderson 1982 MRID 00143540 Anderson 1990a MRID 93206025
Fate in sediment/pond water systems and flooded soil. $t_{1/2}$ of 1 month in anaerobic aquatic systems and flooded soil DPX-5648 readily hydrolyzes to saccharin under sterile or "fresh" conditions. Final metabolic products appear to be naturally occurring aliphatic carboxylic acids.	Dulka 1980b MRID 00078707 [NEW STUDY]
Decomposition of sulfometuron methyl under anaerobic aquatic conditions for 52 weeks in pond water/sediment systems.	Fallon 1989 MRID 42091402 [NEW STUDY]
Calculated $t_{1/2}$ values: 1.0 weeks: Landenberg PA (nonsterile sandy loam) 4.0 weeks: Landenberg PA (autoclaved sandy loam) 1.9 weeks: Bradenton FL (nonsterile sand) 21 weeks: Bradenton FL (autoclaved sand)	McCord 1994 MRID 43188601 [Suppl to Fallon 1989]
Major degradation products: nonsterile systems: free acid sulfometuron methyl and pyrimidine amine autoclaved systems: pyrimidine amine	
In autoclaved systems, hydrolysis occurred in the absence of significant microbial activity; in nonsterile systems, $\leq 0.3\%$ of the radiolabeled parent compound was evolved as $^{14}\text{CO}_2$ at 52 weeks, indicating little mineralization of the radiolabeled decomposition products.	
1 ppm ^{14}C -sulfometuron methyl applied to Landenberg PA and Brandenton FL pond water/sediment test systems and incubated aerobically at $\sim 25^\circ\text{C}$ for 30 or 39 days. pH = 7.6 in Landenberg pond water pH = 8.1 in Brandenton pond water microbial populations > 160 microorganisms/100 mL of pond water from both ponds.	Rhodes 1991 MRID 42091403 [NEW STUDY] Rhodes 1994 MRID 4317403
pH-dependent hydrolysis major route of degradation in aerobic aquatic environments with degradation proceeding more rapidly in the lower pH Landenberg test system ($t_{1/2} = 10$ days, compared with $t_{1/2} = 8$ months for Brandenton test system) significant contribution from microbial degradation degradation products included free acid sulfonamide, sulfonamide, saccharin, pyrimidine amine, and hydroxymethyl pyrimidine sulfometuron methyl.	[Suppl to Rhodes 1991]

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
PHOTOLYSIS	
Aqueous photodegradation of phenyl- ¹⁴ C(U)-sulfometuron methyl and [pyrimidine-2- ¹⁴ C-sulfometuron methyl under simulated sunlight at an initial concentration of approximately 3-5 ppm in sterile pH 5, 7, and 9 buffered solutions. Test solutions exposed to continuous light at a constant temperature of approx. 24°C for 15 days.	Schneiders and Bachmura 1992 MRID 42182401 [NEW STUDY]
Hydrolysis, not photolysis, major degradation route for aqueous systems exposed to light.	Schneiders 1994 MRID 43174101
Degradation rate is pH dependent and follows first order kinetics. pH 5 avg half-life = 8.0-8.4 days pH 7 avg half-life = 142 days pH 9 avg half-life = 128 days.	[Suppl to Schneiders and Bachmura 1992]
Degradation products pH 5 : sulfonamide and pyrimidine amine pH 7 and pH 9: saccharin, pyrimidine urea, and pyrimidine amine.	
Hydrolysis	
Hydrolysis of methyl ester to saccharin. Stable at pH 7 and 9 for 30 days. $t_{1/2}$ of 2 weeks at pH 5.	Harvey et al. 19?? MRID 00071419
	Harvey 1990b MRID 93206022
5 ppm in distilled water. UV Hydrolysis: $t_{1/2}$ of 1-3 days.	Harvey et al. 1980 MRID 00071420
5 ppm in dark sterile buffers pH 5: $t_{1/2}$ 14 days pH 7: 87% remaining at 30 days pH 9: $t_{1/2}$ 92% remaining at 30 days	Brattsten 1987 MRID 41672811
cleavage of sulfonylurea bridge.	
Hydrolysis in sterile, buffered, aqueous solutions at pH 5, 7, and 9 at conc. of 3-5 ppm. First order pH 5: $t_{1/2}$ 8.4 days pH 7: $t_{1/2}$ 113 days pH 9: $t_{1/2}$ 134 days	Schneiders 1993 MRID 42715201
Deg products: pH 5: sulfonamide and pyrimidine amine pH 7 and 9: saccharin and pyrimidine amine	

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
Hydrolysis in sterile and non-sterile soil. Unlike many studies, uses ¹⁴ C-pyrimidine ring labeled SM. Main metabolite 2-amino-4,6-dimethylpyrimidene. [This may be more chemical/hydrolysis than microbial]. Studies relationship of temp to rates.	Cambon et al. 1992
Hydrolysis in aqueous buffer solutions at pH 5, 7, and 9 at concentration of 6 ppm. pH 5: t _{1/2} 13.2 days pH 7: t _{1/2} 43.6 days pH 9: t _{1/2} 60.3 days	Naidu 1990 MRID 41672812 [NEW STUDY]
Degradation products: pH 5: ??? (not specified?) pH 7: pyrimidine amine pH 9: pyrimidine amine	
Bioconcentration	
In bluegill sunfish exposed to 0.01 and 1.0 ppm ¹⁴ C-phenyl-DPX-5648 for 28 days and investigated for accumulation in edible tissue, viscera and liver. In edible tissue : At 0.1 ppm, bioconcentration factor (BCF) after 24 days hours was 0.1 and highest BCF was 0.6 after 21 days of exposure. At 1.0 ppm, BCF after 24 hours was 0.01 and the highest BCF was 0.07 after 10 days of exposure. In liver : At 0.1 ppm, BCF after 24 hours was 0.04 and highest BCF was 1.8 after 7 days of exposure. At 1.0 ppm, BCF after 24 hours was <0.01 and highest BCF was 1.6 after 7 days of exposure. In viscera : At 0.1 ppm, BCF after 24 hours was 0.1 and highest BCF for chronic exposure was 0.7 on exposure days 21 and 28. At 1.0 ppm, BCF after 24 hours was 0.09 and highest BCF was 0.4 after 7 days of exposure.	Harvey 1981a MRID 00146279 Harvey 1990a MRID 93206028
In channel catfish exposed to a water/sediment system with water concentrations ranging from 0.01 to 0.02 ppm ¹⁴ C-phenyl-DPX-564 and investigated for accumulation in edible tissue, viscera and liver. Based on water concentrations: In edible tissue : after 24 hours, BCF was 3 and the highest BCF was 7 after 21 days of exposure. In liver : after 24 hours, BCF was 5 and the highest BCF was 44 after 21 days of exposure. In viscera : after 24 hours, BCF was 3.5 and the highest BCF was 6 after 28 days of exposure.	
K_{ow}	
K _{ow} , octanol/water partition coefficient pH 5: 11 pH 7: 0.346 pH 9: 0.0136 K _{ow} decrease as pH increase because of increasing ionization of SM (pKa 5.3)	Cadwgan 1990a MRID 93206001

