



SERA TR-052-25-03c

Triclopyr
Human Health and Ecological Risk Assessment
Corrected Final Report

Submitted to:
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May 24, 2011
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Error Notes

October 20, 2011

In the original release of the final report (SERA TR-052-25-03a dated May 24, 2011), Tables 2 and 22 incorrectly listed the water solubility of TCP as 100 mg/L. As indicated in Table 1, the correct value, from Knuteson (1999), is 49,000 mg/L. This error was noted by Dr. K. King (U.S. Fish and Wildlife Service). The error has been corrected. While the Gleams-Driver runs were made using the 100 mg/L water solubility, re-runs using the water solubility of 49,000 mg/L yielded results that are indistinguishable from the original runs. Thus, the appendices have not been change. Water solubility is not a sensitive parameter in GLEAMS unless the soil water is saturated. This did not occur in the Gleams-Driver modeling.

July 9, 2016

During an audit of WorksheetMaker (Version 6.00.15), it was noted that the chronic toxicity values of TCP to aquatic invertebrates had been entered incorrectly into the WorksheetMaker database and the aquatic toxicity values of TCP for algae had been omitted. The workbooks (i.e., Attachments 5, 6, and 7) have been corrected. The toxicity values had been correctly designated in the risk assessment.

In addition, the original discussion of concentrations of TCP in water following aquatic applications (both submergent and emergent) was unclear in the original risk assessment and the attachments submitted with the original risk assessment were not fully developed. This has been corrected in the current document. Each of the sections relating to exposure assessments for aquatic applications (i.e., Section 3.2.3.4.6.2.1 for submergent vegetation and Section 3.2.3.4.6.2.2 for emergent vegetation) has been divided into subsections covering triclopyr and TCP. The subsections on triclopyr are identical to the sections in the original risk assessment. The subsection of TCP discusses the methods used to estimate concentrations of TCP in water. These methods have been implemented in new versions of Attachment 6 (emergent vegetation) and Attachment 7 (submergent application) which are provided with this revised risk assessment.

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- Appendix 13: TCP Following Application of Triclopyr TEA

Note: Appendices are included in a separate file.

LIST OF ATTACHMENTS

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- Attachment 2: Terrestrial Applications of Triclopyr BEE
- Attachment 3: Emergent Aquatic Applications of Triclopyr TEA
- Attachment 4: Submergent Aquatic Applications of Triclopyr TEA
- Attachment 5: TCP in Terrestrial Applications of Triclopyr (TEA and BEE)
- Attachment 6: TCP in Emergent Aquatic Applications of Triclopyr TEA
- Attachment 7: TCP in Submergent Aquatic Applications of Triclopyr TEA

Note: All attachments are EXCEL workbooks that are included as separate files.

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADD	attention-deficit disorder
ADHD	attention-deficit hyperactivity disorder
AEL	adverse-effect level
a.e.	acid equivalent
a.i.	active ingredient
APHIS	Animal and Plant Health Inspection Service
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BEE	butoxyethyl ester
BUN	blood urea nitrogen
bw	body weight
CBI	confidential business information
cc	cubic centimeter
cm	centimeter
CNS	central nervous system
DAA	days after application
DAT	days after treatment
DER	data evaluation record
d.f.	degrees of freedom
EC _x	concentration causing X% inhibition of a process
EC ₂₅	concentration causing 25% inhibition of a process
EC ₅₀	concentration causing 50% inhibition of a process
EDTA	ethylenediaminetetra acetic acid
EFED	Environmental Fate and Effects Division (U.S. EPA/OPP)
ExToxNet	Extension Toxicology Network
F	female
FH	Forest Health
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FQPA	Food Quality Protection Act
g	gram
GLP	Good Laboratory Practices
ha	hectare
HED	Health Effects Division (U.S. EPA/OPP)
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IC ₅₀	concentration causing 50% inhibition
IRIS	Integrated Risk Information System
K	potassium (salt)
k _a	absorption coefficient
k _e	elimination coefficient
kg	kilogram
K _{o/c}	organic carbon partition coefficient
K _{o/w}	octanol-water partition coefficient
K _p	skin permeability coefficient

L	liter
lb	pound
LC ₅₀	lethal concentration, 50% kill
LD ₅₀	lethal dose, 50% kill
LOAEL	lowest-observed-adverse-effect level
LOC	level of concern
m	meter
M	male
MATC	maximum acceptable tolerance concentration
MCS	multiple chemical sensitivity
mg	milligram
mg/kg/day	milligrams of agent per kilogram of body weight per day
mL	milliliter
mM	millimole
mPa	millipascal, (0.001 Pa)
MOS	margin of safety
MRID	Master Record Identification Number
MSDS	material safety data sheet
MSMA	monosodium methanearsonate
MW	molecular weight
N/A	not available
NAWQA	USGS National Water Quality Assessment
NCI	National Cancer Institute
NCOD	National Drinking Water Contaminant Occurrence Database
NIOSH	National Institute for Occupational Safety and Health
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NOS	not otherwise specified
NRC	National Research Council
NTP	National Toxicology Program
OM	organic matter
OPP	Office of Pesticide Programs
OPPTS	Office of Pesticide Planning and Toxic Substances
OSHA	Occupational Safety and Health Administration
Pa	Pascal
PBPK	physiologically-based kinetic
ppm	parts per million
PSP	phenolsulfonphthalein
RBC	red blood cells
RED	re-registration eligibility decision
RfD	reference dose
RTU	ready to use
S.A.	South American
SERA	Syracuse Environmental Research Associates
TCDD	2,3,7,8-tetrachlorodibenzo-p-dioxin
TCP	3,5,6-trichloro-2-pyridinol
TEA	triethylamine

TEP	typical end-use product
T.G.I.A.	Technical grade active ingredient
TIPA	Triisopropanolamine
TRED	Tolerance Reassessment Eligibility Decision
UF	uncertainty factor
U.S.	United States
USDA	U.S. Department of Agriculture
U.S. EPA	U.S. Environmental Protection Agency
USGS	U.S. Geological Survey
WCR	water contamination rate
WHO	World Health Organization

COMMON UNIT CONVERSIONS AND ABBREVIATIONS

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

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

CONVERSION OF SCIENTIFIC NOTATION

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

EXECUTIVE SUMMARY

The triethylamine salt (TEA) and the butoxyethyl ester (BEE) of triclopyr are used in Forest Service programs primarily for conifer or hardwood release, noxious weed control, site preparation, and rights-of-way management. Aquatic weed control is a minor use (TEA salt).

Potential risks associated with terrestrial applications are greatest for workers as well as women consuming vegetation contaminated with triclopyr. The central estimates of the HQs indicate that workers will not be subject to hazardous levels of triclopyr during applications of triclopyr TEA at the unit application rate of 1 lb a.e./acre. For triclopyr BEE, the central estimates of the HQs range from 0.7 to 1.2 based on the chronic RfD. At the upper bounds of the estimated exposures for all application methods, the HQs for both triclopyr TEA (HQs = 1.6 to 3) and triclopyr BEE formulations (HQs = 6 to 12) exceed the level of concern (HQ=1), based on the chronic RfD. For a young woman consuming contaminated vegetation, the upper bound HQ is 27 for acute exposures and 6 for longer-term exposures. In addition, some of the central estimates of exposure to triclopyr or TCP involving a young woman consuming contaminated vegetation or fruit also exceed the level of concern. All of these HQs apply to an application rate of 1 lb a.e./acre and will scale proportionately to the application rate. Because triclopyr has been shown to cause adverse developmental effects in mammals, the high HQs associated with terrestrial applications are of particular concern in terms of the potential for adverse reproductive outcomes in humans. Adverse developmental effects in experimental mammals have been observed, however, only at doses that cause frank signs of maternal toxicity. The available toxicity studies suggest that overt and severe toxicity would not be associated with any of the upper bound HQs and this diminishes concern for reproductive effects in humans.

Qualitatively, the risk characterization for ecological effects is parallel in many respects to the risk characterization for human health effects. At an application rate of 1 lb a.e./acre, HQs exceed the level of concern for exposures involving the consumption of contaminated vegetation by mammals and birds. HQs are greatest for large mammals. As with the human health risk assessment, the high HQs suggest the potential for adverse effects, but not overt toxic effects, in large mammals. Based on a very cursory probabilistic assessment, exposures of mammalian wildlife that would be associated with upper bound HQs are probably rare occurrences.

With the exception of aquatic plants, substantial risks to nontarget species (including humans) associated with the contamination of surface water are low, relative to risks associated with contaminated vegetation. Applications of triclopyr BEE in excess of about 1.5 to 3 lbs a.e./acre could be associated with acute effects in sensitive species of fish or invertebrates, in cases of substantial drift or off-site transport of triclopyr via runoff.

1. INTRODUCTION

1.1. Chemical Specific Information

This document provides human health and ecological risk assessments of the environmental consequences of using triclopyr in Forest Service vegetation management programs. These risk assessments update previous USDA Forest Service risk assessments on triclopyr (SERA 1996, 2003).

In the preparation of this risk assessment, an updated literature search of triclopyr was conducted using TOXLINE. In addition, a FOIA has been submitted to the U.S. EPA/OPP for a current list of all registrant submitted studies. Additional sources of information were used including the U.S. EPA Reregistration Eligibility Decision document on triclopyr and related risk assessments (U.S. EPA/OPP 1998a,b,c) as well as a more recent EPA ecological risk assessment on triclopyr (U.S. EPA/OPP 2009a). Other sources of relevant literature were identified through reviews and risk assessments in the open literature (Antunes-Kenyon and Kennedy 2004; Cal EPA 1986; Cessna et al. 2002; Cox 2000; Dost 2003; Dow AgroSciences 2009; ENSR 2007; Ganapathy 1997; Kegley et al. 2008; Neary et al. 1993; NPIC 2002; Petty et al. 2003; Sassaman et al. 1984; Smith and Oehme 1991; Tu et al. 2001; U.S. DOE-BPA 2000; Washington State Dept. Ecology 2004; Wolt et al. 1997). Generally, these reviews are used only to identify published studies to ensure adequate coverage of the literature. In some cases, information taken from reviews is used directly in this risk assessment and this is specifically noted in the text as appropriate.

In the previous Forest Service risk assessment (SERA 2003), 1117 registrant submissions on triclopyr and triclopyr formulations were identified. Of these, 142 submissions—i.e., full copies of the studies submitted to the U.S. EPA—were kindly provided by the U.S. EPA Office of Pesticide Programs. These submissions included all key studies cited in the RED (U.S. EPA/OPP 1998a) as well as some additional studies submitted after the completion of the RED. The U.S. EPA/OPP no longer provides full copies of registrant studies for risk assessments conducted in support of activities outside of U.S. EPA/OPP. Consequently, summaries of the 142 submissions from SERA (2003) are included in the current Forest Service risk assessment and are cited in the bibliography (Section 5) as MRID03.

During the development of this risk assessment, some of the summaries of the MRID studies given in SERA (2003) were found to be incomplete and additional registrant submitted studies of interest were identified. Two sets of requests for registrant submitted studies were made to Dow AgroSciences, one of the registrants for triclopyr. Dow AgroSciences kindly provided 77 submissions, most of which were full studies. These additional submissions are identified in the bibliography as MRID 2003r, MRID10, and MRID11. These studies are cited in the text in standard author and date format. In some cases, information on other registrant-submitted studies is taken from various U.S. EPA/OPP risk assessments. In these cases, the information is designated in the text of the current risk assessment only by MRID number.

The U.S. EPA/OPP is in the process of reviewing the registration of many pesticides (http://www.epa.gov/oppsrrd1/registration_review). The review of triclopyr, however, is not

1 scheduled to begin until 2014, and the U.S. EPA has not yet opened a docket for the registration
2 review (U.S. EPA/OPP 2010, p. 14).

3 **1.2. General Information**

4 This document has four chapters, including the introduction, program description, risk
5 assessment for human health effects, and risk assessment for ecological effects or effects on
6 wildlife species. Each of the two risk assessment chapters has four major sections, including an
7 identification of the hazards, an assessment of potential exposure to this compound, an
8 assessment of the dose-response relationships, and a characterization of the risks associated with
9 plausible levels of exposure.

10
11 This is a technical support document which addresses some specialized technical areas.
12 Nevertheless an effort was made to ensure that the document can be understood by individuals
13 who do not have specialized training in the chemical and biological sciences. Certain technical
14 concepts, methods, and terms common to all parts of the risk assessment are described in plain
15 language in a separate document (SERA 2007a). The human health and ecological risk
16 assessments presented in this document are not, and are not intended to be, comprehensive
17 summaries of all of the available information. The information presented in the appendices and
18 the discussions in chapters 2, 3, and 4 of the risk assessment are intended to be detailed enough
19 to support a review of the risk analyses.

20
21 As discussed in Section 1.1, the current Forest Service risk assessment is an update to previous
22 risk assessments on triclopyr (SERA 1996, 2003). At some point in the future, the Forest
23 Service will update this risk assessment again and welcomes input from the general public and
24 other interested parties on the selection of studies included in the risk assessment. This input is
25 helpful, however, only if recommendations for including additional studies specify why and/or
26 how the new or not previously included information would be likely to alter the conclusions
27 reached in the risk assessments.

28
29 As with all Forest Service risk assessments, almost no risk estimates presented in this document
30 are given as single numbers. Usually, risk is expressed as a central estimate and a range, which
31 is sometimes quite large. Because of the need to encompass many different types of exposure as
32 well as the need to express the uncertainties in the assessment, this risk assessment involves
33 numerous calculations, most of which are relatively simple and are included in the body of the
34 document.

35
36 Some of the calculations, however, are cumbersome. For those calculations, EXCEL workbooks
37 (sets of EXCEL worksheets) are included as attachments to this risk assessment. The worksheets
38 provide the detail for the estimates cited in the body of the document. Documentation for the use
39 of these workbooks is available in SERA (2010a).

40
41 The EXCEL workbooks are an integral part of the risk assessment. The worksheets contained in
42 these workbooks are designed to isolate the large number of calculations from the risk
43 assessment narrative. In general, all calculations of exposure scenarios and quantitative risk
44 characterizations (i.e., HQs) are derived and contained in the worksheets. The rationale for the
45 calculations and the interpretation of the HQs are contained in this risk assessment document.

46

- 1 Seven EXCEL workbooks accompany this risk assessment covering both triclopyr and 3,5,6-
2 trichloro-2-pyridinol (TCP), a major metabolite of triclopyr:
3
4 Attachment 1: Terrestrial Applications of Triclopyr TEA
5 Attachment 2: Terrestrial Applications of Triclopyr BEE
6 Attachment 3: Emergent Aquatic Applications of Triclopyr TEA
7 Attachment 4: Submergent Aquatic Applications of Triclopyr TEA
8 Attachment 5: TCP in Terrestrial Applications of Triclopyr (TEA and BEE)
9 Attachment 6: TCP in Emergent Aquatic Applications of Triclopyr TEA
10 Attachment 7: TCP in Submergent Aquatic Applications of Triclopyr TEA

2. Program Description

2.1 Overview

Triclopyr is used in Forest Service programs primarily for conifer and/or hardwood release, noxious weed control, site preparation, and rights-of-way management. Two forms of triclopyr are used commercially as herbicides: the triethylamine salt (TEA) and the butoxyethyl ester (BEE). As listed in Table 3, the BEE formulations include ready-to-use 13.6% formulations as well as 60.5, 61.6, and 83.9% liquid formulations. The TEA formulations include several 44.4% liquid formulations and one 14% granular formulation. Several TEA formulations are labeled for aquatic applications. Although aquatic applications have limited use in Forest Service programs, they are addressed in the current Forest Service risk assessment.

The most common application method for triclopyr is backpack (selective) foliar applications. Other application methods include ground broadcast foliar application, several non-broadcast application methods (i.e., basal bark, cut stump, and streamline basal bark), and aerial application. While aerial applications are not commonly used in Forest Service programs or projects, aerial applications are encompassed in this risk assessment.

Formulations of triclopyr BEE may be applied at rates of up to 8 lb a.e./acre, and formulations of triclopyr TEA may be applied at rates of up to 9 lb a.e./acre. While the full range of labeled application rates are considered in this risk assessment, the typical application rate in Forest Service programs is 1 lb a.e./acre and rarely exceeds 6 lb a.e./acre. Some aquatic applications are based on target concentrations in water of up to 2.5 mg a.e./L.

Based on Forest Service use statistics for 2004 (the most recent year for which Forest Service pesticide use statistics are available), about 12,500 lbs of triclopyr are used annually in Forest Service programs, and most of this use occurs in the southeastern region of the United States (Forest Service Region 8). The use of triclopyr in Forest Service programs represents only about 1% of the agricultural use of triclopyr.

2.2. Chemical Description and Commercial Formulations

Triclopyr is the common name for [(3,5,6-trichloro-2-pyridinyl)oxy]acetic acid. Triclopyr is the pyridine analogue of 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) and differs from 2,4,5-T only by the presence of a nitrogen (N) atom in the ring structure (Figure 1). Like 2,4,5-T, triclopyr mimics auxin, a plant growth hormone, thus disrupting the normal growth and viability of plants (Section 4.1.2.5). Triclopyr was initially registered as a herbicide in 1979 (U.S. EPA/OPP 1998a).

Two forms of triclopyr are used commercially as herbicides: the triethylamine salt (TEA) and the butoxyethyl ester (BEE). The structures of both of these forms of triclopyr as well as triclopyr acid are also illustrated in Figure 1. Figure 1 also illustrates the structure of 3,5,6-trichloro-2-pyridinol (TCP). 3,5,6-Trichloro-2-pyridinol is a concern in the current risk assessment because it is a major environmental metabolite of triclopyr. As illustrated in Figure 2, 3,5,6-trichloro-2-pyridinol is formed in all relevant environmental media, as a metabolite in plants, soil, and water. While there is little indication that 3,5,6-trichloro-2-pyridinol poses a substantial risk to humans (Section 3.1.15.1), this metabolite is more toxic than triclopyr is to some aquatic organisms

1 (Section 4.1.3), and the risks to aquatic organisms associated with exposures to 3,5,6-trichloro-2-
2 pyridinol are considered quantitatively in this risk assessment.

3
4 Some basic chemical and physical properties of triclopyr acid, triclopyr TEA, and triclopyr BEE
5 are summarized in Table 1. As discussed in several detailed reviews and assessments of
6 triclopyr (e.g., Antunes-Kenyon and Kennedy 2004; Cessna et al 2002; Ganapathy 1997; HSDB
7 2003; Petty et al. 2003; Tu et al. 2001; Washington State Dept Ecology 2004), triclopyr TEA and
8 triclopyr BEE do not persist in the environment. As noted in recent risk assessment on triclopyr
9 by the U.S. EPA/OPP (2009a),

10
11 *Both triclopyr TEA and BEE active ingredients rapidly degrade back to*
12 *triclopyr acid within an aqueous environment. Triclopyr TEA rapidly*
13 *dissociates in water to the triclopyr acid/anion and triethanolamine [sic].*
14 *Triclopyr BEE rapidly hydrolyzes in the environment to the triclopyr*
15 *acid/anion and butoxyethanol. Both triethanolamine and butoxyethanol*
16 *are also rapidly dissipated by microbial degradation, and thus are not*
17 *being evaluated any further in this assessment.*

18 U.S. EPA/OPP 2009a, p. 20

19
20 Note that above quotation incorrectly refers to triethanolamine [N(CH₂CH₂OH)₃] rather than
21 triethylamine [N(CH₂CH₃)₃]. Nonetheless and as discussed further in Section 3.1.15.1, the
22 current risk assessment concurs with the above determination by U.S. EPA/OPP (2009a) and
23 neither triethylamine nor butoxyethanol are assessed quantitatively. In addition, only acute
24 exposures to triclopyr TEA and triclopyr BEE are considered quantitatively. All chronic risks
25 evaluated in the both the human health risk assessment (Section 3) and the ecological risk
26 assessment (Section 4) are based on the longer-term toxicities of triclopyr acid and/or 3,5,6-
27 trichloro-2-pyridinol, the major metabolite of triclopyr. Because 3,5,6-trichloro-2-pyridinol is
28 discussed often and in some detail in the current risk assessment, the abbreviation TCP, which is
29 used extensively in the literature on triclopyr, is used frequently in the current risk assessment to
30 refer 3,5,6-trichloro-2-pyridinol.

31
32 At ambient temperatures, triclopyr acid is a fluffy solid (Budavari et al. 1989) and is readily
33 soluble in water. In aqueous solutions, the hydrogen atom of the carboxylic acid group (**COOH**)
34 may be associated (e.g., **-COOH**) or dissociated (e.g., **-COO⁻ + H⁺**), depending on the pH of the
35 solution. The dissociation constant, or pK_a, for the carboxylic acid group is approximately 3.
36 Thus, at a pH of 3, 50% of the acid is associated and 50% is disassociated. As the acidity of the
37 solution decreases (i.e., the pH of the solution increases), the proportion of triclopyr that is
38 ionized or dissociated increases. The pH of most biological fluids ranges from approximately 5
39 to 9. Thus, within this range of pH, most of the triclopyr acid has a net negative charge (**-COO⁻**).

40
41 The information on the chemical and physical properties of triclopyr acid, triclopyr TEA,
42 triclopyr BEE, and TCP is reasonably consistent; however, there is some variability in the
43 reported properties for these compounds as well as in their normal and expected environmental
44 fate parameters, particularly the reported half-lives in soil and water. The environmental half-
45 lives as well as the K_{oc} and K_{ow} values used quantitatively in the current risk assessment are
46 summarized in Table 2. The rationale for selecting those specific values is provided in the

1 notations to Table 2 and discussed further in the sections in which the values are used—i.e.,
2 Section 3.1.3.2 (Dermal Absorption) for the K_{ow} 's and Section 3.2.3.4.3 (Gleams-Driver
3 Modeling) for the other parameters.

4
5 As discussed in Section 2.4, application rates for triclopyr are expressed in this risk assessment
6 in units of acid equivalents (a.e.) rather than active ingredients (a.i.). For triclopyr, the term
7 active ingredients refers to the TEA salt or BEE ester. Many of the toxicity studies conducted on
8 triclopyr, which are summarized in the appendices to this risk assessment, report exposures in
9 units of a.i. rather than a.e. For the risk characterization, concentrations or doses in units of a.i.
10 are converted to units of a.e. by multiplying the a.i. value by the ratio of the molecular weight of
11 triclopyr acid (256.5 g/mole) to the molecular weight of the a.i. — i.e., 358.67 g/mole for
12 triclopyr TEA or 356.63 g/mole for triclopyr BEE. The specific conversion factors used in this
13 risk assessment are given in Table 1— i.e., 0.719 for triclopyr BEE and 0.715 for triclopyr TEA.

14
15 The number of triclopyr formulations with labeled uses relevant to Forest Service programs
16 continues to grow. When the initial Forest Service risk assessment on triclopyr was conducted,
17 there were only two available formulations, Garlon 3A and Garlon 4 (SERA 1996). The Forest
18 Service risk assessment conducted in 2003 covers six formulations, Garlon 3A, Garlon 4,
19 Forestry Garlon 4, Pathfinder II, Remedy RTU, and Renovate 3. Currently, 19 formulations of
20 triclopyr that might be used in Forest Service programs have been identified (Table 3).

21
22 Many formulations of triclopyr are equivalent to one another in terms of the active ingredient.
23 For example, eight formulations, including Forestry Garlon, Garlon 4, Remedy, Tahoe 4E,
24 Triclopyr 4 Ester R&P, Triclopyr 4E, Triclopyr R&P, and Triquad, contain triclopyr BEE at a
25 nominal concentration of 61.6%, and five formulations, including Garlon 3A, Renovate 3, Tahoe
26 3A, Triclopyr 3A, and Triclopyr 3SL, contain triclopyr TEA at a concentration of 44.4%. Thus,
27 of the 19 formulations identified in Table 3, about 70% (13/19) of the formulations may consist
28 of only two distinguishable groups of formulations—i.e., 61.6% BEE and 44.4% TEA.

29
30 Formulations with the same amount of active ingredient are not necessarily identical. In some
31 cases, the U.S. EPA registration number for the formulation may be useful in assessing the
32 equivalence of formulations. For example, Pathfinder II and Remedy RTU have the same EPA
33 registration number of 62719-176, as indicated in Table 3. Formulations with identical EPA
34 registration numbers may be regarded as equivalent formulations. Similarly, the EPA
35 registration number for Renovate 3 from SePRO is 62719-37-67690. Note that the first two
36 elements of the registration number (i.e., 62719-37) are identical to the registration number for
37 Garlon 3A from Dow AgroSciences. The two-component registration numbers consist of the
38 company identification number followed by the product code. The third element in the three-
39 part registration number for Renovate 3 is the company code for SePRO. This registration
40 number indicates that Renovate 3 is a repackaging of Garlon 3A. In other words, the two
41 formulations are equivalent to one another.

42
43 In considering formulations with unique registration numbers, information on the MSDS for the
44 formulation may be useful in assessing the similarity of the formulations. Appendix 1 (Table 1)
45 provides a summary of the mammalian toxicity information from the MSDS for the formulations
46 included in Table 3. The basis for and significance of the toxicity values are discussed further in

1 Section 3.1 (Hazard Identification for Human Health Effects). The current discussion is
2 concerned only with the apparent similarities among the different formulations.

3
4 Table 1 of Appendix 1 is organized by the type of a.i. (BEE or TEA) and its concentration in the
5 formulation. Accordingly, there are six groups of distinct formulations identified as 13.6% BEE,
6 60.5% BEE, 61.6% BEE, 83.9% BEE, 14% TEA (granular), and 44.4% TEA. In Appendix 1,
7 Table 1, five of the eight formulations specify identical oral LD₅₀ values—i.e., 1581 mg/kg bw
8 for male rats and 1338 mg/kg bw for female rats—and the other three formulations specify the
9 LD₅₀ as >1000 mg/kg bw. The identical LD₅₀ values for five of the eight formulations do not
10 necessarily indicate that the formulations are identical or that five separate bioassays yielded the
11 same LD₅₀ values. Instead, the identical LD₅₀ values indicate that the U.S. EPA/OPP probably
12 allowed data on one formulation to be used to support the registration of other formulations.
13 This general approach is sometimes referred to as *bridging*. If the two formulations are
14 identical—i.e., the same formulation is marketed under different names—data bridging make
15 sense. If the two formulations are substantially different, however, bridging is not permitted, and
16 formulation-specific data are required. For triclopyr formulations, a specific discussion of
17 formulation bridging is not available. While most data on the 61.6% formulations are reasonably
18 similar, Tahoe 4E appears to be distinct in that the MSDS for this formulation indicates that
19 Tahoe 4E is not a skin sensitizer. All other 61.6% formulations indicate that the formulations
20 may cause skin sensitization.

21
22 Substantial differences in the toxicity of the various formulations of triclopyr are likely to be
23 related to differences in the other ingredients, formerly called inerts, in the formulations. The
24 known and disclosed inerts in triclopyr formulations are summarized in Table 4.

25
26 As summarized in Table 4, all of the 61.6% triclopyr BEE formulations contain kerosene, but at
27 differing amounts. Garlon 4 specifies additional ingredients including ethylene glycol and
28 solvent naphtha. Pathfinder II and Remedy RTU are both “ready to use” formulations—i.e.,
29 require no mixing and no addition of surfactants or other adjuvants—and both contain 13.6%
30 triclopyr-BEE and 86.4% inert ingredients. The inert ingredients in these formulations are
31 specified only as “proprietary surfactants”. The liquid formulations of 44.4% triclopyr TEA
32 specify other ingredients as either ethanol (Garlon 3A, Renovate 3, and Tahoe 3A) or
33 ethylenediaminetetraacetic acid (EDTA), which is a chelating agent (Triclopyr 3A, Triclopyr
34 3SL). Triclopyr 3SL also contains ethylene glycol.

35
36 Dow AgroSciences has indicated that kerosene will not be used in Garlon 4 formulations in the
37 future (Jachetta 2011). At this time, it appears that a dearomatized hydrocarbon distillate will be
38 used as an alternative. As of the time that this risk assessment was prepared, however, no new
39 batches of Garlon 4 had been manufactured and a new MSDS for Garlon 4 was not available. As
40 discussed further in Section 3.1.14 (Adjuvants and Other Ingredients), the toxicity of kerosene is
41 considered in the current risk assessment in terms of assessing the currently available toxicity
42 data on Garlon 4 formulations that did contain kerosene.

