APPENDIX C - WILDLIFE

Exposure Groups for Forest Service Sensitive Wildlife

Since exposure data do not exist for most individual wildlife species in the Region, the Forest Service Sensitive wildlife species evaluated in this BA were placed into exposure groups of similar relationship, body size, and food habits. Table C- 1 lists the exposure groups, the exposure scenarios, and the members of each group used for this analysis.

Table C- 1- Exposure groups, exposure scenarios, and species included in each group. Grouping various wildlife species facilitates calculation of estimated exposures to herbicides.

Exposure Group	Exposure Scenarios	Species Included**
Large Herbivore – Mammal	Consumption of 100% contaminated grass	Rocky Mountain bighorn sheep
Small Herbivore – Mammal	Consumption of 100% contaminated leaves and leafy vegetables Direct spray on 50% of body, complete absorption Consumption of water contaminated by an accidental spill.	(Western gray squirrel, pygmy rabbit, Western (Mazama) pocket gopher)*
Carnivore – Mammal	Consumption of an entire days diet of prey that has been directly sprayed on 50% of body surface	California wolverine, (Pacific fisher)
Sm. Insectivore – Mammal	Consumption of an entire day's diet of contaminated insects	(Pacific pallid bat, Townsend's big-eared bat, spotted bat, Pacific fringe-tailed bat, bats, Baird's shrews, Pacific shrews)
Herbivore – Bird	Consumption of 100% contaminated grass	(Western sage grouse1, sharp-tailed grouse, Columbian sharp-tailed grouse)
Insectivore – Bird	Consumption of an entire days diet of contaminated small insects using empirical relationships for residues in vegetation (no data available on concentrations of pesticides in insects)	gray flycatcher, green-tailed towhee, upland sandpiper,(black swift, ash-throated flycatcher, yellow-billed cuckoo, tricolored blackbird, bobolink, greater yellowlegs, yellow rail, bufflehead, harlequin duck)
Predatory Bird	Consumption of an entire day's diet of small mammal prey that has been directly sprayed	American peregrine falcon2, (northern goshawk, ferruginous hawk, great gray owl, greater sandhill crane)
Piscivorous Bird	Consumption of fish contaminated by an accidental spill	(common loon, Clark's grebe, eared grebe, red-necked grebe, horned grebe, least bittern)
Reptiles	None available. Information from literature is used.	striped whipsnake, painted turtle, (Sharptailed snake, California mountain kingsnake, common kingsnake, Northwestern pond turtle)
Amphibians	For sulfometuron methyl, used water concentrations from runoff and percolation estimates. For other herbicides, information from literature is used.	Columbia spotted frog, northern leopard frog, (California slender salamander, Oregon slender salamander, black salamander, Cope's giant salamander, Del Norte salamander, Larch Mountain salamander, Siskiyou Mountain salamander, Van Dyke's salamander, Cascade torrent salamander, Columbia torrent salamander, Olympic torrent salamander, southern torrent (seep) salamander, foothill yellow-legged frog, Oregon spotted frog)

* Sensitive wildlife species within parenthesis are not Umatilla National Forest sensitive species but are included as examples.
**Bolded sensitive species either have been documented or are suspected to occur on the Umatilla National Forest.
1 Most animals will eat more than one type of food. Species were placed in groups that represented the majority of their diet, or the type of diet that would pose the most risk.
2 No scenario is yet available for animals that feed primarily on birds, so exposures from mammal prey are used.

The general effects to wildlife from invasive plant treatments, and treatment and restoration standards are displayed inTable C- 1. For sensitive species, dose estimates for each exposure group were obtained from Forest Service/ SERA risk assessments or calculated in project file worksheets using the Forest Service /SERA exposure scenarios. The exposure estimates were then compared to wildlife toxicity indices. Results of exposure scenarios for birds and mammals are found below in Table C-2 and Table C-3.

When data is insufficient to estimate doses, information from literature is used to evaluate toxic effects. These doses and information from the literature are subsequently used to evaluate effects to the members of each exposure group in conjunction with diet, plausibility of exposure scenario, behavior, etc.

Scientific uncertainty exists in extrapolating laboratory data to specific species and wild conditions. Laboratory species, and soil/air conditions may not accurately reflect in situation scenarios. Herbicides considered in this BA have had comparatively little testing and analysis for amphibians and virtually no data exists for reptiles found in the Region. Also, data is insufficient to evaluate effects to predatory birds that eat primarily birds (i.e. American peregrine falcon), and ducks feeding primarily on aquatic insects (i.e. Harlequin ducks and bufflehead which are not present on the Forest). All these species need to be evaluated at the site-specific scale to determine the likelihood of exposure.

Effects of the Alternatives on Sensitive Wildlife

The invasive plant treatments and restoration projects were designed to reduce or eliminate adverse effects to sensitive species, as required in Treatment and Restoration Standard 22 for all alternatives; however, short-term, minor adverse effects (See individual species discussions) could occur under any alternative from the herbicide treatment methods. There may be some instances where it is most prudent to conduct a project that has a short-term adverse effect in order to provide a long-term beneficial effect to the habitat and Table C- 3display the different herbicides that may be used, with restrictions, in the action alternatives. The No Action Alternative, which continues treatment under the existing 1995 Umatilla EA and the 1994 Wallowa-Whitman EA, is limited to, Glyphosate or Picloram.

Dicamba was originally included in the list of approved herbicides for the Umatilla EA and the 1994 Wallowa-Whitman EA , but was removed from use by the R6 2005 ROD. The exposure scenarios were compiled from the FS and SERA risk assessment found in the R6 2005 FEIS.

Table C- 2. Exposure scenario results from FS/SERA risk assessments for mammals, birds, and honeybees using the typical application rate and upper residue rates.

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
ACUTE EXPOSURES											
Direct spray, bee	--	--	--	--	--	--	--	--	--	--	
Direct spray, sm. mammal	--	--	--	--	--	--	--	--	--	--	*
Consume Contaminated Vegetation											
small mammal	--	--	--	--	--	--	--	--	--	--	--
large mammal	--	--	--	--	--	--	--	--	--	--	*
large bird	--	--	--	--	--	--	--	--	--	*	*
Consume Contaminated Water											
Spill, sm. mammal	--	--	--	--	--	--	--	--	--	--	--

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
Consume Contaminated Insects											
small mammal	--	--	--	--	--	--	--	--	--	--	*
small bird	--	--	--	--	--	--	--	--	--	*	*
Consume Contaminated Prey											
carnivore (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--
CHRONIC EXPOSURES											
Consume Contaminated Vegetation											
small mammal, on site	--	--	--	--	--	--	--	--	--	--	--
lg. mammal, on site	--	--	--	--	--	--	--	--	--	*	--
lg. bird, on site	--	--	--	--	--	--	--	--	--	*	--
Consume Contaminated Water											
small mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects#											
small mammal	--	unk	--	--	--	--	unk	unk	unk	unk	unk
small bird	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
Consume Contaminated Prey											
carnivore (sm. mammal)#	--	--	--	--	--	--	--	--	--	*	--
predatory bird (sm. mammal)#	--	--	--	--	--	--	--	--	--	--	--
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--

-- Exposure scenario results in a dose below the toxicity index
× •Exposure scenario results in a dose that exceeds the toxicity index
*Includes scenario for direct spray of a rabbit-sized mammal.
Data is lacking regarding chronic exposures, so effects are assumed by comparing acute dose vs. chronic NOAEL, and will likely over-estimate actual risk.
unk – unknown; insufficient data to assess risk

Table C- 3. Exposure scenario results from FS/SERA risk assessments for mammals, birds, and honeybees using the highest application rate and upper residue rates.

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
ACUTE EXPOSURES											
Direct spray, bee	--	--	♦	--	--	--	--	--	--	♦	
Direct spray, sm. mammal	--	--	--	--	--	--	--	--	--	--	♦

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
Consume Contaminated Vegetation											
small mammal	--	--	--	--	--	--	--	--	--	--	♦
large mammal	--	--	♦	--	--	--	--	--	--	♦	♦
large bird	--	--	--	--	--	--	--	--	--	♦	♦
Consume Contaminated Water											
Spill, sm. mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects											
small mammal	--	--	♦	--	--	--	♦	--	--	♦	♦
small bird	--	--	♦	--	--	--	--	--	--	♦	♦
Consume Contaminated Prey											
carnivore (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (sm. mammal)	--	--	--	--	--	--	--	--	--	--	♦
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--
CHRONIC EXPOSURES											
Consume Contaminated Vegetation											
small mammal, on site	--	--	--	--	--	--	--	--	--	--	--
lg. mammal, on site	--	--	--	--	--	--	--	--	♦	♦	--
lg. bird, on site	--	♦	♦	--	--	--	--	♦	♦	♦	--
Consume Contaminated Water											
small mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects#											
small mammal	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
small bird	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
Consume Contaminated Prey											
carnivore (sm. mammal)#	--	--	--	--	--	--	--	--	--	♦	♦
predatory bird (sm. mammal)#	--	--	--	--	--	--	--	♦	--	♦	♦
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--

-- Exposure scenario results in a dose below the toxicity index
× •Exposure scenario results in a dose that exceeds the toxicity index
•Includes scenario for direct spray of a rabbit-sized mammal
Data is lacking regarding chronic exposures, so effects are assumed by comparing acute dose vs. chronic NOAEL, which will likely over-estimate actual risk.
unk – unknown; insufficient data to assess risk

In terms of effects to sensitive species, there are no substantial differences between the different standards and PDFs in the alternatives or the alternatives as a whole. Therefore, Table C - 4 summarizes the potential effects to each sensitive species group.

Table C- 4. Potential effects from invasive plant treatment methods to groups of sensitive species

Sensitive Species Group	Potential Effects	Determination
Large herbivorous mammal	Worst-case exposure exceeds toxicity index from ingesting forage that has glyphosate, picloram, sulfometuron methyl, triclopyr, or NPE surfactants if broadcast sprayed. Worst-case herbicide exposure is highly unlikely for non-selective herbicides; more likely for selective herbicides.	MINL* Bighorns utilize cheatgrass. Worst-case exposure can be reduced by project design (Standard 22).
Small herbivorous mammals	Mechanical treatments may reduce cover and increase incidence of cheatgrass in certain habitat. Worst-case exposure exceeds toxicity index from ingesting forage that has been sprayed with triclopyr, or NPE surfactants if broadcast sprayed. Worst-case herbicide exposure is highly unlikely for non-selective herbicides; much more likely for selective herbicides.	MINL. Invasive plants threaten habitat. Short-term adverse effects provide long-term benefit. Worst-case exposure can be reduced by project design (Standard 22).
Carnivorous mammals	Infrequent and short-term disturbance from treatment projects could affect wolverines during breeding season. Worst-case exposure exceeds toxicity index from ingesting prey that has been sprayed with triclopyr. Worst-case herbicide exposure is highly unlikely.	MINL. Invasive plants may degrade habitat for some prey. Short-term adverse effects provide long-term benefit. Worst-case exposure highly unlikely.
Insectivorous mammals	Mechanical treatments may reduce foraging areas over the short-term. Worst-case exposure exceeds toxicity index from ingesting prey that has been sprayed with clopyralid, glyphosate, picloram, sethoxydim, sulfometuron methyl, and triclopyr if broadcast sprayed. Worst-case herbicide exposure is highly unlikely for bats, somewhat more likely for shrews.	MINL. Little overlap between invasive plants and shrew habitat. Bats may forage over large areas, reducing exposure. Worst-case exposure can be reduced by project design (Standard 22).
Herbivorous birds	Mechanical treatments may reduce cover and increase incidence of cheatgrass within grouse habitat. Worst-case exposure exceeds toxicity index from ingesting forage that has been sprayed with clopyralid, glyphosate, picloram, sethoxydim, sulfometuron methyl, and triclopyr if broadcast sprayed. Worst-case herbicide exposure is highly unlikely for non-selective herbicides; much more likely for selective herbicides.	MINL. Invasive plants threaten habitat. Short-term adverse effects provide long-term benefit. Worst-case exposure can be reduced by project design (Standard 22).
Insectivorous birds	Manual and mechanical treatments could trample or harm eggs or young of ground or low-nesting species during the breeding season. Worst-case exposure exceeds toxicity index from ingesting prey that has been sprayed with clopyralid, glyphosate, picloram, sethoxydim, sulfometuron methyl, and triclopyr if broadcast sprayed. Worst-case herbicide exposure is likely for grassland species on large projects.	MINL. Invasive plants threaten habitat for some species. Short-term adverse effects provide long-term benefit. Worst-case exposure can be reduced by project design (Standard 22).
Predatory birds	Manual and mechanical treatments could disturb species during the nesting season or affect their prey base. Worst-case exposure exceeds toxicity index from ingesting prey that has been sprayed with sethoxydim, and triclopyr if broadcast sprayed. Worst-case herbicide exposure is unlikely except aerial spray of grasslands.	MINL. Invasive plants may alter habitat for prey. Short-term adverse effects provide long-term benefit. Worst-case exposure can be reduced by project design (Standard 22).
Piscivorous birds	Manual and mechanical treatments could disturb species during the nesting season. Worst-case exposure does not exceed toxicity index for any herbicide.	MINL. Invasive plants can reduce or eliminate preferred nesting habitat. Short-term adverse effects provide long-term benefit.
Reptiles	Mechanical treatments could trample or harm individuals. Insufficient data to determine potential effects from herbicides.	MINL. Species have extensive distributions. Most adverse effects can be reduced by project design (Standard 22).

Sensitive Species Group	Potential Effects	Determination
Amphibians	Applications or accidental spills of glyphosate or triclopyr, could harm or kill amphibians.	MINL. Little overlap between invasive plants and amphibian habitat, except for riparian weeds. Herbicide exposure can be reduced by project design (Standard 22).
* May Impact, Not likely to adversely impact		

Tables C-5 – C-13 Herbicides

Table C- 5. Herbicides Analyzed in the Region 6 Invasive Plants EIS

Chemical Name	Selectivity	Sample Trade Name
Chlorsulfuron	broad-leaf	Telar, Glean, Corsair
Clopyralid	broad-leaf	Transline, Stinger
Glyphosate	No	RoundUp, Rodeo, Accord, Aquamaster
Imazapic	some broad-leaf & some grasses	Plateau
Imazapyr	No	Arsenal, Chopper, Stalker, Habitat
Metsulfuron methyl	broad-leaf & woody	Escort
Picloram	broad-leaf & woody	Tordon
Sethoxydim	grasses	Poast
Sulfometuron methyl	No	Oust
Triclopyr	broad-leaf & woody	Garlon, Pathfinder, Remedy
* Not selected in the 2005 Record of Decision. Not currently available for use on forests in R6.		

Table C- 6. Herbicide and nonylphenol polyethoxylate application rates to be used to treat invasive plants, including the incidental rates of application of the impurity hexachlorobenzene

Herbicide	Typical Application Rate lb ai/ac*	Lowest Application Rate lb ai/ac	Highest Application Rate lb ai/ac
Chlorsulfuron	0.056	0.0059	0.25
Clopyralid	0.35	0.1	0.5
Glyphosate	2	0.5	7
Imazapic	0.13	0.031	0.19
Imazapyr	0.45	0.03	1.25
Metsulfuron Methyl	0.03	0.013	0.15
Picloram	0.35	0.1	1.0
Sethoxydim	0.3	0.094	0.38
Sulfometuron Methyl	0.045	0.03	0.38
Triclopyr	1.0	0.1	10
Nonylphenol Polyethoxylate	1.67	0.167	6.68
Hexachlorobenzene#	0.000004	0.0000024	0.000012

* pounds of active ingredient per acre
#These application rates reflect the incidental rates of application of the impurity hexachlorobenzene.
Source: USDA Forest Service 2003, SERA 1998, 2001, 2003

Umatilla and Wallowa-Whitman National Forest Herbicide Spray Buffers

Aerial spraying will not be used in municipal watersheds.There are no chemical emergent treatments proposed as part of this project.

Table C- 7. Herbicide Use Buffers – Perennial and Wet Intermittent Streams - Proposed Action

Herbicide	Perennial and Wet Intermittent Stream			
	Aerial	Broadcast	Spot	Hand Select
Aquatic Labeled Herbicides				
Aquatic Glyphosate	300	100	Water's edge	Water's edge
Aquatic Triclopyr-TEA	None Allowed	None Allowed	15	Water's edge
Aquatic Imazapyr*	300	100	Water's edge	Water's edge
Low Risk to Aquatic Organisms				
Imazapic	200	100	15	Bankfull
Clopyralid	200	100	15	Bankfull
Metsulfuron Methyl	None Allowed	100	15	Bankfull
Moderate Risk to Aquatic Organisms				
Imazapyr	300	100	50	Bankfull
Sulfometuron Methyl	None Allowed	100	50	5
Chlorsulfuron	None Allowed	100	50	Bankfull
High Risk to Aquatic Organisms				
Triclopyr-BEE	None Allowed	None Allowed	150	150
Picloram	300	100	50	50
Sethoxydim	300	100	50	50
Glyphosate	300	100	50	50

Table C- 8. Herbicide Use Buffers – Dry Intermittent Streams - Proposed Action

Herbicide	Dry Intermittent Stream			
	Aerial	Broadcast	Spot	Hand/ Select
Aquatic Labeled Herbicides				
Aquatic Glyphosate	100	50	0	0
Aquatic Triclopyr-TEA	None Allowed	None Allowed	0	0
Aquatic Imazapyr*	100	50	0	0
Low Risk to Aquatic Organisms				
Imazapic	100	50	0	0
Clopyralid	100	50	0	0
Metsulfuron Methyl	None Allowed	50	0	0
Moderate Risk to Aquatic Organisms				
Imazapyr	200	50	15	Bankfull
Sulfometuron Methyl	None Allowed	50	15	Bankfull
Chlorsulfuron	None Allowed	50	15	Bankfull
High Risk to Aquatic Organisms				
Triclopyr-BEE	None Allowed	None Allowed	150	150
Picloram	200	100	50	50
Sethoxydim	200	100	50	50

Glyphosate	200	100	50	50

Table C- 9. Herbicide Use Buffers – Lakes and Wetlands - Proposed Action.

Herbicide	Wetlands			
	Aerial	Broadcast	Spot	Hand/ Select
Aquatic Labeled Herbicides				
Aquatic Glyphosate	Not proposed	100**	Water's edge	Water's edge
Aquatic Triclopyr-TEA	None Allowed	None Allowed	15	Water's edge
Aquatic Imazapyr*	Not proposed	100**	Water's edge	Water's edge
Low Aquatic Hazard Rating				
Imazapic	Not proposed	100	15	High water mark
Clopyralid	300	100	15	High water mark
Metsulfuron Methyl	Not proposed	100	15	High water mark
Moderate Aquatic Hazard Rating				
Imazapyr	Not proposed	100	50	High water mark
Sulfometuron Methyl	None Allowed	100	50	5
Chlorsulfuron	None Allowed	100	50	High water mark
Greater Aquatic Hazard Rating				
Triclopyr-BEE	None Allowed	None Allowed	150	150
Picloram	300	100	50	50
Sethoxydim	Not proposed	100	50	50
Glyphosate	Not proposed	100	50	50

*Aquatic Imazapyr (Habitat) may not be used until the risk assessment (currently underway) is completed for inert ingredients and additives.
** If wetland, pond or lake is dry, there is no buffer.

Table C- 10. Buffer width for aerial spraying based upon wind speed.

Buffer width for a 25 foot release height, 7-8 mph winds	Buffer width for a 35 foot release height, 7-8 mph winds	Buffer width for a 50 foot release height, 7-8 mph winds
Designated buffer	Add 1 swath width to buffer	Add 2 swath widths to buffer

Alternatively use low drift technology i.e. nozzle design and/or additives that ensure little to no drift into stream buffers or sensitive areas as directed in PDFs.

Table C- 11. Toxicity indices for mammals used in the effects analysis. Indices represent the most sensitive endpoint from the most sensitive species for which adequate data are available.

Herbicide	Duration	Endpoint	Dose	Species	Effect Noted at LOAEL
Chlorsulfuron	Acute	NOAEL	75 mg/kg	Rabbit	Decreased weight gain at 200 mg/kg
	Chronic	NOAEL	5 mg/kg/day	Rat	Weight changes at 25 mg/kg/day
Clopyralid	Acute	NOAEL	75 mg/kg	Rat	Decreased weight gain at 250 mg/kg
	Chronic	NOAEL	15 mg/kg/day	Rat	Thickening of gastric epithelium at 150 mg/kg/day
Glyphosate	Acute	NOAEL	175 mg/kg	Rabbit	Diarrhea at 350 mg/kg

Herbicide	Duration	Endpoint	Dose	Species	Effect Noted at LOAEL
	Chronic	NOAEL	175 mg/kg/day	Rabbit	Diarrhea at 350 mg/kg
Imazapic	Acute	NOAEL	350 mg/kg	Rabbit	Decreased body weight at 500 mg/kg
	Chronic	NOAEL2	45 mg/kg	Dog	Microscopic muscle effects at 137 mg/kg
Imazapyr	Acute	NOAEL	250 mg/kg	Dog	No effects at highest doses tested
	Chronic	NOAEL	250 mg/kg/day	Dog	No effects at highest doses tested
Metsulfuron methyl	Acute	NOAEL3	25 mg/kg	Rat	Decreased weight gain at 500 mg/kg
	Chronic	NOAEL	25 mg/kg/day	Rat	Decreased weight gain at 125 mg/kg
Picloram	Acute	NOAEL	34 mg/kg	Rabbit	Decreased weight gain at 172 mg/kg
	Chronic	NOAEL	7 mg/kg	Dog	Increased liver weight at 35 mg/kg4
Sethoxydim	Acute	NOAEL	160 mg/kg5	Rabbit	Reduced number of viable fetuses, some dam mortality at 480 mg/kg
	Chronic	NOAEL	9 mg/kg/day	Dog	Mild anemia at 18 mg/kg/day
Sulfometuron methyl	Acute	NOAEL	87 mg/kg	Rat	Decreased body weight at 433 mg/kg
	Chronic	NOAEL	2 mg/kg/day	Rat	Effects on blood and bile ducts at 20 mg/kg/day
Triclopyr6	Acute	NOAEL	100 mg/kg	Rat	Malformed fetuses at 300 mg/kg
	Chronic7	NOAEL	0.5 mg/kg/day	Dog	Effect on kidney at 2.5 mg/kg/day
NPE Surfactants	Acute	NOAEL	10 mg/kg	Rat	Slight reduction of polysaccharides in liver at 50 mg/kg/day
	Chronic	NOAEL	10 mg/kg/day	Rat	Increased weights of liver, kidneys, ovaries, and decreased live pups at 50 mg/kg/day

1 Acute values are based on chronic values; if the dose does not cause an effect over a period of 21 weeks, it is reasonable to assume that it will not cause effects after one day of exposure (SERA 2004 Dicamba).2 Imazapic – NOAEL calculated from a LOAEL of 137 mg/kg/day and application of a safety factor of 3 to extrapolate from a LOAEL to a NOAEL.
3 The acute NOAEL of 24 mg/kg is very close to the chronic NOAEL, so chronic value is used for acute exposures as well.
4 USEPA/OPP 1998
5 Source of the value used by EPA (180 mg/kg) is not well documented, so the lower value of 160 mg/kg from a rabbit study is used as the toxicity index for this analysis.
6 Triclopyr BEE and TEA have equal toxicities to mammals (SERA 2003a).
7 Value taken from Quast et al. 1976 as cited in SERA Triclopyr 2003. This represents an extremely conservative approach, explained in more detail in the write up on triclopyr later in this document.Source: SERA 1998, 2001, 2003, 2004 and USDA FS 2003.

For Table C- 12, categories of the herbicides are simply relative to each other; all 10 of these herbicides are low risk compared to other herbicides, and especially when compared to other pesticides. The categories are based on various criteria. This is general information only and background data should be reviewed before making any conclusions or conducting any analysis regarding these herbicides or NPE-based surfactants.

Table C- 12 Relative Comparison Summary of the 10 Herbicides and NPE Surfactant

Risk Rating	Aquatic[1]	Wildlife[2]	Worker Health[3]	Public Health[4]
LOWEST	clopyralid, imazapic, metsulfuron methyl, NPE-based surfactants	chlorsulfuron, clopyralid imazapic, imazapyr, metsulfuron methyl, sethoxydim	chlorsulfuron, clopyralid, glyphosate, imazapic, imazapyr, metsulfuron methyl, sethoxydim, sulfometuron methyl	chlorsulfuron, metsulfuron methyl, sulfometuron methyl
MODERATE	chlorsulfuron, imazapyr, sulfometuron methyl	glyphosate, picloram	picloram, triclopyr	clopyralid, glyphosate, imazapic, imazapyr, picloram, sethoxydim, triclopyr
HIGHER	sethoxydim, glyphosate, picloram, triclopyr	triclopyr, NPE-based surfactants	NPE-based surfactants	NPE-based surfactants

Aquatics

LOWEST = Under GLEAMS parameters, the concentrations of herbicides in water did NOT exceed level of concern for fish.

MODERATE and HIGHER = some effect to plants, algae, or aquatic insects plausible.

Wildlife

LOWEST = Exposure scenarios result in doses below the toxicity indices for all acute exposures, even at highest application rates.

MODERATE = Exposure scenarios result in doses that exceed the toxicity indices for some acute exposures, but only at highest application rates.

HIGHER = Exposure scenarios result in doses that exceed the toxicity indices for some acute exposures at typical application rates. (Risk of chronic exposure is variable and depends on many factors, including life history of wildlife, and persistence and selectivity of herbicide. Most chronic exposure scenarios are highly unlikely.)

Worker Health: Based on backpack spray applications.

LOWEST = HQ less than 0.1

MODERATE = HQ less than 1.0 but greater than 0.

HIGHER = HQ > 1.0

1 R6 2005 FEIS, Fisheries Biological Assessment
2 R6 2005 FEIS, Appendix P, Summary of Herbicide Effects to Wildlife
3 R6 2005 FEIS, Appendix Q, Human Health Risk Assessment
4 ibid

Public Health:

Based on scenario of public drinking water from a small pond contaminated by an accidental spill of 200 gallons - HQ thresholds same as for WORKER Health

Table C- 13. Summary of exposure scenario results for listed species

SPECIES	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
Gray Wolf	--	--	--	--	--	--	--	--	--	✶1	♦
Bliss R snail3	--	--	--	--	--	--	--	--	--	--	--

-- Exposure scenarios result in a dose below the toxicity index at both the typical and highest application rates.
✶ Exposure scenarios result in a dose that exceeds the toxicity index at the typical and highest application rates.
♦ Exposure scenarios result in a dose that exceeds the toxicity index at the highest application rate only.
1 These scenarios exceed the toxicity index only for assumed chronic exposures, risks are actually unknown, but the chronic exposure scenarios are not plausible.
2 Based on exposure scenario calculations for honeybee
3 Based on water concentrations used to calculate exposure to fish, and information on toxicity to federally listed aquatic invertebrates from analysis used for the BA.
Source: SERA 1998, 2001, 2003, 2004 and USDA FS 2003.

Summary of Herbicide Effects to Wildlife

DRAFT

Prepared by: Shawna L. Bautista, Wildlife Biologist, Invasive Plant EIS

US Forest Service, Region 6 Regional Office, Portland, OR

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Summary of Herbicide Effects to Wildlife

Syracuse Environmental Research Associates (SERA) conducted very comprehensive searches of the literature when preparing the risk assessments, and also evaluated the research papers for quality of methods and analysis used. This document is a summary of toxicity information presented in Forest Service Risk Assessments (SERA 1998, 2001, 2003) and some public literature.

Citation Method Used in This Document

Because a large number of risk assessments produced by SERA are the basis for this document, many of them were produced in the same year, and the inherent difficulty in accurately tracking citations designated by year and lower case letter (e.g. 2003a, 2003b, etc.), For risk assessments produced by SERA, the author and year is followed by the chemical name analyzed in the cited risk assessment. For example, information taken from the glyphosate risk assessment produced by SERA in 2003 is cited as: (SERA 2003 Glyphosate). Information in this report is taken from risk assessments produced by SERA unless otherwise noted.

Herbicides Analyzed

The herbicides included in this summary are those analyzed in the Region 6 Invasive Plant Environmental Impact Statement (EIS) (Table C- 14). These herbicides or formulations are registered for use in forestry applications, right-of-ways, or rangelands and are appropriate for use against invasive plant species in Region 6 of the USDA Forest Service. The mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Table C- 14. Herbicides analyzed and some representative formulation names.