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
<p>K_{ow} (\underline{n}-octanol/water partition coefficient) at two concentrations</p> <p>at high concentration (2.50 mL)</p> <p>pH 5 = 10.2</p> <p>pH 7 = 0.345</p> <p>pH 9 = 0.0134</p> <p>at low concentration (0.250 mL)</p> <p>pH 5 = 11.8</p> <p>pH 7 = 0.346</p> <p>pH 9 = 0.0138</p> <p>results indicate that bioaccumulation potential of sulfometuron methyl in living tissues or other organic matter is low.</p>	<p>Stevenson 1988 MRID 41273601 [NEW STUDY]</p>
<p>Soil Degradation/Transport</p> <p>Soil, Keyport silt loam, 0.12 ppm [120 g/ha] Soil $t_{1/2}$ of about 1 month.</p>	<p>Anderson 1980 MRID 00078701</p>
	<p>Anderson 1990b MRID 93206024.</p>
	<p>Anderson 1994 MRID 43174102 Supplemental note responding to U.S. EPA questions.</p>
<p>^{14}C-SM, Keyport silt loam, 70% NMHC (normal moisture holding capacity), 25°C. 50% of ^{14}C converted to CO_2 after 21 weeks (Fig2). Half-life of parent in soil about 4 weeks at 0.14 ppm or 1.3 ppm. No mineralization in sterile flasks. In sterile flasks, disappearance of parent compound was comparable to non-sterile flasks at 1.3 ppm after 24 weeks (8%) but less so at 0.14 ppm (12% vs 8%). ^{14}C-saccharin was major non-volatile deg product. Over time, unextractable soil residues increased. In soil with 20, 50, or 90% SMHC, more degradation at higher higher moisture levels (Table 5).</p>	<p>Anderson and Dulka 1985</p>
<p>aerobic soil degradation. Keyport silt loam, sterile and nonsterile. 'complete' degradation after 1 year. <i>non-sterile</i> biphasic: $t_{1/2}$ 17 days and 96 days. pyrimidine amine, CO_2, residues incorporated in fulvic and humic acids and inol. humin fractions. <i>sterile</i> $t_{1/2}$ 53 days.</p>	<p>Monson and Hoffman 1990 MRID 42091401</p>

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference															
<p>SM and metabolites from phenyl portion of molecule are mobile in most soils. More so in sandy vs loamy soils and less so in high organic matter soils. As soil pH decreases below 6, SM is protonated and thus less water soluble and less mobile. [Units not specified]</p>	<p>Cadwgan 1990b) MRID 93206026</p>															
<p>Batch equilibrium studies</p> <table border="1"> <thead> <tr> <th data-bbox="203 483 251 504">Soil</th> <th data-bbox="584 483 617 504">K_d</th> <th data-bbox="868 483 917 504">K_{om}</th> </tr> </thead> <tbody> <tr> <td data-bbox="203 514 446 535">Fallsington sandy loam</td> <td data-bbox="584 514 633 535">0.71</td> <td data-bbox="868 514 901 535">51</td> </tr> <tr> <td data-bbox="203 546 389 567">Keyport silt loam</td> <td data-bbox="487 546 535 567">0.97</td> <td data-bbox="771 546 803 567">35</td> </tr> <tr> <td data-bbox="203 577 349 598">Myakka sand</td> <td data-bbox="584 577 625 598">1.0</td> <td data-bbox="868 577 901 598">41</td> </tr> <tr> <td data-bbox="203 609 397 630">Flanagan silt loam</td> <td data-bbox="584 609 633 630">2.85</td> <td data-bbox="868 609 901 630">71</td> </tr> </tbody> </table>	Soil	K _d	K _{om}	Fallsington sandy loam	0.71	51	Keyport silt loam	0.97	35	Myakka sand	1.0	41	Flanagan silt loam	2.85	71	
Soil	K _d	K _{om}														
Fallsington sandy loam	0.71	51														
Keyport silt loam	0.97	35														
Myakka sand	1.0	41														
Flanagan silt loam	2.85	71														
<p>Abstract with K_d values. 0.29. Not very detailed.</p>	<p>Dickens and Wehtje 1986</p>															
<p>K_d values: 0.04-0.6 kg/L at 0-20 cm K_d values: 0.019-0.036 kg/L at 65-95 cm Once herb leaches past the top 10 cm of soil, retardation of herbicides would be slight. Contamination of ground water would depend on rate of decomposition.</p>	<p>Koskinen et al. 1996</p>															
<p>Composition of soils determined, but soil types not classified</p>																
<p>K_d values [units not specified]: sandy clay loam = 0.27; loamy, sand = 0.23; clay loam = 0.68; clay = 0.12 and 0.17</p>	<p>Wehtje et al. 1987</p>															
<p>K_fs/mobility high <i>lots of details</i>.</p>																
<p>Distribution coefficients (K_d values) for leached compounds in Keyport silt loam soil were: 0.121 mL/g (phenyl-labeled compound) 0.114 mL/g (pyrimidine-labeled compound)</p>	<p>Ryan and Atkins 1986 MRID 41680103</p>															
<p>low K_d values indicate that sulfometuron methyl and its eluted metabolites were mobile on water-saturated Keyport silt loam soil.</p>																
<p>lysimeters, various soil types, SM at 42.5 g a.i./ha. Mean concentration in soil water: 0.5 µg/L at 10 cm and 0.4 µg/L at 20 cm. Nothing at 40 or 150 cm. p. 401: 'By 80 d post-treatment, the ¹⁴C- activity was new background level, suggesting that most of the compound had been degraded or irreversibly sorbed into the upper soil layers.' Rainwater acidity had not effect on leaching rate in acid sand soils. Not effected by litter humus.</p>	<p>Stone et al. 1993</p>															
<p>soil adsorption study. for SM, poor correlation with organic matter (r²=0.271) but a better correlation humic matter (r²=0.729) [see Fig. 3, p. 1991.] K_d values ranging from <0.05 at <1% HM to 5-6 with >2% HM.</p>	<p>Strek et al. 1990</p>															