43
44 The only granular formulation of triclopyr, Renovate OTF, contains a different set of other
45 ingredients characterized only as proprietary fiber, proprietary clay, proprietary salt, and titanium
46 dioxide. One or more of these other ingredients may be toxicologically significant. As indicated

1 in Appendix 1, Table 1, the MSDS for Renovate OTF indicates that this formulation may cause
2 sensitization on inhalation exposure. None of the liquid formulations of triclopyr indicates a
3 potential for causing sensitization after inhalation exposures.

4
5 The significance of the other ingredients in triclopyr formulations is discussed further in
6 Section 3.1.14 (Inerts and Adjuvants).

7 **2.3. Application Methods**

8 **2.3.1. Terrestrial Applications**

9 **2.3.1.1. Terrestrial Broadcast Applications**

10 Table 5 provides an overview of the label directions for terrestrial applications of the triclopyr
11 formulations covered in this risk assessment. Except for the ready-to-use formulations (i.e.,
12 Pathfinder II and Remedy RTU), the triclopyr formulations are labeled for ground or aerial
13 broadcast applications.

14
15 The most commonly used application method is backpack (selective) foliar applications. In
16 selective foliar applications, the herbicide sprayer or container is carried by backpack and the
17 herbicide is applied to selected target vegetation. Application crews may treat up to shoulder
18 high brush, which means that chemical contact with the arms, hands, or face is plausible. To
19 reduce the likelihood of significant exposures, application crews are directed not to walk through
20 treated vegetation. Usually, a worker treats approximately 0.5 acres/hour with a plausible range
21 of 0.25-1.0 acres/hour (USDA/FS 1989 p 2-9 to 2-10).

22
23 Broadcast foliar ground applications, which may be conducted occasionally, involve the use of a
24 two- to six-nozzle boom mounted on a tractor or other heavy duty vehicle. With this equipment,
25 workers typically treat 11-21 acres/hour, with the low end of this range representative of a four-
26 wheel drive vehicle in tall grass and the upper end of the range representative of a large
27 bulldozer (USDA/FS 1989 p 2-9 to 2-10).

28
29 As noted in Table 5, several triclopyr formulations are labeled for aerial application. In Forest
30 Service programs, aerial broadcast applications are avoided; nonetheless, aerial applications are
31 included in the current Forest Service risk assessment in the event that aerial applications of
32 triclopyr are considered in specific Forest Service programs or projects. Aerial applications of
33 some triclopyr BEE formulations (i.e., Forestry Garlon, Garlon 4 Ultra, and Triclopyr 4E) are
34 limited to helicopters, while the other triclopyr BEE formulations may be applied by helicopter
35 or fixed wing aircraft. The rationale for limiting the aerial application of some triclopyr BEE
36 formulations to helicopters is not addressed in the available literature on triclopyr. For triclopyr
37 TEA formulations, the product labels generally limit aerial applications to helicopters. Triclopyr
38 3SL may be applied to rice using fixed wing aircraft; however, applications to rice are not
39 relevant to Forest Service activities.

40 **2.3.1.2. Non-Broadcast Applications**

41 Pathfinder II and Remedy RTU are ready-to-use formulations which are not labeled for any form
42 of broadcast application. Instead, these products are labeled for only basal bark and cut stump as
43 well as streamline basal bark applications in the southern United States. These application

1 methods may also be used with some other formulations of triclopyr BEE in which the mixing
2 directions specify the addition of diesel fuel, No. 1 or No. 2 fuel oil, kerosene, or a commercially
3 available basal oil. As discussed in Section 2.2, kerosene will no longer be used in Garlon 4
4 formulations. Nonetheless, kerosene is still permitted as an inert in nonfood use pesticides (U.S.
5 EPA/OPP 2011).

6
7 Basal bark is a low volume application method in which bark at the base of a small tree (usually
8 less than 6 inches in diameter) is wetted with the triclopyr formulation using a backpack sprayer.
9 While the bark is wetted as thoroughly as possible, runoff from the trunk to the ground surface is
10 avoided.

11
12 Cut stump applications, as the name implies, involves cutting down the tree and then applying
13 the triclopyr formulation to the tree stump. The stump is treated by applying the formulation to
14 the cambium as well as to the bark on the stump. As with basal bark applications, the bark is
15 wetted thoroughly; yet, not wetted to the point where the formulation will runoff to the
16 surrounding soil.

17
18 In streamline applications, the herbicide is sprayed directly onto the bark of the lower 2–3 feet of
19 the stem in a horizontal band to one side of the tree. The surfactant in the herbicide formulation
20 allows the active ingredient to spread around the stem. This treatment method is generally used
21 on relatively small trees (e.g., maximum diameters of approximately 4 inches). In these
22 applications, the herbicide sprayer or container is carried by backpack. The nozzle on the wand
23 or gun jet of the backpack sprayer should not be positioned higher than the handlers' waist,
24 reducing the likelihood that the chemical will come into direct contact with the arms, hands, or
25 face of the worker.

26
27 While not specifically noted on the triclopyr labels, triclopyr may be used in hack and squirt
28 applications. Hack and squirt applications are a form of cut surface treatment in which the bark
29 of a standing tree is cut with a hatchet and the herbicide is applied with a squirt bottle. This
30 treatment method is used to eliminate large trees during site preparation, conifer release
31 operations, or rights-of-way maintenance. As with selective foliar applications, a worker usually
32 treats about 0.5 acres/hour with a plausible range of 0.25-1.0 acres/hour (USDA/FS 1989 p 2-9 to
33 2-10).

34
35 In non-broadcast applications, application rates in units of lb a.e./acre may not be regarded as
36 meaningful descriptors of an application in that the areas treated are noncontiguous.
37 Nonetheless, the product labels for Pathfinder II and Remedy RTU have limitations on
38 application rates in units of lb a.e./acre which are identical to the limitations on broadcast
39 applications. Thus, an analysis of a noncontiguous application should be based on the total
40 amount of triclopyr applied and the total area over which the application will be made to
41 approximate an application rate in units of lb a.e./acre.

42 **2.3.2. Aquatic Applications**

43 As summarized in Table 6, several formulations of triclopyr TEA are labeled for aquatic
44 applications. No formulations of triclopyr BEE are labeled for aquatic applications. While not
45 explicitly noted on the product labels, all aquatic applications of triclopyr appear to be limited to
46 the control of aquatic macrophytes rather than algae.

1
2 The specific types of target vegetation differ among the various formulations. Garlon 3A and
3 Triclopyr 3A are labeled only for emergent vegetation along the shores of either standing or
4 flowing bodies of water. As discussed further in Section 2.4.2, the application rates for these
5 types of application are expressed as lbs a.e./acre, essentially identical to application rates used
6 for ground broadcast applications. Renovate 3 and Triclopyr 3SL are labeled for the control of
7 either emergent or submerged aquatic vegetation. For emergent vegetation, application rates are
8 expressed in units of lb a.e./acre and are identical to those for Garlon 3A and Triclopyr 3A. For
9 submerged vegetation, application rates are expressed as target concentrations in units of mg
10 a.e./L. Renovate OTF is labeled for the control of immersed, floating, or submersed vegetation,
11 and all application rates are specified as target concentrations in units of mg a.e./L. The specific
12 target concentrations for the different formulations of triclopyr TEA are discussed further in
13 Section 2.4.2.

14 **2.4. Mixing and Application Rates**

15 **2.4.1. Terrestrial Applications**

16 Foliar applications account for most of the use of triclopyr in Forest Service programs. As
17 discussed further in Section 2.5 (Use Statistics), the most recent use statistics available from the
18 Forest Service are for 2004. These statistics include uses defined by Forest Service region and
19 by management objective. The uses defined by management objective for 2004 are summarized
20 in Table 8. As indicated in Table 8, the major uses of triclopyr in terms of the amount used in
21 Forest Service programs involve conifer release (32%), noxious weed control (27%), site
22 preparation (18 %), mixed hardwood and conifer release (12%), hardwood release (5.5%), and
23 rights-of-way management (4%). All of these management objectives, which account for about
24 98.5% of the use of triclopyr in Forest Service programs, would primarily involve foliar
25 applications.

26
27 The maximum application rates vary according to the treatment site (Table 5). For sites at which
28 grazing may occur, the maximum application rate is 2 lb a.e./acre. For formulations of triclopyr
29 BEE, the maximum application rate at sites where grazing will not occur is 8 lb a.e./acre.
30 Several formulations of triclopyr BEE, however, specify a maximum application rate of 6 lb
31 a.e./acre for forestry sites. Somewhat higher application rates of up to 9 lb a.e./acre may be used
32 with formulations of triclopyr TEA.

33
34 As summarized in Table 8, the average application rate used in Forest Service programs is about
35 1 lb a.e./acre. This unit application rate is used for terrestrial applications in the EXCEL
36 workbooks that accompany this risk assessment. For 2004, the maximum application rate used
37 in any Forest Service program was 6.63 lb a.e./acre. This application was made in Forest 7 of
38 Region 8 (Southern Region) for noxious weed control. The lowest application rate on record for
39 2004 is 0.04 lb a.e./acre. This application was made in Forest 10 of Region 6 (Pacific
40 Northwest) and also was classified as noxious weed control. Albeit speculative, it is likely that
41 the unusually low application rate of 0.04 lb a.e./acre involved a noncontiguous area, as
42 discussed in Section 2.3.1.2.

43
44 Except for the ready-to-use formulations (i.e., Pathfinder II and Remedy RTU), triclopyr
45 formulations will be diluted with a carrier prior to application. For broadcast foliar applications,

1 triclopyr will typically be diluted with water and a surfactant. As noted in Table 5, all
2 formulations of triclopyr recommend the use of a non-ionic surfactant. The specific surfactants
3 that might be used in Forest Service programs have not been identified. Surfactants discussed in
4 the literature include various organosilicone surfactants such as Silwet L-77 (Bollig et al. 1995;
5 Buick et al. 1992; Forester 1998; Jackson et al. 1998; Pline et al. 1998), and alkylphenol
6 ethoxylate-containing surfactants such as R-11(Xie et al. 2005). Abdelghani et al. (1997) discuss
7 the use of Syndets surfactant, which is an ionic surfactant; however, it is not clear that this
8 surfactant is likely to be used in applications associated with Forest Service programs. While it
9 is beyond the scope of the current Forest Service risk assessment on triclopyr to review the
10 toxicity of non-ionic surfactants, the available information on the impact of surfactants on
11 triclopyr is discussed in Section 3.1.14.

12
13 For non-broadcast applications (e.g., streamline or basal bark) of triclopyr BEE, the formulation
14 is mixed with vegetable oils. While diesel fuel, No. 1 or No. 2 fuel oil, or kerosene had been
15 used in the 1990s, these petroleum based oils are no longer used in triclopyr applications.

16
17 For this risk assessment, the extent to which a triclopyr formulation is diluted prior to application
18 primarily influences dermal and direct spray scenarios, both of which depend on ‘field dilution’
19 (i.e., the concentration of triclopyr in the applied spray). In all cases, the higher the
20 concentration of triclopyr, which is equivalent to the lower dilution of the triclopyr formulation,
21 the greater the risk.

22
23 The product labels for Remedy and Triclopyr 4 Ester specify application volumes as low as 2
24 gallons per acre for aerial applications. In general, application volumes of 10-400 gallons per
25 acre are recommended on product labels. For Forest Service programs, however, the upper
26 range on the dilution volume typically will be no more than 40 gallons per acre. A typical
27 dilution rate is 25 gallons per acre. For the current Forest Service risk assessment, the central
28 estimate of the application volume is taken as 25 gallons per acre with a range of 5-40 gallons
29 per acre. Details regarding the calculation of field dilution rates are provided in worksheet A01.

30
31 The selection of application rates and dilution volumes in this risk assessment is intended simply
32 to reflect typical central estimates as well as plausible lower and upper ranges. In the assessment
33 of specific program activities, the Forest Service will use program-specific application rates and
34 application volumes.

35 **2.4.2. Aquatic Applications**

36 Aquatic weed control is a minor use for triclopyr in Forest Service programs. In the 5-year
37 period from 2000 to 2004, only one aquatic application of triclopyr, which involved an
38 application of 3 pounds to 1.8 acres in Region 8, Forest 7, is included in Forest Service pesticide
39 use reports.

40
41 As summarized in Table 6, most formulations of triclopyr TEA labeled for terrestrial
42 applications are also labeled for aquatic application; however, the target vegetation differs
43 among the various formulations. Garlon 3A and Triclopyr 3A are labeled only for emergent
44 aquatic macrophytes. Specific aquatic application rates are not specified on the product labels
45 for these formulations, and the label refers to use rates for terrestrial applications. In other

1 words, the aquatic application rates are expressed in units of lbs a.e./acre in which the acreage
2 refers to the surface area of water to be treated.

3
4 Renovate 3 and Triclopyr 3SL are labeled for either emergent or submerged vegetation. For
5 emergent vegetation, the application rates are expressed in units of lb a.e./acre, as is the case with
6 Garlon 3A and Triclopyr 3A. The label instructions for emergent vegetation specify application
7 rates of 0.5-6 lb a.e./acre, which are identical to the application rates for terrestrial applications.
8 For submerged vegetation, application rates are expressed as target concentrations ranging from
9 0.75 to 2.5 mg a.e./L. The upper bound of this range is also the maximum seasonal application
10 rate.

11
12 All of the above formulations —i.e., Garlon 3A, Triclopyr 3A, Renovate 3, and Triclopyr 3SL—
13 recommend the use of a nonionic surfactant. As discussed in the previous subsection, nonionic
14 surfactants are also recommended in terrestrial applications of these formulations.

15
16 Renovate OTF is labeled for emersed, floating, and submerged aquatic vegetation. This granular
17 formulation is labeled only for aquatic application, and all application rates are expressed as
18 target concentrations—i.e., from 1 to 2.5 mg a.e./L for floating or emersed weeds and from 0.5 to
19 2.5 mg a.e./L for submersed weeds. Unlike the other formulations labeled for aquatic
20 applications, Renovate OTF appears to be a ready-to-use formulation, in that the product label
21 does not include mixing directions or make reference to the use of surfactants.

22
23 For the current risk assessment, applications to both submergent and emergent aquatic vegetation
24 are considered. Similar to the approach taken for terrestrial applications, a unit application rate
25 of 1 lb a.e./acre is used in the EXCEL workbook for applications to emergent aquatic vegetation
26 (Attachment 3), and the consequences of using greater application rates are discussed in the risk
27 characterization (Sections 3.4 and 4.4). For applications to emergent aquatic vegetation, the
28 water depth is an important factor in estimating exposures to nontarget species. Typically,
29 applications to emergent vegetation will be made in relatively shallow water near the shoreline.
30 Based on the product label for Renovate OTF, a water depth of 2 feet with a range of 1-4 feet is
31 used.

32
33 Applications for the control of submergent vegetation (Attachment 4) are based on a nominal
34 target application of 1 mg a.e./L. The consequences of using lower or higher target
35 concentrations are also discussed in the risk characterization (Sections 3.4 and 4.4).

36 **2.5. Use Statistics**

37 Forest Service risk assessments attempt to characterize the use of an herbicide or other pesticide
38 in Forest Service programs relative to its use in agricultural applications. Forest Service
39 pesticide use reports up to the year 2004 are available on the Forest Service web site
40 (<http://www.fs.fed.us/foresthealth/pesticide/reports.shtml>). Information on agricultural use is
41 compiled by the U.S. Geological Survey (<http://water.usgs.gov/nawqa/pnsp/usage/maps/>). In
42 addition, detailed pesticide use statistics compiled by the state of California
43 (<http://www.calepa.ca.gov/>).

44
45 The USDA Forest Service tracks and reports pesticide use by geographical areas referred to as
46 “Regions”. The Forest Service classification divides the United States into nine regions

1 designated from Region 1 (Northern) to Region 10 (Alaska). [Note: There is no *Region 7* in the
2 Forest Service system.] The use of triclopyr in Forest Service regions for the year 2004 (the
3 most recent year for which statistics are available) is illustrated in Figure 3 and detailed further in
4 Table 7. By far, the greatest use of triclopyr occurs in the southeast, referred to by the Forest
5 Service as Region 8 or the Southern region. This region accounted for about 87% of triclopyr
6 use by the Forest Service in 2004. Relatively small amounts were used in Region 4 (about 5%),
7 Region 6 (about 4%), and Region 1 (about 3%). In other regions, the use of triclopyr by the
8 Forest Service is insubstantial, ranging from about 1% in Region 2 to no reported use in Regions
9 3, 9, and 10.

10
11 Triclopyr formulations are used extensively in agriculture. The USGS provides national
12 agricultural use statistics for 2002. As illustrated in Figure 4, about 1,000,000 lbs of triclopyr
13 were applied to pastureland in 2002. Much less triclopyr is applied to other commodities—i.e.,
14 about 150,000 lbs to rice, 100,000 lbs to hay, 2500 lbs to sod, and 10 lbs to blueberries. As
15 noted in Table 7, the total annual use of triclopyr by the Forest Service for 2004 was about
16 12,500 lbs, which is about 1% of the agricultural use [$\approx 12,500 \text{ lbs} \div 1,250,00 \text{ lbs} = 0.01$]. As
17 with Forest Service use, the greatest agricultural use of triclopyr is in the southeast of the United
18 States. Unlike the Forest Service, however, significant amounts of triclopyr are used in the
19 northeast of the United States (Forest Service Region 9).

20
21 More recent use statistics are available for California for the year 2007 (CDPR 2008).
22 According to CDPR (2008, pp. 407-408), the total use of triclopyr BEE was 67,007 lbs. Uses of
23 triclopyr BEE relevant to forest applications include 10,186 pounds applied to timberland and
24 21,029 lbs applied to rights-of-way. Thus, for triclopyr BEE, forestry related uses account for
25 about 46% of the uses of triclopyr BEE [$\approx 31,000 \text{ lbs} \div \approx 67,000 \text{ lbs}$]. For the TEA salt of
26 triclopyr, the total use in California was about 64,030 lbs, similar to the total use of triclopyr
27 BEE. Of this amount, about 8923 lbs or 14% was used in forestry related applications—i.e.,
28 997 lbs to timberland and 7926 lbs to rights-of-way.

3. HUMAN HEALTH

3.1. HAZARD IDENTIFICATION

3.1.1. Overview

The toxicity of triclopyr to mammals is relatively well characterized in numerous standard acute, subchronic, and chronic toxicity studies as well as developmental and reproduction studies required by the U.S. EPA/OPP for pesticide registration. In mammals, the toxicity studies that yield the most sensitive endpoints—i.e., the signs of toxicity that occur at the lowest doses—for triclopyr involve developmental and reproductive effects. For both developmental and reproductive effects, however, adverse effects on offspring, most of which are indicative of delayed growth rather than frank abnormalities, occur at doses associated with maternal toxicity.

Based on histopathology and clinical chemistry data from standard acute, subchronic and chronic toxicity studies on triclopyr, the liver and kidneys are the primary target organs. Like most weak acids, triclopyr is excreted primarily in the kidney by an active transport process. At very high doses, this process may become saturated causing triclopyr to reach toxic levels. At sufficiently high doses, triclopyr may cause toxic effects, including death. Nonetheless, triclopyr has a low order of acute lethal potency. There is no information suggesting that triclopyr causes direct adverse effects on the nervous system, endocrine system, or immune function.

Standard bioassays for carcinogenicity were conducted in both rats and mice. In male rats and mice, no statistically significant dose-related trends in tumor incidence were apparent. Based on pair-wise comparisons (i.e., control group vs an exposed group), statistically significant increases were observed for some tumor types, including benign and/or malignant pheochromocytomas combined as well as skin fibromas, in rats but not mice. In female rats and mice, there was a statistically significant dose-related increase in mammary gland adenocarcinomas. The U.S. EPA/OPP reviewed these studies and determined that the evidence for carcinogenicity is marginal and did not recommend a quantitative dose-response assessment for the carcinogenicity of triclopyr. The current risk assessment defers to this decision.

The major metabolite of triclopyr in both mammals and the environment is 3,5,6-trichloro-2-pyridinol, commonly abbreviated as TCP. Although TCP does not have the phytotoxic potency of triclopyr, this compound is toxic to mammals as well as other species. Based on RfDs derived by the U.S. EPA/OPP (Section 3.3), TCP is more toxic than triclopyr to mammals, and as discussed further in the ecological risk assessment, it is also more toxic than triclopyr to aquatic animals (Section 4.1.3). Consequently, exposures to TCP and its toxicity are considered explicitly in the current risk assessment.

3.1.2. Mechanism of Action

Although the toxicity of triclopyr to mammals is relatively well characterized (as detailed in subsequent sections) and its mechanism of action in plants is understood, its mechanism of action in mammals is unclear.

Studies regarding histopathology and clinical chemistry data on triclopyr suggest that the liver and kidneys are the primary target organs. Like most weak acids, triclopyr is excreted primarily in the kidney by an active transport process (Timchalk and Nolan 1997; Timchalk et al. 1990,

1 1997). At very high doses, this process may become saturated and triclopyr may
2 interfere/compete with the excretion of other weak acids. Under normal conditions of
3 environmental exposures, however, concentrations of weak acids in the body will be far below
4 those required to saturate the active transport process; accordingly, this mechanism of active
5 transport should not play a substantial or significant role in the assessment of potential health
6 effects. For example, at 5 mg triclopyr/kg bw in dogs, triclopyr is associated with a decrease in
7 phenolsulfonphthalein (PSP) excretion, a standard assay for kidney function. This decrease in
8 excretion, however, is due to competition between triclopyr and PSP rather than a direct toxic
9 effect in the kidney (Finco and Cooper 1995). Conversely, many weak acids also bind to protein
10 and this may inhibit secretion. In the monkey, triclopyr tends to increase the secretion of PSP
11 and other compounds, suggesting that triclopyr may compete with these other compounds for
12 protein binding sites (Timchalk et al. 1997). Again, this competition will be significant only at
13 relatively high doses. Since triclopyr is excreted by the kidney and active transport processes are
14 present in the mammalian kidney for triclopyr and many other weak acids, the apparent
15 sensitivity of the kidney to triclopyr may be related to reports of relatively high tissue
16 concentrations of triclopyr in the kidney.

17
18 Triclopyr is the pyridine analogue of 2,4,5-T. Like 2,4,5-T, the toxicity of triclopyr to plants
19 appears to involve the mimicking of auxin growth hormones (Section 4.1.2.5). The mammalian
20 toxicity of 2,4,5-T, particularly the induction of reproductive effects and the toxic effects of
21 2,4,5-T in humans, is related to the contamination of 2,4,5-T with TCDD (2,3,7,8-
22 tetrachlorodibenzo-p-dioxin) which is formed as an impurity in the synthesis of 2,4,5-T from the
23 chlorination of phenols. Because triclopyr is based on a pyridine ring rather than an aromatic
24 ring, the occurrence of TCDD in triclopyr is not plausible.

25 **3.1.3. Pharmacokinetics and Metabolism**

26 **3.1.3.1. General Considerations**

27 Pharmacokinetics involves the quantitative study of the absorption, distribution, and excretion of
28 a compound. Pharmacokinetics is particularly important to this risk assessment on triclopyr.
29 Many of the most plausible and quantitatively most significant exposure assessments (Section
30 3.2) involve dermal exposure, albeit, most of the dose-response assessments (Section 3.3) used to
31 interpret the consequences of dermal exposure involve oral exposure levels. Hence, it is
32 necessary to understand the kinetics of both oral and dermal absorption so that dermal exposure
33 assessments can be appropriately compared with oral dose-response assessments.

34
35 As discussed in Section 3.1.2, triclopyr is a weak acid similar to picloram, clopyralid, and 2,4-D.
36 As with most weak acids, triclopyr is excreted by the kidney via a well-characterized active
37 transport mechanism. Dogs, however, have an impaired ability to excrete weak acids including
38 triclopyr (e.g., Piper et al. 1973; Finco and Cooper 1995; Timchalk and Nolan 1997).
39 Consequently, dogs are more sensitive than humans and other animals to triclopyr. While
40 toxicity studies on dogs are considered in the human health risk assessment, toxicity data on dogs
41 are not used quantitatively in the human health risk assessment but are considered quantitatively
42 in the ecological risk assessment (Section 4.1.2.1).

43
44 Following oral exposure, triclopyr is absorbed and excreted relatively rapidly, with half-times for
45 oral absorption and urinary excretion of 3.61 and 1.1 hours, respectively. Virtually the entire

1 ingested dose of triclopyr is excreted unchanged in the urine, although four minor metabolites
2 are formed. After oral administration of 3 or 60 mg/kg of ¹⁴C-triclopyr acid to rats,
3 approximately 89-95% of the dose was recovered in the urine as unmetabolized triclopyr,
4 indicating that at least this proportion of the administered dose was absorbed. Very little residue
5 was recovered in the feces or carcass (Timchalk et al. 1990). Furthermore, the rapid urinary
6 elimination of triclopyr was observed in cattle after oral exposure to triclopyr, with 86.4% of the
7 administered dose eliminated unchanged in the urine and no residues detected in the milk or
8 feces. In this study, almost the entire administered dose was eliminated in the urine after 24
9 hours (Eckerlin et al. 1987).

10
11 In humans, more than 80% of the dose was recovered unmetabolized in the urine within 48 hours
12 after single oral doses of ¹⁴C-labeled triclopyr acid at 0.1 and 0.5 mg/kg. For these oral
13 exposures, the estimated absorption rate coefficients (k_a) were 0.851 hours⁻¹ at 0.1 mg/kg and
14 0.291 hours⁻¹ at 0.5 mg/kg. The corresponding urinary excretion rates (k_e) were 0.318 hour⁻¹ at a
15 dose of 0.1 mg/kg bw and 0.290 hour⁻¹ at a dose of 0.5 mg/kg bw (Carmichael et al. 1989).

16 **3.1.3.2. Dermal Absorption**

17 Most of the occupational exposure scenarios and many of the exposure scenarios for the general
18 public involve the dermal route of exposure. For these exposure scenarios, dermal absorption is
19 estimated and compared to an estimated acceptable level of oral exposure based on subchronic or
20 chronic toxicity studies in animals. It is, therefore, necessary to assess the consequences of
21 dermal exposure relative to oral exposure and the extent to which triclopyr is likely to be
22 absorbed from the skin surface.

23
24 Two types of dermal exposure scenarios are considered: immersion and accidental spills. In the
25 scenarios involving immersion, the concentration of the chemical in contact with the surface of
26 the skin is assumed to remain constant or at least nearly so. As detailed in SERA (2007), the
27 calculation of absorbed dose for dermal exposure scenarios involving immersion requires an
28 estimate of the dermal permeability coefficient (K_p) expressed in cm/hour, and the rate of
29 absorption is assumed to be essentially constant. In exposure scenarios involving direct sprays
30 or accidental spills where the compound is deposited directly on the skin, the concentration or
31 amount of the chemical on the surface of the skin is assumed to be the limiting factor in dermal
32 absorption. For these scenarios first-order dermal absorption rate coefficients (k_a), expressed as
33 a proportion of the deposited dose absorbed per unit time—e.g., hour⁻¹—are used in the exposure
34 assessment.

35 **3.1.3.2.1. First-Order Dermal Absorption**

36 **3.1.3.2.1.1. Triclopyr BEE**

37 Carmichael et al. (1989) assayed the dermal absorption of triclopyr BEE, formulated as Garlon 4,
38 in five male volunteers. Dermal exposures consisted of placing 0.65-1.1 mL of Garlon 4 on the
39 forearm so that the nominal applied dose was 5 mg triclopyr/kg body weight. The study does not
40 explicitly characterize the dermal loading dose to the skin—i.e., mg/cm² of skin. Based on Table
41 2 in Carmichael et al. (1989, p.435), the average amount of triclopyr BEE applied to the skin of
42 the five subjects was 259 mg/subject with a range from 185 to 345 mg/subject. Taking the
43 average surface area of the male forearm as 0.131 m² or 1310 cm² (EPA/ORD 1997, Table 6-2,
44 p. 6-13), the approximate loading dose to the skin was about 0.2 (0.14 to 0.26) mg/cm².