Chemical Name	Trade Name
Chlorsulfuron	Telar, Glean, Corsair
Clopyralid	Transline, Stinger
Glyphosate	RoundUp, Rodeo, Accord
Imazapic	Plateau
Imazapyr	Arsenal, Chopper, Stalker
Metsulfuron methyl	Escort
Picloram	Tordon
Sethoxydim	Poast
Sulfometuron methyl	Oust
Triclopyr	Garlon, Pathfinder, Remedy

It is not feasible to evaluate specific effects to specific wildlife species at a regional scale. The effects of herbicide use must be evaluated at the site-specific scale before any projects involving herbicide use are authorized. However, it is useful to understand the general and relative risks that proposed herbicides pose to wildlife in the planning area.

The following discussion will provide information on all herbicides considered in the USDA Forest Service, Pacific Northwest Region, Invasive Plant EIS. Refer to the following text box for terms and concepts about potential effects of herbicides.

Terms and acronyms used in this document.

Allometric = pertaining to allometry, the study and measure of growth. In toxicology, the study of the relationship of body size to various processes that may impact how chemicals affect the organism or how the chemicals are transported within the organism.

bioconcentration = the net accumulation of a substance by an aquatic organism as a result of uptake directly from aqueous solution (i.e. water with other stuff mixed in).

bioaccumulation = the net accumulation of a substance by an organism as a result of uptake directly from all environmental sources and from all routes of exposure (primarily from food or water that is ingested).

dose = the actual quantity of a chemical administered to, or absorbed by, an organism.

gavage = a method of dose administration; the substance is placed directly in the stomach..

exposure = the amount of chemical in contact with an animal.

LD_{50} (lethal dose50) - The dose of a chemical calculated to cause death in 50% of a defined experimental animal population over a specified observation period. The observation period is typically 14 days.

LOAEL = Lowest-observed-adverse-effect level; lowest exposure associated with an adverse effect.

NOEL = No-observed-effect level; no effects attributable to treatment.

NOAEL =No-observed-adverse-effect level: An exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered as adverse, or as precursors to adverse effects. In an experiment with several NOAELs, the regulatory focus is primarily on the highest one, leading to the common usage of the term NOAEL as the highest exposure without adverse effects.

NOEC = No-observed-effect concentration; synonymous with NOEL.

Surfactant = surface acting agent; any substance that when dissolved in water or an aqueous solution reduces its surface tension or the interfacial tension between it and another liquid.

Surrogate = a substitute; lab animals are substituted for humans or other wildlife in toxicity testing.

Toxicity index = in this document, it is the dose of herbicide used to determine the potential for an adverse effect to wildlife. It is the lowest dose reported to cause the most sensitive effect in

the most sensitive species tested, and is usually a reported NOAEL for a sub-lethal effect, but may be an LD_{50} (or a portion thereof) when data is lacking.

a.e. = acid equivalent

a.i. = active ingredient

kg = kilogram, equivalent to 1000 grams or 2.2 pounds

g = gram, equivalent to 1000 milligrams or about 0.035 ounce (28 g = 1 ounce)

mg = milligram; 0.001 gram.

mg/L = milligrams per liter; equivalent to ppm.

mg/kg = milligrams per kilogram; equivalent to ppm.

ppm = part(s) per million; equivalent to mg/L and mg/kg.

ppb = part(s) per billion

Herbicides have the potential to adversely affect the environment. The U.S. Environmental Protection Agency (EPA) must register all herbicides prior to their sale, distribution, or use in the United States. In order to register herbicides for outdoor use, the EPA requires the manufacturers to conduct a safety evaluation on wildlife including toxicity testing on representative species of birds, mammals, freshwater fish, aquatic invertebrates, and terrestrial and aquatic plants. An ecological risk assessment uses the data collected to evaluate the likelihood that adverse ecological effects may occur as a result of herbicide use.

The Forest Service conducts its own risk assessments, focusing specifically on the type of herbicide uses in forestry applications. The Forest Service contracts with SERA to conduct human health and ecological risk assessments for herbicides that may be proposed for use on National Forest System lands. The information contained in this EIS relies on these risk assessments. All toxicity data, exposure scenarios, and assessments of risk are based upon information in the SERA risk assessments unless otherwise noted. Typical application rates of herbicides and nonylphenol polyethoxylate (NPE) surfactant used in this analysis can be found inTable C- 15.

Table C- 15. Herbicide and nonylphenol polyethoxylate application rates used to treat invasive plants. Included are the incidental rates of application of the impurity hexachlorobenzene.

Herbicide	Typical Application Rate lb ai/ac*	Lowest Application Rate lb ai/ac	Highest Application Rate lb ai/ac
Chlorsulfuron	0.056	0.0059	0.25
Clopyralid	0.35	0.1	0.5
Glyphosate	2	0.5	7
Imazapic	0.13	0.031	0.19
Imazapyr	0.45	0.03	1.25
Metsulfuron Methyl	0.03	0.013	0.15
Picloram	0.35	0.13	1.0

Herbicide	Typical Application Rate lb ai/ac*	Lowest Application Rate lb ai/ac	Highest Application Rate lb ai/ac
Sethoxydim	0.3	0.094	0.38
Sulfometuron Methyl	0.045	0.03	0.38
Triclopyr	1.0	0.1	10
Nonylphenol Polyethoxylate	1.67	0.167	6.68
Hexachlorobenzene#	0.000004	0.0000024	0.000012

* pounds of active ingredient per acre
#These application rates reflect the incidental rates of application of the impurity hexachlorobenzene.
Source: USDA Forest Service 2003, SERA 1998, 2001, 2003

Inerts, Adjuvants and Impurities

Herbicides are not pure compounds and they contain the active ingredient, impurities, adjuvants, inert ingredients, and may also contain surfactants. The effects of inert ingredients, adjuvants, impurities, and surfactants to wildlife are discussed first, followed by a discussion of the effects of the active ingredients.

Inert compounds are those that are intentionally added to a formulation, but have no herbicidal activity and do not affect the herbicidal activity. Inerts are added to the formulation to facilitate its handling, stability, or mixing. Impurities are inadvertent contaminants in the herbicide, usually present as a result of the manufacturing process. Adjuvants are compounds added to the formulation to improve its performance. They can either enhance the activity of an herbicide's active ingredient (activator adjuvant) or offset any problems associated with its application (special purpose or utility modifiers). Surfactants are one type of adjuvant that makes the herbicide more effective by increasing absorption into the plant, for example.

Inerts and adjuvants, including surfactants, are not under the same registration guidelines as are pesticides. The EPA classifies these compounds into four lists based on the available toxicity information. List 1 contains "inerts of toxicological concern"; List 2 contains "potentially toxic inerts, high priority for testing"; List 3 contains "inerts of unknown toxicity"; and List 4 contains "minimal risk inerts" or "inerts for which EPA has sufficient information to conclude that their current use patterns will not adversely affect public health or the environment." If the compounds are not classified as toxic, then all information on them is considered proprietary and the manufacturer need not disclose their identity. Therefore, inerts and adjuvants generally do not have the same amount of research conducted on their effects, compared to active ingredients.

Inert Ingredient Effects

There is very little data regarding the effects to most wildlife species from inert ingredients contained in the 12 herbicides considered in this EIS. None of the inert ingredients included on EPA's List 2, 3, or 4 need to be disclosed on the herbicide label, despite evidence that some compounds on these lists may cause adverse effects to laboratory animals and humans (Anonymous 1999; Cox 1999; Knight 1997; Knight and Cox 1998; Marquardt et al. 1998). EPA's own website (http://www.epa.gov/opprd001/inerts/) states, "Since neither federal law nor the regulations define the term "inert" on the basis of toxicity, hazard or risk to humans, non-target species, or the environment, it should not be assumed that all inert ingredients are non-toxic." Northwest Coalition for Alternatives to Pesticides (NCAP) obtained the identity of many inert

ingredients through a Freedom of Information Act request; the list of inerts they obtained can be found at http://www.pesticide.org/FOIA/

Many of the inert ingredients are proprietary in nature and have not been tested on laboratory or wildlife species. SERA obtained clearance to access confidential business information (i.e. the identity of proprietary ingredients) and used this information in the preparation of the risk assessment. However, toxicity data to support any assessment of hazard or risk are usually very poor, even when the identity of the inert is known.

Chlorsulfuron – The identity of inerts used in chlorsulfuron are confidential, but SERA reviewed them for preparation of the risk assessment (SERA 2003 Chlorsulfuron). EPA has not classified any of the inerts as toxic. These inert ingredients do not affect the assessment of risk

Clopyralid – Identified inerts include monoethanolamine and isopropyl alcohol, both approved food additives. These inert ingredients do not impact the assessment of risk 5

Glyphosate – There are at least 35 glyphosate formulations that are registered for forestry applications (SERA, 2003-Glyphosate) with a variety of inert ingredients. SERA obtained clearance to access confidential business information (i.e. the identity of proprietary ingredients) and used this information in the preparation of the risk assessment. Surfactants (discussed below) were the only additives identified that impact risk (SERA, 2003-Glyphosate).

Imazapic - The identity of inerts used in imazapic formulations are confidential, but SERA reviewed them for preparation of the risk assessment (SERA, 2003-Imazapic). EPA has not classified any of the inerts as toxic.

Imazapyr – The identity of inerts used in imazapic formulations are confidential, but SERA reviewed them for preparation of the risk assessment (SERA, 2003-Imazapyr). No apparently hazardous materials were identified in the review of inerts. The NCAP website (http://www.pesticide.org/FOIA/picloram.html) identifies only glacial acetic acid, an approved food additive, as an inert ingredient. Isopropanolamine is also present, and it is classified as a List 3 inert.

Metsulfuron methyl - The identity of inerts used in metsulfuron methyl formulations are confidential, but SERA reviewed them for preparation of the risk assessment (SERA, 2003-Metsulfuron methyl). EPA has not classified any of the inerts as toxic.

Picloram – The formulations Tordon K and Tordon 22K contain the following inerts: potassium hydroxide, ethoxylated cetyl ether, alkyl phenol glycol ether, and emulsified silicone oil (NCAP website; www.pesticide.org/FOIA/picloram.html). Potassium hydroxide is an approved food additive. The other compounds are all on EPA's List 4B, inerts of minimal concern. They may also contain the surfactant polyglycol 26-2, which is on EPA's List 3: Inerts of Unknown Toxicity, discussed in the following section. The toxicity data on the formulations encompasses toxic risk from the inerts. Inerts in picloram formulations do not appear to pose a unique toxic risk to wildlife (SERA, 2003-Picloram).

Sethoxydim - The formulation Poast® contains 74 percent petroleum solvent that includes naphthalene. The EPA has placed this naphthalene on List 2 ("agents that are potentially toxic and a high priority for testing"). Petroleum solvents and naphthalene depress the central nervous system and cause other signs of neurotoxicity (SERA, 2001). Poast® has also been reported to cause skin and eye irritation. There is no information suggesting that the petroleum solvent has a substantial impact on the toxicity of sethoxydim to experimental animals, with the important and

notable exception of aquatic animals (SERA, 2001). Poast® is much more toxic to aquatic species than sethoxydim. 6

Sulfometuron methyl - The identity of inerts used in Oust are confidential, but SERA reviewed them for preparation of the risk assessment (SERA, 2003-Sulfometuron). EPA has not classified any of the inerts as toxic. Based on comparison of the toxicities of the active ingredient and the formulation, there is no reason to suspect that Oust contains other ingredients that substantially affect the potential risk to wildlife.

Triclopyr - Formulations contain ethanol (Garlon 3A) or kerosene (Garlon 4), which are known to be neurotoxic. However, the toxicity of these compounds is less than that of triclopyr, so the amount of ethanol and kerosene in these formulations is not toxicologically significant (SERA, 2003-Triclopyr) for wildlife.

Surfactant Effects

Surfactants, or surface-acting agents, facilitate and enhance the absorbing, emulsifying, dispersing, spreading, sticking, wetting, or penetrating properties of herbicides. There is a fair amount of research on the effects of surfactants to terrestrial and aquatic organisms because they are widely used in detergents, cosmetics, shampoos and other products designed for human exposure.

The following information is taken from "Analysis of Issues Surrounding the Use of Spray Adjuvants With Herbicides" (USDA FS, 2002) and "Human and Ecological Risk Assessment of Nonylphenol Polyethoxylate-based (NPE) Surfactants in Forest Service Herbicide Applications" (USDA FS, 2003). Refer to these documents for more complete discussions.

Some glyphosate formulations contain polyethoxylated tallow amine (POEA) surfactant, which is substantially more toxic to aquatic species than glyphosate or other surfactants that may be used with glyphosate (SERA, 2003-Glyphosate, p. 4-14). In the SERA risk assessment, the toxicity of glyphosate is characterized based on the use of a surfactant, either in the formulation or added as an adjuvant in a tank mixture (SERA, 2003-Glyphosate, p. 4-14).

Polyglycol 26-2, used in picloram, will impact mitochondrial function in vitro, but information is insufficient to evaluate risks to wildlife in vivo from field applications at plausible levels of exposure (SERA, 2003-Picloram).

The primary active ingredient in many of the non-ionic surfactants used by the Forest Service is a component known as nonylphenol polyethoxylate (NPE). NPE is found in these commercial surfactants at rates varying from 20 to 80 percent. NPE is formed through the combination of ethylene oxide with nonylphenol (NP), and may contain small amounts of un-reacted NP. The properties of the particular NPE depend upon the number of ethoxylate groups that are attached to the NP. The most common NPE used in surfactants with pesticides is a mixture that has, as a majority, 8-10 ethoxylate groups attached, and can be abbreviated NP9E. NP is a material recognized as hazardous by the U.S. EPA (currently on U.S. EPA's inerts List 1). Both NP and NPE exhibit estrogen-like properties, although they are much weaker than the natural estrogen, estradiol.

Potential effects of NPE were analyzed using exposure scenarios to quantitatively estimate the dose of NPE that birds and mammals may receive if they consumed contaminated vegetation or prey, or if a small mammal was directly sprayed. Each estimated dose was compared to toxicity levels reported from laboratory data and summarized in USDA FS 2003. Data is lacking on the

toxic effects of NP or NPE to birds, with only the median lethal dose (LD_{50}) identified in the literature. Risk to birds is therefore evaluated using the toxicity values from mammals, which introduces additional uncertainty into the conclusions regarding birds. Data for terrestrial invertebrates is lacking or insufficient, so risks cannot be adequately characterized.

NP and NPE are weakly estrogenic in aquatic and terrestrial organisms (1000 to 100,000 times weaker than natural estrogen). NP and NPE are not toxic to soil microbes. NP is highly toxic to many aquatic organisms at low concentrations (currently on U.S. EPA's Inert List 1).

The use of NPE-based surfactants in any of the 12 herbicides considered in this EIS could result in toxic effects to some mammals and birds at typical and high application rates (project file worksheets; USDA, FS 2003). The exposure scenarios and calculated doses used in the analysis represent worst-case scenarios and are not entirely plausible. At the typical application rate, adverse effects could occur to small mammals that may be directly sprayed, large mammals and large birds consuming contaminated vegetation, and small mammals and small birds consuming contaminated insects. At the highest application rate, adverse effects could occur to small mammals that may be directly sprayed, large or small mammals and large birds consuming contaminated vegetation, small mammals and small birds consuming contaminated insects, and a predatory bird consuming a small mammal that has been directly sprayed. No chronic exposures result in plausible risk to mammals or birds.

NP and NPE have been studied for effects to aquatic organisms. NP is more toxic than NP9E, by one to three orders of magnitude (USDA FS, 2003). The toxicities of the intermediate breakdown products, NPEC and others, are intermediate between NP and NPE. In the aquatic environment, the breakdown products NP1EC and NP2EC are likely to be present also. These two metabolites are known to affect vitellogenin (a precursor for egg yolk) production in male fish, but NP, which is a more potent estrogenic compound, did not cause vitellogenin increases in male Xenopus laevis, or leopard frogs (Selcer et al., 2001; cited in USDA FS, 2003).

Mann and Bidwell (2000, 2001) tested several Australian frogs and Xenopus for effects to NP8E. They found that Xenopus was the most sensitive to toxic effects, with an LC_{50} of 3.9 ppm (3.9 mg/L). Similar to studies with herbicides, the LC_{50} values for the frogs are comparable to those for fish (USDA FS, 2003). NP8E inhibited growth at concentrations as low as 1 ppm (Mann and Bidwell, 2000, 2001). Mild narcosis of tadpoles can occur at EC_{50} values as low as 2.3 ppm, and reduced dissolved oxygen content in the water lowered the EC_{50} values by about half as compared to normal oxygen levels. The tadpoles recovered from the narcosis. Malformations in Xenopus occurred at EC_{50} values between 2.8 and 4.6 mg/L.

NP may cause tail resorption with a 14-day NOEC of 25 ppb for Xenopus laevis (Fort and Stover, 1997; cited in USDA FS, 2003). NP also increased the percentage of female Xenopus developing from tadpoles exposed to 22 ppb for 12 weeks, but did not produce this effect at 2.2 ppb.

During operational use of NPE surfactant, ambient levels of NP9E (including a small percentage of NP, NP1EC, and NP2EC) could average 12.5 ppb (range 3.1 to 31.2 ppb). The duration of these exposures from Forest Service use would generally be much shorter than those used in laboratory experiments, due to transport by flowing streams, dilution, and environmental degradation. These levels are not likely to adversely affect amphibians found in the Pacific Northwest for normal operations. However, overspray or accidental spills could produce concentrations of NP9E that could adversely affect amphibians, particularly in small stagnant ponds.

Effects of Impurities

All herbicides likely contain impurities as a result of the synthesis or production process. The toxic effects of impurities are addressed in toxicity tests using the technical grade product, which would contain the impurities.

Hexachlorobenzene is an impurity in the technical grade products of clopyralid and picloram. Hexachlorobenzene is a ubiquitous and persistent chemical in the environment, as it is used or present in a wide variety of manufacturing processes. It has been shown to cause tumors in mice, rats and hamsters, and EPA has classified it as a probable human carcinogen (SERA, 2003-Picloram). The amount of hexachlorobenzene released into the environment from Forest Service use of picloram and clopyralid is inconsequential in comparison to existing background levels and the annual release from manufacturing processes (SERA, 2003-Picloram, pp. 3-25). The use of picloram and clopyralid in remote forest locations could constitute the primary source of localized contamination however. The projected amount of hexachlorobenzene released during invasive plant treatments is calculated to be well below the level that poses a risk to cancer in mammals.

POEA surfactant used in Roundup and Roundup Pro contain 1,4-dioxane as an impurity, which has been classified by EPA as a probable human carcinogen. Based on current toxicity data and an analysis by Borrecco and Neisess (1991), the potential effects of 1,4-dioxane are encompassed by the available toxicity data on the Roundup formulation (SERA, 2003-Glyphosate). Borrecco and Neisess (1991) also demonstrated that the upper limit of risk of cancer from this impurity was less than one in a million.

Triclopyr contains an impurity, 2- butoxyethanol (aka EGBE), that is a major industrial chemical used in a wide variety of industrial and commercial applications. It is known to cause fragile red blood cells in rodents (Borrecco and Neisess 1991). EGBE has been classified as moderately toxic by EPA. Borrecco and Neisess (1991) found that potential doses of EGBE to mammals were less than 0.001 of the lowest LD_{50} and did not substantially increase risk over the risk identified for triclopyr, even under worst case scenarios. Data on toxicity of EGBE to birds was lacking, but the authors conclude that comparative sensitivities between birds and mammals, and the extremely low doses indicated a low risk to birds.

Metabolites

Similar to impurities, the potential health effects of herbicide metabolites are often accounted for in the available toxicity studies, assuming that the toxicological effects of metabolism within the test animal species would be similar to those in other animals. The potential toxic effects of environmental metabolites (those formed as a result of processes outside of the body) may not be accounted for by laboratory toxicity studies.

TCP (3,5,6-trichloro-2-pyridinol) is an environmental metabolite of triclopyr. In mammals, TCP has about the same toxicity as triclopyr. No quantitative estimate of exposure to mammals or birds was calculated in the SERA risk assessment, due to the lack of appropriate data. However, since TCP is as toxic as triclopyr, the risk characterization for triclopyr could be applied to TCP.

Site-specific analysis is necessary to further evaluate the risk of toxic effects from TCP.

Endocrine disruption

Recent information has highlighted the potential for certain synthetic and natural chemicals to affect endocrine glands, hormones, and hormone receptors (endocrine system). The endocrine

system helps control metabolism, body composition, growth and development, reproduction, and many other physiological regulators. An endocrine disrupter is a substance that may exert effects to the body by affecting the availability of a hormone to its target tissue(s) and/or affecting the response of target tissues to the hormone (SERA, 2002). Estrogen is a prominent hormone in animal systems and substances that mimic estrogen or stimulate similar responses in target tissues are referred to as "estrogenic." 10

Scientists have expressed concern regarding estrogenic effects of synthetic chemicals since before the 1970's. The EPA (1997) reports effects of endocrine disruption in animals that "include abnormal thyroid function and development in fish and birds; decreased fertility in shellfish, fish, birds, and mammals; decreased hatching success in fish, birds, and reptiles; demasculinization and feminization of fish, birds, reptiles, and mammals; defeminization and masculinization of gastropods, fish, and birds; decreased offspring survival; and alteration of immune and behavioral function in birds and mammals."

Some of the more noted endocrine glands include gonads, adrenal, pancreas, thyroid, and pituitary. Alteration in endocrine function may affect reproductive output (i.e. feminization, masculization), and therefore, could affect population numbers of affected species.

Many of the known endocrine disrupting contaminants have been banned or are regulated (e.g. DDT/DDE, PCB, TCDD). Some endocrine disrupting compounds are persistent and are still found within the living tissue of wildlife; their decomposition half-life is lengthy, and they are bioaccumulatory and present at high background levels. A local example is the high level of DDT/DDE and PCB that are found within peregrine falcons in the Pacific Northwest (Pagel, unpub. data). Research has suggested that embryonic exposure to endocrine disrupters may cause permanent health effects to adult animals. Some of these effects may include altered blood hormone levels, reduced fecundity, reproductive behavioral alterations, reduced immune function, masculization and feminization, undescended testicles, increased cancer rates, altered bone density and structure, and malformed fallopian female reproductive tract (Kubiak et al., 1989; Colborn and Clement, 1992; White et al., 1994; Fry, 1995; LeBlanc, 1995). Examples of wildlife species that have been adversely affected by endocrine disrupters include wood ducks in Arkansas, wasting and embryonic deformities of Great Lakes piscivorous birds, reproductive abnormalities of snapping turtles, gulls, trout and salmonids, alligators, mink, and Florida panther (Bishop et al. 1991, Colborn, 1991; Facemire et al., 1995; Fox et al., 1978, 1981, 1991 (a, b); Fry and Toone, 1981; Fry et al., 1987; Giesyet et al., 1994; Gilbertson et al., 1991; Guillette et al., 1994, 1995; Kubiak et al., 1989; Mac and Edsall, 1991, 1993; Leatherland, 1993; Peakall and Fox, 1987; White and Hoffman, 1995; and Wren, 1991).

Of the chemicals analyzed in this document, NPE surfactants have been identified as potentially having estrogenic effects (USGS, 1998; Bakke, 2003). Triclopyr and glyphosate have been evaluated for endocrine disrupting effects, and the weight of evidence indicates that these herbicides cause no specific toxic effects on endocrine function (SERA, 2002). One study on glyphosate, Yousef et al. (1995), indicated that there may be some concerns with glyphosate, but the study was poorly conducted and results are not reliable.

Sulfometuron methyl can cause malformations in amphibians (SERA, 2003-Sulfometuron), but whether the malformations are caused by endocrine disruption, cellular toxicity, or other pathway has not been reported.

Synergistic Effects

Certain chemicals may cause synergistic effects in the presence of other chemicals: that is, the total effect of two chemicals may be greater than that suggested by the sum of the effects from the individual components (USEPA, 2000). However, information regarding the existence or potential for synergistic effects from the herbicides discussed in this document is very limited. 11

Some of the herbicides analyzed in this document (e.g. picloram) have been investigated for possible synergistic effects but the study designs were insufficient for the assessment of toxicologic interactions (SERA, 2003-Picloram; p. 3-35) However, data on this potential effect is incomplete and not likely to be obtained in the foreseeable future: the sheer number of potential combinations of contaminants, environmental stressors, and wildlife species make it unfeasible to investigate thoroughly.

USEPA (2000) did state that for exposures at low doses, with low risk for each component in the chemical mixture, that the likelihood of significant interaction (e.g. synergistic effects) is usually considered to be low. Likewise, a report by ATSDR (2004) cited several studies using rats that found no synergistic effects for mixtures of four, eight and nine chemicals at low (sub-toxic) doses. But statistically significant interactions (both syntergistic and antagonistic) have been noted in some studies. Unfortunately, even with excellent data, the uncertainties and complexities of chemical interactions create substantial uncertainty in the risk characterization for chemical mixtures (ATSDR, 2004; USEPA, 2000).

Effects of Active Ingredients and Surrogate Species

Generally, active ingredients have been tested on only a limited number of species and mostly under laboratory conditions. While laboratory experiments can be used to determine acute toxicity and effects to reproduction, cancer rates, birth defect rates, and other effects that must be considered, laboratory experiments do not account for wildlife in their natural environments. This leads to uncertainty in the risk assessment analysis. Environmental stressors can increase the adverse effects of contaminants, but the degree to which these effects may occur for various herbicides is largely unknown. Adverse affects to wildlife health such as lethargy, weight loss, nausea, and fluid loss due to diarrhea or vomiting, can affect their ability to compete for food, locate and/or capture food, avoid or fight off predators, or reproduce. The following analysis relies on these types of effects, when sufficient data exists, rather than lethal doses, to determine the potential for doses to cause an “adverse effect” to wildlife.

FS/SERA risk assessments and published literature are the primary sources of information used to evaluate effects of herbicides to wildlife. First, we discuss field studies found in the published literature regarding potential effects of herbicide use to wildlife. Then, qualitative and quantitative information from the FS/SERA risk assessments and published literature regarding effects of active ingredients are discussed.

Toxicity Data and Exposure Analysis

The FS/SERA risk assessments present the toxicity data from studies conducted to meet EPA registration requirements and from published literature. In addition, exposure of various animals to herbicide is quantitatively estimated to characterize risk from the use of each herbicide.

The Use of Surrogate Species

Most toxicity testing utilizes surrogate species. Surrogate species serve as a substitute for the species of interest, because all species of interest could not be tested. Surrogate species are typically organisms that are easily tested using standardized methods, are readily available, and

inexpensive. Rare species are not tested and the physiological requirements for some organisms prohibit their use in toxicity testing because these requirements cannot be met within the test system. Even when desired species are available (e.g. salmon), researchers may choose a surrogate, like zebrafish (Danio rerio)(aka zebra danio), because test results are more easily discerned with the surrogate, and reproductive capacity allows testing of large numbers of individuals, among other reasons (Scholz, unpublished. proposal, 2003).

However, caution should to be taken when addressing ecological risk and the use of surrogates when analyzing those ecological risks. Some herbicides demonstrate more variation than others in effects among different species, and very limited numbers of species have been tested.

Because of the variation of responses among species, and the uncertainty with regard to how accurately a surrogate species may represent other wildlife, the FS/SERA risk assessments use the most sensitive endpoint from the most sensitive species tested as the toxicity index for terrestrial wildlife. This does not alleviate concerns over interspecies variations in response, however.

Doses and Responses

The likelihood that an animal will experience adverse effects from an herbicide depends on: (1) the inherent toxicity of the chemical, (2) the amount of chemical to which an animal is exposed, (3) the amount of chemical actually received by the animal (dose), and (4) the inherent sensitivity of the animal to the chemical.

The toxicity of the chemical is measured by laboratory tests required by EPA. The amount of chemical to which an animal may be exposed is influenced by several factors, discussed below. When an animal is exposed to a chemical, only a portion of the chemical applied or ingested is actually absorbed or taken in by the animal (the dose). Various absorption rates for wildlife are not available, so some scenarios use the same value for exposure and dose. Also, different species have different susceptibilities to various chemicals. This is discussed more in the section on surrogates.

Factors that Influence Exposure and Dose

The exposure of an animal to an herbicide is greatly influenced by relationships between body size and several physiological, metabolic, and pharmacological processes (allometry). For example, allometric relationship dictates that animals of smaller size have a larger amount of surface area for their mass than larger animals. This relationship greatly influences basic physiological properties, such as food consumption and thermoregulation. Some of the allometric factors that influence exposure to herbicides are detailed below.

Body Weight

Several parameters used to estimate herbicide contact are reported on a "per body weight" basis, expressed in grams (g) or kilograms (kg). For example, both food and water ingestion rates are reported on a per body weight basis (such as gram of fresh food or water per gram of fresh body weight per day). Body weights, in units of mass, are reported as fresh weight that might be obtained by weighing a live animal in the field. Also, body weight data are used in empirical models to calculate some parameters, such as surface area, when there no specific measurements are available. Calculations of "potential dose to animal" use body weight of animals.