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
<p>Field simulation study on percolation and runoff with comparisons to GLEAMS modeling. Application rate of 0.6 kg/ha. Little initial runoff. Generally <1 µg/ml with max of 2.3 µg/ml. Mostly lost from upper root zone by percolation. Rainfall on sandy soil may move most out of 0.1 m of soil quickly. Much slower percolation on clay soil - runoff will be more significant. GLEAMS modeling qualitatively similar but some quant. differences.</p>	Hubbard et al. 1989
<p>Field simulation study. 0.4 kg/ha to 1.2x2.4 m plots. After 24 hrs, simulated rainfall of 69mm/h until 2 mm runoff occurred. 1-2% lost by in runoff regardless of grass cover. Runoff conc.: 0.2-0.5 mg/L max and 0.2-0.09 mg/L mean. [see Table 4, p. 123 for additional details.] Excellent correlations with GLEAMS.</p>	Wauchope et al. 1990
<p>Soil column leaching study to determine mobility of 2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (DPX-5648) in four soils: Keyport silt loam, Fallsington sandy loam, Myakka sand, and Flanagan silt loam.</p>	Dulka (1981) MRID 00078702
<p>Results indicate that DPX-5648 is mobile in sand, sandy loam, and silt loam soils. DPX-5648 shown to be least mobile in soil containing darkly-colored organic matter like Flanagan silt loam and Myakka sand.</p>	
<p>Field study to determine residual levels of methyl 2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (DPX-5648) on soils in Delaware, Mississippi, and North Carolina.</p>	Anderson 1980 MRID 00078705
<p>Residue analysis indicates a rapid breakdown of the intact herbicide under field conditions. In Delaware and Mississippi soils, <5% of intact DPX-5648 remained after 8 weeks;</p>	
<p>Preliminary data from a field simulation study indicates that methyl 2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (DPX-5648) applied at rates of 100-400 g/ha decomposes rapidly in soil with a half time of ≤1 month.</p>	Dulka 1980a MRID 00078706
<p>Major metabolites included 2-(aminosulfonyl) benzoic acid and saccharin.</p>	
<p>[2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl] benzoate, DPX-T5648] applied at 0.2 or 1 ppm to Keyport silt loam, Flanagan silt loam, and Fallsington sandy loam for 1 month exposure to simulated sunlight.</p>	Hardesty 1983 MRID 00137864
<p>>50% degradation of parent compound in 1-2 weeks at each concentration on every soil type</p>	
<p>saccharin was the major degradation product in all treated soil</p>	

Appendix 2: Laboratory and simulation studies on environmental sulfometuron methyl.

Data Summary	Reference
Photodegradation of 2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (sulfometuron methyl) on irradiated or non-irradiated Keyport silt loam for 33 days	Ryan and Atkins 1986 MRID 41420601
t _{1/2} on irradiated soil = 23 days t _{1/2} on irradiated soil = 34 days	Ryan and Atkins 1987
results indicate that photodegradation on soil is not a major decomposition pathway.	MRID 42385707
Identified degradates include sulfonamide, saccharin, and pyrimidine amine, resulting from sulfonylurea bridge cleavage (major aerobic soil metabolism pathway). Aggregate total of minor unidentified degradation products <10.1% only in the sunlight irradiate soil samples.	[The is a supplement to Ryan and Atkins 1986]
Six Brazilian (tropical) soils: K _d values: 0.13-1.18 K _{oc} values: 16-50	Oliveira et al. 2001
This study does not seem especially relevant to our RA in that the objective is to evaluate the compounds for their leaching potential to ground-water in tropical soil.	