1 As detailed in the EXCEL workbooks that accompany this risk assessment, the upper bound of
2 the dermal loading in the exposure scenarios developed in the current risk assessment
3 (Worksheets C03a,b, D01a,b) is about 0.19 mg/cm², which is almost the same as the average
4 loading rate estimated from Carmichael et al. (1989). Thus, the first-order dermal absorption
5 rates that can be calculated from Carmichael et al. (1989) are clearly applicable and relevant to
6 the first-order dermal exposure scenarios developed in this risk assessment.

7
8 Dermal absorption was assayed by measuring the amount of triclopyr excreted in the urine of the
9 male volunteers over a period of 84 hours. As detailed in Carmichael et al. (1989, Table 2, p.
10 453), the urinary excretion of triclopyr was 1.37% with a range of 0.74-2.59%. These data were
11 fit to the pharmacokinetic model developed in the oral dosing phase of the study (as discussed in
12 Section 3.1.3.1), and the dermal absorption was estimated at an average of 1.37% of the dermal
13 dose with a range of 0.95-3.1%. Taking P as the proportion of the absorbed dose over the 8-hour
14 period of exposure (e.g., 0.0137 for the 1.37% absorption), the corresponding first-order dermal
15 absorption rate coefficient (k_a) can be estimated as:

Equation 1

$$k_a = \frac{\ln(1-P)}{t}$$

18
19 The estimated absorbed doses for the five subjects in Carmichael et al. (1989, Table 2) and the
20 corresponding estimated first-order dermal absorption rates (k_a) derived using the above equation
21 are summarized in Table 9 of the current risk assessment. Table 9 also summarizes the
22 derivation of the average k_a with 95% confidence intervals—i.e., 2.1×10^{-3} ($5.0 \times 10^{-4} - 3.7 \times 10^{-3}$)
23 hour⁻¹.

24
25 The only other experimental data on the dermal absorption of triclopyr BEE is the *in vitro* study
26 by Hotchkiss et al. (1992) using flow-through diffusion cells with skin from rats and humans.
27 After 72 hours, the extent of absorption for un-occluded preparations was 3.7% for rat
28 preparations and 0.7% for human preparations. Using occluded preparations, the corresponding
29 values increased to 8.6% for rat preparations and 3.3% for human preparations. The study by
30 Hotchkiss et al. (1992) used skin loading rates of 15 mg/cm². As discussed above, the current
31 risk assessment uses exposure scenarios with maximum loading rates of about 0.19 mg/cm²,
32 which is a factor of about 80 below the loading rates used by Hotchkiss et al. (1992) [15 mg/cm^2
33 $\div 0.19 \text{ mg/cm}^2 \approx 78.95$]. Given the availability of the *in vivo* study by Carmichael et al. (1989)
34 which used loading rates very similar to those considered in the current risk assessment, the *in*
35 *vitro* study by Hotchkiss et al. (1992) is only marginally relevant in quantitatively assessing the
36 dermal absorption rates for triclopyr BEE.

37
38 In the absence of experimental data, Forest Service risk assessments generally estimate first-
39 order dermal absorption rates based on quantitative structure activity relationships (QSAR), as
40 documented in SERA (2007a). The algorithm on which these estimates are based is developed
41 from the analysis of dermal absorption rates for compounds with K_{ow} values ranging from
42 0.0015 to 3,000,000 and molecular weights ranging from 60 to 400 g/mole. Using these methods
43 with the molecular weight (356.63 g/mole) and approximate K_{ow} (20,000) for triclopyr BEE, the
44 estimated first-order dermal absorption rate coefficients are approximately 3.1×10^{-3} ($1.2 \times 10^{-3} -$

1 8.1×10^{-3} hour⁻¹. The calculation of these rates is detailed in Worksheet B03b of Attachment 2
2 (triclopyr BEE formulations).

3
4 As summarized in Table 9, the estimated first-order dermal absorption rate coefficients from
5 Carmichael et al. (1989)—i.e., 2.1×10^{-3} (5.0×10^{-4} – 3.7×10^{-3}) hour⁻¹ – are strikingly similar to the
6 estimated first-order dermal absorption rate coefficients based on the algorithms typically used in
7 Forest Service risk assessments based on quantitative structure activity relationships (QSAR) –
8 i.e., 3.1×10^{-3} (1.2×10^{-3} – 8.1×10^{-3}) hour⁻¹. The selection of either set of values has little impact
9 on estimates of exposure. While experimental measures are typically preferred to estimates
10 based on QSAR, the central estimate and upper bound of the QSAR estimates are modestly
11 higher than those from Carmichael et al. (1989). Consequently, the QSAR estimates of 3.1×10^{-3}
12 (1.2×10^{-3} – 8.1×10^{-3}) hour⁻¹ are used to estimate dermal absorption in all exposure scenarios
13 involving first-order dermal absorption.

14 15 **3.1.3.2.1.2. Triclopyr TEA and Acid**

16 Very little information is available on the dermal absorption of triclopyr acid. As summarized by
17 U.S. EPA/OPP (1998a), 1.5% of a dermal dose (2 g/kg) was absorbed by rabbits. No
18 experimental details of this study are available. The U.S. EPA/OPP typically reports dermal
19 absorption values as a percent and applies the estimates of dermal absorption to exposure periods
20 of 1 day. Using Equation 1, an absorption of 1.5% would correspond to a dermal absorption rate
21 of 6.3×10^{-4} hour⁻¹ [$\ln(1-0.015)/24 \text{ hours} \cong 0.00063 \text{ hour}^{-1}$].

22
23 The TEA salt of triclopyr will dissociate essentially instantaneously in aqueous solutions to
24 triclopyr acid and triethylamine. As discussed further in Section 3.1.14.2, triethylamine is not
25 considered quantitatively in the current risk assessment. Consequently, the first-order dermal
26 absorption rate associated with applications of triclopyr TEA is based on the molecular weight
27 and K_{ow} of triclopyr acid. Using the same algorithm discussed in the previous subsection with
28 the molecular weight (256.47 g/mole) and the approximate K_{ow} (0.35 at pH 7) for triclopyr acid
29 (Table 2), the estimated first-order dermal absorption rate coefficients are approximately
30 8.8×10^{-4} (3.0×10^{-4} – 2.6×10^{-3}) hour⁻¹. The calculation of these rates is detailed in Worksheet
31 B03b of Attachment 1 (triclopyr TEA formulations). The central estimate of the first-order
32 dermal absorption rate using the algorithm is very close to the estimate of 6.3×10^{-4} hour⁻¹ from
33 the rabbit study summarized by U.S. EPA/OPP (1998a). Consequently, the estimated first-order
34 dermal absorption rate coefficients of 8.8×10^{-4} (3.0×10^{-4} – 2.6×10^{-3}) hour⁻¹ are used in all
35 exposure scenarios in which the assumption of first-order dermal absorption is used. These
36 scenarios are discussed further in Section 3.2 (Exposure Assessment).

37 **3.1.3.2.2. Zero-Order Dermal Absorption**

38 Another set of exposure scenarios used in this risk assessment involves the assumption of zero-
39 order absorption (i.e., the dermal absorption rate is constant over time). This type of assumption
40 is reasonable when the skin is in contact with a constant concentration of the pesticide. As
41 discussed further in Section 3.2, this type of exposure scenario is assumed for workers wearing
42 grossly contaminated gloves as well as members of the general public swimming in water
43 contaminated with triclopyr. This type of exposure scenario requires an estimate of dermal
44 permeability (K_p) in units of cm/hour.

1 No experimental data are available on the dermal permeability of triclopyr acids, salts, or esters.
2 In the absence of experimental data, Forest Service risk assessments generally use a QSAR
3 algorithm developed by the EPA (U.S. EPA/ORD 1992, 2007). This approach is discussed in
4 further detail in SERA (2007a). As with the algorithm for estimating the first-order dermal
5 absorption rate coefficient (Section 3.1.3.2.1), the algorithm developed by the U.S. EPA/ORD
6 (1992, 2007) is based on molecular weight and K_{ow} . As with the estimates for first-order
7 absorption, the values for K_{ow} and molecular weight used to implement the algorithms for
8 estimating the K_p are those summarized in Table 2. The algorithm developed by the U.S.
9 EPA/ORD (1992, 2007) is derived from an analysis of 95 organic compounds with K_{ow} values
10 ranging from about 0.0056 to 309,000 and molecular weights ranging from approximately 30 to
11 770. As summarized in Table 2, this range of values for K_{ow} and molecular weight encompass
12 the estimates of the corresponding values for triclopyr acid and triclopyr BEE.

13
14 Details of the implementation of the algorithms are given in Worksheet B03a in the EXCEL
15 workbooks for triclopyr TEA (Attachment 1) and triclopyr BEE (Attachment 2). Note that the
16 workbook for triclopyr TEA (Attachment 1) implements the algorithm for estimating K_p using
17 the molecular weight for triclopyr acid. The rationale for using these values is identical to that
18 given in the previous subsection. The algorithm developed by the U.S. EPA/ORD (1992, 2007)
19 results in an estimated dermal permeability (K_p) of about 2.4×10^{-5} ($1.0 \times 10^{-5} - 5.4 \times 10^{-5}$) cm/hour
20 for triclopyr acid and 1.3×10^{-2} ($6.6 \times 10^{-3} - 2.6 \times 10^{-2}$) cm/hour for triclopyr BEE.

21
22 Dermal exposures to 3,5,6-trichloro-2-pyridinol (TCP) are not likely to be significant in most
23 scenarios, except one which involves a swimmer. As discussed further in Section 3.2.3.4,
24 concentrations of TCP in water will be less than those of triclopyr; however, with respect to
25 potential risk, exposure to TCP in contaminated water cannot be summarily dismissed (Section
26 3.1.15). Since oral exposures to TCP from the consumption of contaminated water are
27 considered, it seems reasonable to consider the potential risk of exposure from swimming in
28 contaminated water. Accordingly, an estimate of the dermal permeability coefficient (K_p) is
29 necessary to assess the potential risk. Like the estimates for triclopyr acid and triclopyr BEE, the
30 dermal permeability coefficient for 3,5,6-trichloro-2-pyridinol is based on the algorithm
31 developed by the EPA (U.S. EPA/ORD 1992, 2007). This algorithm is implemented using a
32 molecular weight of 198.43 and K_{ow} of 100 for TCP (Table 2). Details of the calculations for the
33 K_p of TCP are given in Worksheet B03a of Attachment 5, the EXCEL workbook for TCP.
34 Based on these calculations, the estimated K_p for TCP is 1.5×10^{-2} ($9.4 \times 10^{-3} - 2.4 \times 10^{-2}$) cm/hour.

35 **3.1.3.3. Excretion**

36 Although excretion rates are not used directly in either the dose-response assessment or risk
37 characterization, excretion half-lives can be used to infer the effect of longer-term exposures on
38 body burden, based on the *plateau principle* (e.g., Goldstein et al. 1974, p. 320 ff). Under the
39 assumption of first-order elimination, the first-order elimination rate coefficient (k) is inversely
40 related to the half-life (T_{50}) [$k = \ln(2) \div T_{50}$]. If a chemical with a first-order elimination rate
41 coefficient of k is administered at fixed time interval (t^*) between doses, the body burden after
42 the N^{th} dose ($X_{N\ Dose}$) relative to the body burden immediately following the first dose ($X_{1\ Dose}$) is:
43

Equation 2

$$\frac{X_{N\text{Dose}}}{X_{1\text{Dose}}} = \frac{(1 - (e^{-kt^*})^N)}{1 - e^{-kt^*}}$$

As the number of doses (N) increases, the numerator in Equation 2 approaches a value of 1. Over an infinite period of time, the plateau or steady-state body burden (X_{Inf}) can be calculated as:

Equation 3

$$\frac{X_{Inf}}{X_1} = \frac{1}{1 - e^{-kt^*}} .$$

As discussed in Section 3.1.3.1, the average urinary excretion rate of triclopyr in humans is about 0.3 hour⁻¹ based on the study by Carmichael et al. (1989). For estimating body burden using the plateau principal, whole body excretion rates are generally preferable to urinary excretion rates. Nonetheless, the use of urinary excretion rates is acceptable because triclopyr is eliminated almost exclusively in the urine. An excretion rate of 0.3 hour⁻¹ corresponds to a rate of about 7.2 day⁻¹. Substituting this value into the above equation for the plateau principal, the estimated plateau in the body burden after daily doses over a prolonged period of time would be about 1.00075 [1 ÷ (1 - e^{-7.2})]. In other words, daily doses of triclopyr should not lead to any substantial accumulation in humans over prolonged periods of exposure. As discussed further in Section 3.2.3.5, this assessment is consistent with the lack of bioaccumulation of triclopyr in fish.

3.1.4. Acute Oral Toxicity

One very basic type of acute toxicity information involves time-specific LD₅₀ or LC₅₀ values (i.e., doses or concentrations of a toxicant that result in or are estimated to result in 50% mortality of the test species during a specified exposure or observation period). These values can be viewed as an index of acute lethal potency. Information on the acute oral toxicity of triclopyr formulations is summarized in Appendix 1 and information on the acute oral toxicity of triclopyr acid, the TEA salt of triclopyr, and triclopyr BEE is summarized in Appendix 2.

The acute LD₅₀ values for the triclopyr formulations presented in Appendix 1 are taken from the Material Safety Data Sheets (MSDS) for the different formulations. Several of the acute oral LD₅₀ values for formulations of 66.6% a.i. are identical for rats—i.e., 1581 mg/kg bw for male rats and 1338 mg/kg bw for female rats. Similarly, three formulations of 31.7% triclopyr TEA indicate identical acute oral LD₅₀ values of 2574 mg/kg bw for male rats and 1847 mg/kg bw for female rats. These identical LD₅₀ values for different formulations probably indicate *data bridging*. While the U.S. EPA/OPP generally requires at least acute toxicity data on pesticide formulations, the Agency will sometimes allow toxicity studies on one formulation to support the registration of another formulation. This general approach is sometimes referred to as *bridging*. If the two formulations are identical—i.e., the same formulation is marketed under different names—data bridging makes sense. Although a specific discussion of formulation bridging has not been encountered for triclopyr formulations, the identical acute LD₅₀ values within groups of

1 formulations containing the same active ingredient at the same nominal concentration probably
2 reflect data bridging.

3
4 Typically, acute oral LD₅₀ values given on MSDS are in units of mg formulation/kg bw. Thus,
5 for the acute oral LD₅₀ values for the 61.6% a.i. (44.3% a.e.) formulations of triclopyr BEE, the
6 oral LD₅₀ values of 1581 mg/kg bw for male rats and 1338 mg/kg bw for female rats may be
7 expressed as approximately 700 and 590 mg a.e./kg bw, respectively. Similarly, the rat oral
8 LD₅₀ values for the 44.4% a.i. (≈31.7% a.e.) formulations of triclopyr TEA of 2574 mg/kg bw
9 for male rats and 1847 mg/kg bw for female rats may be expressed as approximately 816 and
10 585 mg a.e./kg bw, respectively.

11
12 All of the toxicity values given on the MSDS for triclopyr formulations should be supported by
13 studies submitted to the U.S. EPA/OPP in support of the registration of the triclopyr
14 formulations. For example, the rat oral LD₅₀ values of 2574 mg/kg bw for male rats and 1847
15 mg/kg bw for female rats for the 44.4% a.i. (≈31.7% a.e.) formulations of triclopyr TEA clearly
16 come from the registrant submission by Mizell and Lomax (1988) (MRID 41443301)
17 summarized in Appendix 2 (Table A2-1). Not all of the LD₅₀ values for formulations given in
18 Appendix 1, however, can be associated with the studies identified on the acute oral toxicity of
19 triclopyr formulations summarized in Appendix 2. This limitation is not unusual and appears to
20 reflect nothing more than the fact the U.S. EPA/OPP does not summarize all acute oral LD₅₀
21 values in the summaries of available registrant-submitted studies (U.S. EPA/OPP 1996b, 1998a).

22
23 In the current risk assessment, acute oral LD₅₀ values are used primarily to compare the toxicity
24 of active ingredients—i.e., triclopyr acid, triclopyr BEE and triclopyr TEA—with corresponding
25 toxicity values on formulations. As discussed further in Section 3.1.14, these comparisons are
26 used to assess the toxicological significance of other ingredients (previously referred to as
27 *inerts*).

28 **3.1.5. Subchronic or Chronic Systemic Toxic Effects**

29 Systemic toxicity encompasses virtually any effects caused by a chemical after it has been
30 absorbed. Certain types of effects, however, are of particular concern to this risk assessment.
31 Such special effects are considered in following subsections and include effects on the nervous
32 system (Section 3.1.6) and immune system (Section 3.1.7), development or reproduction
33 (Section 3.1.8), and carcinogenicity or mutagenicity (Section 3.1.9). This section discusses the
34 remaining studies on systemic toxic effects.

35
36 Studies regarding the chronic and subchronic toxicity of triclopyr are summarized in Appendix 2,
37 Table A2-8. These studies include subchronic, repeated dosing studies conducted as range-
38 finding studies for cancer bioassays (e.g., Tsuda et al. 1987), standard 90-day subchronic studies
39 in rats (Barna-Lloyd et al. 1992; Landry et al. 1984), longer-term studies in dogs (e.g., Quast et
40 al. 1976, 1977, 1988), and lifetime studies in rats (Eisenbrandt et al. 1987) and mice (Tsuda et al.
41 1987). All of the studies summarized in Appendix 5 are unpublished and were submitted to U.S.
42 EPA/OPP to the support the registration of triclopyr.

43
44 The kidney appears to be the most sensitive target organ for triclopyr, and the dog appears to be
45 the most sensitive species. The lowest effect level for triclopyr is 2.5 mg/kg/day in the dog
46 (Quast et al. 1976, 1977, 1988). In Quast et al. (1977), this dose was associated with decreased

1 phenolsulfonphthalein (PSP) urinary excretion as well as reduced absolute and relative kidney
2 weights. As discussed in section 3.1.2, the inhibition of PSP excretion can be attributed to
3 competition between triclopyr and PSP for elimination via active anion transport in the proximal
4 tubule cells of the kidney. In the absence of other toxic effects, the 2.5 mg/kg/day dose in the
5 1977 dog study was classified as a NOEL by U.S. EPA. This determination formed the basis of
6 U.S. EPA's provisional acceptable daily intake of 0.025 mg/kg/day (U.S. EPA 1985)
7 (Section 3.3.3). The NOEL for PBP inhibition in dogs is 0.5 mg/kg/day (Quast et al. 1976).

8
9 In a follow-up study (Quast et al. 1988), the dose of 2.5 mg/kg/day is associated with a
10 statistically significant increase in serum urea nitrogen and creatinine in male dogs. These
11 effects were also evident but more pronounced at 5 mg/kg/day. The NOEL for this effect was
12 0.5 mg/kg/day. Creatinine and urea, which are normal metabolites formed by mammals, are
13 eliminated almost exclusively in the urine. Increases in the levels of these compounds can be
14 caused by impaired kidney function (i.e., decreased glomerular filtration). Although these
15 effects are the most sensitive endpoints available for exposure to triclopyr, they are not
16 particularly sensitive indicators of kidney damage. Usually, before increases in blood urea
17 nitrogen (BUN) or serum creatinine are evident, glomerular filtration must be depressed by 50-
18 70% (Goldstein and Schnellmann 1996).

19
20 One of the considerations in designating the 2.5 mg/kg/day dose as a NOEL in the earlier study
21 (Quast et al. 1977) was that BUN levels were unaffected. In the later study (Quast et al. 1988), a
22 statistically significant increase in BUN levels was noted in male dogs at 2.5 mg/kg/day (57%
23 increase over pre-exposure levels) and 5.0 mg/kg/day (108% increase over pre-exposure levels).
24 The difference between Quast et al. (1977) and Quast et al. (1988) may reflect differences in the
25 durations of the two studies – i.e., about 6 months for Quast et al. (1977) and 1 year for Quast et
26 al. (1988). U.S. EPA (1988a) to classified the dose of 2.5 mg/kg/day from Quast et al. (1988) as
27 an adverse effect level. At the lowest dose, 0.5 mg/kg/day, BUN levels were elevated by 38%
28 over pre-exposure levels, but this increase was not statistically significant. As discussed in
29 Section 3.3., this circumstance resulted in the lowering of a provisional U.S. EPA/OPP RfD to
30 0.005 mg/kg/day using the 0.5 mg/kg/day dose group as the NOEL for effects on kidney
31 function.

32
33 In rodents, kidney effects—hematological and histopathological changes and increased kidney
34 weight—were observed after subchronic exposure to triclopyr doses as low as 70 mg/kg/day for
35 90 days (Barna-Lloyd et al. 1992). Damage was characterized as degeneration of the proximal
36 tubules of the kidneys (≥ 20 mg/kg/day for 90 days) (Landry et al. 1984) and increases in kidney
37 weight (Eisenbrandt et al. 1987, Landry et al. 1984). As discussed further in Section 3.3, the
38 NOAEL for kidney toxicity in rats is 5 mg/kg bw/day from a two generation dietary reproduction
39 study in rats (Vedula et al. 1998) and NOAEL is the basis of the chronic RfD for triclopyr.

40
41 The other general systemic toxic effects of triclopyr are unremarkable. At high doses, signs of
42 liver damage may be apparent as well as decreases in food consumption, growth rate, and gross
43 body weight (Barna-Lloyd et al. 1992; Landry et al. 1984; Quast et al. 1976; Tsuda et al. 1987).

44 **3.1.6. Effects on Nervous System**

45 In severely poisoned animals, virtually any chemical may cause gross signs of toxicity which
46 might be attributed to neurotoxicity—e.g., incoordination, tremors, or convulsions. A direct

1 neurotoxicant, however, is defined as a chemical that interferes with the function of nerves,
2 either by interacting with nerves directly or by interacting with supporting cells in the nervous
3 system. This definition of a direct neurotoxicant distinguishes agents that act directly on the
4 nervous system (direct neurotoxicants) from those agents that might produce neurological effects
5 secondary to other forms of toxicity (indirect neurotoxicants). U.S. EPA has developed a battery
6 of assays to test for neurotoxicity (U.S. EPA/OCSP 2010), and U.S. EPA/OPP requires
7 neurotoxicity studies for pesticides when standard toxicity studies or other considerations such as
8 chemical structure suggest that concerns for effects on the nervous system are credible. In most
9 standard subchronic and chronic rodent bioassays used and accepted by U.S. EPA for pesticide
10 registration brain morphology is assessed. The spinal cord and peripheral nerves (e.g., sciatic
11 nerve) are usually evaluated only if there are other indications of neurotoxicity

12
13 As discussed in Sections 3.1.4, 3.1.5 and 3.1.9, the toxicology of triclopyr has been investigated
14 in acute, subchronic, chronic, developmental, and reproduction studies in mammals. Relatively
15 high doses of triclopyr may produce signs of toxicity that could be associated with neurotoxicity.
16 In a 4-day study, signs of toxicity were not observed in ponies after exposure to 60 mg/kg
17 bw/day; however, ataxia, weakness, and tremors were observed in ponies after exposure to 300
18 mg/kg bw/day (Osweiler 1983). Similarly, tremors were observed in dams exposed to a dose of
19 200 mg/kg bw/day triclopyr acid in a developmental study in rats conducted by Thompson et al.
20 (1979). Given the numerous toxicity studies available on triclopyr, these findings appear to be
21 incidental. While U.S. EPA/OPP (1996b, 1998a) does not explicitly address or evaluate the
22 neurotoxicity of triclopyr, the Agency has not required specific neurotoxicity studies on
23 triclopyr.

24
25 The triclopyr formulations used by the Forest Service contain two inerts which are classified as
26 toxic, ethanol (Garlon 3A) and kerosene (Garlon 4). Both of these agents are neurotoxic. The
27 potential effects of these agents are considered further in Section 3.1.14 (Adjuvants and Other
28 Ingredients).

29 **3.1.7. Effects on Immune System**

30 There is very little direct information on the immunotoxicity of triclopyr. The only studies
31 specifically related to the effects of triclopyr on immune function are skin sensitization studies
32 conducted on triclopyr BEE and the triethylamine salt of triclopyr (Section 3.1.11.2). Skin
33 sensitization was caused by both triclopyr BEE and the triethylamine salt of triclopyr in studies
34 that follow standard protocols accepted by the U.S. EPA/OPP (1998a, p. 6). While these studies
35 support an assessment that triclopyr may cause skin sensitization, they provide no information
36 useful for directly assessing the immune suppressive potential of triclopyr.

37
38 The thymus has an important role in normal immune function and has a considerable capacity to
39 regenerate (Schuurman et al. 1991). An increase in the size of the thymus could be indicative of
40 repair after injury. A field study by Lochmiller et al. (1995) notes increased thymus weights in
41 rabbits in geographical areas treated with triclopyr followed by controlled burn, relative to
42 undisturbed areas. The magnitude of the thymus weight increase was about 56% (Lochmiller et
43 al. 1995, Table 1). No effect on thymus mass, however, was noted in geographical areas treated
44 with triclopyr without a subsequent controlled burn.

1 As noted in the previous discussion on the neurologic effects (Section 3.1.6), the toxicology of
2 triclopyr has been investigated in subchronic, chronic, and multigeneration studies in rodents and
3 in subchronic studies in dogs. In a subchronic feeding study (Tsuda et al. 1987), enlargement of
4 the thymus was observed in mice after a dose of 480 mg/kg bw/day. This observation, however,
5 appears to have been qualitative, and the study does not provide data on thymus weights (U.S.
6 EPA/OPP 1989a, p. 10). None of the other subchronic or chronic studies report changes in
7 lymphoid tissues. In the absence of a consistent pattern of pathology in the thymus or other
8 tissues related to immune function, the observation by Tsuda et al. (1987) of thymus enlargement
9 in treated mice is not a compelling basis for asserting that triclopyr may have a significant impact
10 on immune function in mammals.

11 **3.1.8. Effects on Endocrine System**

12 Assessments of the direct effects of chemicals on endocrine function are most often based on
13 mechanistic studies on estrogen, androgen, or thyroid hormone systems (i.e., assessments on
14 hormone synthesis, hormone receptor binding, or post-receptor processing). The U.S. EPA/OPP
15 has developed a battery of screening assays for endocrine disruption (i.e.,
16 http://www.epa.gov/ocspp/pubs/frs/publications/Test_Guidelines/series890.htm). Triclopyr was
17 not selected as one of the pesticides for which the screening assays are being required (U.S.
18 EPA/OPP 2009b). Inferences concerning the potential for endocrine disruption can sometimes
19 be made from responses seen in standard toxicity tests—i.e., changes in the structure of major
20 endocrine glands (i.e., the adrenal, hypothalamus, pancreas, parathyroid, pituitary, thyroid,
21 ovary, and testis) or changes in growth rates. As with effects on the nervous system and immune
22 function, however, effects on organs associated with endocrine function may be secondary to
23 other toxic effects. Thus, in the absence of information on specific endocrine mechanisms,
24 pathological changes in endocrine tissues do not necessarily indicate a direct effect on endocrine
25 function. In terms of functional effects that have important public health implications, effects on
26 endocrine function would be expressed as diminished reproductive performance or abnormal
27 development.

28
29 Triclopyr has not been tested for activity as an agonist or antagonist of the major hormone
30 systems (e.g., estrogen, androgen, thyroid hormone) in mammals. As discussed further in
31 Section 4.1.3.1, Xie et al. (2005) report a significant induction of plasma vitellogenin in juvenile
32 rainbow trout exposed to mixtures of triclopyr and surfactants. Vitellogenin is a protein
33 associated with eggs yolks and occurs normally in female fish as well as other non-mammalian
34 vertebrates and invertebrates. The induction of vitellogenin in males is suggestive of potential
35 estrogenic activity. Exposure of juvenile trout to triclopyr alone, however, did not result in a
36 significant increase in plasma vitellogenin (Xie et al. 2005, Figure 1). In the abstract to the
37 publication, Xie et al. (2005) state that: *Binary mixtures of TPA with triclopyr also caused*
38 *greater than additive Vtg responses in two middle concentrations when compared to TPA [one of*
39 *the surfactants] or triclopyr alone.* Although certain citations to Xie et al. (2005) in the available
40 literature suggest that triclopyr may be an endocrine disruptor (Kortenkamp 2007), Kramer et al.
41 (2008) discuss a number of concerns with the study, most notably the lack of information
42 regarding the sex of the treated fish and a clear discussion of statistical methods used to support
43 the assertion of greater than additive toxicity. The lack of information on the sex of the fish used
44 in the Xie et al. (2005) study is a serious and substantial criticism. In the absence of this
45 information, the data reported by Xie et al. (2005) cannot be interpreted clearly and are of
46 marginal use in the assessment of potential endocrine effects in fish or other species.