Metabolic Rate

Metabolic rate is not directly calculated in this document, or in the FS/SERA risk assessments, but reported values for various species are used to calculate food consumption requirements. It is

reported on the basis of kilocalories per day for units of body weight (kcal/kg/day). Metabolic rate is closely related to body size, with smaller animals generally having higher metabolic rates than larger animals.

Contact Rate

Exposure involves direct contact with the herbicide, and wildlife may be exposed to herbicides by ingesting the chemical (oral) or by external contact (dermal). Oral exposures may occur from eating contaminated vegetation or prey, drinking contaminated water, or by grooming activities. Dermal exposures may occur from direct spray, or contact with contaminated vegetation or water. These contact routes are influenced by allometric relationships, as well as habitat preferences and feeding behaviors.

Oral Routes

Food ingestion: Small animals generally have higher caloric requirements than large animals, so a small animal ingests a greater amount of food per unit body weight compared to large animals. A 20g mouse, for example, will generally consume an amount of food equal to about 15 percent of its body weight every day, depending on calorie content of the diet. A value of 3.6 g of food consumed per day for a 20g mouse is used in the FS/SERA risk assessments for calculating exposure from contaminated food. This is equivalent to 18 percent of the body weight and is generated from general allometric relationships for food consumption in rodents (US EPA/ORD, 1993, p. 3-6, as cited in SERA, 2003-Glyphosate). This value may underestimate exposure to small mammals that consume primarily vegetation, rather than seeds (SERA, 2003a). Food consumption is calculated from caloric requirements for different sized animals for the various exposure scenarios in the FS/SERA risk assessments.

Dietary composition: Dietary composition is an important consideration in exposure assessments because different foods have varying herbicide residues. Grasses may have substantially higher residues than fruits or other vegetation (Kenaga, 1973; Fletcher et al. 1994; Pfleeger et al., 1996). The FS/SERA risk assessments use data from Siltanen et al. (1981) for concentrations on fruit. Also, small insects may contain higher residues than large insects, based on empirical relationships (Pfleeger et al., 1996). Some herbicides have the potential to bioaccumulate in fish; therefore fish-eating birds may be exposed. Caloric content of various foods, with caloric requirements of animals, is used to estimate daily amount of food consumed based on data from US EPA/ORD 1993 (as cited in SERA, 2003-Glyphosate). In the FS/SERA risk assessments, exposure scenarios use a large herbivore consuming 100 percent grass diet, a large bird consuming grass, a small bird consuming small insects, and a predatory bird consuming contaminated fish (SERA, 2003-Glyphosate, p. 4-14 to 4-15).

Water ingestion: There are well-established relationships between body weight and water consumption across a wide range of mammalian species. Mice, weighing about 20 g (0.02 kg) consume about 0.005 L of water/day (i.e. 0.25 L/kg/day). These values are used in the exposure scenarios for small mammals. Since the body size to volume relationship dictates that smaller animals will receive larger doses for a given exposure, consumption of contaminated water is not calculated for larger animals. Water ingestion is obviously influenced by environmental factors, such as heat and availability. But estimates for the variability in water consumption are not available for wildlife.

Grooming: Birds and mammals may spend a great deal of time grooming fur or feathers. If the animal has been exposed to herbicide, some chemical may be absorbed through the grooming process. However, a study by Gaines (1969, as cited in SERA, 2001) suggests that grooming is

not significant in the toxic response of small mammals. At any rate, the doses received from grooming would be less than those received through contaminated food or direct spray, given the assumptions in the exposure scenarios. See dermal exposure route information below.

Dermal Route

Dermal contact can occur from direct spray or contact with contaminated vegetation or water. Since only a small portion of an applied herbicide would be available as dislodgeable residue on vegetation, or in a water body where it was diluted, dermal exposure is modeled only for direct spray scenarios in FS/SERA risk assessments. The extent of dermal contact for an animal depends on the application rate of the herbicide, the surface area of the animal, and the rate of absorption. Since a larger proportion of a small animal's body would be involved, relative to larger animals, direct spray scenarios are only conducted for a small mammal and a honeybee in FS/SERA risk assessment (SERA, 2001). Skin, fur, and feathers provide some protection from chemicals, and not all of the chemical on an animal will be absorbed. Amphibians may be an exception, since their skin may be much more permeable than the skin of a mammal or bird. In this document, we assume that the skin affords no protection at all (e.g., 100 percent absorption). Scenarios with a different assumption regarding absorption may be found in the various FS/SERA risk assessments. The approach taken here (100 percent absorption) may account for multiple absorption pathways, such as dermal absorption plus that from grooming or preening. However, there is no quantitative data available regarding this assumption. The actual dose received after dermal exposure is also influenced by the specific herbicide considered since different herbicides have different dermal absorption rates and properties (SERA, 2001, section 3.9).

Summary of Exposure Scenarios

An exposure scenario was developed, and a quantitative estimate of dose received by the animal type in the scenario was calculated when enough data was available (SERA, 2001). While it is possible to model exposure in a very large number of non-target animals, highly species-specific exposure assessments are of little use in the absence of species specific dose-response data (SERA, 2001). The exposure assessment should not be more complicated than the dose-response assessment. Therefore, exposure scenarios used in this document are calculated when dose-response data for specific herbicides indicate that one group and/or size of animal may be more sensitive than others. For example, if data indicates that larger mammals may be more sensitive than smaller mammals, separate exposure scenarios have been developed for each. In the absence of such data, only exposures for small mammals may be calculated because they would receive the highest dose per kg body weight.

The exposure scenarios that are used in the Ecological Risk Assessments (SERA, 2001) and/or for this EIS (project file worksheets) are as follows:

Acute Exposures

20 g mammal: A mouse-sized mammal is directly sprayed over 50 percent of body surface area and 100 percent absorption occurs over one day. A "mouse" consumes contaminated vegetation, daily food consumption equal to 18 percent of body weight (a value between seed diet and vegetation diet needs), and one day's diet is 100 percent contaminated. A "mouse" consumes contaminated insects, daily food consumption equals 50 percent of body weight, and one day's diet is 100 percent contaminated. A "mouse" consumes contaminated water (volume water consumed is based on allometric relationship) after spill of 200 gallons into a small pond (with no dissipation or degradation of the herbicide).

5 kg mammal: A fox-sized animal consumes small mammal prey that has been contaminated by direct spray. Daily food consumption equals 8 percent of body weight.

70 kg mammal: A deer-sized animal consumes contaminated grass (grass has higher herbicide residues), daily food consumption is 14.16 kg/day (equal to 20 percent of body weight), and one day's diet is 100 percent contaminated.

4 kg bird: A goose-sized bird consumes contaminated grass and one day's diet is 100 percent contaminated.

10 g bird: A small, passerine-sized bird consumes contaminated small insects and one day's diet is 100 percent contaminated.

Predatory bird: A bird-of-prey consumes fish that has been contaminated by an accidental spill of 200 gal into a small pond. Assumptions used include no dissipation of herbicide, bioconcentration is equilibrium with water, contaminant level in whole fish is used, and upper estimate assumes 15 percent of body weight eaten/day. A spotted-owl sized bird consumes small mammal prey that has been contaminated by direct spray.

Terrestrial invertebrate: A honeybee (0.093g) is directly sprayed and 100 percent absorption occurs over one day.

Chronic Exposures

20 g mammal: A mouse-sized mammal consumes contaminated vegetation for 90 days (upper estimate assumes 20 percent of diet is contaminated), and the herbicide dissipates over time. A "mouse" consumes contaminated ambient water for an extended period.

70 kg mammal: A deer-sized mammal consumes contaminated grass for 90 days (upper estimate assumes 100 percent of diet is contaminated), and the herbicide dissipates over time.

4 kg bird: A goose-sized bird consumes contaminated grass for 90 days (upper estimate assumes 100 percent of diet is contaminated), and herbicide dissipates over time.

Predatory bird: A bird-of-prey consumes fish from contaminated water over a lifetime. Assumptions used include dissipation and degradation of herbicide is considered, bioconcentration is equilibrium with water, contaminant level in whole fish is used, and upper estimate assumes 15 percent of body weight eaten/day.

No data are available to estimate chronic exposures from contaminated insects or mammal prey, so risk from chronic exposure is estimated using the acute dose compared to the chronic toxicity index.

In this document, only the highest ranges of exposure assumptions are included, although a more complete range of possible values is included in the SERA risk assessments. For example, for a given herbicide, residues of the herbicide on vegetation that are reported in the literature will vary between studies and by vegetation type. A range of residue rates is used in the SERA risk assessment worksheets, but only the highest reported rates are used in the data reported here. Only the highest values are used here to reduce length and complexity of this document and also to present a reasonable "worst-case" exposure analysis.

Estimated doses from the above exposure scenarios are compared to toxicity levels from laboratory research. The lowest reported dose that caused the most sensitive effect in the most

sensitive species is used in this analysis to indicate the potential for an adverse effect when that dose is exceeded. These doses are referred to as "toxicity indices" in this document, and NOAEL's are used whenever possible. If available data have not identified a NOAEL, then an LD_{50} or other level may be used. Table C- 16 lists the toxicity indices for mammals and Table C-17 lists the toxicity indices for birds.

Following the tables are summaries of herbicide effects to birds and mammals, reptiles, amphibians, and terrestrial invertebrates based on the results of the analysis and information in the literature. The likelihood that potential adverse effects would occur is then discussed followed by a brief summary of some of the available field studies. The document concludes with detailed descriptions of the exposure scenario results for each scenario and herbicide.

Table C- 16. Toxicity indices for mammals used in the effects analysis. Indices represent the most sensitive endpoint from the most sensitive species for which adequate data are available.

Herbicide	Duration	Endpoint	Dose	Species	Effect Noted at LOAEL
Chlorsulfuron	Acute	NOAEL	75 mg/kg	Rabbit	Decreased weight gain at 200 mg/kg
	Chronic	NOAEL	5 mg/kg/day	Rat	Weight changes at 25 mg/kg/day
Clopyralid	Acute	NOAEL	75 mg/kg	Rat	Decreased weight gain at 250 mg/kg
	Chronic	NOAEL	15 mg/kg/day	Rat	Thickening of gastric epithelium at 150 mg/kg/day
Glyphosate	Acute	NOAEL	175 mg/kg	Rabbit	Diarrhea at 350 mg/kg
	Chronic	NOAEL	175 mg/kg/day	Rabbit	Diarrhea at 350 mg/kg
Imazapic	Acute	NOAEL	350 mg/kg	Rabbit	Decreased body weight at 500 mg/kg
	Chronic	NOAEL[2]	45 mg/kg	Dog	Microscopic muscle effects at 137 mg/kg
Imazapyr	Acute	NOAEL	250 mg/kg	Dog	No effects at highest doses tested
	Chronic	NOAEL	250 mg/kg/day	Dog	No effects at highest doses tested
Metsulfuron methyl	Acute	NOAEL[3]	25 mg/kg	Rat	Decreased weight gain at 500 mg/kg
	Chronic	NOAEL	25 mg/kg/day	Rat	Decreased weight gain at 125 mg/kg
Picloram	Acute	NOAEL	34 mg/kg	Rabbit	Decreased weight gain at 172 mg/kg
	Chronic	NOAEL	7 mg/kg	Dog	Increased liver weight at 35 mg/kg[4]
Sethoxydim	Acute	NOAEL	160 mg/kg[5]	Rabbit	Reduced number of viable fetuses, some dam mortality at 480 mg/kg
	Chronic	NOAEL	9 mg/kg/day	Dog	Mild anemia at 18 mg/kg/day
Sulfometuron methyl	Acute	NOAEL	87 mg/kg	Rat	Decreased body weight at 433 mg/kg
	Chronic	NOAEL	2 mg/kg/day	Rat	Effects on blood and bile ducts at 20 mg/kg/day
Triclopyr[6]	Acute	NOAEL	100 mg/kg	Rat	Malformed fetuses at 300 mg/kg
	Chronic[7]	NOAEL	0.5 mg/kg/day	Dog	Effect on kidney at 2.5 mg/kg/day
NPE Surfactants	Acute	NOAEL	10 mg/kg	Rat	Slight reduction of polysaccharides in liver at 50 mg/kg/day
	Chronic	NOAEL	10 mg/kg/day	Rat	Increased weights of liver, kidneys, ovaries, and decreased live pups at 50 mg/kg/day

1 Small animals are less susceptible than larger animals. NOAEL estimated from LOAEL of 300 mg/kg/day for neurotoxic effects, using safety factor of 10 to extrapolate from a LOAEL to a NOAEL. Identical to observed NOAEL for neurotoxicity in rabbits (Hoberman 1992).
2 Imazapic – NOAEL calculated from a LOAEL of 137 mg/kg/day and application of a safety factor of 3 to extrapolate from a LOAEL to a NOAEL.
3 The acute NOAEL of 24 mg/kg is very close to the chronic NOAEL, so chronic value is used for acute exposures as well.
4 USEPA/OPP 1998
5 Source of the value used by EPA (180 mg/kg) is not well documented, so the lower value of 160 mg/kg from a rabbit study is used as the toxicity index for this analysis (BASF 1980, MRID 00045864 cited in SERA, 2003-Triclopyr).
6 Triclopyr BEE and TEA have equal toxicities to mammals (SERA, 2003a).
7 Value taken from Quast et al. 1976 as cited in SERA Triclopyr 2003. This represents an extremely conservative approach, explained in more detail in the write up on triclopyr later in this document.
Source: SERA 1998, 2001, 2003, 2004 and USDA FS 2003.

Table C- 17. Toxicity indices for birds used in the effects analysis. Indices represent the most sensitive endpoint from the most sensitive species for which adequate data are available.

Herbicide	Duration	Endpoint	Dose	Species	Effects Noted at LOAEL
Chlorsulfuron	Acute	NOAEL	1686 mg/kg	Quail	No significant effects at highest dose
	Chronic	NOAEL	140 mg/kg/day	Quail	No significant effects at highest dose
Clopyralid	Acute	NOAEL	670 mg/kg	Mallard & Quail	No signs of toxicity reported, LOAEL not determined
	Chronic[1]	NOAEL	15 mg/kg/day	Rat	Thickening of gastric epithelium at 150 mg/kg/day
	Chronic	NOAEL	13.6 mg/kg/day[2]	Quail	Neurotoxic effects at 27 mg/kg/day
Glyphosate	Acute	NOAEL	562 mg/kg	Mallard & Quail	No effects at highest dose
	Chronic	NOAEL	100 mg/kg	Mallard & Quail	No effects on reproduction at highest dose
Imazapic	Acute	NOAEL	1100 mg/kg	Quail	No effects at highest dose
	Chronic	NOAEL	113 mg/kg/day	Quail	Decreased weight gain in chicks at 170 mg/kg/day
Imazapyr	Acute	NOAEL	674 mg/kg	Quail	No effects at highest dose
	Chronic	NOAEL	200 mg/kg/day	Mallard & Quail	No effects at highest dose
Metsulfuron methyl	Acute	NOAEL	1043 mg/kg	Quail	No significant effects at highest dose
	Chronic	NOAEL	120 mg/kg/day	Mallard & Quail	No significant effects at highest dose
Picloram	Acute	NOAEL	1500 mg/kg	Chicken & pheasant	No effect to reproduction. LOAEL not reported
	Chronic[3]	NOAEL	7 mg/kg/day	Dog	Increased liver weight at 35 mg/kg/day
Sethoxydim	Acute	NOAEL	>500 mg/kg	Mallard & Quail	No or low mortality at highest doses tested. LOAEL not available.
	Chronic	LOAEL[4]	10 mg/kg/day	Mallard	Decreased number of normal hatchlings at 10 mg/kg/day
Metsulfuron methyl	Acute	NOAEL	1043 mg/kg	Quail	No significant effects at highest dose
	Chronic	NOAEL	120 mg/kg/day	Mallard & Quail	No significant effects at highest dose
Triclopyr BEE[6]	Acute	LD_{50}	388 mg/kg	Quail	50% mortality at 388 mg/kg
	Chronic	NOAEL	10 mg/kg/day	Mallard & quail	Decreased survival of offspring, reduced eggshell thickness at 20 mg/kg/day
Triclopyr TEA	Acute	LD_{50}	535 mg/kg	Quail	50% mortality at 535 mg/kg
	Chronic	NOAEL	10 mg/kg/day	Mallard & Quail	Decreased survival of offspring, reduced eggshell thickness at 20 mg/kg/day
	Chronic[7]	NOAEL	1 mg/kg/day	Rat & dog	Effects on kidney, blood,

Herbicide	Duration	Endpoint	Dose	Species	Effects Noted at LOAEL
					and liver at 5 mg/kg/day
NPE Surfactants[9]	Acute	NOAEL	10 mg/kg	Rat	Slight reduction of polysaccharides in liver at 50 mg/kg/day
	Chronic	NOAEL	10 mg/kg/day	Rat	Increased weights of liver, kidneys, ovaries, and decreased live pups at 50 mg/kg/day

1 Chronic toxicity studies in birds are not available, so the value from mammal studies is used.
2 Higher reported NOAEL for chronic dietary exposure is 92 mg/kg/day, with no signs of neurotoxicity. The lower value from acute exposures is used in FS/SERA risk assessment for chronic exposures as a more protective toxicity index.
3 Chronic toxicity studies in birds are not available, so the value from mammal studies is used.
4 Based on one study in which a NOAEL was not determined, so the LOAEL is used.
5 Birds may be somewhat less sensitive than mammals, but data are limited, so the lower value from mammal studies is used.
6 Unlike in mammals, the toxicities of triclopyr BEE and triclopyr TEA are different for birds, so the indices of the two forms of triclopyr are presented separately
7 Weed Science Society of America 2002.
8 No chronic toxicity data for birds is available; so the mammal chronic value is used.
9 Data on birds is not available in published literature. This information from an unpublished study referred to in USDA FS 2003. Since information is lacking, this value is used for illustrative purposes only and no attempt is made to quantify risk to birds from NPE surfactants.
Source: SERA 1998, 2001, 2003, 2004; USDA FS 2003; and Weed Science Society of America 2002.

Summary of Herbicide Effects to Birds and Mammals

The data available for mammals are derived from numerous studies conducted to meet registration requirements, and primarily on laboratory animals that serve as surrogates. Data for mammals are available for more types of toxicity tests and often on a wider variety of species than are available for birds.

Availability of information on the direct toxicological effects of the 12 herbicides on wild mammals varies by herbicide. Glyphosate has been widely studied, including field applications. Little or no data on wildlife may exist for other herbicides. Herbicides have been tested on only a limited number of species under conditions that may not well-represent populations of free-ranging animals (SERA 1998, 2001, 2003).

Toxicity data available for birds are derived from studies conducted to meet registration requirements, and primarily on domestic birds that serve as surrogates. There are typically fewer types of toxicity studies conducted on birds using a more restricted variety of species than are conducted for mammals. Almost all laboratory data is collected on mallards and northern bobwhite. How the sensitivities of different bird species to herbicides may vary from that reported for mallard and bobwhite is not known.

Tables 5 and 6 summarize the results of exposure scenarios for the 12 herbicides and NPE surfactants considered in this analysis. Chlorsulfuron, imazapic, imazapyr, and metsulfuron methyl do not appear to pose any plausible risk to terrestrial wildlife or bees at either the typical or highest application rates. When an herbicide does pose plausible risk, it is consistently insectivorous and grass-eating animals that are most likely to receive doses above the toxicity index. Direct spray of mammals is a concern only for NPE surfactants at the typical application rate.

Fish-eating birds do not receive a dose above the toxicity index for any herbicide or application rate. Consumption of contaminated water, even as the result of an accidental spill, results in doses well below the toxicity index for all herbicides. For the herbicides considered in this

analysis, birds are less sensitive than mammals to acute exposures. Chronic toxicity data on birds is often limited.

Triclopyr has the highest potential to adversely affect wildlife. Triclopyr TEA and BEE are somewhat more toxic to birds than triclopyr acid. The toxicities of these compounds to mammals show no remarkable differences. Triclopyr can be acutely lethal only at very high doses. However, indications of adverse effects to the kidney can occur at very low doses, at least in dogs. These adverse effects are indicated by increases in blood urea nitrogen and creatinine in dogs, but no histopathological changes to the kidneys were found. Triclopyr exposures exceed the toxicity indices for eight scenarios at the typical application rate, and 12 scenarios at the highest application rate.

Glyphosate, applied at the typical application rate has little potential to adversely affect birds or mammals. An exception might be insectivorous birds that experience chronic exposures. There are no data available on the persistence or degradation of glyphosate residue on insects, so the acute dose is compared to the chronic toxicity index. This is an extremely protective approach and may greatly overestimate risk. However, it is worth noting so that appropriate protective measures may be taken when using glyphosate in the habitat of insectivorous birds. At the highest application rate, glyphosate has the potential to adversely affect large grass-eating mammals, and insectivorous birds and mammals in acute and chronic exposures. Additionally, grass-eating birds may be adversely affected in a chronic exposure. In total, glyphosate exposures exceed the toxicity indices for one scenario at the typical application rate, and eight exposures at the highest application rate.

Clopyralid, applied at the typical application rate has little potential to adversely affect birds or mammals, except for insectivorous birds and mammals. There are no data available on the persistence or degradation of clopyralid residue on insects, so the acute dose is compared to the chronic toxicity index. This is an extremely protective approach and may greatly overestimate risk. However, it is worth noting so that appropriate protective measures may be taken when using clopyralid in the habitat of insectivorous birds and mammals. At the highest application rate, clopyralid may adversely affect grass-eating birds, insectivorous birds and mammals and predatory birds eating small mammal prey for chronic exposures.

The same qualification for chronic exposure to insectivorous animals applies to predatory birds, in that the acute dose is compared to the chronic toxicity index. No acute exposures exceed the toxicity indices. In total, clopyralid exposures exceed the toxicity indices for one exposure at the typical application rate, and four at the highest application rate.

The actual likelihood of exposing specific bird or mammal species depends on the application method, size of treatment area, habitat treated, and season of application, and must be analyzed at the site-specific level. Table C- 18, exposure scenario results from FS/SERA risk assessments for mammals, birds, and honeybees using the typical application rate and upper residue rates.

Table C- 18. Exposure scenario results from FS/SERA risk assessments for mammals, birds, and honeybees using the typical application rate

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
ACUTE EXPOSURES											
Direct spray, bee	--	--	--	--	--	--	--	--	--	--	
Direct spray, sm. mammal	--	--	--	--	--	--	--	--	--	--	*
Consume Contaminated Vegetation											
small mammal	--	--	--	--	--	--	--	--	--	--	--
large mammal	--	--	--	--	--	--	--	--	--	--	*
large bird	--	--	--	--	--	--	--	--	--	*	*
Consume Contaminated Water											
Spill, sm. mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects											
small mammal	--	--	--	--	--	--	--	--	--	--	*
small bird	--	--	--	--	--	--	--	--	--	*	*
Consume Contaminated Prey											
carnivore (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--
CHRONIC EXPOSURES											
Consume Contaminated Vegetation											
small mammal, on site	--	--	--	--	--	--	--	--	--	--	--
lg. mammal, on site	--	--	--	--	--	--	--	--	--	*	--
lg. bird, on site	--	--	--	--	--	--	--	--	--	*	--
Consume Contaminated Water											
small mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects#											
small mammal	--	unk	--	--	--	--	unk	unk	unk	unk	unk
small bird	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
Consume Contaminated Prey											
carnivore (sm. mammal)#	--	--	--	--	--	--	--	--	--	*	--
predatory bird (sm. mammal)#	--	--	--	--	--	--	--	--	--	--	--
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--

*Includes scenario for direct spray of a rabbit-sized mammal.
Data is lacking regarding chronic exposures, so effects are assumed by comparing acute dose vs. chronic NOAEL, and will likely over-estimate actual risk.
unk – unknown; insufficient data to assess risk

Table C- 19. Exposure scenario results from FS/SERA risk assessments for mammals, birds, and honeybees using the highest application rate and upper residue rates.

Animal/Scenario	Chlorsulfuron	Clopyralid	Glyphosate	Imazapic	Imazapyr	Metsulfuron methyl	Picloram	Sethoxydim	Sulfometuron methyl	Triclopyr	NPE Surfactant
ACUTE EXPOSURES											
Direct spray, bee	--	--	♦	--	--	--	--	--	--	♦	
Direct spray, sm. mammal	--	--	--	--	--	--	--	--	--	--	♦
Consume Contaminated Vegetation											
small mammal	--	--	--	--	--	--	--	--	--	--	♦
large mammal	--	--	♦	--	--	--	--	--	--	♦	♦
large bird	--	--	--	--	--	--	--	--	--	♦	♦
Consume Contaminated Water											
Spill, sm. mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects											
small mammal	--	--	♦	--	--	--	♦	--	--	♦	♦
small bird	--	--	♦	--	--	--	--	--	--	♦	♦
Consume Contaminated Prey											
carnivore (sm. mammal)	--	--	--	--	--	--	--	--	--	--	--
predatory bird (sm. mammal)	--	--	--	--	--	--	--	--	--	--	♦
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--
CHRONIC EXPOSURES											
Consume Contaminated Vegetation											
small mammal, on site	--	--	--	--	--	--	--	--	--	--	--
lg. mammal, on site	--	--	--	--	--	--	--	--	♦	♦	--
lg. bird, on site	--	♦	♦	--	--	--	--	♦	♦	♦	--
Consume Contaminated Water											
small mammal	--	--	--	--	--	--	--	--	--	--	--
Consume Contaminated Insects#											
small mammal	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
small bird	--	unk	unk	--	--	--	unk	unk	unk	unk	unk
Consume Contaminated Prey											
carnivore (sm. mammal)#	--	--	--	--	--	--	--	--	--	♦	♦
predatory bird (sm. mammal)#	--	--	--	--	--	--	--	♦	--	♦	♦
predatory bird (fish)	--	--	--	--	--	--	--	--	--	--	--

-- Exposure scenario results in a dose below the toxicity index.
♦ Exposure scenario results in a dose that exceeds the toxicity index.
* Includes scenario for direct spray of a rabbit-sized mammal.
# Data is lacking regarding chronic exposures, so effects are assumed by comparing acute dose vs. chronic NOAEL, which will likely over-estimate actual risk.

Herbicide Effects on Reptiles

There is almost no data available regarding the toxicity of herbicides to reptiles. In a review of pesticide effects to reptiles, Pauli and Money (2000) found very few studies, despite publications stating the need for such research dating back to Hall (1980). The only information available for herbicides included in this document is from two reports concerning 2,4-D. One study investigated the effects of 2,4-D on alligators (Crain et al. 1997, as cited by SERA 1998), and Willemsen and Hailey (1989, cited by Pauli and Money 2000) noted adverse effects to tortoises in Greece after application of 2,4,5-T and 2,4-D. Pauli and Money (2000) concluded, "it is remarkable that no data appear to exist concerning the effects on reptiles of field applications of… modern herbicides (e.g., glyphosate, sulfonylureas)…"

Hall and Henry (1992) stated, "Susceptibility of reptiles to selective pesticides is virtually unknown."

Hall and Clark (1982) found that the green anole lizard (*Anolis carolinenesis*) had a similar sensitivity as mallards and rats to organophosphates. Conversely, reptiles were reported to be more sensitive to some pesticides than birds or mammals (Rudd and Genelly 1956, as cited in Hall 1980). Hall (1980) stated that reptiles are apparently less sensitive than fish. The FS/SERA risk assessments use amphibians and/or fish as surrogates for reptiles. An assumption is made that exposures and doses that are protective of amphibians and fish would also be protective of reptiles. Amphibians and fish have very permeable skin, more so than reptiles, so they are more likely to absorb contaminants from their environment. And their complicated life cycle that includes metamorphosis makes amphibians sensitive indicators for environmental effects (Cowman and Mazanti, 2000). However, the lack of data from reptiles leads to substantial uncertainty in the risk assessment for reptiles, since the response of these animals to doses of herbicide is not known.

Many reptile species would likely be under some cover during the day, when herbicides may be applied. But diurnal reptiles, like lizards, could conceivably be sprayed during applications. Nocturnal and diurnal reptiles could be exposed through contact with contaminated vegetation and soil or ingestion of contaminated prey. Contaminated water or prey could expose aquatic reptiles, but direct spray is not likely. The actual likelihood of exposing reptiles depends on the application method, size of treatment area, habitat treated, and season of application, and must be analyzed at the site-specific level.