Appendix 3: Field Studies on the environmental fate of sulfometuron methyl

Application/Field Conditions	Results	Reference
5 sites (Delaware (1.1 kg/ha), NC (0.91 kg/ha), OR (0.44 kg/ha), Colorado (0.15 kg/hr), and Saskatchewan (0.11 kg/ha). Different times of year.	See Figures 4 through 8, p 601. In eastern soils, about 1% of present after 1 year. In OR and CO soils, 6-12%. In SK, 16% at 43 weeks. After two years, 3% and 5% in OR and SK soils [others not measured.] 9% in CO soil at 78 weeks. All of these measurements refer to parent SM. Eastern soils (4.9-6.4 were more acidic than western soils (5.3-7.4). See Table 1 for other differences.	Anderson and Dulka 1985
lateral soil transport	very little lateral transport at slopes of up to 15% after 1 year. SM moved beyond soil column (70Cm) Detected after >400 d.	Lym and Swenson 1991
0.4 kg/ha as either dispersible granules or pellets in Mississippi (clay) (broadcast aerial) or Florida (sand) (broadcast ground) as dispersible granules (DG) or pellets (P).	Levels in surface water: 23 (P) and 44 (DG) µg/L in Miss. and 5 (P) and 7 (DG) µg/L in Florida. [Pellets were an experimental formulation.] Halftime in soil 5-33 days, in plants 4-11 days	Michael and Neary 1993 additional details in Neary and Michael 1989 data are also in Neary and Michael 1996
see above, FLA	SM not detected in any sediment samples from treated or control waterbodies (?? Limit of Det 1 mg/m ³ [1 mg/1000L or 0.001 mg/L] for water and 0.020 mg/kg for sediment. p. 619) Rain 24 hrs after applic and again 3 days later, 54 mm. Streamflow did not begin until 20 days after treatment. Detected in only 10/185 samples.	Neary and Michael 1989
0, 0.212, and 0.424 g/ha at five sites in Coastal Plain of Georgia. Soil pH 4.8-6.5.	No increase in loblolly pine seedling mortality (Table 2, p. 307) but a marked increase in plants with signs of phytotoxicity (Table 3, p. 308, about 5-73% at low and 20-88% at high rate). Least damage at pH 4.8. Others seem comparable. [Could use for d/r curves but only 2 dose points, 1 d.f.]	Mitchell et al. 1991
Nominal concentrations of 0.10, 0.18, 0.44, or 0.64 µg/mL of ¹⁴ C-sulfometuron methyl on Chino sandy loam, Fargo silt loam, Miaka sand, and Tama silt loam soil.	Weakly to moderately adsorbed on all soils (K _a >0.15-2.1); strong positive correlation between organic matter and K _a ; adsorbed radioactivity was poorly to readily desorbed from all soil types.	Kraut 1993 MRID 42789301

Appendix 3: Field Studies on the environmental fate of sulfometuron methyl

Application/Field Conditions	Results	Reference
Nominal concentrations of 0.10, 0.18, 0.44, or 0.64 µg/mL of INX-X993 (¹⁴ C-pyrimidine amine) on Chino sandy loam, Fargo silt loam, Miaka sand, and Tama silt loam soil.	Weakly to moderately adsorbed on all soils ($K_a > 0.17-3.7$); weak positive correlation between organic matter and K_a ; adsorbed radioactivity was poorly to readily desorbed from all soil types.	Kraut 1993 MRID 42789301
Nominal concentrations of 0.10, 0.18, 0.44, or 0.64 µg/mL of IN-D5803 (¹⁴ C-sulfonamide) on Chino sandy loam, Fargo silt loam, Miaka sand, and Tama silt loam soil.	Decomposes rapidly on to ¹⁴ C-saccharin; consequently, soil desorption could not be characterized further.	Kraut 1993 MRID 42789301
Nominal concentrations of 0.10, 0.18, 0.44, or 0.64 µg/mL of IN-581 (¹⁴ C-saccharin) on Chino sandy loam, Fargo silt loam, Miaka sand, and Tama silt loam soil.	Weakly adsorbed on all soils ($K_a > 0.033-0.27$); poor correlation between organic matter and K_a ; adsorbed radioactivity readily desorbed from all soil types.	Kraut 1993 MRID 42789301
Single soil-directed spray application of DPX-T5648 (Oust) at 9.0 a.i./acre to bare ground at four sites: Greenville, MS, Rochelle, IL, Uvalde TX, and Madera CA. Soils (0-15 cm level) characterized as silty, clay loam, clay, or sandy loam.	The results of the study indicate that sulfometuron methyl dissipates rapidly (generally to ≤ 0.5 ppb after 1 year); the total concentration of metabolite residues (sulfonamide, pyrimidine amine, and saccharin) decreases considerably after 1 year; and sulfometuron methyl and its soil metabolites are immobile.	Trubey 1994b MRID 43212101
Single, soil-directed application of Oust herbicide (formulated as a dispersible granule containing 75% sulfometuron methyl) at rate of 630 g a.i./ha (equivalent to 9.0 oz a.i./acre) at four test sites: Greenville, MS, Rochelle, IL, Uvalde TX, and Madera CA.	Calculated $t_{1/2}$ values ranged from 12-25 days. Sulfometuron methyl residues were below the limit of quantitation (10 ppb) 90 days after treatment at all test sites. The highest concentration of degradation product (2-amino-4,6-dimethylpyrimidine) 40 ppb at the end of the study (day 359 after treatment). Sulfometuron methyl was determined to be immobile (i.e., confined to upper soil depth 0-15 cm) at all field sites throughout the course of the study.	Trubey et al. 1998

Appendix 4: Toxicity of sulfometuron methyl to experimental birds.

Animal	Dose	Response	Reference
Ducks, Mallard, 16 days old at start, 10 per dose	0, 156, 312, 625, 1250, 2500, and 5000 ppm in diet for 9 days. [0, 10.3, 19.5, 39.7, 74.4, 141.3, and 332.5 mg/kg bw, as calculated by the authors based on measured food consumption.]	No mortality. No effects on body weight or food consumption. NOAEL for mortality and toxicity = 5000 ppm	Dudeck and Twigg 1980 MRID 00071414 also summarized by Summers 1990i MRID 93206004
Ducks, Mallard, approximately 9 months old, 5 per dose per sex.	Single gavage doses in carboxymethylcellulose/distilled water: Vehicle, 312, 625, 1250, 2500, and 5000 mg/kg bw. 14 day observation period.	No mortality or signs of toxicity. In males, decreased weight gain at doses of 625 mg/kg and higher. Magnitude of decrease was not dose/related. No consistent effect of body weight in females. NOAEL for decreased weight gain (males only) = 312 mg/kg/day	Dudeck and Bristol 1981a MRID 00078700 Results also reported in Summers 1990j MRID 63206002
Quail, Bobwhite, 15 days old at start, 10 per dose	0, 156, 312, 625, 1250, 2500, and 5000 ppm in diet for 9 days. [0, 1.19, 2.81, 5.00, 9.23, 18.75, and 37.5 mg/kg bw based on measured food consumption.]	Mortality in 5 animals in control group and 1 animal each in the 156, 312, and 2500 ppm dose groups. Lethargy in two animals in the 1250 dose group on observation days 6 and 8. No dose related changes in body weight.	Dudeck and Bristol 1981b MRID 00071415

Appendix 4: Toxicity of sulfometuron methyl to experimental birds.