1
2 As indicated in the following section (Section 3.1.9), extensive data are available on the
3 reproductive and developmental effects of triclopyr; moreover, the current RfD for triclopyr
4 (Section 3.3) is based on a 2-generation reproduction toxicity study in rats (Vedula et al. 1995).
5 Although fetal toxicity and abnormalities have been observed at higher doses (Section 3.1.9),
6 there is no indication in this or any other studies (Appendix 2) that triclopyr caused any of the
7 toxic effects through a mechanism involving endocrine disruption.

8 **3.1.9. Reproductive and Developmental Effects**

9 **3.1.9.1. Developmental Studies**

10 Developmental studies are used to assess whether a compound has the potential to cause birth
11 defects as well as other effects during development or immediately after birth. These studies
12 typically entail gavage administration to pregnant rats or rabbits on specific days of gestation.
13 Teratology assays as well as studies on reproductive function (Section 3.1.9.2) are generally
14 required by the EPA for the registration of pesticides. Very specific protocols for developmental
15 studies are established by U.S. EPA/OPPTS and are available at
16 http://www.epa.gov/opptsfrs/publications/OPPTS_Harmonized.

17
18 Table 10 provides an overview of the developmental studies on triclopyr acid, triclopyr TEA,
19 and triclopyr BEE and includes all of the studies summarized in Appendix 2 (Table 7). Except
20 for the recent study by Carney et al. (2007) and the abstracts of some studies published in the
21 open literature (e.g., Breslin et al. 1996, Breslin and Billington 1995), the studies summarized in
22 Table 10 were submitted by registrants in the support of the registration of triclopyr and are,
23 therefore, unpublished.

24
25 At sufficiently high doses, triclopyr can cause adverse developmental effects including birth
26 defects. A consistent pattern with triclopyr, however, is that adverse developmental effects occur
27 only at doses that are maternally toxic.

28
29 As also summarized in Table 10, developmental studies in rats and rabbits have been conducted
30 using triclopyr acid, triclopyr TEA, and triclopyr BEE. In the studies on rabbits, the three forms
31 of triclopyr appear to be essentially equitoxic. The only substantial difference is the lower
32 maternal NOEC of 10 mg/kg bw/day for triclopyr TEA, compared with the maternal NOAEL of
33 30 mg/kg bw/day in the study by Breslin and Billington (1995). Based on the maternal LOAELs
34 for rabbits, no substantial differences are apparent among the three forms of triclopyr. In the
35 developmental studies on rats, the maternal LOAELs for triclopyr BEE (22-30 mg/kg bw/day)
36 are somewhat lower than those for triclopyr acid (50 mg/kg bw/day) or triclopyr TEA (50-100
37 mg/kg bw/day).

38
39 The recent study by Carney et al. (2007), which involves assays of both triclopyr TEA and BEE,
40 suggests that triclopyr BEE is somewhat more toxic than triclopyr TEA, based on maternal
41 NOAELs and LOAELs. The 22 mg a.e./kg bw/day doses classified as LOAELs in Table 10,
42 however, reflect a relatively mild response—i.e., a transient decrease in body weight which
43 occurred early in the study. In addition, both triclopyr TEA and BEE caused similar effects on
44 maternal body weight gain at a dose of 72 mg a.e./kg bw, while triclopyr BEE caused only a
45 modestly greater decrease in body weight gain at the highest dose tested, 216 mg a.e./kg bw/day

1 (Carney et al. 2007, Figure 1). Most of the various malformations noted in these studies appear
2 to be associated with decreased growth. One possible exception, however, involves eye
3 abnormalities. Based on Table 2 in the study by Carney et al. (2007), the incidence of
4 micropthalmia and anophthalmia (abnormally small or missing eyes) was statistically
5 significant ($p=0.05$) in the high dose group during Study 1; yet, was not observed in the high
6 dose group of Study 2. Although Carney et al. (2007) report that the effect was statistically
7 significant in Study 1, the discussion in the paper suggests that the effect was an aberration.
8 Given the appearance of this effect in control and low-dose groups in Study 2, this assertion
9 appears to be reasonable.

10
11 Overall, the developmental studies in rats and rabbits with triclopyr acid, triclopyr TEA, and
12 triclopyr BEE do not suggest substantial or consistent differences in the developmental effects of
13 the various forms of triclopyr.

14 **3.1.9.2. Reproduction Studies**

15 Multi-generation reproduction studies typically involve dietary exposures of a group of rats or
16 mice referred to as the *parental generation* or P_1 . Male and female animals are selected from
17 this group and mated. Exposure of the female continues through gestation and after delivery.
18 Offspring from the parental generation, typically referred to as F_1 , are then continued on dietary
19 exposure through sexual maturity. The F_1 offspring are mated (and then referred to as the P_2
20 generation) producing an F_2 generation. This is the basic design of a “two-generation” study
21 although variations on this design are sometimes used and occasionally the study is carried over
22 to a third generation. Multi-generation reproduction studies typically focus on effects on
23 reproductive capacity—i.e., the number of young produced and their survival. Teratogenicity
24 studies, which are designed to assess the potential for producing birth defects, typically involve
25 daily gavage exposure of the pregnant female (most often rats or rabbits) during sensitive periods
26 of fetal development.

27
28 As summarized in Appendix 2, Table A2-7, triclopyr acid has been tested in three multi-
29 generation reproduction studies in rats (Beliles and Wosu 1976; Breslin 1990a; Vedula et al.
30 1995). In terms of the current risk assessment, the most significant study is the two-generation
31 reproduction study by Vedula et al. (1995). As detailed in Section 3.3, this study is the basis of
32 the current RfD on triclopyr. In this study, male and female rats were exposed to dietary
33 concentrations of triclopyr resulting in doses of 0, 5, 25, or 250 mg/kg/day, except that the P_1
34 males in the high dose group were exposed only to concentrations resulting in a daily dose of
35 100 mg/kg bw/day. The 5 mg/kg/day dose groups evidenced no adverse effects in parents or
36 offspring. At 25 mg/kg/day, degeneration of renal proximal tubules was observed only in adult
37 animals. At 250 mg/kg/day, parental effects included decreased food consumption and body
38 weights as well as histopathological changes in the liver and kidney. Fetotoxic effects,
39 including decreased pup survival and litter sizes, were noted only at 250 mg/kg/day.

40
41 The NOAEL of 25 mg/kg/day for reproductive effects from Vedula et al. (1995) is supported by
42 Hanley et al. (1983), published in the open literature as Hanley et al. (1984). These investigators
43 conducted a three-generation reproduction study in the same strain of rats in which no adverse
44 effects were observed on offspring at doses of 3, 10, or 30 mg/kg/day. This study appears to be
45 identical to the registrant-submitted study by Breslin (1990a, MRID 41688301). Furthermore,
46 the NOAEL of 25 mg/kg bw/day is also supported by an earlier study, Beliles and Wosu (1976)

1 in which no adverse reproductive effects were observed in rats exposed to doses of up to 30
2 mg/kg bw/day.

3 **3.1.10. Carcinogenicity and Mutagenicity**

4 Information regarding the mutagenicity and carcinogenicity of triclopyr is reviewed in detail in
5 U.S. EPA/OPP (1998a,b,c). Also, a review of the cancer bioassay data on triclopyr (Goodman
6 and Hildebrandt 1996) was submitted to U.S. EPA in support of the registration of this
7 compound.

8
9 Standard bioassays for carcinogenicity have been conducted in both rats (Eisenbrandt et al. 1987)
10 and mice (Tsuda et al. 1987). Details of both studies are summarized in Appendix 2,
11 Table A2-8. In male rats and mice, no statistically significant dose-related trends in tumor
12 incidence were apparent. Based on pair-wise comparisons (i.e., control group vs an exposed
13 group), statistically significant increases were observed for some tumor types, including benign
14 and/or malignant pheochromocytomas combined and skin fibromas, in rats but not mice. In
15 female rats and mice, there was a statistically significant dose-related increase in mammary
16 gland adenocarcinomas.

17
18 The EPA reviewed these studies and determined that the evidence for carcinogenicity is marginal
19 (U.S. EPA/OPP 1998a). This position is articulated briefly in U.S. EPA/OPP (1998a), and
20 because of the importance of this decision to the risk assessment, the position is worth quoting
21 directly:

22
23 *As a result of the August 9, 1995 meeting of the Agency's*
24 *Carcinogenicity Peer Review Committee (CPRC), triclopyr was*
25 *classified as a Group D chemical (not classifiable as to human*
26 *carcinogenicity). This decision was based on increases in mammary*
27 *tumors in both the female rat and mouse, and adrenal*
28 *pheochromocytomas in the male rat, which the majority of the CPRC*
29 *believed to be only marginal. Overall the majority of the CPRC felt that*
30 *the animal evidence was marginal (not entirely negative, but yet not*
31 *convincing). Therefore, the consensus of the CPRC was to classify*
32 *triclopyr as a Group D chemical, based on what was considered only*
33 *marginal response and the absence of additional support from structural*
34 *analogs or genotoxicity.*

35 U.S. EPA/OPP 1998a, p. 18

36
37 A detailed summary of the mutagenicity studies on triclopyr, most of which indicate no
38 mutagenic activity, is provided in Appendix 2, Table A2-9.

39
40 The discussion by Goodman and Hildebrandt (1996) of the potential carcinogenicity of triclopyr
41 is far more detailed and focuses on a re-evaluation of slides from the original studies as well as
42 an assessment of tumor rates in historical controls. Both types of analyses are common and
43 appropriate in the assessment of carcinogenicity data. Based on these analyses, Goodman and
44 Hildebrandt (1996) assert that triclopyr should not be classified as a carcinogen. In terms of the
45 current risk assessment, this position has no impact: The decision stated in EPA/OPP (1998a) to
46 classify triclopyr as Group D is accompanied automatically by a decision not to derive a cancer

1 potency factor for triclopyr; hence, in terms of a risk assessment, the potential carcinogenicity of
2 triclopyr is not considered quantitatively.

3
4 Cox (2000) suggests that since triclopyr has been shown to cause a statistically significant dose-
5 related increase in mammary gland tumors in both mice and rats, the U.S. EPA guidelines for
6 cancer risk assessment indicate that triclopyr should be classified as a carcinogen. Cox (2000)
7 cites the 1984 guidelines issued by U.S. EPA—i.e., FR 49: 46299-46300. The Agency has since
8 issued final guidelines for the classification of potential carcinogenicity (U.S. EPA/RAF 2005).
9 The 1984 guidelines do clearly indicate that a compound will be classified as a carcinogen if it
10 has been shown to cause cancer in two species of laboratory animals. The more recent
11 guidelines, however, are less proscriptive and allow the EPA to exercise substantial judgment
12 based on the nature and quality of the data. In addition, the newer guidelines emphasize the
13 importance of mechanistic considerations in the interpretation of carcinogenicity as well as the
14 development of weight-of-evidence determinations in assessing whether or not compounds
15 should be treated as carcinogens for the purpose of risk assessment.

16
17 Triclopyr has been shown to *cause* the same type of tumors in two species. In addition, while all
18 cancers are a public health concern, the particular tumor type noted in rats and mice (breast
19 cancer) is a common and important form of cancer in humans. Notably, however, none of the
20 dose groups in either rats or mice evidenced a statistically significant pair-wise increase in breast
21 tumors. In other words, the magnitude of the response was not substantial. The other important
22 factor discussed in U.S. EPA/OPP (1998a) is the apparent lack of mutagenic activity of triclopyr.
23 As detailed in U.S. EPA/OPP (1998a), only one study, a dominant lethal assay summarized in
24 detail in Appendix 2 (Table A2-9), indicates any form of mutagenic activity, and the other
25 standard assays for genotoxicity were negative.

26
27 The only other potentially relevant information encountered in the literature is the epidemiologic
28 report by Gambini et al. (1997). This study examines mortality patterns in a cohort of rice
29 farmers in Northern Italy in an effort to determine whether excess risks for cancer might be
30 detected and associated with chemical exposure. The study finds a significantly lower than
31 expected number of deaths, with a slight decrease in overall cancer mortality. The study
32 indicates that in 1990, 210 kg of triclopyr was used in the region of Italy in which the cohort of
33 rice farmers lived.

34
35 While the studies by Eisenbrandt et al. (1987) and Tsuda et al. (1987) could be used to derive
36 cancer potency factors, the current risk assessment defers to the judgment expressed in U.S.
37 EPA/OPP (1998a) and does not quantitatively consider the potential carcinogenic risk of
38 triclopyr. This position is appropriate given the detailed review presented in U.S. EPA/OPP
39 (1998a) and the legislative mandate of U.S. EPA/OPP to determine the carcinogenic risks of
40 pesticides and to regulate their use. In addition, the Group D classification of triclopyr in terms
41 of potential carcinogenicity was recently restated in the Agency's pesticide tolerances for
42 triclopyr (U.S. EPA/OPP 2002a).

43 **3.1.11. Irritation and Sensitization (Effects on the Skin and Eyes)**

44 The U.S. EPA/OPP requires standard assays for skin and eye irritation as well as skin
45 sensitization for both active ingredients and formulations. The material safety data sheets
46 (MSDS) for all triclopyr formulations contain information on these endpoints, which is

1 summarized in Appendix 1 for all of the formulations considered in the current risk assessment.
2 The registrant-submitted studies on these endpoints are summarized in Appendix 2: Table A-3
3 for skin irritation, Table A2-4 for skin sensitization, and Table A2-6 for eye irritation. As with
4 the acute oral toxicity studies discussed in Section 3.1.4, it is not always possible to associate the
5 information on the MSDS with specific registrant studies submitted to the U.S. EPA.
6

7 Exposure to triclopyr formulations may result in irritation to the skin and eyes. Technical grade
8 triclopyr is classified as only slightly irritating (Category IV) (Kuhn 200c). Triclopyr TEA
9 (Garlon 3A) is not a primary skin irritant (Mizell (1988b) is shown in some studies to cause
10 delayed contact sensitizations (Berdasco 1994a; Mizell 1989) but not in others (Berdasco
11 1990a,b). Triclopyr BEE also is shown to cause delayed contact hypersensitivity (Berdasco
12 1994b). Triclopyr BEE causes more severe skin irritation (Van Beeck and Leegwater 1981a)
13 than triclopyr acid or TEA. This may be due to the more rapid absorption of triclopyr BEE.
14

15 Ocular exposure appears to follow a different pattern with triclopyr TEA being much more
16 irritating (Mizell 1988a) than triclopyr acid (Kuhn 2000b) or triclopyr BEE (MRID 40557007, as
17 summarized in U.S. EPA/OPP, 1998a). According to the data summarized in Appendix 1, eye
18 irritation caused by exposure to the 44.4% TEA formulations is characterized variously as
19 *Irreversible/C, Corrosive/Irreversible*, or simply *Corrosive*, and it is not clear whether these
20 brief descriptions from the various MSDS reflect underlying differences in the studies on which
21 these descriptions are based. In addition, it seems likely that the identical descriptions given for
22 eye irritation in the various MSDS for the 44.4% TEA formulations may reflect data bridging
23 rather than essentially identical results from assays of the individual formulations.
24

25 The potential for eye irritation associated with handling 44.4% TEA formulations is clear. In a
26 review of pesticide incidents associated with occupational exposures in California, Maddy et al.
27 (1990) note that the only adverse effect associated with triclopyr involved two cases of eye
28 injury. While eye irritation is not treated quantitatively in the current risk assessment, eye
29 irritation is a clear concern for occupational exposures.

30 **3.1.12. Systemic Toxic Effects from Dermal Exposure**

31 As discussed in Section 3.1.3, triclopyr appears to be more readily absorbed after oral dosing
32 than after dermal application. Accordingly, dermal LD₅₀ values are greater than oral LD₅₀
33 values. As summarized in Appendix 1, the oral definitive LD₅₀ values in rats for the triclopyr
34 formulations considered in this risk assessment range from 1000 to 1581 mg/kg bw. All of the
35 dermal LD₅₀ values are non-definitive. In other words, the reported dermal LD₅₀ values range
36 from >2000 to >5000 mg/kg bw. While *non-definitive* LD₅₀ values are often associated with
37 limit tests (i.e., a single dosing at only one dose level), standard single dose studies involving
38 several different dose levels sometimes result in maximum mortalities that are substantially
39 below 50%, and the dose-response relationship may be such that the LD₅₀ or other comparable
40 value cannot be estimated. In these instances, a non-definitive LD₅₀ is reported in which the
41 *greater than* value is the highest dose or concentration tested. The dermal LD₅₀ values for all
42 triclopyr formulations appear to be based on limit tests, which typically involve single limit
43 doses of either 2000 or 5000 mg/kg bw. All of the acute dermal toxicity studies reviewed in the
44 preparation of this risk assessment (Appendix 2, Table A2-2) involved only a single dose, and no
45 studies report mortality or signs of toxicity other than weight loss.
46

1 Repeated dermal dosing studies on triclopyr are also summarized in Appendix 2, Table A-2.
2 Three of these studies (Van Beeck and Leegwater 1981a,b; Van Beeck et al. 1984) involve
3 applications of Garlon 4—i.e., triclopyr BEE. The only study to report systemic toxic effects is
4 Van Beeck et al. (1984) in which rats received dermal doses of 24, 240, and 480 mg a.i./kg
5 bw/day, 5 days/week for 3 weeks. A significant decrease in food intake and growth was
6 observed in males at all dose levels, and a significant decrease in food efficiency was observed in
7 males at all dose levels and in females at the highest dose. The low dose in this study was
8 equivalent to about 17 mg a.e./kg bw, which is classified as a LOAEL in males and a NOAEL in
9 females. The LOAEL in females (based on food conversion efficiency) was about 170 mg
10 a.e./kg bw/day.

11
12 Comparable 3-week oral toxicity studies on triclopyr BEE are not available. A 13-week oral
13 toxicity study on triclopyr BEE (Barna-Lloyd et al. 1992) reports oral NOAELs of about 20 mg
14 a.e./kg bw in males and 50 mg a.e./kg bw/day in females, with corresponding LOAELs of about
15 50 mg a.e./kg bw/day in males and 250 mg a.e./kg bw/day in females.

16
17 The most reasonable basis for comparing the subchronic dermal data with the oral toxicity data is
18 the NOAELs and LOAELs in female rats. The geometric mean of the NOAELs and LOAELs in
19 female rats from the subchronic dermal study by Van Beeck et al. (1984) is about 54 mg a.e./kg
20 bw/day $[(17 \times 170)^{0.5} \approx 53.759]$. The corresponding value for female rats in the oral toxicity
21 study by Barna-Lloyd et al. (1992) is about 79 mg a.e./kg bw/day $[(50 \times 250)^{0.5} \approx 79.057]$.
22 Based on this comparison of the subchronic dermal and oral toxicity of triclopyr BEE in female
23 rats, the dermal route of exposure appears to be about equally if not modestly more toxic than the
24 oral route of exposure—i.e., $79 \text{ mg a.e./kg bw/day oral} \div 54 \text{ mg a.e./kg bw/day dermal} \approx$
25 $1.46_{\text{oral/dermal}}$. While this comparison is limited and should not be overly interpreted, subchronic
26 dermal exposures to triclopyr BEE are as great a concern as oral exposures.

27
28 One 21-day dermal toxicity single-dose limit study is available in rabbits (MRID 42212701). In
29 this study, which is cited in several EPA assessments (U.S. EPA/OPP 1996b, 1998a, 2002a),
30 increased absolute and relative liver weights were observed in male rabbits after exposure to the
31 limit dose of 1000 mg/kg bw/day as triclopyr BEE. These effects were ...*considered marginal*
32 *and not of toxicological significance* (U.S. EPA/OPP 2002a). There are no corresponding
33 subchronic oral studies on triclopyr acid. Nonetheless, it is apparent that triclopyr acid is less
34 toxic in rabbits than triclopyr BEE is in rats.

35 **3.1.13. Inhalation Exposure**

36 The information regarding the inhalation toxicity of triclopyr, as summarized in Appendix 2
37 (Table A2-5), is limited to three studies involving exposure to technical grade triclopyr, triclopyr
38 BEE, and triclopyr TEA. No mortalities were observed in any of the studies. The only study not
39 summarized in U.S. EPA/OPP (1998a) is the recent report by Carter (2000) on technical grade
40 triclopyr. The reported LC₅₀ of >2.6 mg/L in this study is equivalent to the reported LD₅₀ value
41 of 2.6 mg/L for triclopyr TEA. Based on these results, U.S. EPA/OPP (1998a) classifies
42 inhalation exposures as of no toxicological concern.

43 **3.1.14. Adjuvants and Other Ingredients**

44 At least some formulations of triclopyr TEA contain the triethylamine salt of triclopyr as well as
45 emulsifiers, surfactants, and ethanol (Table 4). Triclopyr BEE formulations contain the

1 butoxyethyl ester (BEE) of triclopyr as well as inerts, including deodorized kerosene. As
2 reviewed in U.S. EPA/OPP (1998a), triclopyr TEA dissociates extremely rapidly to triclopyr
3 acid and triethylamine, and triclopyr BEE hydrolyzes rapidly to triclopyr acid and 2-
4 butoxyethanol. As noted in Section 2.2, formulations of Garlon 4 manufactured after January
5 2011 will not use kerosene. Kerosene is considered in the following discussion because the
6 currently available toxicity data on Garlon 4 does involve formulations that contained kerosene.

7
8 Relatively little information is available on the toxicity of triethylamine. There is an extensive
9 database on the toxicity of 2- butoxyethanol, and much of the available information associated
10 with potential human health effects is reviewed by ATSDR (1998). The acute oral MRL for 2-
11 butoxyethanol is 0.4 mg/kg/day, and the intermediate MRL for 2-butoxyethanol is 0.07
12 mg/kg/day (ATSDR 2002). As detailed further in Section 3.3, the acute MRL for 2-
13 butoxyethanol is on the same order as the acute RfD for triclopyr (1 mg/kg/day), and the
14 intermediate MRL for 2-butoxyethanol is similar to the intermediate and chronic RfD for
15 triclopyr (0.05 mg/kg/day).

16
17 The toxicity of ethanol, which is used in formulations such as Garlon 3A, is extremely well
18 characterized in humans, and the hazards of exposure include intoxication from acute exposure
19 as well as liver cirrhosis and fetal alcohol syndrome (WHO 1988). For chronic exposure, the
20 alcohol contained in Garlon 3A will not be of toxicological significance because of the rapid
21 breakdown of alcohol in the environment and the relatively high levels of alcohol associated with
22 chronic alcohol poisoning. Similarly, alcohol is not likely to pose an acute toxic hazard.
23 Approximately 15 mL of alcohol is contained in 1 oz of an alcoholic beverage containing 50%
24 alcohol (100 proof) [$0.5 \cdot 1 \text{ oz} \cdot 29.6 \text{ mL/oz} \approx 14.8 \text{ mL}$]. This level may cause mild intoxication
25 in sensitive individuals. Each mL of Garlon 3A contains 0.01 mL of ethanol. Therefore, 1480
26 mL, or approximately 1.5 L, of Garlon 3A must be consumed to equal the amount of alcohol
27 contained in 1 oz of an alcoholic beverage. The same amount of Garlon 3A contains 540,000 mg
28 a.e. of triclopyr [$1.5 \text{ L} \cdot 360,000 \text{ mg a.e./L}$]. For a 70 kg man, this dose would equal
29 approximately 770 mg a.e./kg, which is similar to the LD₅₀ for rats. As discussed in the dose-
30 response section (section 3.3), this estimate may be a reasonable approximation of a lethal dose
31 for triclopyr in humans. Thus, compared with the active ingredient, which is triclopyr, the
32 amount of ethanol in Garlon 3A does not appear to be toxicologically significant in terms of
33 potential systemic toxicity. Nonetheless, ethanol is an effective solvent. As detailed in Section
34 3.1.11, some formulations of triclopyr TEA have been associated with severe eye irritation.
35 While somewhat speculative, these irritant effects could be due, at least in part, to ethanol.

36
37 The importance of kerosene to the potential toxicity of Garlon 4 is more difficult to assess.
38 Deodorized kerosene is classified by U.S. EPA as a List 3 Inert. This list contains pesticide
39 inerts that the U.S. EPA considers lacking in toxicological data. The toxicity of kerosene is
40 reviewed in ATSDR (1995). At sufficiently high doses, kerosene can cause many
41 gastrointestinal, central nervous system (CNS), and renal effects. Although some of the effects
42 observed are consistent with the effects observed in mammals given large oral doses of Garlon 4
43 (e.g., diarrhea, lethargy, tremors, etc.), the same effects are observed in animals given triclopyr
44 alone or Garlon 3A.

1 The acute lethal dose of kerosene for humans ranges from approximately 2000 to 12,000 mg/kg;
2 the acute oral LD₅₀ values in experimental mammals range from approximately 16,000 to 23,000
3 mg/kg. As discussed in section 3.3, there is no information regarding the acute lethal potency of
4 triclopyr to humans. In experimental mammals, acute oral LD₅₀ values for triclopyr range from
5 approximately 600 to 1000 mg/kg. Thus, the acute lethal potency of kerosene is approximately
6 16 times less than the acute lethal potency of triclopyr. Given the relative potency of kerosene,
7 the acute effects associated with exposure to Garlon 4 are probably attributable to triclopyr and
8 not to kerosene.

9
10 No monitoring data are available regarding kerosene levels during the application of Garlon 4.
11 Middendorf et al. (1992) monitored triclopyr air levels ranging of approximately 5-15 µg/m³,
12 based on the personal breathing zone air of workers involved in backpack sprays. If kerosene is
13 present at a concentration of ≤20% in Garlon 4, the corresponding concentration of kerosene in
14 the air would be approximately 1-3 µg/m³. The NOAEL for neurological effects in experimental
15 mammals after exposure to kerosene, which ranged from 14 days to 1 year, is approximately 100
16 mg/m³; the NIOSH TLV for petroleum distillates is 350 mg/m³ (ATSDR 1995). Thus, plausible
17 levels of exposure to kerosene during applications of Garlon 4 are approximately 30,000-100,000
18 below the NOEL for kerosene in experimental mammals and a factor of 120,000-350,000 below
19 the TLV for petroleum distillates. Although some components of kerosene are known to be
20 carcinogenic to humans (e.g., benzene), kerosene is not classified as a carcinogen, and
21 quantitative risk assessments have not been conducted on kerosene (ATSDR 1995).

22
23 Inferences concerning the toxicological significance of TEA, BEE, as well as other adjuvants
24 used in triclopyr formulations can also be made based on a comparison of the toxicities of
25 triclopyr acid, triclopyr BEE, triclopyr TEA, and triclopyr formulations. As summarized in
26 Appendix 2, the acute oral LD₅₀ of triclopyr acid is 729 mg a.e./kg bw in male rats and 630 mg
27 a.e./kg bw in female rats. These oral toxicity values are similar to the LD₅₀ values of 828 mg
28 a.e./kg in male rats and 594 mg a.e./kg for exposure to Garlon 3A. Similarly, the acute oral LD₅₀
29 values for triclopyr acid are very close to the reported LD₅₀ of 578 mg a.e./kg bw for exposure to
30 triclopyr BEE. In other words, based on a comparison of the acute oral LD₅₀ values, triclopyr
31 acid rather than the TEA or BEE moieties appears to account for the toxicity of the two active
32 ingredients.

33
34 Taking the oral 578 mg a.e./kg bw for triclopyr BEE, the expected LD₅₀ of a 60.5% a.i. (44.3%
35 a.e.) would be about 1300 mg formulation/kg bw [578 mg a.e./kg bw ÷ 0.443 a.e./formulation ≈
36 1305 mg formulation/kg bw]. As discussed above and detailed in Appendix 1, the lower bound
37 of the reported LD₅₀ values for 60.5% a.i. formulations of triclopyr BEE is 1338 mg
38 formulation/kg bw. In other words, the toxicity of these triclopyr formulations is consistent with
39 the assumption that the agent of concern in the triclopyr BEE formulations is triclopyr rather
40 than the BEE moiety or the other ingredients included in the triclopyr BEE formulations.