Herbicide Effects on Amphibians

Data on toxicity of herbicides to amphibians are limited. Several studies have found that amphibians are less sensitive, or about as sensitive, as fish to some herbicides (Berrill et al. 1994; Berrill et al. 1997; Johnson 1976; Mayer and Ellersieck 1986; Perkins et al. 2000). Consequently, separate dose-response assessments from exposure scenarios have not been created for amphibians in the FS/SERA risk assessments. Available information on toxicity of herbicides to amphibians is summarized below.

Neither the published literature nor the EPA files include data regarding the toxicity of chlorsulfuron, clopyralid, imazapic, imazapyr, metsulfuron methyl, picloram, or sethoxydim to amphibian species. However, data for other aquatic species indicate that chlorsufuron, clopyralid, imazapic, imazapyr, metsulfuron methyl, and picloram have a very low potential to cause any adverse effect in aquatic animals (SERA 2003 Chlorsulfuron; SERA, 2003-Clopyralid; SERA, 2003-Imazapic; SERA, 2003-Imazapyr; SERA, 2003-Metsulfuron methyl; SERA, 2003-Picloram). The formulation Poast is much more toxic to aquatic organisms than sethoxydim.

However, even considering the higher toxicity of Poast, there is no indication that aquatic animals are likely to be exposed to concentrations that would result in toxic effects. There is a substantial limitation to this risk characterization in that no chronic toxicity studies on aquatic animals are available for either sethoxydim or Poast (SERA, 2001 Sethoxydim).

Glyphosate

Glyphosate isopropylamine (IPA), RoundUp and POEA surfactant used in RoundUp have been specifically tested for ability to cause malformations in the frog embryo teratogenesis assay using Xenopus (Perkins et al. 2000). Xenopus is a highly sensitive assay species for determining the teratogenicity of chemicals (Mann and Bidwell 2000, Perkins et al. 2000). No increases in malformations were noted at levels that were not also lethal to the embryos. The RoundUp formulation containing POEA surfactant was 700 times mores toxic than glyphosate IPA. POEA surfactant alone was more toxic than the RoundUp formulation. No statistically significant increases in abnormalities were seen in any groups exposed to POEA at levels that were not also lethal. The 96-hour LC_{50} for glyphosate IPA was 7297 mg a.e./L, and that for RoundUp was 9.3 mg a.e./L. Perkins et al. (2000) calculated that if RoundUp was applied at the highest application rate directly to water 15 cm deep (volumn not specified), the expected environmental contamination was less than the LC_{50} and the LC_5 by a factor of about three.

A study by Smith (2001) looked at effects to western chorus frog (Pseudacris tiseriata) and Plains leopard frog (Rana blairi) from a formulation of glyphosate that contains glyphosate IPA and ethoxylated tallowamine surfactant (Kleeraway Grass and Weed Killer RTU (Monsanto)). Smith exposed 1-week old tadpoles for 24-hours to the following concentrations of Kleeraway: 0.1 (1 part Kleeraway to 9 parts deionized water), 0.1, 0.001, and 0.0001. These concentrations are equivalent to 560 mg a.e./L, 56 mg a.e./L, 5.6 mg a.e./L, and 0.56 mg a.e./L (SERA, 2003-Glyphosate, p. 4-20). Smith reported some mortality at concentrations as low as 0.56 mg a.e./L for both species. Acute exposure to Kleeraway had no effect on growth or development of surviving tadpoles. Results found by Smith are not consistent with other information on the effects of glyphosate or other formulations to amphibians. However, other studies have found that different formulations can have different toxicities to frogs (Mann and Bidwell, 1999). Formulations containing surfactant are known to have much higher toxicity to amphibians than glyphosate. The Forest Service does not use the formulation used in the Smith study.

Bidwell and Gorrie (1995; cited in SERA 2003 Glyphosate) reported 48-hour LC_{50} values of 11.6 mg a.e./L for the Roundup 360 formulation and 121 mg/L for technical grade glyphosate using four species of frogs from western Australia.

At the typical application rate, expected water concentrations for acute and longer-term exposures are well below any reported LC_{50} for amphibians, with the exception of the study by Smith (2001) (SERA, 2003-Glyphosate, Worksheet G03). At the highest application rate, lethal doses could occur from formulations containing surfactant.

Sulfometuron methyl

The effect of sulfometuron methyl to amphibians was investigated in one study using Xenopus (Fort 1998; cited in SERA 2003 Sulfometuron methyl). Results of the study found that sulfometuron methyl exposure can cause moderately severe malformations in these frogs, including miscoiling of the gut, incomplete eye lens formation, abnormal craniofacial development, and decreased tail resorption. The concentration that produced these effects depended upon the length of exposure, with shorter exposures showing no effect at higher

concentrations than longer exposures. The author did not sate whether data were reported in terms of mg of sulfometuron methyl or mg of Oust. The FS/SERA risk assessment assumes that data refer to mg of Oust, to provide the most protection. The NOAEC for malformations for 4-hour exposure is 0.38 mg a.i./L, and that for 30-day exposure is 0.0075. However, exposure to 0.0075 mg a.i./L for 14 days was identified as the LOAEC for tail resorption rate effects. No mortality was observed at concentrations up to 7.5 mg a.i./L.

Unlike the other FS/SERA risk assessments, a quantitative evaluation of exposure and risk from sulfometuron methyl was conducted for amphibians. SERA (2003 Sulfometuron methyl) compared estimated water concentrations for acute and chronic exposures to acute and chronic NOEC values for frogs, from Fort (1998). The estimated exposure is 0.002 of the acute NOEC, and 0.00075 of the chronic NOEC. Therefore, at the typical and highest application rates, there is no basis for asserting or predicting that adverse effects to amphibians are plausible. There is a substantial reservation in that this conclusion is based on data from one species, but other studies have indicated that Xenopus are a sensitive indicator for effects to amphibians (Mann and Bidwell 2000, Perkins et al. 2000).

Triclopyr

Triclopyr BEE is much more toxic to aquatic species that triclopyr TEA or triclopyr acid (SERA 2003 Triclopyr). Triclopyr was specifically tested for ability to cause malformations in the frog embryo teratogenesis assay using Xenopus laevis (Perkins et al. 2000). Xenopus is a highly sensitive assay species for determining the teratogenicity of chemicals (Mann and Bidwell 2000, Perkins et al. 2000). No statistically significant increase in abnormalities were seen in any groups exposed to Garlon 3A or Garlon 4 at levels that were not also lethal to the embryos. Consistent with results for other aquatic species, Garlon 3A, containing triclopyr TEA, was 15 times less toxic than Garlon 4, containing triclopyr BEE. Garlon 4 reduced embryo growth at a concentration below the LC_{50}. Perkins et al. (2000) found that the 96-hour LC_{50} for Garlon 4 was 10 mg a.e./L, and that for Garlon 3A was 159 mg a.e./L. Perkins et al. (2000) calculated that if Garlon 4 was applied at the highest application rate directly to water 15 cm deep (volume not specified), the expected environmental contamination was less than the LC_{50} and the LC_5 by a factor of about four and three, respectively. 30

Berrill et al. (1994) conducted toxicity studies on eggs and tadpoles of leopard frog (Rana pepiens), green frog (Rana clamitans), and bullfrog (Rana catesbeiana) exposed to technical grade triclopyr BEE. The study was conducted in darkness to prevent hydrolysis of triclopyr BEE to tricolopyr acid. Exposure of eggs to concentrations up to 4.6 ppm triclopyr a.e. for 48 hours caused no effect on hatching success, timing, malformations, or subsequent avoidance behavior of tadpoles hatched from exposed eggs (Berrill et al. 1994). Tadpoles were more sensitive; all bullfrog and green frog tadpoles exposed to 2.3 and 4.6 ppm triclopyr a.e. died. Leopard frogs were more tolerant and few died, but all were unresponsive to prodding at 2.3 and 4.6 ppm a.e. About half the bullfrog and most green frog tadpoles became unresponsive to prodding when exposed to 1.1 ppm a.e. Surviving tadpoles recovered after exposure was terminated.

Water concentrations from application of triclopyr acid at the typical application rate are below 1 mg/L (1 ppm), so acute and chronic risks to aquatic animals are low (SERA, 2003-Triclopyr, Worksheet G03). At the highest application rate, acute exposure from runoff could adversely affect responsiveness of some tadpoles, increasing the risk of predation. Despite the difference in toxicity, the conclusion is the same for triclopyr BEE, due to the difference in estimated water concentration.

Herbicide Effects on Invertebrates

Manufacturers are required to conduct toxicity tests on honeybees as part of the registration process. The estimated doses and toxicity values of the herbicides to honey bees are listed in

Table C- 20. The inclusion of other terrestrial invertebrates in toxicity studies varies for each herbicide. However, even the most -studied will include effects on only a small fraction of terrestrial invertebrate species potentially found in any diverse ecosystem. Risk to invertebrates can only be inferred based on the few test species for which data are available.

Effects of chlorsulfuron to terrestrial invertebrates have been studied using a leaf beetle (Gastrophysa polygoni), large whitebutterfly (Pieris brassicae), and nemotodes (SERA, 2003-Chlorsulfuron). Direct spray of first-instar larva and feeding of larva on treated plants did not produce significant changes in mortality, but did delay development of those feeding on treated plants. Placing eggs of the leaf beetle on treated plants significantly decreased survival (Kjaer and Elmegaard, 1996; cited in SERA, 2003-Chlorsulfuron). In another study (Kjaer and Heimbach, 2001), newly hatched larvae of the leaf beetle and whitebutterfly were placed on treated plants and no significant effects on survival or relative growth rates were found. Two species of nematodes (Steinernema carpocapsae and S. feltiae) were exposed to chlorsulfuron in soil and no effect was observed on reproduction, viability, or movement (Rovesti and Desco, 1990; cited in SERA 2003-Chlorsulfuron). A British publication (Tomlin, 2000) reports an LD_{50} > 25mg/kg for honey bees, but it is not clear what research provides the basis for this value.

Clopyralid has been tested on a variety of terrestrial invertebrates. Standard bioassays on honeybees (LD_{50} >90 mg/kg) have been conducted as well as exposure of earthworms to clopyralid in soil (LC_{50} >1000 ppm). Also, Hassan et al. (1994) provided a summary of several bioassays and field trials using a variety of terrestrial invertebrates. Clopyralid produced some mortality in insect parasites, predatory mites, Semiadalia 11-notata (Coccinellidae), Anthocoris nemoralis (Anthocoridae), and Chrysoperla carnea (Chrysopidae). Pekar et al. (2002; cited in SERA 2003 Clopyralid) reported that clopyralid was “harmless” to wild immature spiders (*Theridion impressum*).

There is a low potential for glyphosate to adversely affect terrestrial invertebrates. The honeybee LD_{50} for glyphosate is greater than 1075 mg/kg and the NOEC is 540 mg/kg. Mortality at 134 mg/kg in one study was attributed to equipment failure (SERA, 2003-Glyphosate). Direct foliar spray had no effect on the spider mite (Tetranchys urticae). One-hundred percent mortality to spider mites was reported after application of RoundUp ULTRA at 3.6 kg a.i./ha, but it was attributed to the solution causing the mites to stick to the glass plates. Studies of the effects of glyphosate on the spider Lepthyphantes tenuis resulted in no effects that could be attributed to glyphosate toxicity. No significant effects were noted in studies on rove beetles, butterflies, or terrestrial snail (Helix aspersa). The soil LC_{50} for a worm common in Libya, Aporrectodea caliginosa, is 177-246 mg glyphosate/kg soil (Mohamed et al., 1995; cited in SERA, 2003-Glyphosate).

Table C- 20. Potential herbicide doses for bees in a direct spray scenario, assuming 100% absorption.

Herbicide	Typical Application Rate	Dose for Bee	Toxicity Index for Bee
Chlorsulfuon	0.056 lb/ac	8.98 mg/kg	>25 mg/kg (LD_{50})
Clopyralid	0.35 lb/ac	56.1 mg/kg	909 mg/kg (no mortality)
Glyphosate	2.0 lb/ac	321 mg/kg	540 mg/kg (NOAEC)
Imazapic	0.13 lb/ac	16 mg/kg	387 mg/kg (no mortality)
Imazapyr	0.45 lb/ac	72.1 mg/kg	1000 mg/kg (no mortality)
Metsulfuron Methyl	0.03 lb/ac	4.81 mg/kg	270 mg/kg (NOEC)
Picloram	0.35 lb/ac	56.1 mg/kg	1,000 mg/kg (no mortality)
Sethoxydim	0.3 lb/ac	60.1 mg/kg	107 mg/kg (NOAEL)
Sulfometuron Methyl	0.045 lb/ac	7.21 mg/kg	1,075 mg/kg (NOEC)
Triclopyr BEE	1.0 lb/ac	160 mg/kg	>1,075 mg/kg (LD_{50})
Triclopyr TEA	1.0 lb/ac	160 mg/kg	>1,075 mg/kg (LD_{50})
NP9E	1.67 lbs/ac	268.00 mg/kg	unknown

Source: SERA 1996-2003 and USDA FS 2003.
1 Standard acute toxicity studies using bees were not identified in a complete search of studies submitted to EPA. Tomlin (2000) reports bee LD50 > 25 mg/kg in a British pesticide manual. Another study found no mortality to a leaf-eating beetle directly sprayed at a rate corresponding to 107 lb/ac (SERA 2003 Chlorsulfuron).

The standard acute toxicity study to honeybees is the only study found on the effects of imazapic to terrestrial invertebrates. At 387 mg/kg, mortality was not statistically significant (SERA, 2003-Imazapic).

Imazapyr has a low acute toxicity to bees with an LD_{50} >1000 mg/kg. No information on effects to other terrestrial invertebrates is available.

Standard bioassays on effects of metsulfuron methyl to honeybees reported LD_{50} > 1075 mg/kg and a NOAEL of at least 270 mg/kg. Very high application rates (almost five times higher than the highest labeled application rate) resulted in a 15 percent reduction in eff hatching for rove beetle (Samsoe-Petersen 1995; cited in SERA 2003 Metsulfuron methyl).

Data on the toxicity of picloram to terrestrial invertebrates is available only for the honeybee and the brown garden snail (Helix aspersa). The honeybee LD50 is greater than 1000 mg/kg and dietary concentration of 5000 mg/kg over a 14-day period did not increase mortality for the snail.

For sethoxydim, the honeybee NOAEL is 107 mg/kg. The only other study on invertebrates investigated effects to Mexican bean beetle (Epilachna varivestis) feeding on soybean and lima bean plants treated with the equivalent of 5-6 lbs/acre (15 times higher than the highest labeled application rate). There was a slight increase in days to pupation for larvae, but also significant increases in both the number of egg masses as well as total number of eggs produced by beetles feeding on sethoxydim treated plants (Agnello et al. 1986; cited in SERA 2001 Sethoxydim).

Only two studies are available on the toxicity of sulfometuron methyl to terrestrial invertebrates and they both looked at effects to the honeybee. Sulfometuron methyl has a very low potential to

adversely affect bees, with an acute NOAEL of 1075 mg/kg (SERA, 2001-Sulfometuron methyl). No mortality was reported at the highest doses tested.

Honeybee assays provide the only information on the effects of triclopyr acid and triclopyr TEA to terrestrial invertebrates. In both bioassays, the LD_{50} is greater than 1075 mg/kg (SERA, 2003-Triclopyr). 33

The actual likelihood of exposing invertebrates depends on the application method, size of treatment area, habitat treated, and season of application, and must be analyzed at the site-specific level.

Likelihood these exposures and effects will actually occur

While the above exposure scenarios consider animal sizes, feeding habits, herbicide application rates, and toxicity data, they cannot account for all the variables found in the field during actual applications. Such factors as foliar interception, animal behavior (e.g. nocturnal versus diurnal activity), season of use, and selective application methods can significantly reduce or eliminate actual exposure to herbicides in field conditions. For example, while toxicity of some herbicides could pose a concern for the early stages of amphibian development, an actual application of herbicide occurring after mid-summer, well after this stage of development might be present at a specific location, could significantly reduce risk (Perkins et al., 2000).

Direct spray of small mammals is very unlikely to occur, since they are typically nocturnal and spend the day in burrows, nests, or underneath dense vegetation. Diurnal small mammals, such as ground squirrels, may be active in treatment areas, but would likely seek shelter or move away from the treatment activity. Aerial application could directly spray some diurnal small mammals. The likelihood that a predatory bird or mammal would prey on the same small mammal that had been directly sprayed is remote, and an entire day's diet of contaminated small mammals is very remote.

Direct spray of insects could occur, as they are present in vegetation and would not necessarily flee during treatment operations. However, foliar interception would reduce the actual amount sprayed on almost all insects present. Insectivorous birds may establish territories during the breeding season. If the treatment area involved most of one or several territories, it could be feasible for an insectivorous bird to consume all or most of its daily diet within the treatment area. The young of even herbivorous bird species are highly dependant upon insects for their growth and development. Therefore, while the actual doses received by insectivorous birds may be lower than the exposure scenarios predict, due to foliar interception, application method and other variables, the consumption of contaminated insects by young birds may offset this advantage. Consumption of contaminated insects remains a concern for some herbicides, and likelihood of exposure must be evaluated at the site-specific level. Insectivorous mammals may be less likely to consume a large amount of contaminated invertebrates, because they either forage over very large areas, like bats, or may forage on fossorial invertebrates, like shrews.

Consumption of contaminated grass by large birds or mammals would depend on the habitat-type in the treatment area and whether these animals are likely to forage there. The application method would be very important in determining the amount of exposure. Selective foliar applications to target invasive plants are not likely to lead to exposure. But broadcast foliar applications of large areas, particularly aerial applications, could contaminate forage. Consumption of contaminated vegetation is a substantial concern for some herbicides, but the specific application methods and timing may easily avoid exposure to these animals.

In order to evaluate how actual implementation can influence effects to wildlife, field studies for many of the above herbicides have been conducted.

Field Studies

Field studies can help evaluate the likelihood of population effects to wildlife from herbicides as applied. Some herbicides have been tested in many field studies on several groups of species with results published in open literature, while other herbicides have few or no field studies reported.

Most field studies could only detect changes in population numbers and are not sensitive enough to detect sublethal effects to wildlife. Some studies have investigated sub-lethal effects (e.g. Sullivan et al., 1998). However, sublethal effects that resulted in indirect mortality or other population changes would produce effects that could be detected by most long-term field studies.

Chlorsulfuron

No field studies are available.

Clopyralid

Rice et al. (1997) published results from an 8-year field study that found no significant effects on plant species diversity from the use of clopyralid or picloram. Hassan et al. (1994) reported summary of effects to terrestrial invertebrates in field trials.

Glyphosate

Sullivan et al. (1998) looked at long-term influence of glyphosate treatment in a spruce forest on reproduction, survival, and growth attributes of deer mouse (Peromyscus maniculatus) and southern red-backed vole (Clethrionomys gapperi) populations. For all statistically significant differences in their study (e.g. successful pregnancies, survival), the differences between treated and untreated populations were within the range of natural fluctuations for these small mammal populations over a 5-year period.

Sullivan et al. (1997) investigated the influence of aerial herbicide treatments on small mammal populations 9 and 11 years post-treatment. They found that glyphosate did not adversely affect reproduction, survival, or growth of deer mice or Oregon voles (Microtus oregoni) in coastal forest a decade after application. Species richness and diversity changed little over the decade after treatment and concluded that post-harvest successional change had more impact than that induced by herbicide treatment.

A field study on effects to the spider Lepthyphantes tenuis attributed population decrease to the secondary effects from changes in vegetation (Haughton et al., 2001; cited in SERA, 2003-Glyphosate). Bramble et al. (1997) investigated butterfly diversity and abundance on electric transmission right-of-ways treated with herbicides versus those treated with only mechanical methods. Herbicides used in the right-of-way treatments included a mixture of picloram and triclopyr, a mixture of triclopyr and metsulfuron methyl, a mixture of glyphosate and fosamine, a mixture of triclopyr and imazapyr, and glyphosate alone. They found no significant differences in diversity or abundance of butterflies between herbicide and no-herbicide units.

Cole et al. (1998) found that small mammal capture rates in Oregon forests that were logged, burned and then sprayed with glyphosate did not differ from those that were just logged and burned. Other studies have found that numbers of some species appear to increase or remain the same after treatment with herbicides, while other species decrease (Anthony and Morrison 1985;

Lautenschlager, 1993; Ritchie et al., 1987; Sullivan, 1990a). The same species might show all three responses in different studies with the same herbicide (see Sullivan, 1990a). In these studies, effects to small mammals occurred from habitat changes created by herbicide treatment, rather than from direct effects of herbicides (Santillo et al., 1989; Sullivan 1990a; Sullivan 1990b; Sullivan and Sullivan, 1981).

Santillo et al. (1989) found a substantial decrease in herbivorous insects on glyphosate treated sites, while there was clearcut verses untreated, but no trend between treated and untreated sites for predatory insects. The overall decrease in insect numbers decreased available food for shrews. Cole et al. (1997) sampled amphibians in Oregon clearcuts with and without glyphosate applications. Capture rates did not differ between treated and untreated plots for rough-skinned newt, ensatina, Pacific giant salamander, Dunn's salamander, western redback salamander, and red-legged frog.

Imazapic, Sethoxydim, Sulfometuron methyl

No field studies available.

Imazapyr

Imazapyr was used on a low volume retreatment in the Bramble et al. (1997) study mentioned above (see glyphosate) without apparent adverse effects to butterfly diversity and abundance on electric transmission right-of-ways.

Metsulfuron methyl

Metsulfuron methyl was in one of the mixtures used to treat electric transmission right-of-ways in the Bramble et al. (1997) study mentioned above (see glyphosate), which found no apparent adverse effects to butterfly diversity and abundance.

Picloram

Rice et al. (1997) published results from an 8-year field study that found no significant effects on plant species diversity from the use of clopyralid or picloram. Brooks et al. 1995 studied effects of picloram, imazapyr, and triclopyr mixtures on small mammals and found reduced numbers on sites after herbicide treatments. However, no control site (i.e. non-treated) was used so it is not possible to discern herbicide effects from normal population fluctuations that are common with small mammals. Nolte and Fulbright (1997) studied effects of an aerial application of picloram/triclopyr mixture on small mammals, birds, and rare plants. Effects to animal diversity or plant species richness or evenness were not found.

Picloram was in some of the mixtures used to treat electric transmission right-of-ways in studies by Bramble et al. (1997, 1999). The 1997 study found no significant differences to butterfly diversity and abundance, while the 1999 study found significantly higher diversity and abundance of butterflies on herbicide-treated units than on handcutting units.

Triclopyr

There are a number of field studies reported in the open literature, most of which indicate no or beneficial effects (SERA 2003 Triclopyr). Refer also to the study by Brooks et al. (1995) mentioned above. In contrast, Leslie et al. 1996 found that white-tailed deer avoid areas that used a "brown and burn" technique, where the site is treated with herbicide followed by a prescribed burn. McMurray et al. (1993a; 1993b; 1994) reported no adverse effects to reproductivity in mammals.

Triclopyr was in some of the mixtures used to treat electric transmission right-of-ways in studies by Bramble et al. (1997, 1999). The 1997 study found no significant differences to butterfly diversity and abundance, while the 1999 study found significantly higher diversity and abundance of butterflies on herbicide-treated units than on handcutting units.

Results of Exposure Analysis for Each Herbicide

Calculated doses for each herbicide at typical and highest application rates for each scenario are included in Appendix 1.

Chlorsulfuron

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 1.36 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F02a). This dose is 0.018 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA 2003 Chlorsulfuron, p. 4-27).

At the highest application rate of 0.25 lb/acre, the animal would receive an acute dose of 6.06 mg/kg (project file). This dose is 0.08 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. The estimated dose to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, is 0.11 mg/kg for acute exposure (SERA, 2003-Chlorsulfuron, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.0000074 mg/kg/day (SERA, 2003-Chlorsulfuron, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.0015 of the acute NOAEL, and 0.000001 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

At the highest application rate of 0.25 lb/acre, the acute dose from drinking water contaminated by a spill is 0.495 mg/kg (project file). This dose is 0.007 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 2.72 mg/kg (SERA 2003 Chlorsulfuron, Worksheet F10). This dose is 0.036 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large herbivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27). The chronic NOAEL for mammals in laboratory toxicity tests is 5 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 1.14 mg/kg (SERA,

2003-Chlorsulfuron, Worksheet F11a). This dose is 0.228 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large herbivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are less than the acute NOAEL and equal to the chronic NOAEL for mammals. No exposure exceeds the NOAEL, so no adverse effects are plausible from acute or chronic dietary exposures. The assumptions in the chronic exposure scenario are very unlikely to occur in field conditions, so the weight of evidence suggests that no adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p. 4-28).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.118 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F16a). This dose is 0.0016 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27). Doses to larger mammals would be even lower on a per kg body weight basis.

Chlorsulfuron does not appear to accumulate or persist in animals following either single or multiple doses. The elimination of chlorsulfuron has been studied in rats, goats, cows, and hens (SERA, 2003-Chlorsulfuron). A combination of elimination and metabolism extensively and rapidly eliminated chlorsulfuron and its metabolites from the bodies of all mammalian species studied. The half-life for elimination in rats is less than six hours (Shrivastava, 1979 cited in SERA, 2003-Chlorsulfuron). Therefore, chronic exposures from contaminated mammal prey due to a single application of chlorsulfuron are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of chlorsulfuron over time are plausible.

Estimated doses using the highest application rate (0.25 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p. 4-28).

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.15 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F03). This estimated dose is 0.002 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

The chronic NOAEL for mammals in laboratory toxicity tests is 5 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming the highest residue rates, the animal would receive a chronic dose of 0.013 mg/kg/day (SERA, 2003-Chlorsulfuron, Worksheet F04a). This dose is 0.0026 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Chlorsulfuron, p. 4-28).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 3.89 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet 14a). This dose is 0.052

of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. However, the acute dose is much less than the chronic NOAEL as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous mammals from chronic exposures are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

The estimated dose (17.3 mg/kg) using the highest application rate (0.25 lb/acre) is less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p. 4-28).

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1686 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 4.26 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F12). This dose is 0.0025 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

The chronic NOAEL for birds in laboratory toxicity tests is 140 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 1.79 mg/kg/day (SERA, 2003-Chlorsulfuron, Worksheet F13a). This dose is 0.013 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p.4-28).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of chlorsulfuron in fish was studied in bluegill and channel catfish exposed to ^{14}C-chlorsulfuron for

28 days (Han 1981 and Priester et al., 1991, cited in SERA, 2003 Chlorsulfuron). In the SERA risk assessments, concentrations in viscera are considered to reflect concentration in whole fish. Bioconcentration factors (BCF) for bluegill were <1 L/kg in muscle and 4-6 L/kg in viscera and liver (SERA, 2003-Chlorsulfuron, Appendix 9). BCF for channel catfish were 1.5 L/kg in muscle and < 12 L/kg in viscera and liver (SERA, 2003-Chlorsulfuron, Appendix 9). In both studies, residue levels in live fish dropped 70-90 percent during a two-week cleansing period. No adverse effects on fish were observed during the studies. The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 2.6 L/kg for acute exposure and 12 L/kg for chronic exposure.

The acute NOAEL for birds in laboratory toxicity tests is 1686 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.295 mg/kg (SERA 2003 Chlorsulfuron, Worksheet F08). This dose is 0.00017 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA 2003 Chlorsulfuron, p. 4-27).

The chronic NOAEL for birds in laboratory toxicity tests is 140 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.00009 mg/kg/day (SERA, 2003-Chlorsulfuron, Worksheet F09). This dose is 0.00000064 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-hlorsulfuron, p.4-28).

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 1686 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.181 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F16b). This dose is 0.0001 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Chlorsulfuron does not appear to bioconcentrate or persist in animals following either single or multiple doses. The elimination of chlorsulfuron has been studied in rats, goats, cows, and hens (SERA, 2003-Chlorsulfuron). A combination of elimination and metabolism extensively and rapidly eliminated chlorsulfuron and its metabolites from the bodies of all mammalian species studied. The half-life for elimination in rats is less than six hours (Shrivastava 1979 cited in SERA, 2003-Chlorsulfuron). Therefore, chronic exposures from contaminated mammal prey due to a single application of chlrosulfuron are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p.4-28).

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1686 mg/kg. For exposure scenarios that use the typical application rate of 0.056 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 6.32 mg/kg (SERA, 2003-Chlorsulfuron, Worksheet F14b). This dose is 0.004 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. However, the acute dose is much less than the chronic NOAEL as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects are plausible (SERA, 2003-Chlorsulfuron, p. 4-27).

Estimated doses using the highest application rate (0.25 lb/acre) are much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Chlorsulfuron, p.4-28).

Clopyralid

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For, exposure scenarios that use the typical application rate of 0.35 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 8.49 mg/kg (SERA, 2003-Clopyralid, Worksheet F02a). This estimated dose is 0.10 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Clopyralid, p. 4-23).