Animal	Dose	Response	Reference
Quail, Bobwhite, 14 days old, males and females randomly assigned to dose groups, 10 per dose.	Dietary concentrations of 562, 1000, 1780, 2160, and 5620 ppm for 5 days. Dieldrin used as positive control. Based on the an average body weight of 36.7 g, an average food consumption of 6.7 g/day/bird, the average fractional weight of food consumption per bird = 0.19. Thus, multiplying the concentration in food by 0.19, average daily doses are calculated as 107, 190, 338, 410, and 1068 mg/kg/day	No mortality, overt signs of toxicity, or differences in body weight gain. NOAEL for mortality and toxicity = 5260 ppm	Fink et al. 1981 MRID 00088813 also summarized in Summers 1990k MRID 93206003

Appendix 5: Bioassays of sulfometuron methyl toxicity in terrestrial plants.

Plant	Exposure	Response	Reference															
DIRECT SPRAY																		
Loblolly Pine	greenhouse study. Rates of 0.1, 0.21, and 0.42 kg/ha both foliar and soil as well as combined in fine sandy loam and unclassified loam. No substantial differences in soil types of application methods, so results are combined.	<table border="1"> <thead> <tr> <th>Rate</th> <th>Root length</th> <th># new roots</th> </tr> </thead> <tbody> <tr> <td>0</td> <td>35.0</td> <td>27.8</td> </tr> <tr> <td>0.10</td> <td>20.4</td> <td>18.8</td> </tr> <tr> <td>0.21</td> <td>16.2,</td> <td>15.0</td> </tr> <tr> <td>0.42</td> <td>12.5</td> <td>12.0</td> </tr> </tbody> </table>	Rate	Root length	# new roots	0	35.0	27.8	0.10	20.4	18.8	0.21	16.2,	15.0	0.42	12.5	12.0	Barnes et al. 1990
Rate	Root length	# new roots																
0	35.0	27.8																
0.10	20.4	18.8																
0.21	16.2,	15.0																
0.42	12.5	12.0																
	field application 0.30 kg/ha	inhibition over initial 45 days as in greenhouse study. by end of growing season, biomass accumulation was greater in treated plants because of control of competing weeds.																
leafy spurge	0.105 to 1.12 kg/ha	ineffective control. when combined with auxin herbicides, control was effective.	Beck et al. 1993															
Turnips (<i>Brassica rapa</i>), plant selected because of its sensitivity.	pots, 10 days. four different soils (see table 2, p. 143 for differences in soils). greenhouse study. 10 conc from 0.01-40 µg/kg	<table border="1"> <thead> <tr> <th>EC₅₀ (µg/kg with 95% conf. inter) for growth inhibition in different soils:</th> <th></th> </tr> </thead> <tbody> <tr> <td>Vermiculite:</td> <td>0.12±0.03</td> </tr> <tr> <td>BBA</td> <td>0.19±0.04</td> </tr> <tr> <td>Wendhausen</td> <td>0.17±0.04</td> </tr> <tr> <td>Horotiu</td> <td>0.47±0.16</td> </tr> </tbody> </table>	EC ₅₀ (µg/kg with 95% conf. inter) for growth inhibition in different soils:		Vermiculite:	0.12±0.03	BBA	0.19±0.04	Wendhausen	0.17±0.04	Horotiu	0.47±0.16	Gunther et al. 1989					
EC ₅₀ (µg/kg with 95% conf. inter) for growth inhibition in different soils:																		
Vermiculite:	0.12±0.03																	
BBA	0.19±0.04																	
Wendhausen	0.17±0.04																	
Horotiu	0.47±0.16																	
white mustard, 3 weeks post-emergence [6 true leaves, 50 mm high]	0.25 g/ha, simulated rainfall at 0.5, 1, and 2 hours after treatment. observation at 3 weeks after treatment.	about a 75% reduction in growth relative to controls with 2 hour rainfall. A 64% reduction with 0.5 or 1 hour rainfall. Various adjuvants had minor to moderate effects on 0.5 hour rainfall.	James and Rahman 1992															

Appendix 5: Bioassays of sulfometuron methyl toxicity in terrestrial plants.

Plant	Exposure	Response	Reference
Corn, cucumber, onion, pea, rape, sugar beet, sorghum, soybean, tomato, wheat	Pre- and Post- emergence assays. Oust Herbicide (60% dry flowable) – maximum application rate. Lowest application rates varied with species. The lowest rates used was with tomato – i.e., 0.0000271 oz ai/acre) in pre-emergence assay and 0.000195 oz/acre in post-emergence assay.	Conversion notes: 2.4 oz/acre = 0.5625 lb/acre. 1 oz/acre = 0.0625 lb/acre. Pre-emergence assay: The most sensitive species based on the NOEC, were rape, tomato, sorghum, wheat, and corn with an NOEC of 0.000137 oz ai/acre (0.000086 lb ai/acre). The most tolerant species based on the NOEC were onion, pea, cucumber, and soybean with an NOEC of 0.00412 oz ai/acre (0.00026 lb/acre). [Data from p. 12 of study.] Post-emergence assay: The most sensitive species based on the NOEC is corn with an NOEC of 0.000391 oz ai/acre (0.000024 lb ai/acre). The most tolerant species based on the NOEC is pea with an NOEC of 0.0125 oz ai/acre (0.00078 lb ai/acre). [Data from p. 13 of study.]	McKelvey 1995 MRID 43538501
SUSPENSIONS			
Soybean cells	suspension	EC ₅₀ for growth: 62 µg/L	Scheel and Casida 1985