41
42 As summarized in Appendix 2, Table 1, the oral LD₅₀ of triclopyr TEA (as Garlon 3A) is 828 mg
43 a.e./kg bw in male rats and 594 mg a.e./kg bw in female rats (Mizell and Lomax 1988). These
44 LD₅₀ values are very similar to oral LD₅₀ of triclopyr acid – i.e., 729 mg/kg in male rats and 630
45 mg/kg in female rats. Thus, as with triclopyr BEE (discussed above), the toxicity of this

1 triclopyr formulation is consistent with the assumption that the agent of concern in the triclopyr
2 TEA formulation is triclopyr rather than the TEA moiety.

3 **3.1.15. Impurities and Metabolites**

4 The major metabolite of triclopyr in both mammals and the environment is 3,5,6-trichloro-2-
5 pyridinol, commonly abbreviated as TCP. Although TCP does not have the phytotoxic potency
6 of triclopyr, this compound is toxic to mammals as well as other species. As illustrated in
7 Figure 5, TCP is also a metabolite of the insecticide chlorpyrifos. While a detailed discussion of
8 the toxicity of chlorpyrifos is beyond the scope of the current document, it is worth noting that
9 chlorpyrifos is an organophosphate insecticide that acts by inhibition of cholinesterase (U.S.
10 EPA/OPP 2001b). As also illustrated in Figure 5, chlorpyrifos contains the P=S (phosphorus to
11 sulfur double bond) characteristic of organothiophosphate cholinesterase inhibitors. This
12 structure is not contained in either TCP or triclopyr, and there is no indication that either TCP or
13 triclopyr inhibit cholinesterase.

14
15 In the EPA RED on triclopyr, U.S. EPA/OPP (1998a), the potential hazards associated with
16 exposures to TCP are discussed in some detail (pp. 31 ff). The U.S. EPA estimates dietary
17 exposures at the upper 99.5% level for a young woman (i.e., the most sensitive population in
18 terms of potential reproductive effects) the endpoint of greatest concern for triclopyr. The upper
19 range of acute exposure to triclopyr is estimated at 0.012 mg/kg/day, and the upper range of
20 exposure to chlorpyrifos is estimated at 0.016 mg/kg/day. Thus, based on the assumption that
21 both triclopyr and chlorpyrifos are totally converted to TCP, the total exposure is about 0.028
22 mg/kg/day, which is a factor of about 890 below the level of concern. For chronic exposures,
23 U.S. EPA/OPP (1998a) bases the risk assessment on infants (i.e., individuals at the start of a
24 lifetime exposure). The dietary analysis indicates that the total exposure expressed as a fraction
25 of the RfD is 0.04 for TCP from triclopyr and 0.091 for TCP from chlorpyrifos, for a total of
26 0.131 or a factor of about 7.6 below the level of concern [$1 \div 0.131 = 7.6$]. Based on this
27 assessment, U.S. EPA/OPP (1998a) concludes:

28
29 *...the existing uses of triclopyr and chlorpyrifos are unlikely to result in acute*
30 *or chronic dietary risks from TCP. Based on limited available data and*
31 *modeling estimates, with less certainty, the Agency concludes that existing*
32 *uses of triclopyr and chlorpyrifos are unlikely to result in acute or chronic*
33 *drinking water risks from TCP. Acute and chronic aggregate risks of concern*
34 *are also unlikely to result from existing uses of triclopyr and chlorpyrifos.*

35 – U.S. EPA/OPP 1998a, p. 34.

36
37 These basic conclusions are maintained in the U.S. EPA/OPP (2002a) pesticide tolerance for
38 triclopyr and TCP.

39
40 Notwithstanding the above assessment in U.S. EPA/OPP (1998a, 2002a), this risk assessment
41 does specifically include a consideration of exposures to TCP that may result from specific
42 program activities in the use of triclopyr. This approach is taken because the exposure
43 assessments considered in Forest Service risk assessment (Section 3.2) differ from those used by
44 the U.S. EPA/OPP in dietary and drinking water assessments. In addition and as discussed
45 further in Section 3.3, the acute and chronic RfDs for TCP derived by U.S. EPA/OPP are below
46 the corresponding RfDs derived by the U.S. EPA/OPP. Consequently, oral exposures to TCP

1 which may result from the use of triclopyr in Forest Service programs are addressed in Section
2 3.2.3.8, and the risks that might be associated with these exposures are discussed in Section
3 3.4.3.2.

4 **3.1.16. Toxicological Interactions**

5 The potential for adverse effects in humans or other mammals from the interactions of triclopyr
6 with other compounds is not addressed in the literature, and most inferences that can be made are
7 speculative. In terms of mechanism of action, it is likely that triclopyr would influence and be
8 influenced by other weak acids excreted by the kidney. As discussed in Section 3.1.2, these
9 influences, however, would be significant only at relatively high doses that saturate the active
10 transport processes involved in excretion by the kidney.

3.2. EXPOSURE ASSESSMENT

3.2.1. Overview

All exposure assessments are summarized in Worksheet E01 for workers and Worksheet E03 for the general public in the EXCEL workbooks that accompany this risk assessment. For terrestrial applications as well as aquatic applications for emergent vegetation, all exposure assessments are based on a unit application rate of 1 lb a.e./acre. For aquatic applications to control submergent vegetation, all exposure assessments are based on a target concentration of 1 mg a.e./L. The consequences of varying this application rate are considered in the risk characterization (Section 3.4).

For workers involved in terrestrial applications of triclopyr, three types of application methods are modeled: directed foliar (backpack), broadcast ground spray, and aerial spray. The exposure assessment for workers is substantially different from that in the previous Forest Service risk assessment (SERA 2003). In the previous risk assessment, standard worker exposure rates (mg/kg bw per lb/acre) were used. Reservations with this approach were expressed based on the backpack study by Spencer et al. (2000); however, another backpack study by Middendorf (1992a) involving basal stem applications suggested that the standard worker exposure rates for triclopyr were appropriate. Since the 2003 risk assessment, a backpack foliar study conducted by Krieger et al. (2005), and an earlier backpack foliar study by Middendorf (1992b) were identified. All of these studies involve forestry applications of triclopyr BEE and were sponsored by the Forest Service. Taken together, the studies by Middendorf (1992b), Spencer et al. (2000), and Krieger et al. (2005) clearly suggest that workers involved in backpack foliar applications of triclopyr BEE will be subject to substantially greater exposures than would be anticipated based on the standard methods used in most Forest Service risk assessments. Consequently, the study by Middendorf (1992b) is used directly for estimating exposures to workers involved in backpack foliar applications. This study is also used to adjust exposure rates for workers involved in ground boom and aerial applications of triclopyr BEE formulations. There are no detailed exposure studies of workers applying triclopyr TEA formulations in the available literature. While that lack of worker exposure studies involving triclopyr TEA adds uncertainty to this risk assessment, the differences in dermal absorption rates (which are well-documented for triclopyr BEE) suggest that no adjustments for the worker exposure rates for applications of triclopyr TEA are necessary. Consequently, for applications of triclopyr TEA the standard worker exposure rates used in most Forest Service risk assessments are maintained. The differences in exposures estimated for workers involved in applications of triclopyr TEA and triclopyr BEE have a substantial impact on the risk characterization (Section 3.4.2).

Under normal circumstances, members of the general public should not be exposed to substantial levels of triclopyr as a result of Forest Service activities. Nonetheless, several highly conservative scenarios are developed for this risk assessment. For terrestrial applications of triclopyr, the greatest exposures are associated with the acute and longer-term consumption of contaminated fruit and vegetation. This is typical of any pesticide exposure following foliar application. Exposures associated with dermal contact and the consumption of water (except for an accidental spill) are considerably lower.

3.2.2. Workers

Exposure assessments for workers are summarized in Worksheet E01 of the EXCEL workbooks that accompany this risk assessment: Attachment 1 for terrestrial applications of triclopyr TEA, Attachment 2 for terrestrial applications of triclopyr BEE, and Attachments 3 and 4 for emergent and submergent aquatic applications of triclopyr TEA. These workbooks contain sets of worksheets that detail each exposure scenario discussed in this risk assessment as well as summary worksheets for both workers and members of the general public. Documentation for these worksheets is presented in SERA (2009a). This section on workers and the following section on the general public provide a plain language description of the worksheets and discuss the triclopyr-specific data used in the worksheets.

Two types of worker exposure assessments are considered: general and accidental/incidental. The term *general exposure* is used to designate exposures involving absorbed dose estimates based on handling a specified amount of chemical during specific types of applications. The accidental/incidental exposure scenarios involve specific events that may occur during any type of application. All exposure assessments for workers as well as those for members of the general public and ecological receptors, are based on a unit application rate of 1 lb a.e./acre. The unit application rate is adopted as a convenience. For most exposure scenarios, exposure and consequent risk will scale linearly with the application rate, and the consequences of using lower or higher application rates are considered in the risk characterization (Section 3.4).

3.2.2.1. General Exposures

3.2.2.1.1. Terrestrial Applications

As described in SERA (2007a) and summarized in Table 11 of the current risk assessment, worker exposure rates are expressed in units of mg of absorbed dose per kilogram of body weight per pound of chemical handled. These rates are based on analyses of several different pesticides using a variety of application methods as detailed in SERA (1998). Based on these studies, default exposure rates are estimated for three different types of applications: directed foliar (backpack), boom spray (hydraulic ground spray), and aerial. As summarized in Table 11, the ranges of estimated occupational exposure rates vary substantially among individuals and groups, (i.e., by factors of up to 100). The studies used to develop these exposure rates provided information on estimates for individual workers of both absorbed dose (typically from monitoring urinary excretion) as well as the amount of pesticide that each worker applied. Table 11 also summarizes worker exposure rates that can be derived from three studies involving backpack applications of triclopyr BEE (Middendorf 1992a and 1992b; Krieger et al. 2005; Spencer et al. 2000). With the exception of Krieger et al. (2005), each of these studies provides estimates of both the absorbed dose and the amount of triclopyr applied by individual workers. As discussed further below, the study by Krieger et al. (2005) provides only average exposure rates based on estimates of average absorbed doses and the average amount of triclopyr BEE handled by the workers.

Middendorf (1992a) assayed exposure in groups of backpack workers involved in basal stem applications of Garlon 4. Total absorption was determined by the analysis of triclopyr in the urine over a 5-day post-application collection period. A summary of relevant data from Middendorf (1992a) is given in Table 12. The Middendorf (1992a) study involved 16 workers (designated as Worker A to Worker R) who applied 4-5.6 kg of triclopyr at three different sites.

1 As with most studies of worker exposure which provide individual data, exposure rates among
2 workers varied substantially, with the lowest exposure rate of 0.00015 mg/kg bw per lb applied
3 for Worker A at Site 1 and the highest exposure rate of 0.01428 mg/kg bw per lb applied for
4 Worker H at Site 2 [0.01428 mg/kg bw per lb applied ÷ 0.00015 mg/kg bw per lb applied ≈ 96].
5 As discussed by Middendorf (1992a), a major source of variation involves the use of gloves.
6 As summarized in Table 12, 6 of the 16 workers in the study by Middendorf (1992a) either did
7 not wear gloves during applications. For these workers, the average exposure rate was about a
8 factor of 4 higher than the exposure rate for workers who wore gloves [0.0049 mg/kg per lb
9 applied ÷ 0.00124 mg/kg per lb applied ≈ 3.95]. In terms of the current Forest Service risk
10 assessment, the exposure rates for workers wearing gloves are most relevant because gloves are
11 required in all Forest Service applications of triclopyr.

12
13 As in the SERA (1998) analysis as well as in the analyses of other studies discussed below, both
14 estimated doses (mg/kg bw) and exposure rates (mg/kg bw per lb handled) are expressed as the
15 geometric mean as well as lower and upper bounds based on the fit of the data to a log-normal
16 distribution using STATGRAPHICS Plus (Manugistics 1997). Because all of the data sets are
17 relatively small, the chi-square goodness of fit test to the log-normal distribution could not be
18 conducted, and all measures of goodness of fit are based on the Kolmogorov-Smirnov test in
19 which *p*-values of less than 0.05 indicate a significant lack of fit and *p*-values greater than 0.05
20 are consistent with the hypothesis that the data fit a log-normal distribution. As indicated in
21 Table 12, analyses of both the estimated doses and exposure rates fit the log-normal distribution
22 with *p*-values ranging from 0.68 to 0.96. As indicated in other similar tables discussed below, all
23 data sets discussed in this section are consistent with the log-normal distribution with *p*-values in
24 excess of 0.6 based on the Kolmogorov-Smirnov test. Consequently, the statistical fit to the log-
25 normal distribution is not discussed further.

26
27 As indicated in Table 11 (the summary of all rates), the estimated exposure rates from
28 Middendorf (1992a) for workers wearing gloves are 0.00124 (0.00015 to 0.01) mg/kg bw per lb
29 a.i., and these rates are consistent with the standard rates used for backpack applications in Forest
30 Service risk assessments—i.e., 0.003 (0.0003 to 0.010 mg/kg bw per lb) handled. This
31 consistency is to be expected because Middendorf (1992a) was one of the studies used to
32 develop the exposure rates for the backpack workers in the SERA (1998) analysis.

33
34 The other three backpack studies summarized in Table 11, Middendorf (1992b), Spenser et al.
35 (2000), and Krieger et al. (2005) indicate higher worker exposure rates. Again, the study by
36 Middendorf (1992b) was not included in the SERA (1998) analysis, and the other two studies
37 were published since that analysis. All three studies were funded by and conducted in
38 cooperation with the USDA Forest Service.

39
40 Middendorf (1992b) was conducted with groups of workers involved in directed foliar
41 applications of Garlon 4. This study is summarized in the open literature by Middendorf et al.
42 (1992) and Tharr (1994). As detailed in Table 13, this study involves 22 workers applying
43 Garlon 4 at four different sites with each worker handling between 1.2 and 2.2 lbs a.i. Unlike the
44 study by Middendorf (1992a), worker body weights are not given in Middendorf (1992b). In
45 Table 13, the doses in units of mg/kg bw are based on the total absorbed doses reported for each
46 worker in Middendorf (1992b) divided by an assumed body weight of 83.1 kg, the average body

1 weight of workers in the Middendorf (1992a) study. As with the study by Middendorf (1992a),
2 the variability in the estimated individual exposure rates in the study by Middendorf (1992b) is
3 substantial and spans a factor of about 63—i.e., the lowest exposure rate is about 0.00124 mg/kg
4 bw per lb handled and the highest exposure rate is about 0.078 mg/kg bw per lb handled [0.078
5 \div 0.00124 \approx 63.03].
6

7 The study by Middendorf (1992b) is considered particularly relevant to the current Forest
8 Service risk assessment because the worker practices used in the application are representative of
9 Forest Service programs. As noted in the study,
10

11 *The Forest Service supplied and required all volunteers to wear tightly*
12 *woven, pre-washed, long-sleeved shirts and long pants. All volunteers also*
13 *wore leather boots and a hard hat. Gloves were available for use at each*
14 *site during applications; their use was required when handling the*
15 *concentrate. The clothing met the Forest Service Guidelines.*

16 Middendorf 1992b, p. 11
17

18 Nonetheless, not all workers used the same protective equipment. As summarized in Table 13,
19 three workers at Site 3 – i.e., designated as workers NM, RH, and JJ – did not wear glove during
20 applications and these three workers tended to have relatively high rates of exposure ranging
21 from about 0.01 to 0.066 mg/kg bw per lb handled. In addition, the other two workers at Site 3
22 also had relatively high exposure rates even though these workers wore gloves. The average
23 exposure rate for the workers at Site 3 is about a factor of 4 higher than the average exposure rate
24 that the other sites [0.0236 \div 0.0058 mg/kg bw per lb applied \cong 4.06]. As discussed by
25 Middendorf (1992b), this higher exposure rate appears to be associated with the unusually high
26 brush height at the site:
27

28 *The brush typically ranged from four to 12 feet high on each of the stands*
29 *and was very dense. The stands were described by Forest Service*
30 *representatives as borderline acceptable for treatment. Subsequent*
31 *discussions with Regional Forest Service Representatives suggest that the*
32 *sites may not have been appropriate for directed foliar application based*
33 *on the height of the brush.*

34 Middendorf 1992b, p. 7
35

36 Consequently, and as discussed further below, the data from Site 3 are not viewed as
37 representative of application conditions in Forest Service programs and the data from Site 3 are
38 censored from the derivation of worker exposure rates.
39

40 Spenser et al. (2000) also provides data on the exposure rates for individual workers involved in
41 backpack applications of triclopyr BEE. Data from this study are summarized in Table 11 with
42 additional details provided in Table 14. The exposure rates estimated from Spenser et al. (2000)
43 are 0.015 (0.0042 to 0.052) mg/kg bw per lb handled. These estimated exposure rates are higher
44 than the standard exposure rates by a factor of about 5, based on both the central estimates [0.015
45 \div 0.003 = 5] and upper bounds [0.052 \div 0.010] of exposure rates. A limitation in the Spenser et
46 al. (2000) study involves the urine sampling. While Spenser et al. (2000) attempted to obtain

1 complete urine collections over each 24-hour period, the actual urine collections were highly
2 variable (Spencer et al. 2000, Appendix 1, Table 4), ranging from 30 to 1400 mL. To adjust for
3 incomplete urine collection, Spencer et al. (2000) adjusted all urine volumes to 1400 mL. In
4 other words, urinary excretion was calculated as the pooled concentration of triclopyr in the
5 urine multiplied by 1400 mL and divided by the volume of urine collected from the worker.
6 While the 1400 mL urine volume is a reasonable estimate (ICRP 1975), this approach to
7 correcting for incomplete urine collection would tend to overestimate urinary excretion if the
8 sample was collected during a period of high excretion, such as during or shortly after work, but
9 could underestimate exposure if the urine was collected during a period of low excretion.
10 Nonetheless, the exposure rates from Spenser et al. (2000)—i.e., 0.015 (0.0042 to 0.052) mg/kg
11 bw per lb handle—are strikingly consistent with the rates from Middendorf (1992b)—i.e., 0.0080
12 (0.00088 to 0.073) mg/kg bw per day.

13
14 The third worker exposure study sponsored by the Forest Service, Krieger et al. (2005),
15 monitored the exposure of individuals using backpack sprayers to apply a commercial
16 formulation of triclopyr and 2,4-D (Garlon 4 and 2,4-D LV6) for purposes of conifer release and
17 regeneration in Klamath National Forest in Northern California. The backpack sprayers refilled
18 their tanks approximately every 30 minutes, and treated from 1.1 to 10 acres each day. At the
19 end of 6 days, workers had treated 55 acres of forest with 24 gallons each of the above triclopyr
20 and 2,4-D formulations. Based on the total gallons used and the total number of acres treated,
21 the average triclopyr application rate during the study was 1.75 lb a.e./acre [24 gallons x 4 lb
22 a.e./gallon ÷ 55 acres ≈ 1.7455 lb a.e./acre]. The estimated absorbed dose based on the average
23 amount of triclopyr excreted by each worker as about 0.043 mg/kg bw, and the average amount
24 of triclopyr handled by each worker was 2 lbs. Thus, the estimated worker exposure rate from
25 Krieger et al. (2005) is about 0.0215 mg/kg bw per lb handled. As indicated in Table 11, no
26 lower and upper bounds for exposure rates from Krieger et al. (2005) are given because
27 individual data are not reported in this study. As indicated in Table 11, the central estimate of
28 0.0215 mg/kg bw per lb handled is somewhat greater than the central estimates from the studies
29 by Middendorf (1992b) and Spenser et al. (2000).

30
31 The higher mean exposure rate from the study by Krieger et al. (2005), relative to the studies by
32 Middendorf (1992b) and Spenser et al. (2000), may reflect the difficult terrain in which the study
33 was conducted as well as worker practices. As noted by Krieger et al. (2005, p. 8), the ... *rugged*
34 *terrain was uneven and slopes ranged from 10% to 50%*. As also noted by Krieger et al. (2005,
35 p. 7), the workers in this study were possibly contaminated by using their feet and legs to beat a
36 path through sprayed vegetation. In contrast, Forest Service crews in the study by Middendorf
37 (1992b) were required to walk only through untreated vegetation at all times —i.e., spraying to
38 the left or right or spraying from behind themselves. The higher exposure rates from the
39 Middendorf (1992b) study, relative to the Middendorf (1992a) study, probably reflect the type of
40 backpack application. The Middendorf (1992a) study involved basal stem applications, while
41 the study by Middendorf (1992b) involved directed foliar applications. In general, basal stem
42 applications should entail less worker exposure than foliar applications.

43
44 Regardless of the differences among the studies by Middendorf (1992b), Spenser et al. (2000),
45 and Krieger et al. (2005), these three Forest Service sponsored studies suggest that the standard
46 exposure rates used in most Forest Service risk assessments may not be appropriate for backpack

1 applications of triclopyr BEE. To further explore the magnitude of the differences, comparisons
2 may be made between the estimated daily doses—i.e., mg/kg bw/day rather than exposure rates
3 in mg/kg bw/day per lb handled—based on the standard methods used in Forest Service risk
4 assessments for worker exposure studies involving triclopyr and occupational exposure estimates
5 based on methods typically used by the U.S. EPA.

6
7 These comparisons are presented in Table 15, which is divided into three sections. The upper
8 section provides estimates of absorbed daily doses for workers based on standard methods used
9 in most Forest Service risk assessments. The estimated doses are based on an application rate of
10 1 lb/acre using the worker exposure rates given in upper section of Table 11 and standard
11 assumptions about the number of acres a worker will treat in a single day. The values for the
12 number of acres treated per day are given Attachments 1 and 2—i.e., Worksheet C01a for
13 backpack applications, Worksheet C01b for ground boom broadcast applications, and Worksheet
14 C01c for aerial applications.

15
16 The middle section of Table 15 provides the estimated absorbed doses for workers from the
17 available field studies. These studies include Middendorf (1992a,b), Spencer et al. (2000),
18 Krieger et al. (2005) as well as the study by Gosselin et al. (2005). Note that the comparison
19 given in this section of Table 15 provides gross estimates of absorbed doses in mg/kg bw/day
20 which are not normalized for either application rate or the amount of triclopyr applied by each
21 worker.

22
23 The study by Gosselin et al. (2005) involves both backpack (eight workers) and ground boom
24 applications (two workers) of Garlon 4. In this study, urine collection consisted of only a 22-
25 hour sample taken at the end of a 5-day workweek. Gosselin et al. (2005) corrected for the
26 incomplete collection of triclopyr using the pharmacokinetic data from the study by Carmichael
27 et al. (1989). Estimates of the mean absorbed doses for each of the 10 workers are presented in
28 Table 16. As summarized in Table 15, the estimated absorbed doses were 0.115 (0.024 to 0.552)
29 mg/kg bw for backpack workers and 0.200 (0.103 to 0.339) mg/kg bw for the boom spray
30 workers.

31
32 While the Gosselin et al. (2005) appears to be a well-conducted study and provides a reasonable
33 and detailed pharmacokinetic analysis, it does not specify the average amount of triclopyr
34 handled by workers or the application rate of triclopyr used in either the backpack or boom spray
35 applications, which is a major limitation. Moreover, Gosselin et al. (2005) do not indicate that
36 workers wore protective goggles, long sleeved shirts, or rubber gloves, all of which are required
37 in Forest Service applications. In the conduct of the current Forest Service risk assessment, an
38 email query was sent to the corresponding author of this study (gaetan.carrier@umontreal.ca);
39 however, no response was received. In the absence of information on the amounts handled or at
40 least the application rate used in the study, the estimates of absorbed doses reported by Gosselin
41 et al. (2005) are of limited use in assessing the worker exposure rates for backpack applications
42 used in most Forest Service risk assessments.

43
44 As discussed above, lower and upper bounds are not presented in Table 15 for Krieger et al.
45 (2005), because individual estimates of estimated doses are not reported. Nonetheless, the

1 average estimate of the absorbed dose given by Krieger et al. (2005) is 0.043 mg/kg bw, which is
2 virtually identical to the rate of 0.048 mg/kg bw from Spenser et al. (2000).

3
4 The last approach used in assessing the standard Forest Service exposure rates involves a
5 comparing those rates with worker exposure rates used by U.S. EPA's Office of Pesticide
6 Programs (U.S. EPA/OPP). Worker exposure assessments conducted by U.S. EPA/OPP are
7 typically based on the Pesticide Handler Exposure Database (PHED), Version 1.1. As discussed
8 in SERA (2007a, Section 3.2.2), PHED is a deposition-based approach to estimating worker
9 exposure. In this type of model, the exposure dose is estimated from air concentrations and skin
10 deposition monitoring data. Using these estimates, the absorbed dose can be calculated if
11 estimates are available on absorption rates for inhalation and dermal exposure.

12
13 When available, occupational exposure assessments made by U.S. EPA/OPP are compared
14 directly to occupational exposure assessments used in Forest Service risk assessments. For
15 triclopyr, however, U.S. EPA/OPP (1998a,b) does not provide occupational exposure
16 assessments for triclopyr based on the following rationale:

17
18 *Short-term and intermediate-term dermal and inhalation exposure*
19 *assessments are not required because there are no toxicological endpoints*
20 *of concern. At this time, no chronic risk assessment is required for handler*
21 *exposures to triclopyr, since none of the current handler exposure*
22 *scenarios is likely to result in chronic exposure.*

23 U.S. EPA/OPP 1998a, p. 25.

24
25 In the absence of a U.S. EPA/OPP exposure assessment, the general U.S. EPA/OPP methods are
26 used. As summarized in Table 17, general exposure rates, given as mg/lb handled, have been
27 developed from PHED for 37 different exposure scenarios (Keigwin 1988). For each scenario,
28 both dermal rates and inhalation rates are given. When exposure assessments are compared with
29 oral toxicity data, estimates of dermal absorption are used. In the current analysis, the first-order
30 dermal absorption rates for triclopyr BEE are taken from Carmichael et al. (1989). The
31 Carmichael et al. (1989) study is discussed in U.S. EPA/OPP (1998a, p. 16), and there is little
32 doubt that U.S. EPA/OPP would use this study in any occupational exposure assessment of
33 triclopyr BEE. For the comparisons developed in Table 15, the following scenarios are selected:
34 Scenario 34 (Liquid/open pour/backpack) for backpack applications, Scenario 13 (Groundboom
35 applications, open cab) for ground boom applications, and Scenario 07 (Aerial-fixed wing,
36 enclosed cockpit/liquid) for aerial applications. These scenarios are highlighted in Table 17 with
37 bold font. The specifics of the implementation of the PHED methods are detailed in custom
38 worksheets (specified in Table 15) that are included in the EXCEL workbook for triclopyr BEE
39 (Attachment 2).

40
41 As summarized in Table 15 of the current risk assessment, the exposure assessments based on
42 PHED are lower than the exposure assessments based on the standard Forest Service exposure
43 rates. Based on the upper bound estimates of exposures, the Forest Service rates are greater by
44 factors of about 2.7 for backpack workers, 30 for boom spray workers, and 26 for aerial spray
45 workers.