At the highest application rate of 0.5 lb/acre, the animal would receive an acute dose of 12.1 mg/kg (project file). This dose is 0.2 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 2.33 mg/kg for acute exposure (SERA, 2003-Clopryalid, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.00067 mg/kg/day (SERA, 2003-Clopyralid, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.03 of the acute NOAEL, and 0.00004 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Clopyralid, p. 4-23).

At the highest application rate of 0.5 lb/acre, the acute dose from drinking water contaminated by a spill is 3.32 mg/kg (project file). This dose is 0.04 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 70 kg mammal consumed contaminated

vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 17.0 mg/kg (SERA, 2003-Clopyralid, Worksheet F10). This dose is 0.2 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large herbivorous mammals are plausible (SERA, 2003-Clopyralid, p. 4-23).

The chronic NOAEL for mammals in laboratory toxicity tests is 15 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 8.95 mg/kg/day (SERA, 2003-Clopyralid, Worksheet F11a). This dose is 0.6 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large herbivorous mammals are plausible (SERA, 2003-Clopyralid, p. 4-23).

Estimated doses using the highest application rate (0.50 lb/acre) are less than the acute and chronic NOAELs for mammals, although only marginally so for the chronic NOAEL. Since both doses are still below the NOAEL, there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Clopyralid, p. 4-23).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.734 mg/kg (SERA, 2003-Clopyralid, Worksheet F16a). Doses to a large mammal would be even lower on a per kg body weight basis. This dose is 0.02 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Clopyralid, p. 4-23).

Clopyralid does not appear to accumulate in animal tissues. The elimination and metabolism of clopyralid has been studied in rats, hens, lambs, and goats (SERA, 2003-Clopyralid). These animals rapidly excreted largely unmetabolized clopyralid. The half-life for elimination in rats is three hours (Dow AgroSciences 1998 cited in SERA, 2003-Clopyralid). Therefore, chronic exposures from contaminated mammal prey due to a single application of clopyralid are unlikely to cause any adverse effect. In addition, the acute dose is less than the chronic NOAEL of 15 mg/kg/day for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of clopyralid over time are plausible. 44

Estimated doses using the highest application rate (0.50 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Clopyralid, p. 4-23).

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.938 mg/kg (SERA 2003 Clopyralid, Worksheet F03). This estimated dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA 2003 Clopyralid, p. 4-23).

The chronic NOAEL for mammals in laboratory toxicity tests is 15 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest

residue rates, the animal would receive a chronic dose of 0.0987 mg/kg/day (SERA 2003 Clopyralid, Worksheet F04a). This estimated dose is 0.007 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA 2003 Clopyralid, p. 4-23).

Estimated doses using the highest application rate (0.50 lb/acre) are than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Clopyralid, p. 4-23).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 75 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 24.3 mg/kg (SERA 2003 Clopyralid, Worksheet 14a). This dose is 0.30 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA 2003 Clopyralid, p. 4-23).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. The acute dose is greater than the chronic NOAEL (15 mg/kg/day), so adverse effects to insectivorous mammals appear plausible from chronic dietary exposures. The dose is less than the chronic LOAEL of 150 mg/kg/day, however. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however

The estimated dose (34.7 mg/kg) using the highest application rate (0.50 lb/acre) is less than the acute NOAEL, but greater than the chronic NOAEL for mammals. The dose is less than the chronic LOAEL of 150 mg/kg/day, however. No adverse effects are plausible from acute exposures, but adverse effects to insectivorous mammals are plausible from chronic dietary exposures.

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 670 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 26.6 mg/kg (SERA, 2003-Clopyralid, Worksheet F12). This dose is 0.04 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Clopyralid, p. 4-23).

There is no chronic toxicity index available for effects of clopyralid to birds, so the mammal chronic NOAEL will be used. In acute dietary exposures, the bird NOAEL is about a factor of nine above the mammal NOAEL, suggesting that birds are less sensitive than mammals to clopyralid. The chronic NOAEL for mammals in laboratory toxicity tests is 15 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 14.0 mg/kg/day (SERA, 2003-Clopyralid, Worksheet F13a). This estimated dose is 0.90 of the chronic NOAEL for mammals, and birds appear to be less sensitive to clopyralid than mammals,

so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Clopyralid, p. 4-23).

Estimated doses using the highest application rate (0.50 lb/acre) are less than the acute NOAEL for birds, but greater than the chronic NOAEL for mammals. The chronic dose is less than the chronic LOAEL of 150 mg/kg/day, however. No adverse effects are plausible from acute exposures, but adverse effects to large herbivorous birds appear plausible from chronic dietary exposures. However, the assumptions in the chronic exposure scenario are very unlikely to occur in field conditions, so the weight of evidence suggests that no adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Clopyralid, p. 4-23).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. Clopyralid does not appear to bioconcentrate, based on one study in sunfish (Bidlack 1982 as cited in SERA, 2003-Clopyralid). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 1 L/kg for acute and chronic exposures.

The acute NOAEL for birds in laboratory toxicity tests is 670 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 2.38 mg/kg (SERA, 2003-Clopyralid, Worksheet F08).

This dose is 0.004 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Clopyralid, p. 4-23). There is no chronic toxicity index available for effects of clopyralid to birds, so the mammal chronic NOAEL will be used. In acute dietary exposures, the bird NOAEL is about a factor of nine above the mammal NOAEL, suggesting that birds are less sensitive than mammals to clopyralid. The chronic NOAEL for mammals in laboratory toxicity tests is 15 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.000683 mg/kg/day (SERA 2003 Clopyralid, Worksheet F09). This estimated dose is 0.00005 of the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Clopyralid, p. 4-23).

Estimated doses using the highest application rate (0.50 lb/acre) are less than the acute NOAEL for birds and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 670 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 1.13 mg/kg (SERA 2003 Clopyralid, Worksheet F16b). This is 0.002 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Clopyralid does not appear to bioconcentrate, based on one study in sunfish (Bidlack 1982 as cited in SERA 2003 Clopyralid). The elimination and metabolism of clopyralid has been studied

in rats, hens, lambs, and goats ((SERA, 2003-Clopyralid). These animals rapidly excreted largely unmetabolized clopyralid. The half-life for elimination in rats is three hours (Dow AgroSciences, 1998 cited in SERA, 2003). Therefore, chronic exposures from contaminated mammal prey due to a single application of clopyralid are unlikely to cause any adverse effect. In addition, the acute dose is less than the chronic NOAEL for birds, so there is no basis for asserting/predicting that adverse effects from repeated acute exposures from multiple applications of clopyralid over time are plausible.

Estimated doses using the highest application rate (0.50 lb/acre) are less than the acute NOAEL for birds, and the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 670 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 39.5 mg/kg (SERA, 2003-Clopyralid, Worksheet F14b). This dose is 0.06 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Clopyralid, p. 4-23).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. The acute dose is greater than the chronic NOAEL (15 mg/kg/day) for mammals, so adverse effects to insectivorous birds appear plausible from chronic dietary exposures. The dose is less than the chronic LOAEL of 150 mg/kg/day, however.

The estimated dose (56.4 mg/kg) using the highest application rate (0.50 lb/acre) is less than the acute NOAEL for birds but greater than the chronic NOAEL for mammals.

The dose is less than the chronic LOAEL of 150 mg/kg/day, however. No adverse effects are plausible from acute exposures, but adverse effects to insectivorous birds appear plausible from chronic dietary exposures.

Glyphosate

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. For, exposure scenarios that use the typical application rate of 2 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 48.5 mg/kg (SERA, 2003-Glyphosate, Worksheet F02a). This estimated dose is 0.3 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

At the highest application rate of 7 lb/acre, the animal would receive an acute dose of 170 mg/kg (project file). This dose is 0.97 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 5.32 mg/kg for acute exposure (SERA, 2003-Glyphosate, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.00234 mg/kg/day (SERA 2003 Glyphosate, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.03 of the acute NOAEL, and 0.00001 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

At the highest application rate of 7 lb/acre, the acute dose from drinking water contaminated by a spill is 18.6 mg/kg (project file). This dose is 0.1 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 97.1 mg/kg (SERA, 2003-Glyphosate, Worksheet F10). This dose is 0.6 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

The chronic NOAEL for mammals in laboratory toxicity tests is 175 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 53.2 mg/kg/day (SERA, 2003-Glyphosate, Worksheet F11a). This dose is 0.3 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

Estimated doses using the highest application rate (7 lb/acre) result in doses greater than the acute and equal to the chronic NOAEL for mammals. The acute dose is equal to a LOAEL that resulted in some mortality to pregnant rabbits. Thus, while the acute dose to herbivorous mammals at the highest application rate is well below the LD_{50} (2,000 mg/kg), mortality in some animals would be plausible (SERA, 2003-Glyphosate, p. 4-44).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 4.2 mg/kg (SERA, 2003-Glyphosate, Worksheet F16a). Doses to a large mammal would be even lower on a per kg body weight basis. This dose is 0.024 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

Glyphosate does not appear to accumulate or persist in animal tissues. Only about 30 percent of ingested glyphosate is absorbed from the gastrointestinal tract (several studies by Davies 1996 cited in SERA, 2003-Glyphosate). The glyphosate that is absorbed is distributed widely

throughout the body, and then efficiently excreted. More than 97 percent of the administered dose is excreted unchanged, and glyphosate does not substantially concentrate or persist in any tissue (SERA, 2003-Glyphosate, p. 3-5). These conclusions are consistent with data from a field study that measured glyphosate residues in several small mammal species after an aerial application in Oregon (Newton et al. 1984). Newton et al. (1984) found that residues in small mammals were below 1 mg/kg for deermice and shrews, and below 2 mg/kg for voles, three days after treatment. Therefore, chronic exposures from contaminated mammal prey due to a single application of glyphosate are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of glyphosate over time are plausible.

The estimated dose using the highest application rate (7 lb/acre) is much less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 2.11 mg/kg (SERA, 2003-Glyphosate, Worksheet

F03). This estimated dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

The chronic NOAEL for mammals in laboratory toxicity tests is 175 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.231 mg/kg/day (SERA 2003-Glyphosate, Worksheet F04a). This estimated dose is 0.001 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

Estimated doses using the highest application rate (7 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 175 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 139 mg/kg (SERA, 2003-Glyphosate, Worksheet 14a). This dose is 0.793 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to small insectivorous mammals are plausible (SERA, 2003-Glyphosate, p. 4-43).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous mammals from chronic exposures are plausible.

The estimated dose (486 mg/kg) using the highest application rate (7 lb/acre) is greater than the acute and chronic NOAELs for mammals, so adverse effects to insectivorous mammals are plausible. This dose also exceeds the acute and chronic LOAEL (350 mg/kg) for diarrhea in mammals. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however. (Check Newton et al 1984 paper).

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 562 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 152 mg/kg (SERA, 2003-Glyphosate, Worksheet F12). This dose is 0.3 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Glyphosate, p. 4-43).

The chronic NOAEL for birds in laboratory toxicity tests is 100 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 83.2 mg/kg/day (SERA, 200X-Name, Worksheet F13a). This estimated dose is 0.8 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Glyphosate, p. 4-43).

Estimated doses using the highest application rate (7 lb/acre) are less than the acute NOAEL, but greater than the chronic NOAEL for birds. LOAEL's are not reported for birds in the sources I reviewed, presumably because of a lack of toxic responses in laboratory tests. No adverse effects are plausible from acute exposures, but adverse effects to large herbivorous birds appear plausible from chronic dietary exposures, based on dose exceeding the NOAEL. The assumptions in the chronic exposure scenario are unlikely to occur in field conditions, particularly because glyphosate is a non-selective herbicide and would kill most forage species at this application rate, making the forage unavailable or unpalatable. However, some monitored values for glyphosate residues on vegetation (Newton et al. 1994) are higher than those used in the SERA risk assessments. Therefore, the higher residue rates may offset the lack of forage availability, and adverse effects to herbivorous birds are plausible.

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The EPA uses a BCF for whole fish of 0.52 L/kg based on a study by Forbis (1989 as cited in SERA, 2003-Glyphosate) and corroborated by Chamberlain et al. (1996, as cited in SERA, 2003). Therefore, exposure scenarios in the SERA risk assessment use a whole-fish BCF of 0.52 L/kg for acute and chronic exposures.

The acute NOAEL for birds in laboratory toxicity tests is 562 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 2.83 mg/kg (SERA, 2003-Glyphosate, Worksheet F08). This dose is 0.005 of the acute NOAEL, so there is no basis for

asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Glyphosate, p. 4-43).

The chronic NOAEL for birds in laboratory toxicity tests is 100 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.00125 mg/kg/day (SERA, 2003-Glyphosate, Worksheet F09). This estimated dose is 0.00001 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible.

Estimated doses using the highest application rate (7 lb/acre) are much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 562mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 6.46 mg/kg (SERA, 2003-Glyphosate, Worksheet F16b). This is 0.0115 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Glyphosate does not appear to accumulate or persist in animals. Only about 30 percent of ingested glyphosate is absorbed from the gastrointestinal tract (several studies by Davies 1996 cited in SERA, 2003-Glyphosate). The glyphosate that is absorbed is distributed widely throughout the body, and then efficiently excreted. More than 97 percent of the administered dose is excreted unchanged, and glyphosate does not substantially concentrate or persist in any tissue (SERA 2003 Glyphosate, p. 3-5). These conclusions are consistent with data from a field study that measured glyphosate residues in several small mammal species after an aerial application in Oregon (Newton et al., 1984). Newton et al. (1984) found that residues in small mammals were below 1 mg/kg for deermice and shrews, and below 2 mg/kg for voles, three days after treatment.

Therefore, chronic exposures from contaminated mammal prey due to a single application of glyphosate are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of glyphosate over time are plausible.

Estimated doses using the highest application rate (7 lb/acre) are less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 562 mg/kg. For exposure scenarios that use the typical application rate of 2 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 226 mg/kg (SERA, 2003-Glyphosate, Worksheet F14b). This dose is 0.4 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Glyphosate, p. 4-43).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. However, the acute dose is greater than the chronic NOAEL for birds. LOAEL's are not reported for birds in the sources I reviewed, presumably because of a lack of toxic responses in laboratory tests. Adverse effects to insectivorous birds appear plausible from chronic dietary exposures, based on dose exceeding the NOAEL.

The estimated dose using the highest application rate (7 lb/acre) is greater than the acute and chronic NOAELs for birds, so adverse effects to insectivorous birds appear plausible at the highest application rate.

Imazapic

Small Mammal Directly Sprayed

For, exposure scenarios that use the typical application rate of 0.1 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 2.42 mg/kg (SERA, 2003-Imazapic, Worksheet F02a). This estimated dose is 0.007 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

At the highest application rate of 0.19 lb/acre, the animal would receive an acute dose of 4.36 mg/kg (project file). This dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The acute NOAEL for mammals in laboratory toxicity tests is 350 mg/kg. The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 0.665 mg/kg for acute exposure (SERA, 2003-Imazapic, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.000000439 mg/kg/day (SERA, 2003-Imazapic, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis.

These doses are 0.002 of the acute NOAEL, and 0.000000009 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

At the highest application rate of 0.19 lb/acre, the acute dose from drinking water contaminated by a spill is 1.26 mg/kg (project file). This dose is 0.004 of the acute NOAEL.

The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 350 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 4.86 mg/kg (SERA, 2003-Imazapic, Worksheet F10). This dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA 2003 Imazapic, p. 4-21).

The chronic NOAEL for mammals in laboratory toxicity tests is 45 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 0.929 mg/kg/day (SERA, 2003-Imazapic, Worksheet F11a). This dose is 0.02 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

Estimated doses using the highest application rate (0.1875 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 20030-Imazapic, p. 4-21).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 350 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.21 mg/kg (SERA, 2003-Imazapic, Worksheet F16a). Doses to a large mammal would be even lower on a per kg body weight basis. This dose is 0.0006 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003=-Imazapic, p. 4-21).

Imazapic does not appear to accumulate or persist in animals following either single or multiple doses. The elimination of imazapic has been studied in rats, hens, and goats (Afzal, 1994; Cheng, 1993; Gatterdam 1993a,b; Kao 1993a,b; Sharp and Thalacker, 1999; all as cited in SERA, 2003-Imazapic). A combination of elimination and metabolism extensively and rapidly eliminated imazapic and its metabolites from the bodies of all species studied.

Therefore, chronic exposures from contaminated mammal prey due to a single application of imazapic are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of imazapic over time are plausible.

The estimated dose using the highest application rate (0.1875 lb/acre) is less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 350 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.268 mg/kg (SERA, 2003-Imazapic, Worksheet F03). This estimated dose is 0.0008 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

The chronic NOAEL for mammals in laboratory toxicity tests is 45 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.0102 mg/kg/day (SERA, 2003-Imazapic, Worksheet F04a). This estimated dose is 0.0002 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

Estimated doses using the highest application rate (0.1875 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 350 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 6.94 mg/kg (SERA, 2003-Imazapic, Worksheet 14a). This dose is 0.02 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA, 2003-Imazapic, p. 4-21).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL for mammals as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous mammals from chronic exposures are plausible.

The estimated dose using the highest application rate (0.1875 lb/acre) is less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1100 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 7.6 mg/kg (SERA, 2003-Imazapic, Worksheet F12). This dose is 0.007 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Imazapic, p. 4-21).

The chronic NOAEL for birds in laboratory toxicity tests is 113 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 1.45 mg/kg/day (SERA, 2003-Imazapic, Worksheet F13a). This estimated dose is 0.01 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Imazapic, p. 4-21).

Estimated doses using the highest application rate (0.1875 lb/acre) are less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Imazapic, p. 4-21).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of imazapic in fish was studied in bluegill sunfish exposed to ^{14}C-labeled imazapic for 28 days (Robinson, 1994, cited in SERA, 2003-Imazapic). In the SERA risk assessments, concentrations in viscera are considered to reflect concentration in whole fish. Bioconcentration factors (BCF) for bluegill were 0.11 L/kg in whole fish, indicating that the concentration of imazapic in the fish

was less than the concentration of imazapic in the water (SERA, 2003-Imazapic). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 0.11 L/kg for acute and chronic exposures because of the rapid time it takes to reach a steady state and the very low BCF.

The acute NOAEL for birds in laboratory toxicity tests is 1100 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.0749 mg/kg (SERA, 2003-Imazapic, Worksheet F08). This dose is 0.00007 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Imazapic, p. 4-21).

The chronic NOAEL for birds in laboratory toxicity tests is 113 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.0000000495 mg/kg/day (SERA, 200X-Worksheet F09). This estimated dose is 0.0000000004 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Imazapic, p. 4-21).

Estimated doses using the highest application rate (0.1875 lb/acre) also result in exposures much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 1100 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.323 mg/kg (SERA, 2003-Imazapic, Worksheet F16b). This is 0.0003 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible (SERA, 2003-Imazapic, p. 4-21).

Imazapic does not appear to accumulate or persist in animals following either single or multiple doses. The elimination of imazapic has been studied in rats (Cheng 1993), hens

(Afzal, 1994; Gatterdam, 1993a,b), and goats (Kao 1993a,b; Sharp and Thalacker, 1999; cited in SERA, 2003-Imazapic). A combination of elimination and metabolism extensively and rapidly eliminated imazapic and its metabolites from the bodies of all species studied. Therefore, chronic exposures from contaminated mammal prey due to a single application of imazapic are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of imazapic over time are plausible.

The estimated dose using the highest application rate (0.1875 lb/acre) is less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1100 mg/kg. For exposure scenarios that use the typical application rate of 0.1 lb/acre, if a 10 g bird consumed contaminated insects

on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 11.3 mg/kg (SERA, 2003-Imazapic, Worksheet F14b). This dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Imazapic, p. 4-21).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL for birds as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous birds from chronic exposures are plausible.

The estimated dose using the highest application rate (0.1875 lb/acre) is less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapic, p. 4-21).

Imazapyr

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 250 mg/kg. For, exposure scenarios that use the typical application rate of 0.45 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 10.9 mg/kg (SERA, 2003-Imazapyr, Worksheet F02a). This estimated dose is 0.04 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

At the highest application rate of 1.25 lb/acre, the animal would receive an acute dose of 30.3 mg/kg (project file). This dose is 0.1 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 1.22 mg/kg for acute exposure (SERA, 2003-Imazapyr, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.0000659 mg/kg/day (SERA 2003 Imazapyr, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.005 of the acute NOAEL, and 0.0000003 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

At the highest application rate of 1.25 lb/acre, the acute dose from drinking water contaminated by a spill is 3.39 mg/kg (project file). This dose is 0.005 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario. The acute NOAEL for mammals in laboratory toxicity tests is 250 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.944 mg/kg (SERA, 2003-Imazapyr, Worksheet F16a). (Doses to a large mammal would be even lower on a per kg body weight basis). This dose is 0.004 of the acute NOAEL, so there is no

basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

Imazapyr does not appear to accumulate or persist in animals following either single or multiple doses (SERA, 2003-Imazapyr, p. 3-2). The elimination of imazapyr has been studied in rats and lactating goats and the studies reported that it is rapidly excreted, unchanged, in urine and feces (Mallipudi et al., 1983; and Zdybak, 1992 as cited in SERA, 2003-Imazapyr). No metabolites were identified. Therefore, chronic exposures from contaminated mammal prey due to a single application of imazapyr are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of imazapyr over time are plausible.

Estimated doses using the highest application rate (1.25 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-mazapyr, p. 4-25).

The acute NOAEL for mammals in laboratory toxicity tests is 250 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 21.9 mg/kg (SERA, 2003-Imazapyr, Worksheet F10). This dose is 0.09 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

The chronic NOAEL for mammals in laboratory toxicity tests is 250 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 10.6 mg/kg/day (SERA, 200X-Name, Worksheet F11a). This dose is 0.04 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

Estimated doses using the highest application rate (1.25 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4.25).

Medium Carnivorous Mammal; Large Herbivorous Mammal; Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 250 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 1.21 mg/kg (SERA, 2003-Imazapyr, Worksheet F03). This estimated dose is 0.005 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

The chronic NOAEL for mammals in laboratory toxicity tests is 250 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.117 mg/kg/day (SERA, 2003-Imazapyr, Worksheet F04a). This estimated dose is 0.0005 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible.

Estimated doses using the highest application rate (1.25 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 250 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 31.2 mg/kg (SERA, 2003-Imazapyr, Worksheet 14a). This dose is 0.1 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA, 2003-Imazapyr, p. 4-25).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL for mammals as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous mammals from chronic exposures are plausible.

The estimated dose using the highest application rate (1.25 lb/acre) is less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 674 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 34.2 mg/kg (SERA, 2003-Imazapyr, Worksheet F12). This dose is 0.05 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Imazapyr, p. 4-25).

The chronic NOAEL for birds in laboratory toxicity tests is 200 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 16.5 mg/kg/day (SERA, 2003-Imazapyr, Worksheet F13a). This estimated dose is 0.08 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Imazapyr, p. 4-25).

Estimated doses using the highest application rate (1.25 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of imazapyr in fish was studied in bluegill sunfish exposed to ^{14}C-labeled imazapyr for 28 days (McAllister et al., 1985, cited in SERA, 2003-Imazapyr). In the SERA risk assessments, concentrations in viscera are considered to reflect concentration in whole fish. Bioconcentration factors (BCF) for bluegill were 0.5 L/kg, indicating that the concentration of imazapyr in the fish

was less than the concentration of imazapyr in the water (SERA, 2003-Imazapyr, p. 3-20). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 0.5 L/kg for acute and chronic exposures.

The acute NOAEL for birds in laboratory toxicity tests is 674 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.625 mg/kg (SERA, 2003-Imazapyr, Worksheet F08). This dose is 0.0009 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Imazapyr, p. 4-25).

The chronic NOAEL for birds in laboratory toxicity tests is 200 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.0000338 mg/kg/day (SERA, 2003-Imazapyr, Worksheet F09). This estimated dose is 0.0000002 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible.

Estimated doses using the highest application rate (1.25 lb/acre) are less than the acute and chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 674 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 1.45 mg/kg (SERA, 2003-Imazapyr, Worksheet F16b). This is 0.002 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Imazapyr does not appear to accumulate or persist in animals following either single or multiple doses (SERA, 2003-Imazapyr, p. 3-2). The elimination of imazapyr has been studied in rats and lactating goats and the studies reported that it is rapidly excreted, unchanged, in urine and feces (Mallipudi et al., 1983; and Zdybak, 1992 as cited in SERA, 2003-Imazapyr). No metabolites were identified. Therefore, chronic exposures from contaminated mammal prey due to a single application of imazapyr are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of imazapyr over time are plausible.

The estimated dose using the highest application rate (1.25 lb/acre) is less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 674 mg/kg. For exposure scenarios that use the typical application rate of 0.45 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 50.8 mg/kg (SERA, 2003-Imazapyr, Worksheet F14b). This dose is 0.08 of the acute NOAEL, so

there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Imazapyr, p. 4-25).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL for birds as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous birds from chronic exposures are plausible.

The estimated dose using the highest application rate (1.25 lb/acre) is less than the acute and chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Imazapyr, p. 4-25).

Metsulfuron METHYL

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 25 mg/kg. For, exposure scenarios that use the typical application rate of 0.03 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 0.727 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F02a). This estimated dose is 0.03 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

At the highest application rate of 0.15 lb/acre, the animal would receive an acute dose of 3.64 mg/kg (project file). This dose is 0.1 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 0.0443 mg/kg for acute exposure (SERA, 2003-Metsulfuron methyl, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.00000176 mg/kg/day (SERA 2003 Metsulfuron methyl, Worksheet F07). Doses to a larger mammal would be even lower on a per kg body weight basis. These doses are 0.002 of the acute NOAEL, and 0.00000007 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26, 4-27).

At the highest application rate of 0.15 lb/acre, the acute dose from drinking water contaminated by a spill is 0.222 mg/kg (project file). This dose is 0.009 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 25 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 1.46 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F10). This dose is 0.06 of the acute NOAEL, so there is no basis for asserting

or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26). The chronic NOAEL for mammals in laboratory toxicity tests is 25 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 0.613 mg/kg/day (SERA, 2003-Metsulfuron methyl, Worksheet F11a). This dose is 0.02 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-27).

Estimated doses using the highest application rate (0.15 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Metsulfuron methyl, p. 4-27).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 25 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.0629 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F16a). Doses to a large mammal would be even lower on a per kg body weight basis. This dose is 0.003 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

Metsulfuron methyl does not appear to accumulate or persist in animal tissues. The elimination of metsulfuron methyl has been studied in rats, hens cows, and goats (SERA 2003 Metsulfuron methyl, citing Charlton and Bookhart, 1996; USEPA, 1998; Hershberger and Moore, 1985; Hundley, 1985; Hunt, 1984). A combination of elimination of the unchanged compound and metabolism rapidly eliminated metsulfuron methyl from the bodies of all species studied. The half-life for elimination in all species is one day or less (SERA, 2003-Metsulfuron methyl, p. 3-3). Therefore, chronic exposures from contaminated mammal prey due to a single application of metsulfuron methyl are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of metsulfuron methyl over time are plausible. The estimated dose using the highest application rate (0.15 lb/acre) is less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-etsulfuron methyl, p. 4-27).

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 25 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.0804 mg/kg (SERA, 200- Metsulfuron methyl, Worksheet F03). This estimated dose is 0.003 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

The chronic NOAEL for mammals in laboratory toxicity tests is 25 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.00676 mg/kg/day (SERA, 2003-Metsulfuron methyl, Worksheet F04a). This estimated dose is 0.0003 of the chronic NOAEL, so

there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-27).

Estimated doses using the highest application rate (0.15 lb/acre) are less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Metsulfuron methyl, p. 4-27).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 25 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 2.08 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet 14a). This dose is 0.08 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is less than the chronic NOAEL as well, and chronic doses are much lower than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous mammals from chronic exposures are plausible.

Estimated doses using the highest application rate (0.15 lb/acre) also result in an exposure less than the acute and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1043 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 2.28 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F12). This dose is 0.002 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

The chronic NOAEL for birds in laboratory toxicity tests is 120 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 0.96 mg/kg/day (SERA, 2003-Metsulfuron methyl, Worksheet F13a). This estimated dose is 0.008 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Metsulfuron methyl, p. 4-27).

Estimated doses using the highest application rate (0.15 lb/acre) are less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Metsulfuron methyl, p. 4-27).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of metsulfuron methyl in fish was studied in bluegill sunfish exposed to ^{14}C-metsulfuron methyl for 28 days (Han 1982, cited in SERA, 2003-Metsulfuron methyl). In the SERA risk assessments, concentrations in viscera are considered to reflect concentration in whole fish. Bioconcentration factors (BCF) reported for bluegill viscera were 0.21 L/kg after 24 hours and the highest BCF reported was 2.11 L/kg after 14 days (SERA, 2003-Metsulfuron methyl, Appendix 8). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 0.21 L/kg for acute exposure and 2.11 L/kg for chronic exposure.