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
FRESH WATER SPECIES			
FISH			
Minnow, Fathead, 6 weeks old, 10 per dose group.	0.75 to 12.5 mg/L nominal; 0.6 to 7.3 mg/L measured average concentration. 96 hour exposure and observation period.	No mortality. No signs of toxicity were reported. 96-hour LC ₅₀ > 7.3 mg/L NOEC (mortality and toxicity) = 7.3 mg/L	Muska and Driscoll 1982 MRID 00126600
Minnow, Fathead, embryos and larvae	0, DMF ¹ control, 0.15, 0.3, 0.6, 1.2, and 2.5 mg/L [nominal] for 30 days post-hatch. Mean measured concentrations in exposed groups were 0.06, 0.14, 0.32, 0.65, and 1.17 mg/L.	No significant effects on embryo hatch or larval survival or growth. NOEC for all effects = 1.17 mg/L	All appear to be the same study Muska and Driscoll 1982 MRID 00126600 Driscoll 1984 MRID 00143539 Comments by Summers 1990l MRID 42385704 Summarized by Summers 1990m MRID 93206007
Sunfish, Bluegill, 3.4 cm mean length, 0.99 g mean weight, 10 animals per concentration.	0, DMF ¹ Control, 0.125, 1.25, 12.5 ppm for 96 hours, static, no aeration. DMF ¹ used for stock solution because of poor solubility of test material.	1 of 10 fish at 1.25 ppm died by 48 hours. No mortality in other groups. No signs of toxicity reported. 96-hour LC ₅₀ > 12.5 ppm NOEC (mortality and toxicity) = 12.5 ppm Summers Comment: Problems with solubility and use of DMF as vehicle.	Muska and Hall 1980 MRID 00071417 Comments by Summers 1990n MRID 42385701 summarized by Summers 1990o MRID 93206005

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
Sunfish, Bluegill, 1.5-2.6 cm mean length, 0.07-0.42 g mean weight, 30 animals per concentration.	0 and 150 mg/L, pH adjusted and unadjusted, aeration. [nominal conc. was verified by analysis.]	No mortality or sublethal signs of toxicity. LC ₅₀ > 150 mg/L NOEC (mortality and toxicity) = 150 mg/L	Brown 1994a MRID 43501801 [this was missing from fiche and fax by U.S. EPA]
Trout, Rainbow, 4.3 cm mean length and 1.27 g mean weight, 10 animals per concentration.	0, DMF ¹ Control, 0.125, 1.25, 12.5 ppm for 96 hours, static, no aeration. DMF ¹ used for stock solution because of poor solubility of test material.	No mortality in any groups. No signs of toxicity. 96-hour LC ₅₀ > 12.5 ppm NOEC (mortality and toxicity) = 12.5 ppm Summers Comment: Problems with solubility and used of DMF as vehicle.	Muska and Trivits 1980b MRID 00071416 Comments by Summers 1990p MRID 42385702 Summarized by Summers 1990q MRID 93206006
Trout, Rainbow, fingerlings, 32. to 4.8 cm, 0.47 to 1.79 g, 15 animals per replicate, 2 replicates per concentration.	148 mg/L adjusted to pH 9 to ensure solubility. Duration of 96 hours with observations at 24, 48, 72, and 96 hours. Static, no aeration. Used unadjusted water control and pH 9 adjusted water control.	No mortality in any groups. 96-hour LC ₅₀ > 148 mg/L NOEC (mortality and toxicity) = 148 mg/L	Brown 1994b MRID 43501802
AQUATIC INVERTEBRATES			
Daphnia magna, <24 hours old, 2 replicates per concentration, 10 animals per replicate	0, DMF ¹ Control, 0.125, 1.25, 12.5 ppm for 48 hours, static, no aeration. DMF ¹ used for stock solution because of poor solubility of test material.	No mortality in exposed groups except for 1 animal at 0.125 ppm. One animal also died in DMF control. No signs of acute toxicity observed. 48-hour LC ₅₀ > 12.5 mg/L NOEC (mortality and toxicity) = 12.5 mg/L	Muska and Trivits 1980a MRID 00071418 Comments by Summers 1990r MRID 42385703 Summarized by Summers 1990s MRID 93206007

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
Daphnia magna, <24 hours old, 2 replicates per concentration, 10 animals per replicate	Nominal concentrations ranging from 1000 mg/L to 10000 mg Oust/L. Exposure period was 48 hours	48-hour LC ₅₀ = 8500 (CI 6500-12200) mg Oust DF/L. In terms of a.i., LC ₅₀ = 6375 mg a.i./L)	Wetzel 1984 MRID 00145514
<i>Oust Dispersible Granule (75 DF)</i>		NOAEC no mortality or toxicity = 2400 Oust mg/L (1800 mg a.i./L). LOAEC = 3200 mg Oust/L (2400 mg a.i./L)	
Daphnia magna, seven replicates with 1 adult per replicate and 3 replicates with 5 adults/replicate per exposure level.	Nominal concentrations of 0.1, 0.39, 1.6, 6.3, 25, and 100 mg/L. Mean measured concentrations of 0.076, 0.4, 1.5, 6.1, 24, and 97 mg/L. Exposure period was 21 days.	Number of neonates per surviving adult significantly reduced at 24 mg/L but not at 97 mg/L or any of the other lower concentrations. No significant effect on adult survival or length at any concentration.	Baer 1990 MRID 41672806
		Although there was no clear dose-response effect, the authors give an NOEC for reproductive effects = 6.1 mg/L	
Daphnia magna, <24 hours old, 8 animals per replicate, 4 replicates per concentration.	Unadjusted water, pH 9 adjusted water, and 150 mg a.i./L for 48 hours. [150 mg/L was both nominal and measured value.]	No effects in any test animals exposed to SM. 48-hour LC ₅₀ > 150 mg/L NOEC (mortality and toxicity) = 150 mg/L 1/32 test animals in pH adjusted water was immobile at 48 hours.	Brown 1994b MRID 43501803