1 Overall, the comparisons in Table 15 suggest that the typical exposure scenarios used in Forest
2 Service risk assessments are more conservative than those based on PHED. Nonetheless, several
3 of the other worker exposure studies involving applications of triclopyr BEE (i.e., Spenser et al.
4 2000; Krieger et al. 2005; Gosselin et al. 2005) yield higher estimates of absorbed doses than
5 those that would typically be used in the Forest Service risk assessment.
6

7 The worker exposure rates used in the current Forest Service risk assessment are summarized in
8 Table 18. Of the studies by Middendorf (1992b), Spenser et al. (2000), and Krieger et al. (2005),
9 the study by Middendorf (1992b) appears to be most relevant. As discussed above, this study
10 involves relatively complete urine collections and, excluding the data from Site 3, application
11 conditions in the Middendorf (1992b) study are most representative of those likely to be made in
12 Forest Service programs. Consequently, the exposure rates from Middendorf (1992b) are used
13 explicitly for directed foliar applications of triclopyr BEE.
14

15 Studies from which to estimate worker exposure rates are not available for ground boom or aerial
16 applications of triclopyr BEE. As discussed above and summarized in Table 15, Gosselin et al.
17 (2005) report much higher absorbed doses in two workers involved in boom spray applications
18 of triclopyr BEE. Based on central estimates, the dose estimate from Gosselin et al. (2005)—i.e.,
19 0.2 mg/kg bw—is a factor of about 10 higher than the estimated dose based on standard Forest
20 Service exposure rates and application assumptions—i.e., 0.022 mg/kg bw. The study by
21 Gosselin et al. (2005), however, is not directly useful because it does not report the amount of
22 triclopyr that was applied or handled by the workers.
23

24 In the absence of additional information, exposure levels for workers involved in boom spray
25 and aerial applications of triclopyr BEE are assumed to be higher by the same ratio as that for
26 backpack workers, based on the data from Middendorf (1992b). As noted in Table 18, the ratio
27 of rates from Middendorf (1992b) to standard Forest Service rates for directed foliar spray are
28 approximately 1.9, 2.9, and 3.9, based on the central estimate, lower bound, and upper bound
29 values. These ratios are used to adjust rates for ground boom and aerial applications of triclopyr
30 BEE based on the standard rates for these application methods. For example, the central
31 estimate typically used for ground boom applications is 0.0002 mg/kg bw per lb handled. Based
32 on the Middendorf (1992b) study of backpack applications, the central estimate for triclopyr
33 BEE is about a factor of 1.9 higher than the standard Forest Service rate. Thus, for triclopyr
34 BEE, the central estimate for ground boom applications is taken as 0.00053 mg/kg bw per lb
35 handled [0.0002 mg/kg bw per lb handled x 1.9 ≈ 0.00038 mg/kg bw per lb handled]. All
36 adjusted values given in Table 18 are similarly calculated and rounded.
37

38 As discussed in Section 3.1.3.2.1, the first-order dermal absorption rate coefficients for triclopyr
39 BEE, 3.1×10^{-3} ($1.2 \times 10^{-3} - 8.1 \times 10^{-3}$) hour^{-1} , are about 3.5 times greater than those for triclopyr
40 acid, i.e., 8.8×10^{-4} ($3.0 \times 10^{-4} - 2.6 \times 10^{-3}$) hour^{-1} . This relationship suggests that exposures for
41 workers applying triclopyr TEA are likely to be less than those for workers applying triclopyr
42 BEE. While that lack of worker exposure studies involving triclopyr TEA adds uncertainty to
43 this risk assessment, the differences in dermal absorption rates (which are well documented for
44 triclopyr BEE) suggest that adjustments to the worker exposure rates for triclopyr TEA are
45 unnecessary. Consequently, for applications of triclopyr TEA, the standard worker exposure
46 rates used in most Forest Service risk assessments are maintained. The Abdelghani (1995) study

1 addresses worker exposure to triclopyr TEA (as Garlon 3A), but does not provide sufficient
2 information to estimate worker exposure rates, except to report that the maximum estimated
3 exposure for any worker was 0.00061 mg/kg bw. As summarized in Worksheet E01
4 (Attachment 1), this estimate is at the lower range of estimated exposures for workers applying
5 triclopyr TEA, in this risk assessment.

6 **3.2.2.1.2. Aquatic Applications**

7 The literature on triclopyr does not include data regarding workers exposed to aquatic
8 applications. There is, however, a study on worker exposure rates during aquatic applications of
9 2,4-D (Nigg and Stamper 1983). This study involves the application of a liquid formulation of
10 2,4-D by airboat handguns to control water hyacinths. The absorbed doses of 2,4-D were
11 assayed in four workers as total urinary elimination over a 24-hour period. The estimated
12 occupational exposure rates for the 2,4-D workers were 0.0009 (0.0004-0.002) mg/kg body
13 weight per lb handled.

14
15 To estimate worker exposure rates for triclopyr applications, the estimated occupational exposure
16 rates for the 2,4-D workers are used with the estimated amount of triclopyr handled. As
17 specified in Worksheets C01 of Attachment 3 (emergent applications), the amount handled is
18 calculated as the product of the application rate (lbs a.e./acre) and the number of acres of surface
19 water to be treated. For this exposure scenario, the unit application rate of 1 lb a.e./acre is used,
20 and the worker is assumed to apply triclopyr to a 10-acre area. These inputs can be modified in
21 Worksheet A01 of Attachment 3. The consequences of using different application rates and
22 treating different surface areas are discussed in the risk characterization. A similar approach is
23 taken for submergent applications (Attachment 4, Worksheet C01) except that the amount
24 handled is based on the target concentration and the volume of water to be treated.

25
26 Using 2,4-D data to estimate worker exposures to triclopyr adds uncertainty to the risk
27 assessment. In the absence of a worker exposure study involving aquatic applications of
28 triclopyr, there is no alternative approach to reduce this uncertainty.

29 **3.2.2.2. Accidental Exposures**

30 Although typical occupational exposures are likely to involve multiple routes of exposure (i.e.,
31 oral, dermal, and inhalation), dermal exposure is generally the predominant route for herbicide
32 applicators (Ecobichon 1998; van Hemmen 1992). Typical multi-route exposures are
33 encompassed by the methods used in Section 3.2.2.1 on general exposures. Accidental
34 exposures, on the other hand, are most likely to involve splashing a solution of herbicide into the
35 eyes and may also involve various dermal exposure scenarios.

36
37 Quantitative exposure scenarios for ocular exposures are not developed in this or other Forest
38 Service risk assessments. As discussed in Section 3.1.11.3 (Ocular Effects), ocular exposures to
39 some formulations of triclopyr, particularly formulations of triclopyr TEA, may cause moderate
40 to severe eye damage. This effect is considered qualitatively in the risk characterization for
41 workers (Section 3.4.2).

42
43 Accidental dermal exposure to triclopyr is considered quantitatively in this risk assessment. The
44 two types of modeled dermal exposure include direct contact with a pesticide solution and
45 accidental spills of the pesticide onto the surface of the skin. Two exposure scenarios are

1 developed for each of the two types of dermal exposure, and the estimated absorbed dose for
2 each scenario is expressed in units of mg chemical/kg body weight. Both sets of exposure
3 scenarios are summarized in Worksheet E01 in the attachments that accompany this risk
4 assessment. Worksheet E01 references other worksheets which provide detailed calculations.
5

6 Exposure scenarios involving direct contact with triclopyr solutions are characterized either by
7 immersion of the hands in a field solution for 1 hour or wearing pesticide contaminated gloves
8 for 1 hour. The assumption that the hands or any other part of a worker's body will be immersed
9 in a chemical solution for a prolonged period of time may seem unreasonable; however, it is
10 quite plausible that the gloves or other articles of clothing worn by a worker may become
11 contaminated with a pesticide. For these exposure scenarios, the key assumption is that wearing
12 gloves grossly contaminated with a chemical solution is equivalent to immersing the hands in the
13 pesticide solution. In both cases, the chemical concentration in contact with the skin and the
14 resulting dermal absorption rate are essentially constant.
15

16 For both scenarios (hand immersion and contaminated gloves), the assumption of zero-order
17 absorption kinetics is appropriate—i.e., because the concentration of the pesticide in contact with
18 the skin is constant, or nearly so, the rate of absorption will be constant. For these types of
19 exposures, the rate of absorption is estimated, based on the dermal permeability coefficient (K_p).
20 Details regarding the derivation of the K_p value for triclopyr TEA and triclopyr BEE are
21 provided in 3.1.3.2.2. The amount of the pesticide absorbed per unit time depends directly on
22 the concentration of the chemical in solution. As discussed in Section 2.4.1, the current risk
23 assessment uses an application volume of 25 gallons/acre with a range of 4-40 gallons/acre,
24 which encompasses the potential range of application rates to be used in ground and aerial
25 applications.
26

27 Exposure scenarios involving chemical spills onto the skin are characterized by a spill on to the
28 lower legs as well as a spill on to the hands, and both scenarios are based on the assumption that
29 a certain amount of the chemical adheres to the skin. The absorbed dose is then calculated as the
30 product of the amount of chemical on the surface of the skin (i.e., the amount of liquid per unit
31 surface area multiplied by the surface area of the skin over which the spill occurs and the
32 chemical concentration in the liquid), the first-order absorption rate coefficient (k_a), and the
33 duration of exposure. Estimates of the first-order absorption rate coefficients are discussed in the
34 hazard identification for both triclopyr BEE (Section 3.1.3.2.1.1) and triclopyr TEA (Section
35 3.1.3.2.1.2). As discussed in these sections, the estimated first-order dermal absorption rate
36 coefficient for triclopyr BEE is well supported by Carmichael et al. (1989).
37

38 Numerous exposure scenarios could be developed for direct contact or accidental spills by
39 varying the amount or concentration of the chemical on, or in contact with, the skin surface, the
40 surface area of the affected skin, and the duration of exposure. The impact of these variables on
41 the risk assessment is discussed further in the risk characterization (Section 3.4.2).

1 **3.2.3. General Public**

2 **3.2.3.1. General Considerations**

3 **3.2.3.1.1. Likelihood and Magnitude of Exposure**

4 The chances that members of the general public will be exposed to triclopyr in Forest Service
5 applications are highly variable. In some Forest Service applications, triclopyr could be applied
6 in recreational areas, including campgrounds, picnic areas, and trails. Because of the
7 conservative exposure assumptions used in the current risk assessment, neither the probability of
8 exposure nor the number of individuals who might be exposed has a substantial impact on the
9 risk characterization presented in Section 3.4. As noted in Section 1 (Introduction) and detailed
10 in SERA (2007a, Section 1.2.2.2), the exposure assessments developed in this risk assessment
11 are based on *Extreme Values* rather than a single value. Extreme value exposure assessments, as
12 the name implies, bracket the most plausible estimate of exposure (referred to statistically as the
13 central or maximum likelihood estimate) with lower and upper bounds of credible exposure
14 levels.

15
16 This Extreme Value approach is essentially an elaboration on the concept of the *Most Exposed*
17 *Individual* (MEI), sometime referred to as the *Maximum Exposed Individual*. As this name
18 implies, exposure assessments that use the MEI approach attempt to characterize the extreme but
19 still plausible upper limit on exposure. This common approach to exposure assessment is used
20 by U. S. EPA, other government agencies, and the International Commission on Radiological
21 Protection (e.g., ATSDR 2002; ICRP 2005; Payne-Sturges et al. 2004). In the current risk
22 assessment, all upper bounds on exposure are intended to encompass exposures to the MEI.

23
24 In addition to this upper bound MEI value, the Extreme Value approach used in this risk
25 assessment provides a central estimate of exposure as well as a lower bound on exposure.
26 Although not germane to assessing the upper bound risk, using the central estimate and
27 especially the lower bound estimate is not intended to lessen concern. To the contrary, the
28 central and lower estimates of exposure are used to assess the prospect of mitigation—e.g.,
29 protective measures to limit exposure. If lower bound exposure estimates exceed a level of
30 concern (which is not the case in the current risk assessment), there is strong indication that the
31 pesticide cannot be used in a manner that will lead to acceptable risk.

32
33 In addition to concern for the most exposed individual, there is concern for individuals who may
34 be more sensitive than most members of the general population to triclopyr exposure. This
35 concern is considered in the dose-response assessment (Section 3.3) which bases exposures on
36 the most sensitive endpoint in the most sensitive species and uses an uncertainty factor for
37 sensitive individuals. Atypical sensitivities—i.e., special conditions that might increase an
38 individual’s sensitivity to a particular agent—are also considered separately in the risk
39 characterization (Section 3.4.4).

40 **3.2.3.1.2. Summary of Assessments**

41 The exposure scenarios developed for the general public are summarized in Worksheet E03 of
42 the EXCEL workbook that accompanies this risk assessment. As with the worker exposure
43 scenarios, details about the assumptions and calculations used in these assessments are given in
44 the detailed calculation worksheets in the EXCEL workbook (Worksheets D01–D11).

1
2 As summarized in Worksheet E03, the kinds of exposure scenarios developed for the general
3 public include acute accidental, acute non-accidental, and longer-term or chronic exposures. The
4 accidental exposure scenarios assume that an individual is exposed to the compound of concern
5 either during or shortly after its application. As well, the nature of the accidental exposures is
6 intentionally extreme. Non-accidental exposures involve dermal contact with contaminated
7 vegetation as well as the consumption of contaminated fruit, vegetation, water, or fish. The
8 longer-term or chronic exposure scenarios parallel the acute exposure scenarios for the
9 consumption of contaminated fruit, water, or fish. All of the non-accidental exposure scenarios
10 are based on levels of exposure to be expected in the routine uses of triclopyr at a unit
11 application rate of 1 lb a.e./acre. The upper bounds of the exposure estimates for the non-
12 accidental scenarios involve conservative assumptions intended to reflect exposure for the MEI
13 (*Most Exposed Individual*). The impact of lower or higher application rates on the risk
14 characterization is discussed in Section 3.4.

15 **3.2.3.2. Direct Spray**

16 Direct sprays involving ground applications are modeled similarly to accidental spills for
17 workers (Section 3.2.2.2). In other words, the scenarios assume that an individual is sprayed
18 with a chemical solution, some of which remains on the skin and is absorbed by first-order
19 kinetics. Two direct spray scenarios are included in this risk assessment: one for a young child
20 (D01a) and the other for a young woman (D01b).

21
22 The exposure scenario involving the young child assumes that a naked child is sprayed directly
23 with a chemical during a ground broadcast application and is completely covered (i.e., 100% of
24 the surface area of the body is exposed). This exposure scenario is intentionally extreme. As
25 discussed in Section 3.2.3.1.1, the upper limits of this exposure scenario are intended to represent
26 the *Extreme Value* upper limits of exposure for the *Most Exposed Individual* (MEI).

27
28 The exposure scenario involving the young woman (Worksheet D01b) is somewhat less extreme,
29 but more credible. In this scenario, it is assumed that the woman is accidentally sprayed over the
30 feet and lower legs. The preference for using a young woman rather than an adult male in many
31 of the exposure assessments relates to concerns for both the *Most Exposed Individual* (MEI) as
32 well as the most sensitive individual. Based on general allometric considerations, the smaller the
33 individual, the greater will be the chemical doses per unit body weight (e.g., Boxenbaum and
34 D'Souza, 1990). In general, the body size of a female is smaller than that of males. Thus, in
35 direct spray exposure scenarios, females are subject to somewhat higher doses than males. More
36 significantly, reproductive effects are a major concern in all Forest Service risk assessments.
37 Consequently, exposure levels for a young woman of reproductive age are used in order to better
38 assess the potential for adverse effects in the population at risk from potential reproductive
39 effects—i.e., the most exposed and the most sensitive individual.

40
41 For this exposure scenario, assumptions are made regarding the surface area of the skin and the
42 body weight of the individual, as detailed in Worksheet A03. The rationale for and sources of
43 the specific values used in these and other exposure scenarios is given in the documentation for
44 the worksheets (SERA 2009a) as well as the documentation for the preparation of Forest Service
45 risk assessments (SERA 2007a). The first-order absorption dermal absorption rates are identical
46 to those used in the similar worker exposure scenarios (Section 3.2.2.2).

1 **3.2.3.3. Dermal Exposure from Contaminated Vegetation**

2 The exposure scenario involving contaminated vegetation assumes that the herbicide is sprayed
3 at a given application rate and that a young woman comes in contact with the sprayed vegetation
4 or with other contaminated surfaces on the same day (Worksheet D02). This exposure scenario
5 depends on estimates of dislodgeable residue (the estimated amount of the chemical which could
6 be released from the vegetation, expressed in units of pesticide mass/surface area of vegetation),
7 and dermal transfer rates (i.e., the rate at which the chemical is transferred from the contaminated
8 vegetation to the surface of the skin). Dermal transfer rates are reasonably consistent for a
9 number of pesticides (Durkin et al.1995).

10
11 Dislodgeable residues may vary according to the pesticide, the formulation, and the site-specific
12 conditions. In the absence of chemical-specific data, dislodgeable residues are taken as 10% of
13 the nominal application rate in most Forest Service risk assessments. A registrant submitted
14 study (McCormick and Robb 2000, MRID 45249901) assayed dislodgeable residues of triclopyr
15 after applications of Grandstand, a triclopyr TEA formulation, at a nominal application rate of
16 0.375 lb a.e./acre ($\approx 4.203 \mu\text{g}/\text{cm}^2$ [1 lb a.e./acre = $11.21 \mu\text{g}/\text{cm}^2$]). On the day of application,
17 average dislodgeable residues ranged from 0.382 to $0.774 \mu\text{g}/\text{cm}^2$, which is equivalent to about
18 0.09 to 0.18 of the nominal application rate. Based on this study, the proportion of triclopyr
19 available as dislodgeable residue on vegetation is taken as 0.15 (0.1 to 0.2).

20
21 This exposure scenario assumes both a contact period of 1hour and that the chemical is not
22 effectively removed by washing within 24 hours of exposure. Other estimates used in this
23 exposure scenario involve estimates of body weight, skin surface area, and first-order dermal
24 absorption rates. The specific values for each of these estimates are provided in Worksheet D02
25 together with the references for each value.

26 **3.2.3.4. Contaminated Water**

27 **3.2.3.4.1. Accidental Spill**

28 **3.2.3.4.1.1. Triclopyr**

29 The accidental spill scenario assumes that a young child consumes contaminated water shortly
30 after an accidental spill of a field solution into a small pond. The specifics of this scenario are
31 given in Worksheet D05. Because this scenario is based on the assumption that exposure occurs
32 shortly after the spill, no dissipation or degradation is considered. Since this exposure scenario is
33 based on assumptions that are somewhat arbitrary and highly variable, it may overestimate
34 exposure. The actual chemical concentrations in the water will vary according to the amount of
35 compound spilled, the size of the water body into which the chemical is spilled, the time at
36 which water consumption occurs relative to the time of the spill, and the amount of contaminated
37 water consumption. To reflect the variability inherent in this exposure scenario, a spill volume
38 of 100 gallons (range of 20-200 gallons) is used to reflect plausible spill events. The triclopyr
39 concentrations in the field solution are also varied to reflect the plausible range of concentrations
40 in field solutions—i.e., the material that might be spilled—using the same values as in the
41 accidental exposure scenarios for workers (Section 3.2.2.2). Based on these assumptions, the
42 estimated concentration of triclopyr in a small pond ranges from about 0.23 to about 18 mg
43 a.e./L, with a central estimate of about 1.6 mg a.e./L (Worksheet D05).

3.2.3.4.1.2. TCP

As discussed in the previous subsection, accidental spills of triclopyr will result in peak concentrations of about 1.6 (0.23 to 18) mg a.e./L. While no data are available on the concentrations of TCP in water following an accidental spill of triclopyr, ample data are available on concentrations of TCP in water following aquatic applications of triclopyr. These data are clearly relevant to an assessment of TCP exposures following an accidental spill because the intentional application of triclopyr to water is essentially equivalent to a spill of triclopyr into water. The monitoring studies from the open literature as well as studies submitted to the U.S. EPA have been reviewed in some detail (Cessna et al. 2002; Ganapathy 1997; Knuteson 1999; Petty et al. 2003).

The most relevant data in terms of assessing the plausibility of significant exposures to TCP following an accidental spill are from a series of pond studies in which triclopyr was applied at target concentrations of 2.5 mg a.e./L and triclopyr and TCP concentrations in pond water and sediment were assayed for 42 days beginning immediately after application and 42 days later. These studies are reviewed by Petty et al. (2003), published in the open literature, and by Knuteson (1999), an unpublished registrant submission provided by Dow AgroSciences during the preparation of the current risk assessment and which contains the most detailed data. Table 19 summarizes the maximum triclopyr and TCP concentrations monitored in pond water and sediment. As shown in Table 19, the average of the monitored maximum concentrations of triclopyr in the seven ponds was 2.5 mg a.e./L, identical to the intended application rate. The maximum monitored concentration of TCP was only 0.02 mg/L. Based on the maximum monitored concentrations of triclopyr and TCP at each of the ponds, the maximum concentration of TCP was below the maximum concentration of triclopyr by factors of about 235 (137 to 586). As detailed in Knuteson (1999), the maximum concentrations of triclopyr and TCP occurred at different times, as would be expected. Thus, the TCP concentrations can be considered reasonable *worst-case* estimates based on field data representative of likely Forest Service applications.

The data from Petty et al. (2003) are consistent with an unpublished registrant-submitted study on triclopyr applications to rice in which the concentration of TCP was ...*typically two to three orders of magnitude less than that of triclopyr* (Cessna et al. 2002, p. 26). As discussed below (Section 3.2.3.4.5), the observations from the pond and rice studies are consistent with the much lower concentrations of TCP, relative to triclopyr, modeled in numerous Gleams-Driver simulations, which yield estimates of triclopyr in surface water that are consistent with monitoring studies.

For the current risk assessment, the estimated peak concentrations of TCP in pond water following an accidental spill are taken as the range of nominal peak concentrations for triclopyr – i.e., 1.6 (0.23 to 18) mg a.e./L as detailed in the previous subsection – divided by the factors of 235 (137 to 586). As a conservative approximation, the maximum concentration of TCP is calculated by dividing the upper bound concentration of triclopyr by the lower bound adjustment factor of 137. The minimum concentration of TCP is based on the minimum concentration of triclopyr divided by the upper bound adjustment factor. The concentrations of TCP in a pond following an accidental spill are estimated at about 0.0077 (0.0004 to 0.13) mg/L. Details of these calculations are given in Worksheet B04b of Attachment 5.

3.2.3.4.2. Accidental Direct Spray of or Drift to a Pond or Stream

Scenarios involving direct spray or drift are less severe but more plausible than the accidental spill scenario described in the previous subsection. The concentrations of triclopyr in a small pond (Worksheet 10a) and a small stream (Worksheet D10b) are based on standard estimates of drift adapted from AgDrift for four application methods: aerial, high boom ground broadcast, low boom ground broadcast and backpack applications. As discussed in SERA (2010a), on Tier 1 analyses for aerial and ground broadcast applications using AgDRIFT Version 2.0.05. AgDRIFT permits very detailed modeling of drift based on the chemical and physical properties of the applied product, the configuration of the aircraft, wind speed, and temperature for aerial applications. The generic estimates used in the current risk assessment are intended to be conservative, and more refined estimates of drift would be appropriate in any site-specific application,

If a 1-meter deep pond is directly sprayed with triclopyr at a unit application rate of 1.0 lb a.e./acre, the peak concentration in the pond would be about 0.11 mg/L, equivalent to 110 µg/L or 110 ppb (Worksheet D10a). This concentration is a factor of about 164 below 18 mg a.e./L, the upper bound of the central estimate of the concentration in pond water after an accidental spill (Section 3.2.3.4.1, Worksheets D05). Based on the Tier 1 estimates of drift, triclopyr concentrations in a small pond contaminated by drift would range from about 0.000035 mg/L (35 part per trillion) to 0.025 mg/L (25 part per billion), depending on the application method and the distance of the pond from the treated site.

For the stream scenario, the resulting water concentrations depend on the surface area of the stream and the rate of water flow in the stream. The stream modeled using Gleams-Driver (Section 3.2.3.4.3) is about 6 feet wide (1.82 meters), and it is assumed that the pesticide is applied along a 1038 foot (316.38 meters) length of the stream with a flow rate of 710,000 L/day. Using these values, the concentration in stream water after a direct spray is estimated at about 0.09 mg/L (90 parts per billion). Much lower concentrations, ranging from about 0.00003 mg/L (30 part per trillion) to 0.02 mg/L (20 parts per billion) are estimated based on drift at distances of 25-900 feet (Worksheet D10b).

3.2.3.4.3. Gleams-Driver Modeling

The Forest Service developed a program, Gleams-Driver, to estimate expected peak and longer-term pesticide concentrations in surface water. Gleams-Driver serves as a preprocessor and postprocessor for GLEAMS (Knisel and Davis 2000). GLEAMS (Groundwater Loading Effects of Agricultural Management Systems) is a field scale model developed by the USDA/ARS and has been used for many years in Forest Service and other USDA risk assessments. Gleams-Driver offers the option of conducting general exposure assessments using site-specific weather files from Cligen, a climate generator program developed and maintained by the USDA Agricultural Research Service (<http://horizon.nserl.purdue.edu/Cligen>). Details concerning the use of Gleams-Driver are given in SERA (2007b). Gleams-Driver is used in the current risk assessment to model concentrations of triclopyr BEE, triclopyr acid, and TCP (3,5,6-trichloro-2-pyridinol) in a small stream and small pond.

3.2.3.4.3.1. Inputs to Gleams-Driver

The generic site parameters used in the Gleams-Driver runs are summarized in Table 20, and additional details are available in the documentation for Gleams-Driver (SERA 2007b). For each

1 site modeled, simulations were conducted using clay (high runoff, low leaching potential), loam
2 (moderate runoff and leaching potential), and sand (low runoff, high leaching potential) soil
3 textures. Neither GLEAMS nor PRZM/EXAMS (discussed further in Section 3.2.3.4.4)
4 explicitly accommodate buffers. Consequently, all Gleams-Driver simulations as well as the
5 PRZM/EXAMS modeling discussed in Section 3.2.3.4.4 do not incorporate buffers.

6
7 The locations of the generic sites selected for modeling include a total of nine sites, as
8 summarized in Table 21. As discussed in SERA (2007b), these locations are standard sites for
9 the application of Gleams-Driver in Forest Service risk assessments and are intended to represent
10 combinations of precipitation (dry, average, and wet) and temperature (hot, temperate, and cool).
11 For each site, Gleams-Driver was used to simulate 100 applications at a unit application rate of
12 1 lb/acre, and each of the simulations was followed for a period of about 1½ years after
13 application.

14
15 Two formulations of triclopyr are represented in the GLEAMS-Driver simulations. One consists
16 of application of triclopyr BEE with sequential degradation products of triclopyr acid and TCP
17 (Garlon 4). The second is application of a salt formulation which instantaneously transforms
18 triclopyr TEA to triclopyr acid upon mixing, with a single degradation product TCP (Garlon 3).
19 Neither triethylamine (formed by the dissociation of triclopyr TEA) nor butoxyethanol (formed
20 by the hydrolysis of triclopyr BEE) are modeled. This approach is consistent with the one taken
21 by in U.S. EPA/OPP (1998a, 2009a):

22
23 *Triclopyr BEE rapidly hydrolyzes in the environment to the triclopyr*
24 *acid/anion and butoxyethanol. Both triethanolamine [sic] and*
25 *butoxyethanol are also rapidly dissipated by microbial degradation, and*
26 *thus are not being evaluated any further in this assessment.*

27 U.S. EPA/OPP 2009a, p. 20.
28

29 In addition, the EPA assessments, U.S. EPA/OPP (1998a, 2009a), do not quantitatively consider
30 ambient water levels of triclopyr BEE and TCP. Triclopyr BEE will rapidly degrade to triclopyr
31 and butoxyethanol. Nonetheless, potential levels of triclopyr BEE in ambient water are
32 quantitatively considered in the current risk assessment. While triclopyr BEE (distinct from
33 triclopyr) is not a concern in the human health risk assessment, triclopyr BEE is much more toxic
34 than triclopyr to aquatic organisms. Accordingly, the current risk assessment makes a clear
35 distinction between plausible exposures to triclopyr BEE and triclopyr acid, as discussed in
36 further detail in the ecological risk assessment (Section 4). The EPA does not model TCP
37 because it does not consider TCP to be an agent of toxicological concern (U.S. EPA/OPP 2009a).
38 As discussed further in Section 4.1.3 (hazard identification for aquatic organism), the current risk
39 assessment considers TCP to be an agent of concern for some groups of aquatic organisms.
40 Consequently, the formation of TCP in surface water following applications of triclopyr BEE
41 and triclopyr TEA is considered quantitatively in the current risk assessment.