The acute NOAEL for birds in laboratory toxicity tests is 1043 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.00954 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F08). This dose is 0.000009 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

The chronic NOAEL for birds in laboratory toxicity tests is 120 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.0000038 mg/kg/day (SERA, 2003-Metsulfuron methyl, Worksheet F09). This estimated dose is 0.00000003 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible (SERA 2003 Metsulfuron methyl, p. 4-27).

Estimated doses using the highest application rate (0.15 lb/acre) are much less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 1043mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.097 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F16b). This is 0.00009 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

Metsulfuron methyl does not appear to accumulate or persist in animal tissues. The elimination of metsulfuron methyl has been studied in rats, hens cows, and goats (SERA, 2003-Metsulfuron methyl, citing Charlton and Bookhart, 1996; USEPA, 1998; Hershberger and Moore, 1985; Hundley, 1985; Hunt, 1984). A combination of elimination of the unchanged compound and metabolism rapidly eliminated metsulfuron methyl from the bodies of all species studied. The half-life for elimination in all species is one day or less (SERA, 2003-Metsulfuron methyl, p. 3-3). Therefore, chronic exposures from contaminated mammal prey due to a single application of metsulfuron methyl are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of metsulfuron methyl over time are plausible.

The estimated dose using the highest application rate (0.15 lb/acre) is less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1043 mg/kg. For exposure scenarios that use the typical application rate of 0.03 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 3.38 mg/kg (SERA, 2003-Metsulfuron methyl, Worksheet F14b). This dose is 0.003 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Metsulfuron methyl, p. 4-26).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. However, the acute dose is less than the chronic NOAEL for birds as well, and chronic doses are much less than acute doses. Therefore, there is no basis for asserting or predicting that adverse effects to insectivorous birds from chronic exposures are plausible.

The estimated doses using the highest application rate (0.15 lb/acre) is less than the acute and chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Picloram

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 34 mg/kg. For, exposure scenarios that use the typical application rate of 0.35 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 8.49 mg/kg (SERA, 2003-Picloram, Worksheet F02a). This estimated dose is 0.2 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Picloram, p. 4-29).

At the highest application rate of 1 lb/acre, the animal would receive an acute dose of 24.2 mg/kg (project file). This dose is 0.7 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 0.887 mg/kg for acute exposure (SERA, 2003-Picloram, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.000205 mg/kg/day (SERA, 2003-Picloram, Worksheet F07).

Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.03 of the acute NOAEL, and 0.00003 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Picloram, p. 4-29).

At the highest application rate of 1 lb/acre, the acute dose from drinking water contaminated by a spill is 2.53 mg/kg (project file). This dose is 0.07 of the acute NOAEL. The chronic dose is also

below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 34 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 17.0 mg/kg (SERA, 2003-Picloram, Worksheet F10). This dose is 0.5 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Picloram, p. 4-29). The chronic NOAEL for mammals in laboratory toxicity tests is 7 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 2.18 mg/kg/day (SERA 2003 Picloram, Worksheet F11a). This dose is 0.3 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Picloram, p. 4-29).

Estimated doses using the highest application rate (1 lb/acre) are greater than the acute NOAEL and about equal to the chronic NOAEL for mammals. The acute dose (48.6 mg/kg) is less than the acute LOAEL for decreased weight gain in rabbits (USEPA/OPP, 1998). No adverse effects are plausible from chronic exposures, but adverse effects to large herbivorous mammals may be plausible from acute dietary exposures.

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 34 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.734 mg/kg (SERA, 2003-Picloram, Worksheet F16a). Doses to a larger mammal would be even lower on a per kg body weight basis. This dose is 0.0216 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA, 2003-Picloram, p. 4-29).

Picloram does not appear to accumulate or persist in animals. The elimination of picloram has been studied in humans, rats, dogs, and cattle (SERA 2003 Picloram). In humans, over 75 percent of the administered picloram was eliminated after six hours and over 90 percent was eliminated after 72 hours (SERA, 2003-Picloram citing Nolan et al. 1984). Therefore, chronic exposures from contaminated mammal prey due to a single application of picloram are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of carnivorous mammals over time are plausible.

The estimated dose using the highest application rate (1 lb/acre) is less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 34 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.938 mg/kg (SERA, 2003-Picloram, Worksheet F03). This estimated

dose is 0.03 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Picloram, p. 4-29).

The chronic NOAEL for mammals in laboratory toxicity tests is 7 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.024 mg/kg/day (SERA, 2003-Picloram, Worksheet F04a). This estimated dose is 0.003 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to small herbivorous mammals are plausible (SERA, 2003-Picloram, p. 4-29).

Estimated doses using the highest application rate (1 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 34 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 24.3 mg/kg (SERA, 2003-Picloram, Worksheet 14a). This dose is 0.714 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible (SERA, 2003-Picloram, p. 4-29).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. The acute dose is greater than the chronic NOAEL (7 mg/kg), and near the chronic LOAEL (35 mg/kg/day) for increased liver weight. So adverse effects to insectivorous mammals appear plausible from chronic dietary exposures. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however.

The estimated dose (69.4 mg/kg) using the highest application rate (1 lb/acre) is greater than the acute and chronic NOAELs for mammals. It is less than the acute LOAEL for decreased weight gain, but is almost twice the chronic LOAEL for increased liver weight. So adverse effects to insectivorous mammals appear plausible from acute or chronic dietary exposures.

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1500 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 26.6 mg/kg (SERA, 2003-Name, Worksheet F12). This dose is 0.02 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Picloram, p. 4-29).

There is no chronic toxicity index available for effects of picloram to birds, so the mammal chronic NOAEL will be used. Since the acute NOAEL for birds is greater than the acute NOAEL for mammals, the use of the chronic figure from mammals is likely to over-estimate risk to birds. The chronic NOAEL for mammals in laboratory toxicity tests is 7 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the

highest residue rates and 100 percent of diet is contaminated, results in a dose of 3.41 mg/kg/day (SERA, 2003-Picloram, Worksheet F13a). This estimated dose is 0.5 of the chronic NOAEL, so there is no/ basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Picloram, p. 4-29).

Estimated doses using the highest application rate (1 lb/acre) are less than the acute NOAEL for birds, but greater than the chronic NOAEL for mammals. The chronic dose is less than the chronic LOAEL for mammals. No adverse effects are plausible from acute exposures, but adverse effects to large herbivorous birds appear plausible from chronic dietary exposures, based on dose exceeding the NOAEL. Since picloram does not kill grass, herbicide residues on grass may be more available for chronic ingestion than non-selective herbicides.

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of picloram in fish was studied in bluegill and channel catfish exposed to ^{14}C-picloram for 28 days (Bidlack 1980a,b cited in SERA, 2003-Picloram). Only trace amounts of ^{14}C-picloram were recovered in the fish, so the BCF for picloram appears to be substantially less than one (SERA 2003 Picloram). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 1 L/kg for acute and chronic exposures, which will over-estimate exposure.

The acute NOAEL for birds in laboratory toxicity tests is 1500 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.908 mg/kg (SERA, 2003-Picloram, Worksheet F08). This dose is 0.0006 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Picloram, p. 4-29).

There is no chronic toxicity index available for effects of picloram to birds, so the mammal chronic NOAEL will be used. Since the acute NOAEL for birds is greater than the acute NOAEL for mammals, the use of the chronic figure from mammals is likely to over-estimate risk to birds. The chronic NOAEL for mammals in laboratory toxicity tests is 7 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.000214 mg/kg/day (SERA, 2003-Picloram, Worksheet F09). This estimated dose is 0.00003 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Picloram, p. 4-29).

Estimated doses using the highest application rate (1 lb/acre) are less than the acute NOAEL for birds and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 1500 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 1.13 mg/kg (SERA 2003 Picloram, Worksheet F16b). This is

0.000754 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible (SERA, 2003-Picloram, p. 4-29).

Picloram does not appear to accumulate or persist in animals. The elimination of picloram has been studied in humans, rats, dogs, and cattle (SERA, 2003-Picloram). In humans, over 75 percent of the administered picloram was eliminated after six hours and over 90 percent was eliminated after 72 hours (SERA, 2003-Picloram citing Nolan et al. 1984). Therefore, chronic exposures from contaminated mammal prey due to a single application of picloram are unlikely to cause any adverse effect. In addition, the acute dose is less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of picloram over time are plausible.

The estimated dose using the highest application rate (1 lb/acre) is less than the acute NOAEL for birds and chronic NOAEL mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 1500 mg/kg. For exposure scenarios that use the typical application rate of 0.35 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 39.5 mg/kg (SERA, 2003-Picloram, Worksheet F14b). This dose is 0.03 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Picloram, p. 4-29).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is greater than the chronic NOAEL for mammals, so adverse effects to insectivorous birds appear plausible from chronic dietary exposures.

The estimated dose using the highest application rate (1 lb/acre) is less than the acute NOAEL for birds, but greater than the chronic NOAEL for mammals. The acute dose (113 mg/kg) is also greater than the chronic LOAEL for mammals (35 mg/kg/day), so adverse effects to insectivorous birds appear plausible from chronic dietary exposures.

Sethoxydim

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 160 mg/kg. For, exposure scenarios that use the typical application rate of 0.30 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of

7.27 mg/kg (Project file, Sethoxdim Worksheet F02a). This estimated dose is 0.05 and 0.005 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA 2001 Sethoxydim, p. 4-19).

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 0.997 mg/kg for acute exposure (Project file, Sethoxdim Worksheet F05). If a small mammal consumes contaminated water over time,

accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.0000527 mg/kg/day (Project file, Sethoxdim Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.006 of the acute NOAEL, and 0.000006 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA 2001 Sethoxydim, p. 4-19).

At the highest application rate of 0.375 lb/acre, the acute dose from drinking water contaminated by a spill is 0.997 mg/kg (project file). This dose is 0.006 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal 79

The acute NOAEL for mammals in laboratory toxicity tests is 160 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percent of the diet contaminated, it would receive an acute dose of 14.6 mg/kg (Project file, Sethoxdim Worksheet F10). This dose is 0.09 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2001-Sethoxydim, p. 4-19).

The chronic NOAEL for mammals in laboratory toxicity tests is 9 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 0.701 mg/kg/day (Project file, Sethoxdim Worksheet F11a). This dose is 0.08 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2001-Sethoxydim, p. 4-19).

Estimated doses using the highest application rate (0.375 lb/acre) are less the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2001 Sethoxydim, p. 4-19).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 160 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.629 mg/kg (Project file,

Sethoxdim Worksheet F16a). Doses to a large mammal would be even lower on per kg body weight basis. This dose is 0.004 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible (SERA 2001 Sethoxydim, p. 4-19).

There is no information in the risk assessment (SERA 2001 Sethoxydim) on accumulation or elimination of sethoxydim in mammals. Therefore, the potential for chronic exposures from contaminated mammal prey due to a single application of sethoxydim cannot be deduced. However, the acute dose is less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of sethoxydim over time are plausible.

Estimated doses using the highest application rate (0.375 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 160 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.804 mg/kg (Project file, Sethoxdim Worksheet F03). This estimated dose is 0.005 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2001-Sethoxydim, p. 4-19).

The chronic NOAEL for mammals in laboratory toxicity tests is 9 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.00773 mg/kg/day (Project file, Sethoxdim Worksheet F04a). This estimated dose is 0.0009 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2001-Sethoxydim, p. 4-19).

Estimated doses using the highest application rate (0.375 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2001-Sethoxydim, p. 4-19).

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 160 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 20.8 mg/kg (Project file, Sethoxdim Worksheet 14a). This dose is 0.10 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible.

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is greater than the chronic NOAEL and the chronic LOAEL (18 mg/kg/day) for mild anemia. So adverse effects to insectivorous mammals appear plausible from chronic dietary exposures. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however.

The estimated dose using the highest application rate (0.375 lb/acre) is less than the acute NOAEL, but greater than the chronic NOAEL for mammals, so adverse effects to insectivorous mammals are plausible from chronic dietary exposures.

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 500 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is

contaminated, it would receive an acute dose of 22.8 mg/kg (Project file, Sethoxdim Worksheet F12). This dose is 0.05 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2001-Sethoxydim, p. 4-19).

The chronic LOAEL for birds in laboratory toxicity tests is 10 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 1.10 mg/kg/day (Project file, Sethoxdim Worksheet F13a). This estimated dose is 0.1 of the chronic LOAEL. If we apply the standard EPA conversion for extrapolating from a LOAEL to a NOAEL, the NOAEL becomes 1 mg/kg, and the dose is equal to the chronic NOAEL. At this dose, adverse reproductive effects to large grass-eating birds are not likely.

Estimated doses using the highest application rate (0.375 lb/acre) are less than the acute NOAEL and chronic LOAEL. But the estimated dose is greater than the extrapolated chronic NOAEL for birds, so adverse effects to grass-eating birds is plausible from chronic dietary exposures at the highest application rate.

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of sethoxydim in fish was studied in bluegill and catfish. Bioconcentration factors (BCF) for catfish were 0.71 L/kg in muscle and 0.75 L/kg in whole fish (SERA, 2001-Sethoxydim, Appendix 3). BCF for bluegill sunfish were substantially higher, measuring 7 L/kg in muscle and 21 L/kg in whole fish (SERA, 2001-Sethoxydim, Appendix 3). The BCF for acute exposure is calculated using the elimination half-life of sethoxydim residue in fish, to adjust for the expected bioconcentration after one day (SERA, 2001-Sethoxydim, p. 3-16). The exposure scenarios in the SERA risk assessment use a whole-fish BCF of 3.6 L/kg for acute exposure and 21 L/kg for chronic exposure.

The acute NOAEL for birds in laboratory toxicity tests is 500 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 3.68 mg/kg (Project file, Sethoxdim Worksheet F08). This dose is 0.007 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2001-Sethoxydim, p. 4-19).

The chronic LOAEL for birds in laboratory toxicity tests is 10 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.00113 mg/kg/day (Project file, Sethoxdim Worksheet F09). This estimated dose is 0.0001 of the chronic LOAEL. If we apply the standard EPA safety factor for extrapolating from a LOAEL to a NOAEL, the NOAEL becomes 1 mg/kg. The dose is 0.001of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible (SERA, 2001-Sethoxydim, p. 4-19).

Estimated doses using the highest application rate (0.375 lb/acre) also result in exposures less than the acute and extrapolated chronic NOAELs for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 500 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.97 mg/kg (Project file, Sethoxdim Worksheet F16b). This is 0.002 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

There is no information in the risk assessment (SERA, 2001-Sethoxydim) on accumulation or elimination of sethoxydim in mammals. Therefore, the potential for chronic exposures from contaminated mammal prey due to a single application of sethoxydim cannot be deduced. However, the acute dose is less than the chronic LOAEL, and the extrapolated NOAEL, for birds, so there is no basis for asserting/predicting that adverse effects from repeated acute exposures from multiple applications of sethoxydim over time are plausible.

The estimated dose using the highest application rate (0.375 lb/acre) is less than the acute NOAEL and less than the chronic LOAEL. The dose (1.21 mg/kg) is greater than the extrapolated chronic NOAEL for birds. Therefore, adverse effects to predatory birds appear plausible from chronic dietary exposures at the highest application rate, base on dose exceeding an extrapolated chronic NOAEL.

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 500 mg/kg. For exposure scenarios that use the typical application rate of 0.30 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 33.8 mg/kg (Project file, Sethoxdim Worksheet F14b). This dose is 0.07 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2001-Sethoxydim, p. 4-19).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is 3 times greater than the chronic LOAEL for birds, so adverse effects to reproduction of insectivorous birds are expected from chronic dietary exposures. The estimated dose using the highest application rate (0.375 lb/acre) is less than the acute NOAEL, but 4 times greater than the chronic LOAEL for birds. Therefore, adverse effects to reproduction of insectivorous birds are expected from chronic dietary exposures at the highest application rate.

Sulfometuron Methyl

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 87 mg/kg. For, exposure scenarios that use the typical application rate of 0.045 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 1.09 mg/kg (SERA 2003 Sulfometuron methyl, Worksheet F02a). This estimated dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

At the highest application rate of 0.38 lb/acre, the animal would receive an acute dose of 9.21 mg/kg (project file). This dose is 0.1 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible at any application rate.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 0.122 mg/kg for acute exposure (SERA 2003 Sulfometuron methyl, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.461 mg/kg/day (SERA 2003 Sulfometuron methyl, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.001 of the acute NOAEL, and 0.0000002 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible (SERA 2003 Sulfometuron methyl, p. 4-30 and 4-31).

At the highest application rate of 0.38 lb/acre, the acute dose from drinking water contaminated by a spill is 1.03 mg/kg (project file). This dose is 0.01 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 87 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 2.19 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet F10). This dose is 0.03 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

The chronic NOAEL for mammals in laboratory toxicity tests is 2 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 0.35 mg/kg/day (SERA, 2003-Sulfometuron methyl, Worksheet F11a). This dose is 0.2 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

Estimated doses using the highest application rate (0.38 lb/acre) are less than the acute NOAEL, but greater than the chronic NOAEL for mammals. The chronic dose (2.95 mg/kg) is less than the chronic LOAEL (20 mg/kg/day) for effects to blood and bile ducts. No adverse effects are plausible from acute exposures, but adverse effects to large herbivorous mammals appear plausible from chronic dietary exposures, based on dose exceeding the chronic NOAEL. However, the assumptions in the chronic exposure scenario are very unlikely to occur in field conditions, so the weight of evidence suggests that no adverse effects are plausible using typical or worst-case exposure assumptions (SERA 2003 Sulfometuron methyl, p. 4-31).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 87 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it

would receive an acute dose of 0.0944 mg/kg (SERA, 2003 Sulfometuron methyl, Worksheet F16a). Doses to a larger mammal would be even lower on a per kg body weight basis. This dose is 0.001 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible SERA, 2003 -ulfometuron methyl, p. 4-30.

Sulfometuron methyl is eliminated fairly rapidly and does not appear to accumulate in animal tissues (SERA, 2003-Sulfometuron methyl). The metabolism of sulfometuron methyl has been studied in lactating goats and rats. Goats eliminated 94-99 percent in the urine (Keoppe and Mucha, 1991 cited in SERA, 2003-Sulfometuron methyl). The half-life for metabolism in rats is 28 hours after a gavage dose of 16 mg/kg and 40 hours after a dose of 3000 mg/kg (DuPont, 1989 cited in SERA, 2003-Sulfometuron methyl). Therefore, chronic exposures from contaminated mammal prey due to a single application of sulfometuron methyl are unlikely to cause any adverse effect. In addition, the acute dose is much less than the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of sulfometuron methyl over time are plausible.

The estimated dose using the highest application rate (0.38 lb/acre) is less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 87 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.121 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet F03). This estimated dose is 0.001 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

The chronic NOAEL for mammals in laboratory toxicity tests is 2 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.00386 mg/kg/day (SERA, 2003-Sulfometuron methyl, Worksheet F04a). This estimated dose is 0.002 of the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible (SERA, 2003-Sulfometuron methyl, p. 4-31).

Estimated doses using the highest application rate (0.38 lb/acre) are less than the acute and chronic NOAELs for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 87 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 3.12 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet 14a). This dose is 0.04 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammal insectivores are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. The acute dose is greater than the chronic NOAEL (2

mg/kg/day), but less than the chronic LOAEL (20 mg/kg/day) for effects to blood and bile ducts. So adverse effects to insectivorous mammals appear plausible from chronic dietary exposures, based on dose exceeding the chronic NOAEL. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however.

The estimated dose (26.4 mg/kg) using the highest application rate (0.38 lb/acre) is less than the acute NOAEL. But the acute dose is greater than the chronic NOAEL and the chronic LOAEL (20 mg/kg/day) for effects to blood and bile ducts. No adverse effects are plausible from acute exposures, but adverse effects to insectivorous mammals are plausible, and may be expected, from chronic dietary exposures at the maximum application rate.

Large Herbivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 312 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 3.42 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet F12). This dose is 0.01 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

There is no chronic toxicity index available for effects of sulfometuron methyl to birds, so the mammal chronic NOAEL will be used (acute toxicities of sulfometuron methyl to mammals and birds are of similar magnitude (SERA 2003 Sulfometuron methyl, p. 4-24)). The chronic NOAEL for mammals in laboratory toxicity tests is 2 mg/kg/day. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 0.547 mg/kg/day (SERA, 2003-Sulfometuron methyl, Worksheet F13a). This estimated dose is 0.3 of the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects to large grass-eating birds are plausible (SERA, 2003-Sulfometuron methyl, p. 4-31).

Estimated doses using the highest application rate (0.38 lb/acre) are less than the acute NOAEL for birds, but greater than the chronic NOAEL for mammals. The chronic dose (4.62 mg/kg/day) is less than the chronic LOAEL for mammals. No adverse effects are plausible from acute exposures, but adverse effects to large herbivorous birds appear plausible from chronic dietary exposures, based on dose exceeding a NOAEL. However, the assumptions in the chronic exposure scenario are very unlikely to occur in field conditions, so the weight of evidence suggests that no adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Sulfometuron methyl, p. 4-31).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of sulfometuron methyl in fish was studied in bluegill sunfish and channel catfish exposed to ^{14}C-sulformeturon methyl for 28 days (Harvey, 1981, cited in SERA, 2003-Sulfometuron methyl, p. 3-21). In the SERA risk assessments, concentrations in viscera are considered to reflect concentration in whole fish. No bioaccumulation occurred in either muscle or viscera of bluegill.

Bioconcentration Factors (BCF) for viscera of channel catfish after one day of exposure was 3.5 L/kg, and 6 L/kg after 28 days (SERA, 2003-Sulfometuron methyl, Appendix 2). Therefore, exposure scenarios in the SERA risk assessment use a whole-fish BCF of 3.5 L/kg for acute exposure and 6 L/kg for chronic exposure.

The acute NOAEL for birds in laboratory toxicity tests is 312 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 0.437 mg/kg (SERA, 200X, Worksheet F08). This dose is 0.001 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

There is no chronic toxicity index available for effects of sulfometuron methyl to birds, so the mammal chronic NOAEL will be used (acute toxicities of sulfometuron methyl to mammals and birds are of similar magnitude (SERA, 2003-Sulfometuron methyl, p. 4-24)).

The chronic NOAEL for mammals in laboratory toxicity tests is 2 mg/kg/day. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.000003 mg/kg/day (SERA, 200X-Worksheet F09). This estimated dose is 0.000001 of the chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible (SERA 2003 Sulfometuron methyl, p. 4-31).

Estimated doses using the highest application rate (0.38 lb/acre) also result in exposures much less than the acute NOAEL for bird and chronic NOAEL for mammals, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Sulfometuron methyl, p. 4-31).

Large Predatory Bird

The acute NOAEL for birds in laboratory toxicity tests is 312 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 0.145 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet F16b). This is 0.0005 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Sulfometuron methyl does not appear to accumulate in animal tissues. The elimination of this herbicide has been studied in lactating goats and rats (SERA, 2003-Sulfometuron methyl). Goats eliminated 94-99 percent in the urine (Keoppe and Mucha 1991 cited in SERA, 2003-Sulfometuron methyl). The half-life for metabolism in rats is 28 hours after a gavage dose of 16 mg/kg and 40 hours after a dose of 3000 mg/kg (DuPont, 1989 cited in SERA, 2003-Sulfometuron methyl). Therefore, chronic exposures from contaminated mammal prey due to a single application of sulfometuron methyl are unlikely to cause any adverse effect. In addition, the acute dose is less than the chronic NOAEL for mammals, so there is no basis for asserting/predicting that adverse effects from repeated acute exposures from multiple applications of sulfometuron methyl over time are plausible.

Estimated doses using the highest application rate (0.38 lb/acre) are less than the acute NOAEL for birds and chronic NOAEL for mammals, so there is no basis for asserting or predicting that

adverse effects are plausible using typical or worst-case exposure assumptions (SERA, 2003-Sulfometuron methyl, p. 4-30 and 4-31).

Small Insectivorous Bird

The acute NOAEL for birds in laboratory toxicity tests is 312 mg/kg. For exposure scenarios that use the typical application rate of 0.045 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 5.08 mg/kg (SERA, 2003-Sulfometuron methyl, Worksheet F14b). This dose is 0.02 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous birds are plausible (SERA, 2003-Sulfometuron methyl, p. 4-30).

There is no chronic toxicity index available for effects of sulfometuron methyl to birds, so the mammal chronic NOAEL will be used (acute toxicities of sulfometuron methyl to mammals and birds are of similar magnitude (SERA, 2003-Sulfometuron methyl, p. 4-24)). Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. The acute dose is greater than the chronic NOAEL for mammals (2 mg/kg/day), but less than the chronic LOAEL (20 mg/kg/day) for mammals. So adverse effects to insectivorous birds appear plausible from chronic dietary exposures, based on an acute dose exceeding a chronic NOAEL.

The estimated dose using the highest application rate (0.38 lb/acre) is less than the acute NOAEL for birds, but greater than the chronic NOAEL for mammals. The acute dose (42.9 mg/kg/day) is also two times greater than the chronic mammal LOAEL for effects to blood and bile ducts. No adverse effects are plausible from acute exposures, but adverse effects to insectivorous birds are plausible, and may be expected, from chronic dietary exposures at the maximum application rate.

Triclopyr

Toxicity indices and doses are the same for triclopyr acid and triclopyr BEE for mammals, but they differ for birds. The EPA has used two different values for a reference dose on the effects of triclopyr to mammals. The FS/SERA risk assessment (2003 Triclopyr) relies on a chronic toxicity index (NOEL of 5 mg/kg/day) from a rat reproduction study. In this analysis, we will use a lower value from a 1-year feeding study of dogs (chronic NOEL of 0.5 mg/kg/day; Quast et al. 1976, cited in SERA, 2003-Triclopyr). Dogs were not considered by EPA to be a good model for human health effects, because they do not excrete weak acids as well as other animals (see Timchalk and Nolan 1997; Timchalk et al. 1997). Canids are, however, relevant for concerns about effects to wildlife. It may be argued that the use of the 0.5 mg/kg/day value for the toxicity index in this analysis is overly cautious, because it represents competition for excretion rather than a toxic effect (Timchalk et al. 1997), and because it is being applied to other animals besides canids. However, it meets the criteria for providing a data-based worst-case analysis for potential effects to wildlife, and is therefore consistent with the criteria for choice of other indices used in this analysis.

Small Mammal Directly Sprayed

The acute NOAEL for mammals in laboratory toxicity tests is 100 mg/kg. For, exposure scenarios that use the typical application rate of 1 lb/acre, if a small mammal is directly sprayed, and 100 percent absorption is assumed, the animal would receive an acute dose of 24.2 mg/kg (SERA, 2003-Triclopyr, Worksheet F02a). This estimated dose is 0.2 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible.

At the highest application rate of 10 lb/acre, the animal would receive an acute dose of 242 mg/kg (project file). This dose is greater than the acute NOAEL but less than the acute LOAEL for malformed fetuses, although not substantially; therefore adverse effects are plausible from direct spray at the highest application rate, based on dose exceeding the NOAEL.

Small Mammal Drinking Contaminated Water

The estimated doses to a small mammal from drinking water contaminated by an accidental spill, assuming the highest levels of contamination, are 2.66 mg/kg for acute exposure (SERA, 2003-Triclopyr, Worksheet F05). If a small mammal consumes contaminated water over time, accounting for dissipation, degradation, and other processes, the animal would receive a chronic dose of 0.00732 mg/kg/day (SERA, 2003-Triclopyr, Worksheet F07). Doses to a large mammal would be even lower on a per kg body weight basis. These doses are 0.03 of the acute NOAEL, and 0.01 of the chronic NOAEL, respectively, so there is no basis for asserting or predicting that adverse effects to mammals are plausible.

At the highest application rate of 10 lb/acre, the acute dose from drinking water contaminated by a spill is 26.6 mg/kg (project file). This dose is 0.3 of the acute NOAEL. The chronic dose is also below the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to mammals are plausible, even in a worst-case scenario.

Large Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 100 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a 70 kg mammal consumed contaminated vegetation on site shortly after application, assuming the highest residue rates and 100 percents of the diet contaminated, it would receive an acute dose of 48.6 mg/kg (SERA, 2003-Triclopyr, Worksheet F10). This dose is 0.5 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to large grass-eating mammals are plausible.