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
<p>Four field collected species, 48 duration, no carrier, OUST, acclimated for 96 hrs, pH 8.0-8.5</p> <p>Although not specified, it appears that all values are given in terms of the formulation, not a.i.</p>	<p>Group Diaptomus sp. Eucyclops sp. Alonella sp. Cypria sp.</p>	<p>LC₅₀: Diaptomus sp: 1315 mgOust/L [986 mg a.i./L] Eucyclops sp: 1230 mg/L [922 mg a.i./L] Alonella sp: 802 mg/L [601 mg a.i./L] Cypria sp: 2241 mg/L [1680 mg a.i./L] [see Table on p. 390 for d/r data from 100 to 2500 mg/L.]</p> <p>LOEC: Alonella sp: 100 Oust mg/L [75 mg a.i./L] Cypria sp: 100 mg/L [75 mg a.i./L]</p>	<p>Naqvi and Hawkins 1989.</p>
<p>Crayfish, juvenile, <i>Procambarus clarkii</i>, 3-3.4 cm, 1.1-1.5 g) collected, OUST</p>	<p>Acclimated for 96 hrs., exposure period of 24 hrs., pH 6.8±0.1.</p>	<p>LC₅₀ 12,174 mg Oust /L (11,980-12,359) [9130 mg a.i./L]</p> <p>Although not specified, it appears that LC₅₀ values are given in terms of the formulation, not a.i.</p>	<p>Naqvi et al. 1987</p>
<p>ALGAE</p>			
<p>Freshwater Algae, <i>Senenstrum capricornutum</i></p>	<p>0.63, 1.3, 2.5, 5.0, 10, 20 µg/L for 120 hours.</p>	<p>EC₅₀ 4.6(2.6-8.2) µg a.i./L for reduction in cell density relative to controls. [See Table 2 for details. Looks like stimulation of growth at 0.63 µg/L at 120 hours. Some stimulation at higher conc. - up to 2.5 µg/L at 72 hours.]</p>	<p>Hoberg 1990 MRID 41680102</p>

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
Anabaen flos-aquae, freshwater algae	for Navicula pelliculosa: Nominal concentrations of 13, 25, 50, 100, and 200 µg/L for 120 hours.	Results in Anabaen flos-aquae: EC ₂₅ for Cell Density 17 (8.8-76) µg/L EC ₅₀ for Cell Density 65 (31-93) µg/L	Thompson 1994 MRID 43538502
Navicula pelliculosa, freshwater diatom	For Navicula pelliculosa: 370 µg a.i./L – range finding experiment	EC ₅₀ for Growth Rate 167 (157-182) µg/L Results in Navicula pelliculosa: no inhibition observed	
3 strains of Chlorella (1-wildtype and 2 mutant strains)	0.3 µM [~110 µg/L]	estimated EC ₅₀ for growth inhibition = 0.25 µM (91 µg/L).	Landstein et al. 1993
Macrophytes			
<i>Hydrilla verticillata</i> (water thyme), aquatic angiosperm, rooted aquatic plant - see description on rational for using on p. 509	OUST 1 µg/L to 1000 µg/L for 7 days. Unclear whether results are expressed in terms of µg OUST/L or µg a.i./L. Assumption that values are expressed in terms of mg Oust/L.	see Fig. 3 p. 512, growth and peroxidase activity. Eye-fit on EC ₅₀ for growth of about 10 µg/L (7.5 µg a.i./L). NOEC = 1 µg Oust/L (0.75 µg a.i./L). Higher EC ₅₀ for induction of peroxidase activity.	Byl et al. 1994
<i>Lemna gibba</i> , (species of duckweed known as ‘fat duckweed;’) macrophyte	0, 0.13, 0.207, 0.323, 0.590, and 1.045 µg a.i./L for 14 days	FronD Counts EC ₂₅ 0.344(0.305-0.358) µg/L EC ₅₀ 0.462 (0.436-0.493) µg/L NOEC 0.207 µg/L Biomass EC ₂₅ 0.451 (0.360-0.534) µg/L EC ₅₀ 0.785 (0.663-0.982) µg/L NOEC 0.323 µg/L	Kannuck and Sloman 1995 MRID 43538503

Appendix 6: Toxicity to fish, aquatic invertebrates, and aquatic plants. [sulfometuron methyl technical unless specified as Oust or Oust XP].

Animal	Exposure	Response	Reference
SALT WATER SPECIES			
Minnow, Sheepshead, juvenile, 20 per level	0, 15, 25, 40, and 60 and 100 mg/L nominal. Measured averages of 0, 8.2, 14.4, 21.7, 29.8, and 45 mg/L. Static, unaerated. 99.1% purity.	No mortality in any group. Insoluble material observed in test chambers.	Ward and Boeri 1990a MRID 41672803
Eastern oyster, embryos and larvae, 2 treatment replicates per concentration and 4 replicates for control. Approx. 30,000 embryos per replicate.	Measured average concentrations of 0, 8.5, 13.9, 22.2, 27.8, and 38.2 mg/L for 48 hours. Unaerated, static.	No concentration related changes in number of animals or number of animals with abnormalities.	Ward and Boeri 1990b MRID 41672805
Shrimp, Mysid, <24 hours old, 20 per replicate, 2 replicates/dose	Measured mean concentrations of 0, 9.8, 15.6, 23.2, 31.5, and 44.8 mg/L for 96 hours. Unaerated, static.	No mortality. Insoluble material observed in test chambers.	Ward and Boeri 1990c MRID 41672804
Skeletonema costatum, marine diatom	410 µg a.i./L	-7.3% growth relative to controls	Thompson 1994 MRID 43538502
<i>Myriophyllum sibiricum</i>	Sulfometuron methyl (Oust) at 0.56 kg/ha (maximum label rate) 0.37 (expected environmental concentration) static exposure for 14 days. Results indicated as <i>inhibitory concentration</i> (IC)	Results indicate that rooted aquatic macrophytes are highly sensitive to sulfometuron methyl. Shoot growth: IC ₂₅ = 0.00016 mg a.e./L IC ₅₀ = 0.00037 mg a.e./L Root number: IC ₂₅ = 0.00028 mg a.e./L IC ₅₀ = 0.00039 mg a.e./L Root dry mass: IC ₂₅ = 0.00006 mg a.e./L IC ₅₀ = 0.00012 mg a.e./L	Roshon et al. 1999