42
43 Table 22 summarizes the chemical-specific values used in Gleams-Driver simulations. For the
44 most part, the chemical properties used in the Gleams-Driver simulations are taken from U.S.
45 EPA/OPP (2009a). The EPA modeling efforts are discussed below (Section 3.2.3.4.4). In the
46 current risk assessment, most of the model input values are based on the environmental fate

1 studies submitted to the EPA by registrants as well as standard values for GLEAMS modeling
2 recommended by Knisel and Davis (2000). The notes to Table 22 indicate the sources of the
3 chemical-specific values used in the GLEAMS modeling.
4

5 Some of the chemical specific parameters used in Gleams-Driver modeling are based on
6 distributions rather than single values. This approach differs from the approach used in the
7 modeling done by U.S. EPA/OPP (2009a). As summarized in Table 1, soil K_{oc} and sediment K_d
8 values for triclopyr are highly variable, which is a common characteristic for many pesticides. In
9 the Gleams-Driver simulations, the values for soil K_{oc} and sediment K_d are represented by the
10 triangular distributions based on the central estimates and ranges of values given in Table 22.
11 The reported foliar half-lives for triclopyr BEE and triclopyr acid are also highly variable and are
12 modeled with a uniform distribution. No information is available on the foliar half-life of TCP,
13 which is assumed to be identical to that of triclopyr.
14

15 As also summarized in Table 1, the amount of information on the soil half-life of triclopyr BEE,
16 triclopyr acid, and TCP is substantial. All of the information on triclopyr BEE indicates that it is
17 hydrolyzed rapidly in soil. As noted in Footnote 3 in Table 22, the selection of a half-life of 0.2
18 days for triclopyr BEE is conservative, in that much more rapid degradation rates are reported in
19 U.S. EPA/OPP (2009a). The value of 0.2 days is probably a substantial overestimate of the soil
20 half-life of triclopyr BEE. As a conservative assumption—i.e., an assumption that will tend to
21 increase the modeled concentrations of triclopyr BEE in surface water—the soil half-life of 0.2
22 days is maintained as a constant. While the reported half-lives of triclopyr acid and TCP are less
23 variable than the corresponding values for soil K_{oc} and sediment K_d , the soil half-lives for
24 triclopyr acid and TCP are also modeled using a triangular distribution. As summarized in
25 Table 19, the sediment monitoring data from Petty et al. (2003), suggest that the estimates of
26 sediment binding for TCP based on K_{oc} and the assumption of 1% organic carbon will
27 substantially underestimate the binding of TCP to pond sediment. Consequently, the
28 concentrations of TCP in sediment and pond water (Table 19) are used to estimate K_d values of
29 7.5 (5 to 11.3) for TCP in pond sediment.
30

31 **3.2.3.4.3.2. Results from Gleams-Driver**

32 Table 23 summarizes the results for the Gleams-Driver runs as well as other modeling efforts
33 and monitoring data, discussed further in the following subsections. Details of the results for the
34 Gleams-Driver runs are provided in Appendices 9 through 13, as specified in Table 23. Note
35 that all results from the Gleams-Driver runs are expressed as the median value with approximate
36 95% empirical limits. In other words, the two extreme lower and upper values from the 100
37 simulations at each site are dropped, and the lowest and highest remaining values are used for the
38 lower and upper bound estimates reported in Table 23 as well as the more detailed values
39 reported in Appendices 9 through 13.
40

41 In all sets of simulations – i.e., with each simulation consisting of 9 locations with 3 soil textures
42 per location – the expected concentrations of triclopyr BEE, triclopyr, and TCP in surface water
43 were zero even at the upper bounds for locations with little rainfall (Table 21). This is to be
44 expected and is a common finding in Gleams-Driver modeling of pesticides. GLEAMS tracks
45 the movement of pesticides in a field due to precipitation and subsequent transport of the
46 pesticide in sediment, runoff, and percolation, all of which are a function of water flow. If there

1 is no water flow, GLEAMS will not predict offsite losses of the pesticide. For arid or at least
2 relatively dry regions, any substantial contamination of surface would most likely be due to drift,
3 as discussed in Section 3.2.3.4.2.

4
5 Even in locations with moderate to heavy rainfall, many individual simulations – i.e., sets of 100
6 Gleams-Driver runs at a specific location with a specific soil texture – lead to lower bound
7 estimates and sometimes central estimates of concentrations in surface water that are zero or
8 nearly so. This again is a common pattern in Gleams-Driver simulations and reflects years with
9 low to moderate rainfall. For example, Appendix 10, Table A10-7 summarizes the results for
10 estimates of triclopyr acid in a small pond following the application of a triclopyr BEE
11 formulation at an application rate of 1 lb a.e./acre. For dry locations, the central and lower
12 bound estimates of triclopyr in surface water for all three locations are zero. This is also the case
13 for sites with average rainfall and sand soil textures as well as sites with loam soil textures and
14 cool temperatures. Even in locations with relatively high rainfall, the lower bounds of the
15 estimated concentrations of triclopyr in a small pond are zero for all sites with moderate
16 temperatures as well as sites with warm temperatures and loamy or sand soil textures. In other
17 words, over the course of the 100 simulations conducted for each location and soil type, at least
18 three years are sufficiently dry to yield empirical 95% lower bounds of zero for the concentration
19 of the pesticide in the pond. Lastly, it is worth noting that GLEAMS outputs pesticide losses in a
20 text rather than binary format. While scientific notation is sometimes used, zero values are
21 typically given in fixed decimal notation out to six places – i.e., “0.000000”. As discussed
22 further in Section 3.2.3.4.6, this leads to some instability and apparently erratic estimates of
23 lower bound concentrations that need to be addressed in selection lower bound non-zero
24 estimates of concentrations of chemicals in surface water from Gleams-Driver simulations.

25
26 Median and upper bound values are much easier to interpret. For comparisons among the three
27 compounds modeled in the Gleams-Driver – i.e., triclopyr BEE, triclopyr acid, and TCP – the
28 discussion will focus on the more stable median values. As would be expected from the rapid
29 hydrolysis of triclopyr BEE, concentrations of triclopyr BEE are much less than those for
30 triclopyr acid. Median peak concentrations of triclopyr span a narrow range of about 3 to 4 µg/L
31 with somewhat higher concentrations in ponds relative to streams and somewhat higher
32 concentrations following applications of triclopyr TEA compared to triclopyr BEE. Both of
33 these patterns are intuitive. Triclopyr TEA is assumed to instantaneously convert to triclopyr
34 acid while peak concentrations of triclopyr acid following triclopyr BEE applications are lower
35 because of the short but still finite time required for triclopyr BEE to degrade to triclopyr acid.
36 Concentrations of triclopyr acid in ponds are estimated to be higher than those in streams
37 because stream water is turned over daily whereas pond water is not (except in possible but
38 highly unlikely cases of extremely high rainfall).

39
40 Peak concentrations of triclopyr BEE in ponds are below those for triclopyr acid by a factor of
41 about 70 [$3.34 \mu\text{g/L} \div 0.047 \mu\text{g/L} \approx 71.06$]. In streams, however, the peak concentrations of
42 triclopyr BEE are below those for triclopyr acid by a factor of only about 6.8 [$2.80 \mu\text{g/L} \div 0.41$
43 $\mu\text{g/L} \approx 6.829$]. This difference primarily reflects the much higher concentrations of triclopyr
44 BEE modeled for streams relative to ponds – i.e. a factor of about 8.72 [$0.41 \mu\text{g/L}$ in streams \div
45 $0.047 \mu\text{g/L}$ in ponds].

1 As discussed further in Section 3.2.3.4.5, the available monitoring studies (Norris et al. 1987;
2 Smith and McCormack 1988) on triclopyr are sparse but they do suggest that the estimates of the
3 concentrations of triclopyr in streams from Gleams-Driver are plausible. No monitoring studies,
4 however, report detections of triclopyr BEE in streams. The monitoring study by Norris et al.
5 (1987) does not specify the form of triclopyr that was used. The study by Smith and
6 McCormack (1988) did involve an application of triclopyr BEE. In Table 1 of Smith and
7 McCormack (1988, p. 106), concentrations of triclopyr in stream water are reported at about 9 to
8 48 ppb ($\mu\text{g/L}$) following an application of triclopyr at 1.9 kg a.e./ha (≈ 1.7 lb a.e./acre). The
9 reported concentrations, normalized to a 1 lb a.e./acre application rate, are about 5.3 to 20 $\mu\text{g/L}$
10 in the area with a buffer zone. While not explicitly stated in the publication, the concentrations
11 reported by Smith and McCormack (1988) are almost certainly in units of $\mu\text{g a.e./L}$ and probably
12 reflect triclopyr acid rather than triclopyr BEE.
13

14 While the reasonableness of the high peak concentrations of triclopyr BEE modeled in streams
15 relative to ponds cannot be assessed without monitoring studies, these high peak concentrations
16 in streams do not have a substantial impact on the risk assessment. As discussed further in
17 Section 3.2.3.4.6, the peak modeled concentrations of triclopyr BEE in streams are close to the
18 concentrations in streams that might be expected due to drift. Thus, even if the peak stream
19 concentrations were discounted, the upper bound concentrations of triclopyr BEE in surface
20 water would be adjusted to consider drift.
21

22 Longer-term concentrations of triclopyr modeled in surface water are about a factor of two below
23 peak concentrations for ponds and a factor of about 70 below peak concentrations for streams.
24 These comparisons are based on the median values but differences based on upper bound values
25 are only somewhat less. This difference in the relationship between peak and longer term
26 concentrations in ponds and streams is intuitive and relates to the daily water replacement in
27 streams as discussed above.
28

29 Longer-term water concentrations of triclopyr BEE in surface water are extremely low. As
30 summarized in Table 23, the peak concentrations are less than 0.1 ppb with central estimates of
31 longer-term concentrations in surface water of about central estimates at or below 2 ppt (i.e.,
32 0.0018 $\mu\text{g/L}$ or 0.0000018 mg/L). These concentrations are considered in this risk assessment
33 but are insubstantial.
34

35 Concentrations of TCP (3,5,6-trichloro-2-pyridinol) are substantially below those of triclopyr for
36 peak and longer-term exposures. Based on median peak concentrations, TCP levels are below
37 those of triclopyr by factors of about 3 to 10. Based on longer-term concentrations, the
38 differences are greater with TCP concentrations being lower than those for triclopyr by factors of
39 about 40 in ponds. The pattern in streams, however, is different. For triclopyr, the longer-term
40 concentrations are substantially higher in ponds than in streams. For TCP, there are no
41 substantial differences between pond and streams concentrations.
42

43 The plausibility of the modeled concentrations of TCP is difficult to assess. As discussed in
44 Section 3.2.3.4.6.2 (Aquatic Applications), there is a substantial body of monitoring data on
45 concentrations of triclopyr and TCP in surface water following aquatic applications and these
46 data are used directly in the exposure assessment for aquatic applications to emergent vegetation.

1 Monitoring data on TCP in surface water following terrestrial applications of triclopyr, however,
2 are limited.

3
4 Table 26 (discussed further in Section 3.2.3.4.6) provides a summary of the water contamination
5 rates used in the current risk assessment. The bottom part of this table provides rates for
6 triclopyr and TCP based on monitoring data following aquatic applications. Based on the
7 monitoring data from aquatic applications, the ratio of the peak concentration of triclopyr to the
8 peak concentration of TCP is about 3.3 [0.18 mg triclopyr/L ÷ 0.055 TCP/L] and the ratio of the
9 longer-term concentrations is about 1.2 [0.0059 mg triclopyr/L ÷ 0.005 TCP/L]. As summarized
10 in Table 23 (Gleams-Driver modeling), the ratios of triclopyr to TCP based on the central
11 estimates of modeled peak concentrations are about 8 for ponds [3.34 ppb ÷ 0.42 ppb] and 3.2 for
12 streams [2.8 ppb ÷ 0.88 ppb]. Based on the longer-term modeled concentrations, the ratios of
13 triclopyr to TCP are about 33 for ponds [1.37 ppb ÷ 0.041 ppb] and 1.3 for streams [0.037 ppb ÷
14 0.0293 ppb]. The Gleams-Driver comparisons are all based on triclopyr BEE modeling but the
15 values for triclopyr TEA are very similar.

16 **3.2.3.4.4. Other Modeling Efforts**

17 To estimate pesticide concentrations in ambient water, the U.S. EPA typically uses either Tier 1
18 screening models such as GENEEC or PRZM/EXAMS, a more refined Tier 2 modeling system.
19 GENEEC was used in two assessments by U.S. EPA/OPP, the RED for triclopyr (U.S. EPA/OPP
20 1998a) and an ecological risk assessment of triclopyr (U.S. EPA/OPP 2004). PRZM/EXAMS
21 was used in U.S. EPA/OPP's recent analysis of risks to the California Red-legged Frog (U.S.
22 EPA/OPP 2009a). As a Tier 1 model, GENEEC is intended to be very conservative—i.e., more
23 likely to overestimate than underestimate chemical concentrations in water. PRZM/EXAMS, on
24 the other hand, is a modeling system generally intended to provide more realistic concentrations
25 of chemicals in surface water.

26
27 U.S. EPA/OPP (2004) used GENEEC to estimate a peak concentration of 19 µg/L in a small
28 pond. The upper bound estimate of the peak stream concentration of triclopyr BEE based on the
29 Gleams-Driver modeling is about 17 µg/L. Given the very different input assumptions used in
30 the GENEEC and Gleams-Driver modeling as well as the structural difference between the two
31 models, this correspondence is probably coincidental. For a small pond, the Gleams-Driver
32 modeling estimates upper bound peak concentrations of triclopyr BEE of about 3 µg/L,
33 substantially below the estimated concentration of 19 µg/L from GENEEC. This difference
34 appears to be attributable to the longer half-lives used by U.S. EPA/OPP (2004) in the GENEEC
35 modeling, which appear to reflect the half-lives of triclopyr acid rather than triclopyr BEE.

36
37 For triclopyr acid, the U.S. EPA/OPP (1998a) application of GENEEC result in estimated peak
38 concentrations of about 30 µg/L and longer-term concentrations of about 20 µg/L. The Gleams-
39 Driver simulations for triclopyr acid resulted in lower central estimates (i.e., about 3 to 5 µg/L)
40 but substantially higher upper bound estimates (i.e., about 60 to 220 µg/L).

41
42 A summary of the U.S. EPA/OPP modeling using PRZM/EXAMS is given in Table 23, and
43 additional details are given in Table 24 of the current risk assessment, which is a minor
44 reformatting of Table 3-3 from U.S. EPA/OPP (2009a, pp. 62-63). The summary of all
45 PRZM/EXAMS simulations (Table 24) is based on a range of application rates of up to 20
46 lbs/acre and up to 17 applications with application intervals of 21 days. These modeling efforts

1 are summarized for the sake of completeness, but many of the application rates and schedules
2 used by the U.S. EPA/OPP are much higher and more frequent than would be typical in Forest
3 Service programs (Section 2).

4
5 A subset of the scenarios from Table 24 that are relevant to forestry applications is given in
6 Table 25. This table specifies the application rate for each scenario, the number of applications,
7 and the application interval. In addition, the concentrations reported by the EPA (Table 23) are
8 divided by the application rates to crudely approximate water contamination rates at a
9 normalized rate of 1 pound per acre—i.e., $\mu\text{g/L}$ per lb/acre applied. Note that the scenarios for
10 nonagricultural rights-of-way and one of the two scenarios for rangeland pastures in Table 25
11 involve only a single application.

12
13 In terms of the normalized water contamination rates for forestry simulations, the scenario used
14 by U.S. EPA/OPP for rangeland pastures estimates a peak concentration of $3 \mu\text{g/L}$ per lb/acre.
15 As summarized in Table 23, this concentration is similar to the central estimates of the peak
16 concentrations of triclopyr from Gleams-Driver simulations for ponds (≈ 3.3 to $4.6 \mu\text{g/L}$ per
17 lb/acre) and streams (≈ 2.8 to $3.9 \mu\text{g/L}$ per lb/acre). The PRZM/EXAMS modeling for rights-of-
18 way leads to a maximum estimated water contamination rate of about $240 \mu\text{g/L}$ per lb/acre,
19 which is virtually identical to the upper bound concentration of $221 \mu\text{g/L}$, based on the Gleams-
20 Driver simulations for the application of triclopyr TEA to a small pond. Given the differences
21 between the PRZM/EXAMS and Gleams-Driver simulations, these maximum estimated water
22 contamination rates are strikingly concordant. Similar patterns of consistency between Gleams-
23 Driver and PRZM/EXAMS simulations in estimates of upper bound concentrations are
24 frequently noted in Forest Service risk assessments.

25 **3.2.3.4.5. Monitoring Data**

26 Monitoring data are available on triclopyr concentrations in surface water from aquatic
27 applications (e.g., Knuteson 1999; Petty et al. 2003; Siemering et al 2008). These monitoring
28 studies are not directly useful in assessing the modeling of terrestrial applications of triclopyr, as
29 discussed in the previous subsections. The monitoring data associated with aquatic applications
30 of triclopyr are discussed further in Section 3.2.3.4.6.2.

31
32 While triclopyr has been used for many years, there are only a few monitoring studies associated
33 with terrestrial applications of triclopyr that report pesticide concentrations in water. As
34 summarized in Table 23, triclopyr concentrations ranging from about 0.0002 to $0.42 \mu\text{g/L}$ are
35 reported in general surveys of pesticide concentrations in ambient surface water (Rawn et al.
36 1999; Woudneh et al. 2007). The U.S. Geological survey has an extensive program for
37 monitoring pesticides in water but monitoring data on triclopyr are not included in the most
38 recent USGS report that is available (Gilliom et al. 2007). While very low monitored
39 background concentrations of triclopyr are consistent with the lower bounds of estimated
40 concentrations of triclopyr in ponds and streams based on Gleams-Driver simulations, these
41 monitored concentrations cannot be associated with terrestrial applications of triclopyr. and the
42 correspondence with Gleams-Driver simulations is incidental.

43
44 As also summarized in Table 23, two monitoring studies involving terrestrial applications of
45 triclopyr in forests are available in which concentrations of triclopyr in streams can be associated
46 with specific and well-characterized applications of triclopyr (Norris et al. 1987; Smith and

1 McCormack 1988). In terms of maximum concentrations in streams at a normalized application
2 rate of 1 lb a.e./acre, the reported concentrations span a narrow range from about 31 to 33 μg
3 a.e./L. These peaks occurred shortly after application and were probably due to drift or direct
4 spray. As summarized at the top of Table 23, the modeled concentrations of triclopyr in small
5 ponds or streams resulting from drift at 25 feet or direct spray range from about 20 to 100 $\mu\text{g}/\text{L}$
6 per lb/acre. Both Norris et al. (1987) and Smith and McCormack (1988) also report
7 concentrations in surface water that were delayed and clearly associated with rainfall. These
8 concentrations range from about 4 to 30 $\mu\text{g}/\text{L}$ per lb/acre. The lower bound of this range is
9 consistent with the median estimates from Gleams-Driver for a small pond (3.3 $\mu\text{g}/\text{L}$) and a
10 small stream (2.8 $\mu\text{g}/\text{L}$). The monitored upper bound of 30 $\mu\text{g}/\text{L}$ per lb/acre (Smith and
11 McCormack 1988) is encompassed by the range of the upper bound estimates from Gleams-
12 Driver of 142 $\mu\text{g}/\text{L}$ per lb/acre for a small pond and 62 $\mu\text{g}/\text{L}$ per lb/acre for a small stream.
13
14 Neither Norris et al. (1987) nor Smith and McCormack (1988) discuss characteristics of the
15 streams and ponds that would permit a detailed evaluation of Gleams-Driver; furthermore, such
16 an effort would be beyond the scope of the current risk assessment. Nonetheless, the consistency
17 of the estimated water contamination rates from Gleams-Driver with the forestry monitoring in
18 Norris et al. (1987) and Smith and McCormack (1988) suggest that the estimated water
19 contamination rates from Gleams-Driver are credible.

20 **3.2.3.4.6. Concentrations in Water Used for Risk Assessment**

21 **3.2.3.4.6.1. Terrestrial Applications**

22 Table 26 summarizes the concentrations of triclopyr BEE, triclopyr acid, and TCP (3,5,6-
23 trichloro-2-pyridinol) in surface water used in the current risk assessment for terrestrial
24 applications of either triclopyr BEE or TEA formulations. The concentrations of triclopyr BEE
25 are used only in the EXCEL workbook (Attachment 2) for triclopyr BEE formulations.
26 Concentrations of triclopyr in surface water associated with aquatic applications are discussed in
27 Section 3.2.3.4.5.2.
28

29 The concentrations given in Table 26 are specified as water contamination rates (WCRs)—i.e.,
30 the concentrations in water expected at a normalized application rate of 1 lb a.e./acre, converted
31 to units of ppm or mg/L per lb a.e./acre. In the previous tables discussing concentrations in
32 water, units of exposure are expressed as ppb or $\mu\text{g}/\text{L}$, as a matter of convenience. In Table 26,
33 however, ppb is converted to ppm because ppm (i.e., mg/L) is the unit of measure used in the
34 EXCEL workbooks for contaminated water exposure scenarios in both the human health and
35 ecological risk assessments. The WCR are entered in Worksheet B04 in each of the EXCEL
36 workbooks that accompany this risk assessment. The values in Worksheet B04 are linked to the
37 appropriate scenario-specific worksheets in the EXCEL workbooks. While all values below are
38 given as water contamination rates expressed as mg/L per lb/acre applied, they are discussed as
39 simple concentrations expressed as mg/L for the sake of brevity. Finally, all concentrations of
40 triclopyr BEE and triclopyr are expressed as mg a.e./L, and all concentrations of TCP are
41 expressed as mg TCP/L.
42

43 Most of the concentrations used in the current risk assessment are based on modeling using
44 Gleams-Driver. As discussed in previous subsections, the Gleams-Driver modeling is
45 reasonably consistent with other modeling efforts as well as the relevant monitoring data.
46 Nonetheless, the water contamination rates derived in this section are based on a wide variety of

1 sites and weather conditions. This approach is intended to encompass worse-case exposures. As
2 detailed in Appendices 9 through 15, much lower water contamination rates would be expected
3 in some regions, particularly areas with low rates of rainfall and high temperatures. As detailed
4 in SERA (2010a), local weather and site conditions should be considered in applying or
5 modifying the rates discussed below to assess potential exposures for any particular locality. In
6 addition, as is true all Forest Service risk assessments, the pesticide concentrations in water are
7 based on the assumption that a body of surface water is close to the application site. This
8 assumption will not hold in all areas where triclopyr may be applied.

9
10 Note that the same estimates of the concentrations of triclopyr acid and TCP in surface water are
11 used for applications of both triclopyr BEE and triclopyr TEA. While somewhat lower
12 concentrations for triclopyr acid and TCP following applications triclopyr BEE formulations
13 could be made, the differences between the simulations are minor relative to the site-specific
14 differences discussed in the previous paragraph.

15 16 **3.2.3.4.6.1.1. Triclopyr BEE**

17 For all acute exposures in both the human health and ecological risk assessments, the
18 concentrations of triclopyr BEE in surface water are taken as 0.0004 (1.5×10^{-7} to 0.03) mg a.e./L.
19 The central estimate is taken as the median of the Gleams-Driver simulations for a small stream
20 (0.41 $\mu\text{g/L}$ as summarized in Table 23 and detailed in Appendix 5, Table A9-5).

21
22 The upper bound peak concentration of 0.03 mg a.e./L is based on the field study by Smith and
23 McCormack (1988) in which triclopyr was monitored in a stream immediately after an
24 application of triclopyr BEE for conifer release. While the concentration of 0.03 mg a.e./L
25 reported by Smith and McCormack (1988) may have been due to an accidental spray, this
26 concentration is very close to the concentration of triclopyr BEE that could be expected in a
27 small stream due to drift – i.e., 0.025 mg a.e./L (Section 3.2.3.4.2). As discussed in Section
28 3.2.3.4.3.2 and summarized in Table 23, the peak concentration of 0.03 mg a.e./L may
29 substantially overestimate concentrations that might occur in a small pond (≈ 0.003 mg a.e./L) but
30 is similar to the upper bound concentrations modeled by Gleams-Driver for a small stream
31 (≈ 0.017 mg a.e./L).

32
33 The selection of lower bounds for peak water concentrations is somewhat arbitrary. In areas of
34 low rainfall, it is plausible that a stream would not be subject to any contamination by triclopyr
35 BEE. As discussed below, this is also the case for triclopyr acid and TCP. For the current risk
36 assessment, a concentration of 1.5×10^{-7} mg/L is used. This concentration is the lowest non-zero
37 central estimate of peak concentrations of triclopyr BEE in a small pond—i.e., Appendix 9,
38 Table A9-7, wet and cool locations with sandy soil textures.

39
40 No monitoring data are available on longer-term concentrations of triclopyr BEE in water. The
41 lack of monitoring data is consistent with the rapid breakdown of triclopyr BEE to triclopyr and
42 butoxyethanol. For the current risk assessment, the longer-term concentrations of triclopyr BEE
43 in surface water are taken as 2×10^{-6} (2×10^{-11} to 7×10^{-5}) mg a.e./L. The central estimate is taken
44 as the median of the longer-term concentrations of triclopyr BEE in small streams from the
45 Gleams-Driver modeling—i.e., 0.0018 or 1.8×10^{-3} $\mu\text{g a.e./L}$, which rounded to one significant
46 decimal is equivalent to 2×10^{-6} mg a.e./L. The upper bound of 7×10^{-5} mg a.e./L is taken directly

1 from the upper bound of the longer-term concentrations of triclopyr BEE modeled in small
2 streams using Gleams-Driver—i.e., 0.07 µg a.e./L, as summarized in Table 23 and detailed in
3 Appendix 9, Table A9-6. The lower bound of 2×10^{-11} mg a.e./L is also taken directly from the
4 Gleams-Driver modeling, in this case the lowest non-zero value for any of the longer-term
5 concentrations of triclopyr BEE in a small pond—i.e., 2.4×10^{-8} µg a.e./L for a stream in an area
6 with average rainfall, moderate temperature, and predominantly sand soil textures, as
7 summarized in Appendix 9, Table A9-6. As with all lower bound estimates, longer-term
8 concentrations of triclopyr BEE in water are estimated to be zero in many of the simulations,
9 particularly in areas with low rates of rainfall. Thus, the selection of a non-zero lower bound is
10 somewhat arbitrary but has no impact on the risk assessment in terms of the risk characterization.
11

12 **3.2.3.4.6.1.2. Triclopyr Acid**

13 As discussed in Section 3.2.3.4.4, the Gleams-Driver simulations conducted for the current risk
14 assessment and the PRZM/EXAMS simulations are reasonably concordant in terms of the upper
15 bound estimates of the concentrations of triclopyr in surface water. This is not the case,
16 however, for the central estimates. Based on Gleams-Driver simulations of applications of either
17 triclopyr BEE or triclopyr TEA, the central estimates of the peak concentrations of triclopyr are
18 similar in both ponds (3.3 to 4.5 µg/L) and streams (2.8 to 3.9 µg/L). As summarized in
19 Table 23 and detailed further in Tables 24 and 25, the PRZM/EXAMS estimates are substantially
20 higher with an average concentration of 106 µg/L and a geometric mean of about 60 µg/L. As
21 summarized in Table 23, the arithmetic mean of 106 µg/L is consistent with a direct spray of a
22 small pond or stream and the geometric mean is substantially above estimates of worst-case drift
23 to a small pond or stream over a distance of 25 feet from the application site. In Forest Service
24 risk assessments, estimates of drift are incorporated into upper bound or worst-case estimates of
25 pesticide concentrations in water. This approach is taken because it is standard Forest Service
26 procedure to attempt to minimize drift in any application of pesticides. Consequently, while the
27 U.S. EPA/OPP modeling using PRZM/EXAMS may reflect worst-case exposures, the
28 concentrations estimated in U.S. EPA/OPP (2009a) are not incorporated into the central
29 estimates (i.e., most likely to occur) of exposures in the current Forest Service risk assessment.
30

31 With the above considerations, the estimated peak concentrations of triclopyr (i.e., water
32 contamination rates) in surface water are taken as 0.005 (1×10^{-9} to 0.24) mg/L per lb/acre. The
33 central estimate of 0.005 mg/L is based on the median estimates from the Gleams-Driver
34 modeling for triclopyr concentrations in a small pond following the application of triclopyr TEA,
35 as summarized in Table 23—i.e., 4.55 µg/L is rounded to one significant figure and converted to
36 a concentration in units of mg/L.
37

38 The peak upper bound of 0.24 mg/L is based on the upper bound estimate from U.S. EPA/OPP
39 (2009a), as summarized in Table 23 and detailed further in Tables 24 and 25. Specifically, the
40 upper bound concentration of 0.24 mg/L is based on the EPA modeling of the application of
41 triclopyr to rights-of-way at a rate of 12 lb/acre and a resulting modeled peak concentration of
42 2,929.6 µg/L, which is equivalent to a water contamination rate of $[2,929.6 \text{ µg/L} \div 12 \text{ lb/acre} \approx$
43 $244.13 \text{ µg/L per lb/acre} \approx 0.24 \text{ mg/L per lb/acre}]$. As discussed in Section 3.2.3.4.5, this
44 estimated peak concentration of triclopyr in U.S. EPA/OPP (2009a) is virtually identical to the
45 peak concentration from Gleams-Driver simulations (i.e., 221 µg/L from the application of
46 triclopyr TEA). The use of either the PRZM/EXAMS or the Gleams-Driver maximum peak

1 values has no impact on the risk assessment. Nonetheless, Forest Service risk assessments
2 generally use assumptions and exposures that are at least as conservative as those used by the
3 U.S. EPA/OPP, unless there is a compelling basis for doing otherwise. The differences between
4 the peak estimates from Gleams-Driver and peak PRZM/EXAMS are insubstantial and offer no
5 compelling basis for rejecting the estimated peak concentrations from U.S. EPA/OPP. It may be
6 noted that the concentrations in the range of 200 µg/L are higher than the concentrations
7 estimated from the direct spray of a small pond (110 µg/L) and small stream (90 ug/L). This
8 comparison, however, is not a substantial issue. It is not uncommon for peak concentrations
9 estimated from Gleams-Driver as the result of off-site transport to exceed concentrations
10 estimated from a direct spray.