The chronic NOAEL for mammals in laboratory toxicity tests is 0.5 mg/kg/day. Chronic exposure from the consumption of contaminated vegetation for 90 days at the treatment site, assuming the highest residue rates, results in a dose of 32.0 mg/kg/day (SERA, 2003-Triclopyr, Worksheet F11a). This dose is greater than the chronic NOAEL and 13 times greater than the LOAEL of 2.5 mg/kg for effects to kidneys. Adverse effects to grass-eating mammals are plausible and of substantial concern with the use of triclopyr (SERA, 2003-Triclopyr, p. 4-28).

Estimated doses using the highest application rate (10 lb/acre) are greater than the acute and chronic NOAELs for mammals. The acute dose is 486 mg/kg; which also exceeds the acute LOAEL for malformed fetuses. The chronic dose is 320 mg/kg; which exceeds the chronic LOAEL for effects to kidneys. Adverse effects to reproduction and internal organs of grass-eating mammals are plausible with acute and chronic exposures at the highest application rate. The potential for adverse effects are of substantial concern with the use of triclopyr (SERA, 2003-Triclopyr, p. 4-28).

Medium Carnivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 100 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a 5 kg mammal consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 2.10 mg/kg (SERA 2003 Triclopyr, Worksheet F16a). Doses to a larger mammal would be even lower on a per kg body weight basis. This dose is 0.021 of the

acute NOAEL, so there is no basis for asserting or predicting that adverse effects to carnivorous mammals are plausible.

Triclopyr acid and triclopyr BEE do not appear to accumulate or persist in animals. The elimination of triclopyr has been studied in rats and cattle (SERA 2003 Triclopyr). A study by Timchalk et al. (1990) found that the half-life for elimination in rats is 3.6 hours and that virtually all the ingested dose of triclopyr is excreted unchanged in the urine; although four minor metabolites are formed. In cattle, over 86 percent of the ingested dose was eliminated unchanged in the urine and almost all the dose was eliminated after 24 hours (Eckerlin et al. 1987, cited in SERA 2003). Therefore, chronic exposures from contaminated mammal prey due to a single application of triclopyr are unlikely to cause any adverse effect. However, the acute dose is greater than the chronic NOAEL for mammals, but slightly less than the chronic LOAEL, so adverse effects to carnivorous mammals appear plausible from chronic dietary exposures.

The estimated dose using the highest application rate (10 lb/acre) is less than the acute NOAEL, but greater than the chronic LOAEL for effects to kidneys of mammals. No adverse effects are plausible from acute exposures, but adverse effects to carnivorous mammals appear plausible from chronic dietary exposures at the maximum application rate.

Small Herbivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 100 mg/kg. If a small mammal consumes contaminated vegetation shortly after application, assuming the highest residue rates, the acute dose received is 0.495 mg/kg (SERA, 2003-Triclopyr, Worksheet F03). This estimated dose is 0.005 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible.

The chronic NOAEL for mammals in laboratory toxicity tests is 0.5 mg/kg/day. If a small mammal consumes contaminated vegetation at the treatment site for 90-days, assuming highest residue rates, the animal would receive a chronic dose of 0.0652 mg/kg/day (SERA, 2003-Triclopyr, Worksheet F04a). This estimated dose is 0.1 the chronic NOAEL, so there is no basis for asserting or predicting that adverse effects to herbivorous mammals are plausible.

Estimated doses using the highest application rate (10 lb/acre) are less than the acute NOAEL, but slightly greater than the chronic NOAELs for mammals. The chronic dose (0.65 mg/kg/day) is less than the chronic LOAEL (2.5 mg/kg/day) for effects to kidneys. No adverse effects are plausible from acute exposures, but adverse effects to herbivorous mammals appear plausible from chronic dietary exposures at the maximum application rate, based on dose exceeding a NOAEL.

Small Insectivorous Mammal

The acute NOAEL for mammals in laboratory toxicity tests is 100 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a small mammal consumes contaminated insects shortly after application, assuming the highest residue rates, the acute dose received is 69.4 mg/kg (SERA, 2003-Triclopyr, Worksheet 14a). This dose is 0.694 of the acute NOAEL, so there is no basis for asserting or predicting that adverse effects to insectivorous mammals are plausible.

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is much greater than the chronic

LOAEL for mammals, so adverse effects to insectivorous mammals appear plausible from chronic dietary exposures. The exposure scenario uses residue rates from small insects, which are substantially higher than those for large insects, and assumes that 100 percent of the daily diet is composed of insects that have been directly sprayed. For bats, in particular, the scenario is unlikely to occur in the field. It seems more plausible for shrews and small fossorial insectivores, however.

The estimated dose (694 mg/kg) using the highest application rate (10 lb/acre) is much greater than the acute and chronic NOAELs for mammals. The acute dose is more than two times greater than the acute LOAEL for malformed fetuses and more than 200 times greater than the chronic LOAEL for effects to kidneys. Therefore, adverse effects to insectivorous mammals may be expected if they feed on insects contaminated with triclopyr applied at the highest application rate.

Large Herbivorous Bird

Triclopyr BEE is slightly more toxic to birds in acute exposures than triclopyr acid. For triclopyr acid, the acute LD_{50} for birds in laboratory toxicity tests is 535 mg/kg and for triclopyr BEE the acute LD_{50} is 388 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a 4 kg bird consumed contaminated grass on site shortly after application, assuming the highest residue rates and 100 percent of diet is contaminated, it would receive an acute dose of 76.0 mg/kg (SERA 2003 Triclopyr, Worksheets F12). This dose is 0.1 of the acute LD_{50} for triclopyr acid and 0.2 of the acute LD_{50} for triclopyr BEE. Since the acute exposure scenario for bird is based on an LD_{50} rather than an acute NOAEL, the FS/SERA risk assessments base the level of concern on 0.1 of the LD_{50} (SERA, 2003-Triclopyr), a factor used by EPA as a result of data analysis and modeling conducted by their Office of Pesticide Programs (Urban and Cook, 1986). Therefore, acute exposure from triclopyr acid is equal to the level of concern and that from triclopyr BEE is greater than the level of concern (SERA 2003 Triclopyr). Adverse effects to grass-eating birds are plausible and of substantial concern with the use of triclopyr (SERA, 2003-Triclopyr, p. 4-28).

The chronic NOAEL for birds in laboratory toxicity tests is 10 mg/kg/day for both triclopyr acid and triclopyr BEE. Chronic exposure from the consumption of contaminated grass for 90 days at the treatment site, assuming the highest residue rates and 100 percent of diet is contaminated, results in a dose of 50.1 mg/kg/day (SERA, 2003-Triclopyr, Worksheets F13a). This estimated dose is greater than the chronic NOAEL and more than two times greater than the chronic LOAEL for decreased survival of offspring. The assumptions in the chronic exposure scenario are unlikely to occur in field conditions, however, adverse effects reproduction of grass-eating birds are plausible and of substantial concern with the use of triclopyr (SERA, 2003-Triclopyr, p. 4-28).

At the highest application rate (10 lb/acre), the acute dose is 760 mg/kg, which is greater than the acute LD_{50} for birds, for both triclopyr acid and triclopyr BEE. Mortality could be expected for birds feeding on vegetation contaminated with triclopyr applied at the highest application rate. In the case of the chronic exposures, the estimated dose (501 mg/kg/day) is much greater than the chronic LOAEL for decreased survival of offspring. Adverse effects, including mortality and decreased reproduction, to grass-eating birds are plausible and of substantial concern with the use of triclopyr (SERA, 2003-Triclopyr, p. 4-28).

Large Fish-eating Bird

Many chemicals may be concentrated or partitioned from water into the tissues of animals in the water. This process is referred to as bioconcentration. The potential for bioconcentration of triclopyr in fish was studied in bluegill sunfish exposed to ^{14}C-triclopyr (Rick et al., 1996; and Lickly and Murphy, 1987; cited in SERA 2003 Triclopyr). Bioconcentration factors (BCF) of triclopyr and its metabolites (primarily TCP) for bluegill were 0.83 L/kg for whole fish, which is the figure used in the exposure scenarios in the SERA risk assessment for acute and chronic exposures.

Triclopyr BEE is slightly more toxic to birds in acute exposures than triclopyr acid. For triclopyr acid, the acute LD_{50} for birds in laboratory toxicity tests is 535 mg/kg and for triclopyr BEE the acute LD_{50} is 388 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a predatory bird consumed fish from a pond contaminated by an accidental spill, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive an acute dose of 2.26 mg/kg (SERA, 2003-Triclopyr, Worksheet F08). This dose is 0.004 of the acute LD_{50} for triclopyr acid, and 0.006 of the acute LD50 for triclopyr BEE. Since the acute exposure scenario for bird is based on an LD_{50} rather than an acute NOAEL, the FS/SERA risk assessments base the level of concern on 0.1 of the LD_{50} (SERA, 2003-Triclopyr), a factor used by EPA as a result of data analysis and modeling conducted by their Office of Pesticide Programs (Urban and Cook 1986). The resultant values are much less than the level of concern, so there is no basis for asserting or predicting that adverse effects to fish-eating birds are plausible.

The chronic NOAEL for birds in laboratory toxicity tests is 10 mg/kg/day for both triclopyr acid and triclopyr BEE. If a predatory bird consumed fish contaminated by runoff for a lifetime, assuming the highest concentrations in fish and highest intake on a body weight basis, it would receive a chronic dose of 0.00623 mg/kg/day (SERA, 2003-Triclopyr, Worksheet F09). This estimated dose is 0.0006 of the chronic NOAEL, so there is no basis for asserting/predicting that adverse effects to fish-eating birds are plausible.

Estimated doses using the highest application rate (10 lb/acre) are less than 0.1 of the acute LD_{50} and the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects are plausible using typical or worst-case exposure assumptions.

Large Predatory Bird

Triclopyr BEE is slightly more toxic to birds in acute exposures than triclopyr acid. For triclopyr acid, the acute LD_{50} for birds in laboratory toxicity tests is 535 mg/kg and for triclopyr BEE the acute LD_{50} is 388 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a 0.6 kg bird consumed small mammal prey that has been contaminated by direct spray, assuming 100 percent absorption for the prey, it would receive an acute dose of 3.23 mg/kg (SERA, 2003-Triclopyr, Worksheet F16b). This is 0.00604 of the acute LD_{50} for triclopyr acid and 0.00833 of the acute LD_{50} for triclopyr BEE. Since the acute exposure scenario for bird is based on an LD_{50} rather than an acute NOAEL, the FS/SERA risk assessments base the level of concern on 0.1 of the LD_{50} (SERA, 2003-Triclopyr), a factor used by EPA as a result of data analysis and modeling conducted by their Office of Pesticide Programs (Urban and Cook, 1986).

The resultant values are much less than the level of concern, so there is no basis for asserting or predicting that adverse effects to predatory birds are plausible.

Triclopyr acid and triclopyr BEE do not appear to accumulate or persist in animals. The elimination of triclopyr has been studied in rats and cattle (SERA, 2003-Triclopyr). A study by Timchalk et al. (1990) found that the half-life for elimination in rats is 3.6 hours and that virtually all of the ingested dose of triclopyr is excreted unchanged in the urine, although four minor metabolites are formed. In cattle, over 86 percent of the ingested dose was eliminated unchanged in the urine and almost all of the dose was eliminated after 24 hours (Eckerlin et al., 1990). Therefore, chronic exposures from contaminated mammal prey due to a single application of triclopyr are unlikely to cause any adverse effect. In addition, the acute dose is less than the chronic NOAEL for birds, so there is no basis for asserting or predicting that adverse effects from repeated acute exposures from multiple applications of predatory birds over time are plausible.

Estimated doses using the highest application rate (10 lb/acre) are less than 0.1 of the LD_{50} for both triclopyr acid and triclopyr BEE, although only marginally so for triclopyr BEE (acute dose of 32.3 vs. 38.8 for 0.1 of the LD_{50}). The acute dose (32.3 mg/kg) is greater than the bird chronic LOAEL (20 mg/kg) for decreased survival of offspring, so adverse affects to predatory birds are plausible from triclopyr at the highest application rate.

Small Insectivorous Bird

Triclopyr BEE is slightly more toxic to birds in acute exposures than triclopyr acid. For triclopyr acid, the acute LD_{50} for birds in laboratory toxicity tests is 535 mg/kg and for triclopyr BEE the acute LD_{50} is 388 mg/kg. For exposure scenarios that use the typical application rate of 1 lb/acre, if a 10 g bird consumed contaminated insects on site shortly after application, assuming the highest residue rates, it would receive an acute dose of 113 mg/kg (SERA 2003 Triclopyr, Worksheet F14b). This dose is 0.2 of the acute LD_{50} for triclopyr acid, and 0.3 of the LD_{50} for triclopyr BEE. Since the acute exposure scenario for bird is based on an LD_{50} rather than an acute NOAEL, the FS/SERA risk assessments base the level of concern on 0.1 of the LD_{50} (SERA 2003 Triclopyr), a factor used by EPA as a result of data analysis and modeling conducted by their Office of Pesticide Programs (Urban and Cook 1986). Therefore, the acute dose is two times greater than the level of concern for triclopyr acid, and three times greater than the level of concern for triclopyr BEE (but less than both LD_{50}s). Adverse effects to insectivorous birds are plausible, assuming the highest residue rates.

Data on degradation of herbicide residues from insects is not available, so no chronic exposure scenario has been developed. Residue on insects likely declines over time, but the extent of decline has not been quantified. However, the acute dose is five times greater than the chronic LOAEL for decreased survival of offspring in birds, so adverse effects to insectivorous birds may be expected from chronic dietary exposures.

Estimated dose from contaminated insects, assuming the highest residue rates, at the highest application rate (10 lb/acre) is 1,130 mg/kg. This dose is two times greater than the LD_{50} for triclopyr acid and three times greater than the LD_{50} for triclopyr BEE. Mortality is expected if insectivorous birds feed exclusively within the treatment area on contaminated insects.

Literature Cited for Summary of Herbicide Effects to Wildlife

Anonymous 1999. Are "inert" ingredients in pesticides really benign? J. Pesticide Reform 19 (2): 8

Anthony, R.G., and Morrison, M.L. 1985. Influence of glyphosate herbicide on small-mammal populations in western Oregon. Northwest Science 59(3):159-68.

ATSDR (Agency for Toxic Substances and Disease Registry). 2004. Guidance manual for the assessment of joint toxic action of chemical mixtures. Available from U.S. Department of Health and Human Services, Public Health Service, ATSDR, Division of Toxicology.

Berrill, M., S. Bertram, and B. Pauli. 1997. Effects of Pesticides on amphibian embryos and larvae. Hepetological Conservation 1: 233-245.

Berrill, M., S. Bertram, B., L. McGillivray, M. Kolohon, and B. Pauli. 1994. Effects of low concentrations of forest-use pesticides on frog embryos and tadpoles. Environmental Toxicol. and Chemistry 13(4): 657-664.

Borrecco, J.E., and J. Neisess. 1991. Risk Assessment for the Impurities 2-Butoxyethanol and 1,4-Dioxane found in Garlon 4 and Roundup Herbicide Formulations. Forest Pest Management, Report No. R91-2. Pacific Southwest Region. 33 pp.

Bramble, W.C.; R.H. Yahner; and W.R. Byrnes. 1997. Effect of herbicides on butterfly populations of an electric transmission right-of-way. J. Arboriculture 23(5): 196-206.

Brooks, J.J.; J.L. Rodrigue; M.A. Cone; K.V. Miller; B.R. Chapman; and A.S. Johnson. 1995. Small mammal and avian communities on chemically-prepared sites in the Georgia sandhills. USDA Forest Service, Gen. Tech. Rep.SRS-S. pp. 21-23.

Calabrese, E.J. and L.A. Baldwin.1993. Performing Ecological Risk Assessments. Lewis Publishers. Ann Arbor, Michigan.257pp.

Chamberlain, K, AA, Evan, RH Bromilow. 1996. 1-octanol/waqter partition coefficient (Kow) and pka for ionisable pesticides measured by a ph-metric method. Pest Sci. 47(3): 265-271.

Cole, E.C., McComb, W.C., Newton, M., Leeming JP, and Chambers, C. L. 1998. Response of small Mammals to Clearcutting, Burning, and Glyphosate Application in the Oregon Coast Range. Journal of Wildlife Management 62(4):1207-16.

Cowman, D.F. and L.E. Mazanti. Ecotoxocology of "new generation" pesticides to amphibians. Pp. 233-268 In D.W. Sparling, G. Linder, and C.A. Bishop, eds. Ecotoxicology of Amphibians and Reptiles. Society of Environmental Toxicology and Chemistry. Pensacola, Florida. 904pp.

Cox, C. 1999. Inert ingredients in pesticides: who's keeping secrets? J. Pesticide Reform 19 (3): 2-7.

Dow AgroSciences. 1998. Clopyralid: A North American Technical Profile. Dow AgroSciences LLC. Indianapolis, Indiana. Cited in SERA 2003 Clopyralid.

Durkin, P. Personal communication. Syracuse Environmental Research Associates, Inc. Fayetteville, New York.

Eckerlin, R.H., J.G. Ebel Jr., G.A. Maylin, T.V. Muscato, W.H. Gutenmann, C.A. Bache, and D.J. Lisk. 1987. Excretion of triclopyr herbicide in the bovine. Bull Environ Contam Toxicol. 39(3): 443-447.

Fletcher, J.S., J.E. Nellessen, and T.G. Pfleeger. 1994. Literature review and evaluation of the EPA food-chain (Kenaga) nomogram, an instrument for estimating pesticide residue on plants. Environ. Toxicol. and Chemistry 13(9): 1383-1391.

Francis, B.M., R.L. Lampan, R.L. Metcalf. 1985. Model ecosystem studies of the environmental fate of five herbicides used in conservation tillage. Arch Environ Contam Toxicol. 14:693-704.

Gaines, TB. 1969. Acute toxicity of pesticides. Tox. Appl. Pharamacol. 14: 515-534 (cited in SERA 2001)

Hall, R.J. 1980. Effects of environmental contaminants on reptiles: a review. USDI Fish and Wildlife Service, Special Scientific Report, Wildlife No. 228. Washington, D.C.12pp.

Hall, R.J. and D.R. Clark, Jr. 1982. Responses of the iguanid lizard Anolis carolinensis to four organophosphorus pesticides. Environmental Pollution (Series A) 28: 45-52.

Hall, R.J. and P.F.P. Henry. 1992. Review: Assessing effects of pesticides on amphibians and reptiles: status and needs. Herpetological J. 2: 65-71.

Johnson, D.R., and Hansen, R.M. 1969. Effects of range treatment with 2,4-D on rodent populations. Journal of Wildlife Management 33: 1125-132.

Knight, H. 1997. Hidden toxic “inerts”: a tragicomedy of errors. J. Pesticide Reform 17 (2): 10-11

Knight, H. and C. Cox. 1998. Worst kept secrets: toxic inert ingredients in pesticides. unpub. report downloaded from: http://www.pesticide.org/ActiveInertsRpt.pdf on January 14, 2004. Northwest Coalition for Alternatives to Pesticides. Eugene, Oregon. 19 pp.

Lautenschlager, R.A. 1993. Response of wildlife to forest herbicide applications in northern coniferous ecosystems. Canadian Journal of Forest Resources 23:2286-99. 104 Preventing and Managing Invasive Plants Final Environmental Impact Statement April 2005 DRAFT

Leslie, D.M. Jr.; R.B. Sper; R.L. Lochmiller; and D.M. Engle. 1996. Habitat use by white-tailed deer on cross timbers rangeland following brush management. J. Range Manage. 49(5): 401-406.

Mann, R.M. and J.R. Bidwell. 1999. The toxicity of glyphosate and several glyphosate formulations to four species of southwestern Australian frogs. Arch. Environ. Contam. Toxicol. 36: 193-199.

Mann, R.M. and J.R. Bidwell. 2000. Application of the FETAX protocol to assess the developmental toxicity of nonylphenol ethoxylate to Xenopus laevis and two Australian frogs. Aquatic Toxicology 51: 19-29.

Mann, R.M. and J.R. Bidwell. 2001. The acute toxicity of agricultural surfactants to the tadpoles of four Australian and two exotic frogs. Environmental Pollution 114: 195-205.

Marquardt, S., C. Cox, and H. Knight. 1998. Toxic Secretes: "inert" ingredients in pesticides 1987-1997. Northwest Coalition for Alternatives to Pesticides. Unpub. report downloaded from http://www.pesticide.org/inertsreport.pdf on January 14, 2004.

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

Nolte, K.R., and T.E. Fulbright. 1997. Plant, small mammal, and avian diversity following control of honey mesquite. J. Range Manage. 50(2): 205-212.

Norris, R.F., and M. Kogan. Interactions between weeds, arthropod pests, and their natural enemies in managed ecosystems. Weed Science 48:94-158.

Pauli, B.D., and S. Money. 2000. Ecotoxicology of pesticides in reptiles. pp. 269-324 In D.W. Sparling, G. Linder, and C.A. Bishop, eds. Ecotoxicology of Amphibians and Reptiles. Society of Environmental Toxicology and Chemistry. Pensacola, Florida. 904pp.

Perkins, P.J., H.J. Boermans, and G.R. Stephenson. 2000. Toxicity of glyphosate and triclopyr using the frog embryo teratogenesis assay – Xenopus. Environmental Toxicol. and Chemistry 19(4): 940-945.

Pfleeger, T.G.; A. Fong; R. Hayes; H. Ratsch; and C. Wickliff. 1996. Field evaluation of the EPA (Kenaga) nomogram, a method for estimating wildlife exposure to pesticide residue on plants. Environmental Toxicology and Chemistry 15(4):535-543.

Rice, P.M.; J.C. Toney; D.J. Bedunah; and C.E. Carlson. 1997. Plant community diversity and growth form responses to herbicide applications for control of Centaurea maculosa. J. Applied Ecol. 34(6): 1397-1412.

Ritchie, D.C., Harestad, A.S., and Archibald, R. 1987. Glyphosate treatment and deer mice in clearcut and forest. Northwest Science 61(3):199-202.

Roberts, B.L., and H.W. Dorough. 1984. Relative toxicities of chemicals to the earthworm Eisenia foetida. Environ. Toxicol. Chem. 3: 67-78.

Santillo, D.J., D.M. Leslie, Jr., and P.W. Brown. 1989. Responses of small mammals and habitat to glyphosate application on clearcuts. J. Wildlife Manage. 53(1): 164-172.

Scholz, N.L. 2003. Evaluating the effects of forestry herbicides on early life history stages of fish. Unpublished proposal submitted to Forest Service Pesticide Impact Assessment Program, 2003. National Oceanographic and Atmospheric Administration, Northwest Fisheries Science Center. Seattle, Washington.

SERA (Syracuse Environmental Research Associates, Inc.). 1998. 2,4-Dichlorophenoxyacetic acid Formulations – Human Health and Ecological Risk Assessment - Final Report. SERA TR 95-21-09-01d. September 20, 1998. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2001. Sethoxydim [Poast] – Human Health and Ecological Risk Assessment - Final Report. SERA TR 01-43-01-01c. October 31, 2001. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2001. Preparation of Environmental Documentation and Risk Assessments. SERA MD 2001-01a. July 18, 2001. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Chlorsulfuron - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 02-43-18-01b. September 5, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Clopyralid - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 02-43-17-03a. August 27, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Dicamba - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 03-43-17-06a. October 31, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Glyphosate - Human Health and Ecological Risk Assessment - Final Report. SERA TR 02-43-09-04a. March 1, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Imazapic - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 02-43-17-04a. September 26, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Imazapyr - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 03-43-17-05a. October 13, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Metsulfuron Methyl - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 03-43-17-01a. September 23, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Picloram - Human Health and Ecological Risk Assessment – Final Report. SERA TR 03-43-16-01b. June 30, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Sulfometuron Methyl - Human Health and Ecological Risk Assessment – Peer Review Draft. SERA TR 03-43-17-02b. October 19, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

SERA (Syracuse Environmental Research Associates, Inc.). 2003. Triclopyr - Human Health and Ecological Risk Assessment – Final Report. SERA TR 02-43-13-03b. March 15, 2003. Syracuse Environmental Research Assoc., Inc. Fayetteville, New York.

Serota, D., C. Burns, G. Burdock, et. al. 1983. Subchronic toxicity study in rats – 2,4-Dichlorophenoxyacetic acid (2,4-D). Project No. 2184-102 Final report. Unpublished study received October 14, 1983 under unknown admin. No. Prepared by Hazleton Laboratories America, by 2,4-D Task Force, Washington, DC. MRID No. 00131304. [Contains confidential business information and is cited in SERA 1998, but not available to the USDA Forest Service).

Siltanen, H., C. Rosenberg, M. Raatikainen, and T. Raatikainen. 1981. Triclopyr, glyphosate and phenoxyherbicide residues in cowberries, bilberries and lichen. Bull. Environ. Contam. Toxicol. 27(5): 731-737.

Smith, G.R. 2001. Effects of acute exposure to a commercial formulation of glyphosate on the tadpoles of two species of Anurans. Bull. Environ. Contam. Toxicol. 67: 483-488. 107

Sullivan, T.P. 1990a. Demographic responses of small mammal populations to a herbicide application in coastal coniferous forest: population density and resiliency. Can. J. Zool. 68: 874-883.

Sullivan, T.P. 1990b. Influence of forest herbicide on deer mouse and Oregon vole population dynamics. J. Wildlife Manage. 54(4): 566-576.

Sullivan, T.P.; C. Nowotny; and R.A. Lautenschlager. 1998. Silvicultural use of herbicide in sub-boreal spruce forest: implications for small mammal population dynamics. Journal of Wildlife Management 62(4): 1196-206.

Sullivan, T.P., and D.S. Sullivan. 1981. Responses of a deer mouse population to a forest herbicide application: reproduction, growth, and survival. Can. J. Zool. 59:1148-1154.

Sullivan, T.P., Sullivan, D.S., Lautenschlager, R.A., and Wagner, R. G. 1997. Long-term influence of glyphosate herbicide on demography and diversity of small mammal communities in coastal coniferous forest. Northwest Science 71(1): 6-17.

Timchalk, C., D.R. Finco, and J.F. Quast. 1997. Evaluation of renal function in rhesus monkeys and comparison to beagle dogs following oral administration of the organic acid triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid). Fundamentals and Applied Toxicol. 36: 47-53.

Timchalk, C., M.D. Dryzga, and P.E. Kastl. 1990. Pharmacokinetics and metabolism of triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid) in Fischer 344 rats. Toxicology 62: 71-87.

Timchalk, C., and R.J. Nolan. 1997. Pharmacokinetics of triclopyr (3,5,6-trichloro-2-pyridinyloxyacetic acid) in the beagle dog and rhesus monkey: perspective on the reduced capacity of dogs to excrete this organic acid relative to the rat, monkey, and human. Toxicol. and Applied Pharmacol. 144: 268-278.

Urban, D.J, and N.J. Cook. Hazard Evaluation Division, Standard Evaluation Procedure, Ecological Risk Assessment. unpublished report. US EPA, Office of Pesticide Programs, Hazard Evaluation Division. Washington, D.C.

US EPA/ORD (Environmental Protection Agency/Office of Research and Development). 1993. Wildlife Exposure Factors Handbook. Volumes 1 and 2. EPA/600/R-93/187a,b. Available NTIS: PB94-174778 and PB94-174779.

USEPA (Environmental Protection Agency). 1997. Status of data for 2,4-D. Accessed at: http://www.epa.gov/ngispgm3/.

USEPA (Environmental Protection Agency). 2000. Supplementary guidance for conductiong Health Risk Assessment of chemical mixtures. EPA/630/r-00/002. Washington, D.C. 143 pp. + appendices.

USDA Forest Service. 2002. Analysis of Issues Surrounding the Use of Spray Adjuvants with Herbicides. David Bakke. Pacific Southwest Region. Vallejo, California.

USDA Forest Service. 2003. Human and Ecological Risk Assessment of NPE Surfactants in Forest Service Herbicide Applications, 2003. David Bakke. Pacific Southwest Region. Vellego, California.

Weed Science Society of America. 2002. Herbicide Handbook. 8th edition. W.K. Vencill (ed.). Weed Science Society of America. Lawrence, Kansas. 493pp.

Yu et al. 1975 bioconcentration of dicamba.

APPENDIX 1 of Summary of Herbicide Effects to Wildlife

Estimated doses for each exposure scenario for 12 herbicides.

The upper estimate used for this analysis includes worst-case assumptions such as highest residue rates, highest food intake, etc.