¹ **SUMMERS (DuPont) COMMENT ON SOLUBILITY:** Because of its toxicity to aquatic species, DMF (dimethyl formamide) is not one of the EPA preferred solvents. The use of the solvent limits the test concentration since SEP limits the solvent to 0.5 ml/L. The pka of sulfometuron methyl is 5.2. Under unbuffered normal aquatic test conditions, the sol. of SM is < 12.5 ppm at both pH 5 and 7. Under highly buffered conditions, the sol. at pH 7 is 244 ppm at 25°C.

Appendix 7: Toxicity of sulfometuron methyl to amphibians

Animal	Exposure	Response	Reference
African Clawed frog (<i>Xenopus laevis</i>), blastulae stage embryos, 20/dose group	4-day organogenesis (tail resorption) test at concentrations of 0.001, 0.005, 0.01, 0.025, 0.05, 0.075, 0.1, 0.5, 1.0, or 10.0 mg/L sulfometuron methyl formulated as Oust	NOAEC = 0.5 mg/L (mortality and malformation) [Note: malformations were observed at 0.1 mg/L, but they were not statistically significant].	Fort 1998 MRID 44682601
		LOAEC = 1.0 mg/L (mortality and malformation 96-hour LC ₅₀ >10.0 mg/L 96-hour EC ₅₀ = 0.94 mg/L (confidence interval = 0.92-0.96 mg/L) Treatment-related malformations included miscoiling of the gut at concentrations >0.5 mg/L; malformations of the eye (incomplete lens formation) and abnormal craniofacial development at concentrations >1.0 mg/L.	Fort et al. 1999a [Published abstract of Fort et al. 1999b] Fort et al. 1999b [Appears to be identical to CBI study by Fort 1998]
African Clawed frog (<i>Xenopus laevis</i>), blastulae stage embryos, 20/dose group	30-day organogenesis/limb development with 96-hour renewal at concentrations of 0.001, 0.005, 0.01, 0.05, 0.1, 0.5, 1.0, or 10.0 mg/L sulfometuron methyl formulated as Oust	NOAEC = 0.01 mg/L	Fort 1998 MRID 44682601
		LOAEC = 0.05 mg/L Treatment-related malformations included selective reduction deficiencies distal to the femur.	Fort et al. 1999a [Published abstract of Fort et al. 1999b] Fort et al. 1999b [Appears to be identical to CBI study by Fort 1998]

Appendix 7: Toxicity of sulfometuron methyl to amphibians

Animal	Exposure	Response	Reference
African Clawed frog (<i>Xenopus laevis</i>), blastulae stage embryos, 20/dose group	14-day tail resorption (thyroid disruption) with 96-hour renewal at concentrations of 0.1, 1, 10, 100, or 1000 µg/L	NOAEC = 0.001 mg/L LOAEC = 0.01 mg/L Addition of 100 µg/L thyroxin partially reversed the inhibitory effects of sulfometuron methyl.	Fort 1998 MRID 44682601 Fort et al. 1999a [Published abstract of Fort et al. 1999b] Fort et al. 1999b [Appears to be identical to CBI study by Fort 1998]

Appendix 8. Effects of Sulfometuron methyl on microorganisms and microbial populations in soil

Application	Observations	Reference
<p>2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (DPX-5648) applied to Keyport silt loam or Fallsington sandy loam soil at 0.098 ppm or 0.98 ppm.</p>	<p>No significant effect on the rate of nitrate production in either Keyport silt loam or Fallsington sandy loam soil, compared with untreated controls.</p>	<p>Anderson and Berg 1980? MRID 00071421</p>
<p>2-[[[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl] amino]sulfonyl]-benzoate (DPX-5648) applied to fresh Fallsington sandy loam or Flanagan silt loam at 0.098 ppm or 0.98 ppm (oven dried soil basis).</p>	<p>No effect on cellulose, protein or starch metabolism.</p> <p>No treatment related effects were observed in the rate of evolution of ¹⁴CO₂ from soil to which was added ¹⁴C-cellulose as a source of microbial carbon.</p> <p>Recovery of ¹⁴C-algal protein as ¹⁴CO₂ was 31% in Flanagan silt loam and 34% in Fallsington sandy loam after 32 days in both treated and untreated soils.</p> <p>Recovery of ¹⁴C-starch as ¹⁴CO₂ was 55% in Flanagan silt loam and 35% in Fallsington sandy loam after 34 days in both treated and untreated soils.</p>	<p>Anderson and Berg 1980? MRID 00071421</p>
<p>2-[(4,6-dimethyl-2-pyrimidinyl) aminocarbonyl]amino]sulfonyl]-benzoic acid, methyl ester (DPX-5648) applied to Keyport silt loam, Sharkey clay oam, Renohill sandy loam, or Cheney silt loam at a concentration of 0, 1, 100, or 1000 µg/g.</p>	<p>No effects on soil microorganism populations during the 30-day period following treatment. No effect on the distribution of fungal genera recovered from treated soil.</p>	<p>Hadley et al. 19?? MRID 00071422</p>
<p>Yearly applications of OUST at rates of 56.7 and 113.4 g a.i./ha for 4 years to Christmas tree plantation sites</p>	<p>Except for one sample obtained during the 4-year period, Oust treated soil did not have a significantly lower total bacterial biomass compared to control soil. Sporadic increased in inorganic nitrogen observed in Oust treated fields. No sign of toxicity to trees</p>	<p>Arthur and Wang 1999</p>