Worksheet G01 (modified): Summary of Exposure Scenarios for Terrestrial Animals

Chlorsulfuron / Typical Application Rate. Only the Upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	1.36E+00	1.36E+00	1.36E+00	F02a
bee, 100% absorption	8.98E+00	8.98E+00	8.98E+00	F02b
Contaminated vegetation				
small mammal	7.00E-02	7.00E-02	1.50E-01	F03
large mammal	9.63E-01	9.63E-01	2.72E+00	F10
large bird	1.51E+00	1.51E+00	4.26E+00	F12
Contaminated water				
small mammal, spill	1.11E-02	2.22E-03	1.11E-01	F05
Contaminated insects				
small mammal	1.30E+00	1.30E+00	3.89E+00	F14a
small bird	2.11E+00	2.11E+00	6.32E+00	F14b
Contaminated prey				
predatory mammal (small mammal)	1.17E-01	1.17E-01	1.17E-01	F16a
predatory bird (small mammal)	1.81E-01	1.81E-01	1.81E-01	F16b
predatory bird (fish)	1.97E-02	1.97E-03	2.95E-01	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	2.95E-03	1.47E-03	1.26E-02	F04a
large mammal, on site	1.22E-01	4.05E-02	1.14E+00	F11a
large bird, on site	1.90E-01	6.34E-02	1.79E+00	F13a
Contaminated water				
small mammal	4.92E-06	8.20E-07	7.38E-06	F07
Contaminated fish				
predatory bird	4.03E-05	3.36E-06	9.07E-05	F09

Chlorsulfuron / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	6.06E+00	6.06E+00	6.06E+00	F02a
bee, 100% absorption	4.01E+01	4.01E+01	4.01E+01	F02b
Contaminated vegetation				
small mammal	3.13E-01	3.13E-01	6.70E-01	F03
large mammal	4.30E+00	4.30E+00	1.21E+01	F10
large bird	6.73E+00	6.73E+00	1.90E+01	F12
Contaminated water				
small mammal, spill	4.95E-02	9.89E-03	4.95E-01	F05
Contaminated insects				
small mammal	5.78E+00	5.78E+00	1.73E+01	F14a
small bird	9.40E+00	9.40E+00	2.82E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	5.25E-01	5.25E-01	5.25E-01	F16a
predatory bird (small mammal)	8.08E-01	8.08E-01	8.08E-01	F16b
predatory bird (fish)	8.79E-02	8.79E-03	1.32E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.32E-02	6.58E-03	5.64E-02	F04a
large mammal, on site	5.43E-01	1.81E-01	5.11E+00	F11a
large bird, on site	8.50E-01	2.83E-01	8.00E+00	F13a
Contaminated water				
small mammal	2.20E-05	3.66E-06	3.29E-05	F07
Contaminated fish				
predatory bird	1.80E-04	1.50E-05	4.05E-04	F09

Clopyralid / Typical Application Rate. Only upper exposure estimates are used in this document

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	8.49E+00	8.49E+00	8.49E+00	F02a
bee, 100% absorption	5.61E+01	5.61E+01	5.61E+01	F02b
Contaminated vegetation				
small mammal	4.38E-01	4.38E-01	9.38E-01	F03
large mammal	6.02E+00	6.02E+00	1.70E+01	F10
large bird	9.42E+00	9.42E+00	2.66E+01	F12

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Contaminated water				
small mammal, spill	4.65E-01	1.11E-01	2.33E+00	F05
Contaminated insects				
small mammal	8.10E+00	8.10E+00	2.43E+01	F14a
small bird	1.32E+01	1.32E+01	3.95E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	7.34E-01	7.34E-01	7.34E-01	F16a
predatory bird (small mammal)	1.13E+00	1.13E+00	1.13E+00	F16b
predatory bird (fish)	3.18E-01	3.79E-02	2.38E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.77E-02	7.04E-03	9.87E-02	F04a
large mammal, on site	7.29E-01	1.94E-01	8.95E+00	F11a
large bird, on site	1.14E+00	3.03E-01	1.40E+01	F13a
Contaminated water				
small mammal	3.59E-04	5.12E-05	6.66E-04	F07
Contaminated fish				
predatory bird	2.45E-04	1.75E-05	6.83E-04	F09

Clopyralid / Highest Application Rate. Only upper exposure estimates are used in this document

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	1.21E+01	1.21E+01	1.21E+01	F02a
bee, 100% absorption	8.01E+01	8.01E+01	8.01E+01	F02b
Contaminated vegetation				
small mammal	6.25E-01	6.25E-01	1.34E+00	F03
large mammal	8.60E+00	8.60E+00	2.43E+01	F10
large bird	1.35E+01	1.35E+01	3.80E+01	F12
Contaminated water				
small mammal, spill	6.65E-01	1.58E-01	3.32E+00	F05
Contaminated insects				
small mammal	1.16E+01	1.16E+01	3.47E+01	F14a
small bird	1.88E+01	1.88E+01	5.64E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	1.05E+00	1.05E+00	1.05E+00	F16a
predatory bird (small mammal)	1.62E+00	1.62E+00	1.62E+00	F16b

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
predatory bird (fish)	4.54E-01	5.41E-02	3.41E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	2.52E-02	1.01E-02	1.41E-01	F04a
large mammal, on site	1.04E+00	2.77E-01	1.28E+01	F11a
large bird, on site	1.63E+00	4.33E-01	2.00E+01	F13a
Contaminated water				
small mammal	5.12E-04	7.32E-05	9.52E-04	F07
Contaminated fish				
predatory bird	3.50E-04	2.50E-05	9.75E-04	F09

Glyphosate / Typical Application Rate: Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	4.85E+01	4.85E+01	4.85E+01	F02a
bee, 100% absorption	3.21E+02	3.21E+02	3.21E+02	F02b
Contaminated vegetation				
small mammal	8.57E-01	8.57E-01	2.11E+00	F03
large mammal	3.44E+01	3.44E+01	9.71E+01	F10
large bird	5.38E+01	5.38E+01	1.52E+02	F12
Contaminated water				
small mammal, spill	2.66E+00	1.06E+00	5.32E+00	F05
Contaminated insects				
small mammal	4.63E+01	4.63E+01	1.39E+02	F14a
small bird	8.E+01	7.52E+01	2.26E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	4.20E+00	4.20E+00	4.20E+00	F16a
predatory bird (small mammal)	6.46E+00	6.46E+00	6.46E+00	F16b
predatory bird (fish)	9.45E-01	1.89E-01	2.83E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	4.69E-02	2.35E-02	2.31E-01	F04a
large mammal, on site	5.65E+00	1.88E+00	5.32E+01	F11a
large bird, on site	8.84E+00	2.95E+00	8.32E+01	F13a
Contaminated water				
small mammal	2.93E-04	2.93E-05	2.34E-03	F07

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Contaminated fish				
predatory bird	1.04E-04	5.20E-06	1.25E-03	F09

Glyphosate / Highest Application Rate: Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	1.70E+02	1.70E+02	1.70E+02	F02a
bee, 100% absorption	1.12E+03	1.12E+03	1.12E+03	F02b
Contaminated vegetation				
small mammal	3.00E+00	3.00E+00	7.38E+00	F03
large mammal	1.20E+02	1.20E+02	3.40E+02	F10
large bird	1.88E+02	1.88E+02	5.32E+02	F12
Contaminated water				
small mammal, spill	9.31E+00	3.72E+00	1.86E+01	F05
Contaminated insects				
small mammal	1.62E+02	1.62E+02	4.86E+02	F14a
small bird	3.E+02	2.63E+02	7.90E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	1.47E+01	1.47E+01	1.47E+01	F16a
predatory bird (small mammal)	2.26E+01	2.26E+01	2.26E+01	F16b
predatory bird (fish)	3.31E+00	6.61E-01	9.92E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.64E-01	8.21E-02	8.07E-01	F04a
large mammal, on site	1.98E+01	6.59E+00	1.86E+02	F11a
large bird, on site	3.09E+01	1.03E+01	2.91E+02	F13a
Contaminated water				
small mammal	1.02E-03	1.02E-04	8.20E-03	F07
Contaminated fish				
predatory bird	3.64E-04	1.82E-05	4.37E-03	F09

Imazapic / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+00	2.42E+00	2.42E+00	F02a
bee, 100% absorption	1.60E+01	1.60E+01	1.60E+01	F02b
Contaminated vegetation				
small mammal	1.25E-01	1.25E-01	2.68E-01	F03
large mammal	1.72E+00	1.72E+00	4.86E+00	F10
large bird	2.69E+00	2.69E+00	7.60E+00	F12
Contaminated water				
small mammal, spill	2.42E+00	2.42E+00	2.42E+00	F05
Contaminated insects				
small mammal	2.31E+00	2.31E+00	6.94E+00	F14a
small bird	3.76E+00	3.76E+00	1.13E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E-01	2.10E-01	2.10E-01	F16a
predatory bird (small mammal)	3.23E-01	3.23E-01	3.23E-01	F16b
predatory bird (fish)	1.67E-02	5.00E-03	7.49E-02	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	8.02E-04	1.20E-04	1.02E-02	F04a
large mammal, on site	3.31E-02	3.31E-03	9.29E-01	F11a
large bird, on site	5.18E-02	5.18E-03	1.45E+00	F13a
Contaminated water				
small mammal	2.93E-07	1.46E-07	4.39E-07	F07
Contaminated fish				
predatory bird	2.20E-08	5.50E-09	4.95E-08	F09

Imazapic / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	4.36E+00	4.36E+00	4.36E+00	F02a
bee, 100% absorption	2.89E+01	2.89E+01	2.89E+01	F02b
Contaminated vegetation				
small mammal	2.25E-01	2.25E-01	4.82E-01	F03
large mammal	3.10E+00	3.10E+00	8.74E+00	F10
large bird	4.85E+00	4.85E+00	1.37E+01	F12

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Contaminated water				
small mammal, spill	4.21E-01	2.53E-01	1.26E+00	F05
Contaminated insects				
small mammal	4.16E+00	4.16E+00	1.25E+01	F14a
small bird	6.77E+00	6.77E+00	2.03E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	3.78E-01	3.78E-01	3.78E-01	F16a
predatory bird (small mammal)	5.82E-01	5.82E-01	5.82E-01	F16b
predatory bird (fish)	3.16E-02	9.49E-03	1.42E-01	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.44E-03	2.16E-04	1.84E-02	F04a
large mammal, on site	5.95E-02	5.95E-03	1.67E+00	F11a
large bird, on site	9.32E-02	9.32E-03	2.62E+00	F13a
Contaminated water				
small mammal	5.27E-07	2.64E-07	7.91E-07	F07
Contaminated fish				
predatory bird	3.96E-08	9.90E-09	8.91E-08	F09

Imazapyr / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	1.09E+01	1.09E+01	1.09E+01	F02a
bee, 100% absorption	7.21E+01	7.21E+01	7.21E+01	F02b
Contaminated vegetation				
small mammal	5.63E-01	5.63E-01	1.21E+00	F03
large mammal	7.74E+00	7.74E+00	2.19E+01	F10
large bird	1.21E+01	1.21E+01	3.42E+01	F12
Contaminated water				
small mammal, spill	5.98E-01	2.99E-01	1.22E+00	F05
Contaminated insects				
small mammal	1.04E+01	1.04E+01	3.12E+01	F14a
small bird	1.69E+01	1.69E+01	5.08E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	9.44E-01	9.44E-01	9.44E-01	F16a
predatory bird (small mammal)	1.45E+00	1.45E+00	1.45E+00	F16b
predatory bird (fish)	2.04E-01	5.11E-02	6.25E-01	F08

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	2.13E-02	6.66E-03	1.17E-01	F04a
large mammal, on site	8.80E-01	1.83E-01	1.06E+01	F11a
large bird, on site	1.38E+00	2.87E-01	1.65E+01	F13a
Contaminated water				
small mammal	6.59E-06	6.59E-07	6.59E-05	F07
Contaminated fish				
predatory bird	2.25E-06		1.13E-07	

Imazapyr / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Upper	Lower	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	3.03E+01	3.03E+01	3.03E+01	F02a
bee, 100% absorption	2.E-01	2.E-01	2.E-01	F02b
Contaminated vegetation				
small mammal	1.56E+00	1.56E+00	3.35E+00	F03
large mammal	2.15E+01	2.15E+01	6.07E+01	F10
large bird	3.37E+01	3.37E+01	9.50E+01	F12
Contaminated water				
small mammal, spill	1.66E+00	8.31E-01	3.39E+00	F05
Contaminated insects				
small mammal	2.89E+01	2.89E+01	8.67E+01	F14a
small bird	4.70E+01	4.70E+01	1.41E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	2.62E+00	2.62E+00	2.62E+00	F16a
predatory bird (small mammal)	4.04E+00	4.04E+00	4.04E+00	F16b
predatory bird (fish)	5.68E-01	1.42E-01	1.73E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	5.92E-02	1.85E-02	3.24E-01	F04a
large mammal, on site	2.44E+00	5.09E-01	2.93E+01	F11a
large bird, on site	3.83E+00	7.97E-01	4.59E+01	F13a
Contaminated water				
small mammal	1.83E-05	1.83E-06	1.83E-04	F07
Contaminated fish				
predatory bird	6.25E-06	3.13E-07	9.38E-05	F09

Metsulfuron methyl / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	7.27E-01	7.27E-01	7.27E-01	F02a
bee, 100% absorption	4.81E+00	4.81E+00	4.81E+00	F02b
Contaminated vegetation				
small mammal	3.75E-02	3.75E-02	8.04E-02	F03
large mammal	5.16E-01	5.16E-01	1.46E+00	F10
large bird	8.08E-01	8.08E-01	2.28E+00	F12
Contaminated water				
small mammal, spill	1.11E-02	1.11E-03	4.43E-02	F05
Contaminated insects				
small mammal	6.94E-01	6.94E-01	2.08E+00	F14a
small bird	1.13E+00	1.13E+00	3.38E+00	F14b
Contaminated prey				
predatory mammal (small mammal)	6.29E-02	6.29E-02	6.29E-02	F16a
predatory bird (small mammal)	9.70E-02	9.70E-02	9.70E-02	F16b
predatory bird (fish)	1.59E-03	7.95E-05	9.54E-03	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.58E-03	7.89E-04	6.76E-03	F04a
large mammal, on site	6.51E-02	2.17E-02	6.13E-01	F11a
large bird, on site	1.02E-01	3.40E-02	9.60E-01	F13a
Contaminated water				
small mammal	8.78E-07	4.39E-07	1.76E-06	F07
Contaminated fish				
predatory bird	1.27E-06	3.17E-07	3.80E-06	F09

Metsulfuron methyl / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	3.64E+00	3.64E+00	3.64E+00	F02a
bee, 100% absorption	2.40E+01	2.40E+01	2.40E+01	F02b
Contaminated vegetation				
small mammal	1.88E-01	1.88E-01	4.02E-01	F03
large mammal	2.58E+00	2.58E+00	7.28E+00	F10

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
large bird	4.04E+00	4.04E+00	1.14E+01	F12
Contaminated water				
small mammal, spill	5.54E-02	5.54E-03	2.22E-01	F05
Contaminated insects				
small mammal	3.47E+00	3.47E+00	1.04E+01	F14a
small bird	5.64E+00	5.64E+00	1.69E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	3.15E-01	3.15E-01	3.15E-01	F16a
predatory bird (small mammal)	4.85E-01	4.85E-01	4.85E-01	F16b
predatory bird (fish)	7.95E-03	3.97E-04	4.77E-02	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	7.89E-03	3.95E-03	3.38E-02	F04a
large mammal, on site	3.26E-01	1.09E-01	3.07E+00	F11a
large bird, on site	5.10E-01	1.70E-01	4.80E+00	F13a
Contaminated water				
small mammal	4.39E-06	2.20E-06	8.78E-06	F07
Contaminated fish				
predatory bird	6.33E-06	1.58E-06	1.90E-05	F09

Picloram / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.E-01	2.E-01	2.E-01	F02a
bee, 100% absorption	6.E-02	6.E-02	6.E-02	F02b
Contaminated vegetation				
small mammal	1.E-02	1.E-02	3.E-02	F03
large mammal	2.E-01	2.E-01	5.E-01	F10
large bird	6.E-03	6.E-03	2.E-02	F12
Contaminated water				
small mammal, spill	5.E-03	1.E-03	3.E-02	F05
Contaminated insects				
small mammal	2.38E-01	2.38E-01	7.14E-01	F14a
small bird	9.E-03	9.E-03	3.E-02	F14b
Contaminated prey				
predatory mammal (small mammal)	2.16E-02	2.16E-02	2.16E-02	F16a
predatory bird (small mammal)	7.54E-04	7.54E-04	7.54E-04	F16b
predatory bird (fish)	7.E-05	1.E-05	6.E-04	F08

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	8.E-04	4.E-04	3.E-03	F04a
large mammal, on site	3.E-02	1.E-02	3.E-01	F11a
large bird, on site	5.E-02	2.E-02	5.E-01	F13a
Contaminated water				
small mammal	7.E-06	7.E-07	3.E-05	F07
Contaminated fish				
predatory bird	5.E-06	3.E-07	3.E-05	F09

Picloram / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+01	2.42E+01	2.42E+01	F02a
bee, 100% absorption	1.60E+02	1.60E+02	1.60E+02	F02b
Contaminated vegetation				
small mammal	1.25E+00	1.25E+00	2.68E+00	F03
large mammal	1.72E+01	1.72E+01	4.86E+01	F10
large bird	2.69E+01	2.69E+01	7.60E+01	F12
Contaminated water				
small mammal, spill	4.43E-01	1.33E-01	2.53E+00	F05
Contaminated insects				
small mammal	2.31E+01	2.31E+01	6.94E+01	F14a
small bird	3.76E+01	3.76E+01	1.13E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E+00	2.10E+00	2.10E+00	F16a
predatory bird (small mammal)	3.23E+00	3.23E+00	3.23E+00	F16b
predatory bird (fish)	3.03E-01	4.54E-02	2.60E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.60E-02	8.01E-03	6.87E-02	F04a
large mammal, on site	6.61E-01	2.20E-01	6.22E+00	F11a
large bird, on site	1.04E+00	3.45E-01	9.74E+00	F13a
Contaminated water				
small mammal	1.46E-04	1.46E-05	5.86E-04	F07
Contaminated fish				
predatory bird	1.00E-04	5.00E-06	6.00E-04	F09

Sethoxydim / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	7.27E+00	7.27E+00	7.27E+00	F02a
bee, 100% absorption	4.81E+01	4.81E+01	4.81E+01	F02b
Contaminated vegetation				
small mammal	3.75E-01	3.75E-01	8.04E-01	F03
large mammal	5.16E+00	5.16E+00	1.46E+01	F10
large bird	8.08E+00	8.08E+00	2.28E+01	F12
Contaminated water				
small mammal, spill	3.99E-01	6.21E-02	9.97E-01	F05
Contaminated insects				
small mammal	6.94E+00	6.94E+00	2.08E+01	F14a
small bird	1.13E+01	1.13E+01	3.38E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	6.29E-01	6.29E-01	6.29E-01	F16a
predatory bird (small mammal)	9.70E-01	9.70E-01	9.70E-01	F16b
predatory bird (fish)	9.81E-01	7.63E-02	3.68E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.80E-03	9.02E-04	7.73E-03	F04a
large mammal, on site	7.44E-02	2.48E-02	7.01E-01	F11a
large bird, on site	1.17E-01	3.88E-02	1.10E+00	F13a
Contaminated water				
small mammal	3.51E-05	8.78E-07	5.27E-05	F07
Contaminated fish				
predatory bird	5.04E-04	6.30E-06	1.13E-03	F09

Sethoxydim/ Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	9.09E+00	9.09E+00	9.09E+00	F02a
bee, 100% absorption	6.01E+01	6.01E+01	6.01E+01	F02b
Contaminated vegetation				
small mammal	4.69E-01	4.69E-01	1.00E+00	F03
large mammal	6.45E+00	6.45E+00	1.82E+01	F10
large bird	1.01E+01	1.01E+01	2.85E+01	F12

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Contaminated water				
small mammal, spill	3.99E-01	6.21E-02	9.97E-01	F05
Contaminated insects				
small mammal	8.67E+00	8.67E+00	2.60E+01	F14a
small bird	1.41E+01	1.41E+01	4.23E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	7.87E-01	7.87E-01	7.87E-01	F16a
predatory bird (small mammal)	1.21E+00	1.21E+00	1.21E+00	F16b
predatory bird (fish)	9.81E-01	7.63E-02	3.68E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	2.25E-03	1.13E-03	9.66E-03	F04a
large mammal, on site	9.30E-02	3.10E-02	8.76E-01	F11a
large bird, on site	1.46E-01	4.86E-02	1.37E+00	F13a
Contaminated water				
small mammal	4.39E-05	1.10E-06	6.59E-05	F07
Contaminated fish				
predatory bird	6.30E-04	7.88E-06	1.42E-03	F09

Sulfometuron methyl / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	1.09E+00	1.09E+00	1.09E+00	F02a
bee, 100% absorption	7.21E+00	7.21E+00	7.21E+00	F02b
Contaminated vegetation				
small mammal	5.63E-02	5.63E-02	1.21E-01	F03
large mammal	7.74E-01	7.74E-01	2.19E+00	F10
large bird	1.21E+00	1.21E+00	3.42E+00	F12
Contaminated water				
small mammal, spill	4.43E-02	1.44E-02	1.22E-01	F05
Contaminated insects				
small mammal	1.04E+00	1.04E+00	3.12E+00	F14a
small bird	1.69E+00	1.69E+00	5.08E+00	F14b
Contaminated prey				
predatory mammal (small mammal)	9.44E-02	9.44E-02	9.44E-02	F16a
predatory bird (small mammal)	1.45E-01	1.45E-01	1.45E-01	F16b
predatory bird (fish)	1.06E-01	1.72E-02	4.37E-01	F08

Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	9.00E-04	4.50E-04	3.86E-03	F04a
large mammal, on site	3.71E-02	1.24E-02	3.50E-01	F11a
large bird, on site	5.81E-02	1.94E-02	5.47E-01	F13a
Contaminated water				
small mammal	2.64E-07	6.59E-08	4.61E-07	F07
Contaminated fish				
predatory bird	1.08E-06	1.35E-07	2.84E-06	F09

Sulfometuron methyl / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	**Lower**	**Upper**	**Worksheet**
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	9.21E+00	9.21E+00	9.21E+00	F02a
bee, 100% absorption	6.09E+01	6.09E+01	6.09E+01	F02b
Contaminated vegetation				
small mammal	4.75E-01	4.75E-01	1.02E+00	F03
large mammal	6.54E+00	6.54E+00	1.85E+01	F10
large bird	1.02E+01	1.02E+01	2.89E+01	F12
Contaminated water				
small mammal, spill	3.74E-01	1.22E-01	1.03E+00	F05
Contaminated insects				
small mammal	8.79E+00	8.79E+00	2.64E+01	F14a
small bird	1.43E+01	1.43E+01	4.29E+01	F14b
Contaminated prey				
predatory mammal (small mammal)	7.97E-01	7.97E-01	7.97E-01	F16a
predatory bird (small mammal)	1.23E+00	1.23E+00	1.23E+00	F16b
predatory bird (fish)	8.95E-01	1.45E-01	3.69E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	7.60E-03	3.80E-03	3.26E-02	F04a
large mammal, on site	3.14E-01	1.05E-01	2.95E+00	F11a
large bird, on site	4.91E-01	1.64E-01	4.62E+00	F13a
Contaminated water				
small mammal	2.23E-06	5.56E-07	3.89E-06	F07
Contaminated fish				
predatory bird	9.12E-06	1.14E-06	2.39E-05	F09

Triclopyr acid / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+01	2.42E+01	2.42E+01	F02a
bee, 100% absorption	1.60E+02	1.60E+02	1.60E+02	F02b
Contaminated vegetation				
small mammal	3.30E-01	3.30E-01	4.95E-01	F03
large mammal	1.72E+01	1.72E+01	4.86E+01	F10
large bird	2.69E+01	2.69E+01	7.60E+01	F12
Contaminated water				
small mammal, spill	5.32E-01	3.32E-01	2.66E+00	F05
Contaminated insects				
small mammal	2.31E+01	2.31E+01	6.94E+01	F14a
small bird	3.76E+01	3.76E+01	1.13E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E+00	2.10E+00	2.10E+00	F16a
predatory bird (small mammal)	3.23E+00	3.23E+00	3.23E+00	F16b
predatory bird (fish)	3.02E-01	9.42E-02	2.26E+00	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62E-02	6.20E-03	6.52E-02	F04a
large mammal, on site	2.52E+00	6.46E-01	3.20E+01	F11a
large bird, on site	3.95E+00	1.01E+00	5.01E+01	F13a
Contaminated water				
small mammal	4.39E-03	1.17E-03	7.32E-03	F07
Contaminated fish				
predatory bird	2.49E-03	3.32E-04	6.23E-03	F09

Triclopyr acid / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+02	2.42E+02	2.42E+02	F02a
bee, 100% absorption	1.60E+03	1.60E+03	1.60E+03	F02b
Contaminated vegetation				
small mammal	3.30E+00	3.30E+00	4.95E+00	F03
large mammal	1.72E+02	1.72E+02	4.86E+02	F10
large bird	2.69E+02	2.69E+02	7.60E+02	F12

Contaminated water				
small mammal, spill	5.32E+00	3.32E+00	2.66E+01	F05
Contaminated insects				
small mammal	2.31E+02	2.31E+02	6.94E+02	F14a
small bird	3.76E+02	3.76E+02	1.13E+03	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E+01	2.10E+01	2.10E+01	F16a
predatory bird (small mammal)	3.23E+01	3.23E+01	3.23E+01	F16b
predatory bird (fish)	3.02E+00	9.42E-01	2.26E+01	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62E-01	6.20E-02	6.52E-01	F04a
large mammal, on site	2.52E+01	6.46E+00	3.20E+02	F11a
large bird, on site	3.95E+01	1.01E+01	5.01E+02	F13a
Contaminated water				
small mammal	4.39E-02	1.17E-02	7.32E-02	F07
Contaminated fish				
predatory bird	2.49E-02	3.32E-03	6.23E-02	F09

Triclopyr BEE / Typical Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	Lower	Upper	Worksheet
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+01	2.42E+01	2.42E+01	F02a
bee, 100% absorption	1.60E+02	1.60E+02	1.60E+02	F02b
Contaminated vegetation				
small mammal	3.30E-01	3.30E-01	4.95E-01	F03
large mammal	1.72E+01	1.72E+01	4.86E+01	F10
large bird	2.69E+01	2.69E+01	7.60E+01	F12
Contaminated water				
small mammal, spill	5.32E-01	3.32E-01	2.66E+00	F05
Contaminated insects				
small mammal	2.31E+01	2.31E+01	6.94E+01	F14a
small bird	3.76E+01	3.76E+01	1.13E+02	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E+00	2.10E+00	2.10E+00	F16a
predatory bird (small mammal)	3.23E+00	3.23E+00	3.23E+00	F16b
predatory bird (fish)	3.02E-01	9.42E-02	2.26E+00	F08

Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62E-02	6.20E-03	6.52E-02	F04a
large mammal, on site	2.52E+00	6.46E-01	3.20E+01	F11a
large bird, on site	3.95E+00	1.01E+00	5.01E+01	F13a
Contaminated water				
small mammal	4.39E-03	1.17E-03	7.32E-03	F07
Contaminated fish				
predatory bird	2.49E-03	3.32E-04	6.23E-03	F09

Triclopyr BEE / Highest Application Rate. Only upper exposure estimates are used in this document.

Scenario	Dose (mg/kg/day)			
	Typical	**Lower**	**Upper**	**Worksheet**
Acute/Accidental Exposures				
Direct spray				
small animal, 100% absorption	2.42E+02	2.42E+02	2.42E+02	F02a
bee, 100% absorption	1.60E+03	1.60E+03	1.60E+03	F02b
Contaminated vegetation				
small mammal	3.30E+00	3.30E+00	4.95E+00	F03
large mammal	1.72E+02	1.72E+02	4.86E+02	F10
large bird	2.69E+02	2.69E+02	7.60E+02	F12
Contaminated water				
small mammal, spill	5.32E+00	3.32E+00	2.66E+01	F05
Contaminated insects				
small mammal	2.31E+02	2.31E+02	6.94E+02	F14a
small bird	3.76E+02	3.76E+02	1.13E+03	F14b
Contaminated prey				
predatory mammal (small mammal)	2.10E+01	2.10E+01	2.10E+01	F16a
predatory bird (small mammal)	3.23E+01	3.23E+01	3.23E+01	F16b
predatory bird (fish)	3.02E+00	9.42E-01	2.26E+01	F08
Longer-term Exposures				
Contaminated vegetation				
small mammal, on site	1.62E-01	6.20E-02	6.52E-01	F04a
large mammal, on site	2.52E+01	6.46E+00	3.20E+02	F11a
large bird, on site	3.95E+01	1.01E+01	5.01E+02	F13a
Contaminated water				
small mammal	4.39E-02	1.17E-02	7.32E-02	F07
Contaminated fish				
predatory bird	2.49E-02	3.32E-03	6.23E-02	F09