11
12 The lower bound peak concentration of 1×10^{-9} mg/L is a composite estimate. This estimate is
13 based on the lowest non-zero peak concentration modeled for a small pond—i.e., the central
14 estimate for a pond in an area of high rainfall and cool temperatures with predominantly loamy
15 soil texture, following the application of triclopyr BEE (Appendix 10, Table A10-7).

16
17 The longer-term concentrations of triclopyr are taken as 0.002 (2×10^{-10} to 0.2) mg/L. As with the
18 peak concentrations, the central estimate of 0.002 mg/L is based only on the Gleams-Driver
19 simulations, specifically the central estimate of the longer-term concentration of 1.91 µg/L
20 following applications of triclopyr TEA is rounded to one significant place and converted to
21 units of mg/L.

22
23 The upper bound longer-term concentration of 0.2 mg/L is based on the upper bound of the
24 PRZM/EXAMS simulations involving forestry related sites. As summarized in Table 22 and
25 detailed in Table 25, the U.S. EPA/OPP (2009a) modeling of a rights-of-way application leads to
26 a 60-day time-weighted average concentration of 204 µg/L. The maximum longer-term
27 concentration based on the Gleams-Driver simulations is 93 µg/L. This difference does not
28 suggest an inconsistency in the PRZM/EXAMS and Gleams-Driver modeling. As a convention,
29 the longer-term concentrations modeled using Gleams-Driver are based on the maximum 1-year
30 time-weighted average. This duration is also used in many EPA risk assessments. The annual
31 average concentration of 93 µg/L modeled by Gleams-Driver is from the simulation of a small
32 pond in an area with high rainfall, cool temperatures, and predominantly sandy soil texture (see
33 Appendix 12, Table A12-8). Examination of the detailed Gleams-Driver outputs for this site
34 yields a maximum 60-day time-weighted average concentration of 225.1 µg/L, virtually identical
35 to the concentration of 204 µg/L noted in U.S. EPA/OPP (2009a) for the rights-of-way
36 application. Both concentrations round to 0.2 mg/L.

37
38 In considering the duration period for time-weighted applications, it should be noted that the
39 differences between the annual average and the 60-day average is not substantial—i.e., about a
40 factor of 2. In terms of biological relevance, the duration for the time-weighted average will
41 vary according to species. For humans and most species of wildlife mammals and birds, the
42 annual TWA-concentration is appropriate for assessing chronic toxicity. For many aquatic
43 invertebrates (e.g., daphnids) chronic studies are typically conducted for 3 weeks; hence, a
44 maximum 21-day TWA would be most appropriate. Similarly, most egg-to-fry studies are
45 conducted for about a 30-day period. For these studies a 30-day TWA might be suggested. As a
46 simplification, the annual TWA is used in Forest Service risk assessments, and its potential

1 impact on risks to aquatic organisms is addressed in the risk characterization for these species
2 (Section 4.4.3).

3
4 The lower bound of the longer-term concentration of triclopyr is taken as 2×10^{-10} mg/L. As with
5 all of the lower bound concentrations, this selection is somewhat arbitrary. The specific value is
6 the lowest non-zero central estimate following the application of a triclopyr TEA formulation—
7 i.e., 1.87×10^{-7} µg/L for an area with average rainfall, warm temperatures, and predominantly
8 sandy soil textures.

9 10 **3.2.3.4.6.1.3. TCP**

11 There is a substantial body of monitoring data for TCP concentrations in surface water following
12 aquatic applications of triclopyr (Section 3.2.3.4.1.2). Conversely, there appear to be no
13 monitoring data for TCP concentrations in surface following terrestrial applications of triclopyr.
14 Accordingly, the TCP concentrations in surface water as a result of terrestrial applications of
15 triclopyr are estimated entirely from the Gleams-Driver modeling.

16
17 As discussed in Section 3.2.3.4.3 and summarized in Table 23, the ratio of longer-term
18 concentrations of TCP to longer-term concentrations of triclopyr in ponds estimated using
19 Gleams-Driver is a factor of about 30. Based on peak concentrations, however, the differences
20 between concentrations of TCP and triclopyr are relatively modest. Moreover, there are no
21 substantial differences between the concentrations of triclopyr and TCP modeled in streams for
22 either peak or longer-term concentrations.

23
24 For the current risk assessment, the peak concentrations of TCP are taken as 0.0009 (1×10^{-8} to
25 0.03) mg/L. The central estimate is taken from the concentration of TCP in streams—i.e., 0.96
26 µg/L following applications of triclopyr BEE or 0.86 µ/L following applications of triclopyr
27 TEA—rounded to one significant place. Similarly, the upper bound concentration of 0.03 mg/L
28 is based on the upper bound concentration of about 26.5 µg/L following applications of triclopyr
29 BEE. The lower bound concentration is based on the lowest non-zero lower bound of about
30 0.00001 µg/L modeled for ponds in locations with average rainfall, cool temperatures, and
31 predominantly sandy soil textures following the application of triclopyr BEE (Appendix 11,
32 Table A11-7).

33
34 The longer-term concentrations of TCP are taken as 5×10^{-5} (3×10^{-12} - 0.002) mg/L. As with the
35 peak concentrations, the central and upper bound of the longer-term concentrations are based on
36 the Gleams-Driver simulations of the small pond following applications of triclopyr TEA. The
37 lower bound of the longer-term concentration is the lowest non-zero concentration modeled in
38 any of the Gleams-Driver simulations.

39 40 **3.2.3.4.6.2. Aquatic Applications**

41 As summarized in Table 24, U.S. EPA/OPP (2009a) uses a concentration of 2500 µg/L (2.5
42 mg/L) for aquatic applications of triclopyr TEA. This is the maximum labeled target
43 concentration of triclopyr for the treatment of submerged vegetation. Triclopyr, however, is also
44 registered for the treatment of emergent vegetation, and the application rates for these treatments
45 are expressed in lbs a.e./acre. Thus, separate exposure assessments are derived in the current risk
46 assessment for submergent and surface applications of triclopyr to water.

1
2 **3.2.3.4.6.2.1. Applications for Submergent Vegetation**

3 **3.2.3.4.6.2.1.1. Triclopyr**

4 In the EXCEL workbook for applications to submergent vegetation (Attachment 4), the target
5 concentration of 1 mg a.e./L is used as the central estimate as well as the upper and lower bounds
6 of peak concentration. The use of lower or higher target concentrations, from 0.75 to 2.5 mg
7 a.e./L, as specified on the product labels, is discussed in the risk characterization (Sections 3.4
8 and 4.4). Thus, for acute exposures, estimated doses of triclopyr given in Worksheet in B04a
9 (Attachment 4) are based on a concentration of 1 mg a.e./L.

10
11 While the estimated peak concentration of 1 mg a.e./L for a nominal treatment concentration of
12 1 mg a.e./L may seem intuitive, this approach assumes instantaneous mixing. While
13 instantaneous mixing will often be a reasonable assumption, this will not always be the case.
14 Knuteson (1999) provides a detailed summary of several aquatic applications of triclopyr to
15 ponds and lakes conducted at nominal target concentrations of 2.5 mg a.e./L. For small ponds,
16 the maximum concentration of triclopyr shortly after application was typically close to the
17 nominal target concentration, about 2.0-3.0 mg a.e./L (Figures 5-6 and Figure 10 in Knuteson
18 1999). For applications to bays in lakes (presumably involving shoreline treatments),
19 concentrations of up to about 4 mg a.e./L were monitored within hours after application. These
20 minor excursions above the nominal target concentration may occur occasionally in any direct
21 application of a pesticide to water. These excursions are not considered explicitly in the
22 exposure assessment. Excursions substantially above the nominal application rate are
23 encompassed by a consideration of accidental spills. As detailed in Worksheet B04b of
24 Attachment 4 (submergent applications of triclopyr), the central estimate of the concentration of
25 triclopyr in water following an accidental spill is about 5.2 mg/L.

26
27 Longer-term concentrations of triclopyr in treated waters will depend on various, but difficult to
28 generalize, site-specific factors. As discussed in Section 3.2.3.4.1, the U.S. EPA/OPP (2009a)
29 includes estimated dissipation half-lives in surface waters of 0.5 to 3.5 days. As detailed in
30 Table 1, these estimates are reasonably consistent with field dissipation half-lives reported in
31 several published studies following aquatic applications of triclopyr TEA (Fox et al. 2002;
32 Getsinger et al. 1989,1996; Green et al. 1989; Hautman et al. 1997c; Petty et al. 2001, 2003;
33 Solomon et al. 1988; Turner et al. 1994). Consequently, the half-lives of 0.5 to 3.5 days with a
34 central estimate of 2 days are used to estimate the longer-term concentrations for triclopyr in
35 surface water. Details of these calculations are given in Worksheet B04a of Attachment 3. As
36 summarized in Table 26, the longer-term concentrations are taken as about 0.032 (0.0080 to
37 0.064) mg a.e./L.

38
39 **3.2.3.4.6.2.1.2. TCP**

40 The method used to estimate of the concentrations of TCP in water following applications for the
41 control of submergent vegetation is essentially identical to the approach taken for an accidental
42 spill (Section 3.2.3.4.1.2). As with the accidental spill, triclopyr is added directly to water. The
43 only difference between an accidental spill and the application for the control of submergent
44 aquatic vegetation is that the former is (by definition) accidental and the latter is intentional. The
45 concentrations of triclopyr in surface water from Worksheet B04a of Attachment 4 (triclopyr,
46 submergent vegetation) are entered into Worksheet B04a of Attachment 7 (TCP, submergent

1 vegetation). The concentrations of triclopyr in water are divided by the factors of 235 (137 to
2 586) to estimate corresponding concentrations of TCP as discussed in Section 3.2.3.4.1.2.

3.2.3.4.6.2.2. Applications for Emergent Vegetation

3.2.3.4.6.2.2.1. Triclopyr

6 Applications for emergent vegetation are based on a unit application rate of 1 lb a.e./acre to a
7 body of water with a depth of 2 (1-4) feet. The peak concentrations are estimated based on a
8 simple dilution model. As summarized in Table 26, these applications will result in initial peak
9 concentrations of 0.18 (0.09-036) mg a.e./L, and details of these calculations are given in
10 Worksheet B04a of Attachment 3. Based on the aquatic dissipation half-lives of 2 (0.5 to 3.5)
11 days discussed above in the exposure assessment for submergent vegetation, the longer-term
12 concentrations of triclopyr are estimated at 0.0059 (0.00074 to 0.021) mg a.e./L, and these
13 calculations are also detailed in Worksheet B04a of Attachment 3.

3.2.3.4.6.2.2.2. TCP

16 Estimates of the concentration of TCP in water following applications of triclopyr for the control
17 of emergent vegetation are essentially identical to those used for submergent vegetation (Section
18 3.2.3.4.6.2.1.2). The concentrations of triclopyr in surface water from Worksheet B04a of
19 Attachment 3 (triclopyr, emergent vegetation) are entered into Worksheet B04a of Attachment 6
20 (TCP, emergent vegetation). The concentrations of triclopyr in water are divided by the factors
21 of 235 (137 to 586) to estimate corresponding concentrations of TCP in water as discussed in
22 Section 3.2.3.4.1.2.

3.2.3.5. Oral Exposure from Contaminated Fish

24 Many chemicals may be concentrated or partitioned from water into the tissues of animals or
25 plants in the water. This process is referred to as bioconcentration. The concentration of the
26 pesticide in fish (C_F) is taken as the product of the concentration of the chemical in water (C_W)
27 and the bioconcentration factor (BCF):

Equation 4

$$C_{Fish_{mg/kg}} = C_{W_{mg/L}} \times BCF_{L/kg}$$

31 Bioconcentration is measured as the ratio of the concentration in the organism to the
32 concentration in the water. For example, if the concentration in the organism is 5 mg/kg and the
33 concentration in the water is 1 mg/L, the BCF is 5 L/kg [5 mg/kg ÷ 1 mg/L]. As with most
34 absorption processes, bioconcentration depends initially on the duration of exposure but
35 eventually reaches steady state. Details regarding the relationship of bioconcentration factor to
36 standard pharmacokinetic principles are provided in Calabrese and Baldwin (1993).

38 This risk assessment includes three sets of exposure scenarios for the consumption of
39 contaminated fish, and each set includes separate estimates for the general population and
40 subsistence populations. These exposure scenarios consist of one set for acute exposures
41 following an accidental spill (Worksheets D08a and D08b), another set for acute exposures based
42 on expected peak concentrations (Worksheets D09c and D09d), and the third set for chronic
43 exposures based on estimates of longer-term concentrations in water (Worksheets D09a and
44 D09b). The two worksheets in each of these three sets are intended to account for different rates
45 of wild-caught fish consumption in both general and subsistence populations. Details of

1 exposure scenarios involving the consumption of contaminated fish are provided in Section
2 3.2.3.5 of SERA (2007a).

3 **3.2.3.5.1. Triclopyr**

4 Triclopyr acid and triclopyr BEE have a relatively low potential for bioconcentration. While
5 triclopyr BEE is highly lipophilic, it will be rapidly degraded in water and will not
6 bioconcentrate in fish. Because of the low potential for either triclopyr TEA or triclopyr BEE to
7 accumulate in fish, the EPA waived the requirements for bioconcentration studies in fish for both
8 triclopyr TEA and triclopyr BEE (U.S. EPA/OPP 1996c).

9
10 Barron et al. (1990) determined BCF values in red swamp crayfish (*Procambarus clarki*)
11 exposed to triclopyr. At a concentration of 1 mg/L, the BCF values were 0.51 in whole crayfish
12 and 0.099 in tail muscle. At a concentration of 2.5 mg/L, the corresponding values were 1 and
13 0.2, respectively. Barron et al. (1989b, 1991) investigated the pharmacokinetics and metabolism
14 of triclopyr (BEE) in yolk-sac fry of the coho salmon (*Oncorhynchus kisutch*) and found that the
15 accumulation of triclopyr BEE was limited in the fish due to rapid hydrolysis of triclopyr BEE to
16 triclopyr acid, which was the principal metabolite in fish and water, accounting for over 99% of
17 total residue. No TCP was detected in any residue or in test water.

18
19 In a bioconcentration study of triclopyr in bluegill sunfish (*Lepomis macrochirus*), however,
20 Rick et al. (1996) note that TCP was a major metabolite, accounting for 16.3% of the residues
21 and that an additional 26.4% of the residues was a base-labile conjugate of TCP. The BCF
22 values for triclopyr were 0.19 for total body and 0.06 for muscle. Based on total residue
23 (triclopyr and metabolites), the BCF values were 0.83 for total body and 0.06 for muscle.

24
25 In another laboratory study on blue gill sunfish exposed to triclopyr (¹⁴C-labeled on the pyridine
26 ring) at 2.5 mg/L for 96 hours, whole body residue were 2.33 mg/kg (BCF ≈ 1 L/kg) and levels
27 in edible flesh were 0.13 mg/kg (BCF=0.05 L/kg) (Lickly and Murphy 1987). As in the study by
28 Rick et al. (1996), TCP accounted for a substantial proportion of the residues, about 15-26%
29 (Lickly and Murphy 1987, Table 5, p. 217).

30
31 In a field study, no detectable levels of triclopyr were found in fish after an application of
32 Garlon 3A at a target concentration of 2.5 mg a.e./L. Modest levels of bioconcentration,
33 however, were noted in crayfish and clams (BCF ≤ 4 L/kg) with rapid decreases in tissue levels
34 as water levels decreased (Woodburn et al. 1993b).

35
36 For this risk assessment, the BCF values are taken from Rick et al. (1996) based on total
37 residues—i.e., a BCF of 0.06 L/kg for triclopyr and triclopyr metabolites in edible tissue. As
38 detailed further in the risk characterization, this approach is used to consider the potential effects
39 of both triclopyr and TCP. The lower BCF for edible tissue is used under the assumption that
40 individuals will consume only the edible muscle. In the ecological risk assessment, the whole-
41 body BCF of 0.83 L/kg is used, based on the assumption that fish-eating mammals and birds will
42 consume the entire fish.

43
44 For both the acute and longer-term exposure scenarios involving the consumption of
45 contaminated fish, the water concentrations of triclopyr used are identical to the concentrations
46 used in the contaminated water scenarios, as summarized in Table 26.

3.2.3.5.2. TCP

Bioconcentration studies on TCP are not available. As reviewed by Calabrese and Baldwin (1993, pp. 12-24), there are various algorithms for estimating bioconcentration factors in fish based on either the K_{ow} (increasing BCF with increasing K_{ow}) or water solubility (decreasing BCF with increasing water solubility) of the compound. As summarized in Table 2, the estimated K_{ow} for TCP is substantially greater than the K_{ow} for triclopyr (i.e., 1000 vs 0.35). Similarly, the water solubility of TCP is somewhat less than that of triclopyr (i.e., 100 vs 400 mg/L). Thus, it would be expected that the BCF for TCP would be greater than that for triclopyr. Based on the algorithm currently recommended by the U.S. EPA, the bioconcentration factor for triclopyr is about 3.1 and the bioconcentration factor of TCP is about 61 (U.S. EPA/OPPTS 2011). As discussed above, however, the study by Rick et al. (1996) reports measured fish BCFs for triclopyr of 0.83 in whole fish and 0.06 in fillet. Thus, the BCF of 3.1 for triclopyr from the EPA's algorithm seems to be an overestimate.

As discussed further in Section 3.2.3.4.1, the peak concentration of TCP following the aquatic application of triclopyr at a target concentration of 2.5 mg a.e./L is about 0.7 (0.6 to 1) mg/L, and this estimate is consistent with a substantial body of monitoring data from Knuteson (1999). Based on the estimated BCF of 61 L/kg in U.S. EPA/OPPTS (2011), the expected peak residues of TCP in fish would be about 42.7 (36.6 to 61) mg/L. Knuteson (1999), however, also provides biomonitoring of the concentration of TCP in fish following the aquatic application of triclopyr at target concentrations of about 2.0-3.0 mg a.e./L. The maximum concentration of TCP found in fish viscera was about 0.4 mg/L. Thus, the BCF of 61 for TCP cited in U.S. EPA/OPPTS (2011) appears to be a substantial overestimate.

Although Knuteson (1999) reports the half-life of TCP in fish, these half-lives involve exposures to varying concentrations of TCP in water and cannot be used in a kinetic model to estimate the BCF (e.g., Calabrese and Baldwin 1993). In addition, no data are available on the kinetics of absorption of TCP by fish, which is required to estimate the BCF analytically.

As an alternative, the simplifying assumption could be made that the BCF for TCP is identical to that of triclopyr. Under this assumption and using the estimated peak concentrations of TCP in water of 0.7 (0.6 to 1) mg/L following the aquatic application of triclopyr at a target application rate of 2.5 mg a.e./L, the expected concentration of TCP in fish would be about 0.6 (0.5 to 0.83) mg TCP/kg fish [$0.83 \text{ L/kg} \times 0.7 \text{ (0.6 to 1) mg/L} \approx 0.581 \text{ (0.498 to 0.83) mg/L}$]. As discussed above, these estimated concentrations are above the peak concentrations in fish reported by Knuteson (1999) but the degree of the overestimate is modest.

The assumption that the bioconcentration factor for TCP is identical to that of triclopyr is consistent with the available data, and the estimate of 0.83 L/kg for whole fish seems at least somewhat conservative. For the current risk assessment, the BCF for TCP in whole fish is taken as 0.83 L/kg and the BCF for fillet is taken as 0.06 L/kg, the same values used for triclopyr from the study by Rick et al. (1996).

As detailed in Section 3.2.3.4.2, the maximum TCP concentration in water following an accidental spill is expected to be 0.13 mg/L. For the accidental spill scenario, the most extreme exposure is the consumption of contaminated fish by subsistence populations. This scenario is

1 detailed in Worksheet D08b of the EXCEL workbooks that accompany this risk assessment.
2 Applying the BCF of 0.06 for TCP to this scenario with the upper bound concentration of 0.13
3 mg/L results in an upper bound of the estimated dose of about 0.00009 mg/kg bw, which is far
4 below the estimated dose of TCP, 0.015 mg/kg bw, for a small child consuming water at some
5 point after an accidental spill. Using the acute RfD of 0.025 mg/kg bw, the upper bound HQ for
6 an individual consuming fish would be 0.004, below the level of concern by a factor of 250.
7 This cursory analysis supports the assertion in Section 3.2.3.4.2 that oral exposures to TCP
8 associated with the contamination of surface water are insubstantial.

9 **3.2.3.6. Dermal Exposure from Swimming in Contaminated Water**

10 Some geographical sites maintained by the Forest Service or Forest Service cooperators include
11 surface water in which members of the general public might swim. To assess the potential risks
12 associated with swimming in contaminated water, an exposure assessment is developed for a
13 young woman swimming in surface water for 1 hour (Worksheet D11). Conceptually and
14 computationally, this exposure scenario is virtually identical to the contaminated gloves scenario
15 used for workers (Section 3.2.2.2)—i.e., a portion of the body is immersed in an aqueous
16 solution of the compound at a fixed concentration for a fixed period of time.

17
18 As in the corresponding worker exposure scenario, the 1-hour period of exposure is somewhat,
19 but not completely, arbitrary, given that longer periods of exposure are plausible. Nonetheless,
20 the 1-hour period is intended as a unit exposure estimate. In other words, the exposure and
21 consequently the risk will increase linearly with the duration of exposure, as indicated in
22 Worksheet D11. Thus, a 2-hour exposure would lead to a HQ that is twice as high as that
23 associated with an exposure period of 1 hour. In cases in which this or other similar exposures
24 approach a level of concern, further consideration is given to the duration of exposure in the risk
25 characterization (Section 3.4).

26
27 In Forest Service risk assessments, the ingestion of water during swimming is not considered
28 explicitly. U.S. EPA/OPP (2003) uses a model for swimming exposures based on essentially the
29 same approach to dermal absorption used in Worksheet D11. The EPA model, however,
30 incorporates the assumption that an adult will consume water while swimming at a rate of 50
31 mL/hour. This assumption is based on data from ingestion rates in swimming pools. Based on
32 more recent studies of water ingestion while swimming in pools (Dorevitch et al. 2010; Dufour
33 et al. 2006), the EPA assumption of 50 mL/hour is a plausible upper bound.

34 **3.2.3.7. Oral Exposure from Contaminated Vegetation (Triclopyr)**

35 Applications of triclopyr associated with Forest Service programs will not involve crop
36 treatment. Under normal circumstances and in most types of applications, it is extremely
37 unlikely that humans will consume substantial amounts of vegetation contaminated with
38 triclopyr. Nonetheless, any number of accidental or incidental scenarios could be developed
39 involving either spraying of crops, gardens, or edible wild vegetation. Again, in most instances
40 and particularly for longer-term scenarios, treated vegetation would probably show signs of
41 damage from exposure to triclopyr (Section 4.3.2.4), thereby reducing the likelihood of
42 consumption which might lead to significant levels of human exposure.

43
44 Notwithstanding the above reservations, all forest service risk assessments involving foliar
45 applications currently include two sets of standard exposure scenarios: one for the consumption

1 of contaminated fruit and the other for the consumption of contaminated broadleaf vegetation.
2 These scenarios are detailed in EXCEL workbooks for foliar applications (Attachments 1 and 2)
3 in Worksheets D03a (fruit) and D03b (broadleaf vegetation) for acute exposure and Worksheets
4 D04a (fruit) and D04b (broadleaf vegetation) for longer-term exposure. This is a change in
5 procedure from the previous Forest Service risk assessment on triclopyr (SERA 2003) which
6 considered only exposure scenarios for the consumption of contaminated fruit.

7
8 The pesticide concentration on contaminated fruit and vegetation is estimated using the empirical
9 relationships between application rate and concentration on different types of vegetation
10 (Fletcher et al. 1994). The rates provided by Fletcher et al. (1994) are based on a reanalysis of
11 data originally compiled by Hoerger and Kenaga (1972) and represent estimates of pesticide
12 concentration in different types of vegetation (mg chemical/kg vegetation) after a normalized
13 application rate of 1 lb a.i./acre. Although the human health risk assessments conducted by the
14 EPA do not consider this exposure scenario, the residue rates recommended by Fletcher et al.
15 (1994) are used by U.S. EPA/OPP in their recent ecological risk assessment of triclopyr (U.S.
16 EPA/OPP 2009a, p. 53).

17
18 The residue rates recommended by Fletcher et al. (1994) are given in Table 27 of the current
19 Forest Service risk assessment. Fletcher et al. (1994) and Hoerger and Kenaga (1972) provide
20 only central and upper bound estimates of residue rates. Accordingly, the lower bound estimates
21 in Table 27 are made under the assumption that the ratio of the central estimate to the upper
22 bound estimate is identical to the ratio of the lower bound estimate to the central estimate (i.e.,
23 the variability is log-symmetrical).

24
25 In the previous Forest Service risk assessment on triclopyr, the study by Siltanen et al. (1981)
26 was used to adopt lower residue rates for triclopyr on fruit than the rates recommended by
27 Fletcher et al. (1994). Siltanen et al. (1981) monitored levels of triclopyr on cowberries and
28 bilberries after backpack sprays of Garlon 3A at application rates of 0.25, 0.75, and 2.25 kg
29 a.e./ha [\approx 0.22, 0.67, and 2 lbs/acre]. As illustrated in Figure 6 of the current risk assessment,
30 residue data on cowberries were monitored for 98 days after application at a rate of 0.75 kg
31 a.i./ha (\approx 0.48 lb a.e./acre). Although some scatter is apparent, the time-course of the residue data
32 are consistent with first-order dissipation ($p=0.0096$). Based on exponential regression, the
33 estimate time-zero residues are about 1.97 (0.69 to 5.64) mg/kg. Siltanen et al. (1981) do not
34 explicitly state whether the residues are reported as a.i. or a.e. Assuming that the residues are
35 reported as a.e., the residue rates may be estimated at about 4.1 (1.4 to 11.7) mg/kg per lb/acre.
36 While these rates are somewhat lower than the residues rates on fruit recommended by Fletcher
37 et al. (1994)—i.e., 7 (3.2 to 15) mg/kg per lb/acre—that difference is not substantial. The
38 application of any pesticide to fruit is essentially a physical deposition, and initial rates should be
39 reasonably constant for all pesticides. The initial analysis by Hoerger and Kenaga (1972) as well
40 as the reanalysis by Fletcher et al. (1994) reflect a careful consideration of numerous studies.
41 Consequently, the current risk assessment uses the modestly higher standard residue rates
42 recommended by Fletcher et al. (1994) rather than the rates which can be derived from the
43 triclopyr study by Siltanen et al. (1981).

44
45 While initial residues on fruit and other commodities are likely to be the same or nearly so for
46 most pesticides, the dissipation of residues will clearly vary among pesticides and different types

1 of vegetation. As discussed in Section 3.2.3.4.3. (Gleams-Driver Modeling) and summarized in
2 Tables 1 and 2, the foliar dissipation of triclopyr acid is relatively rapid with foliar half-lives of
3 6.2 (2.6 to 15) days. The study by Siltanen et al. (1981), however, suggests substantially longer
4 half-lives on cowberries. Based on the exponential regression illustrated in Figure 6, the first-
5 order dissipation coefficients (k) are estimated at about 0.0257 (0.00948 to 0.0419) days⁻¹,
6 corresponding to half-lives of about 26.9 (16.5 to 73.1) days [$t_{50} = \ln(2) \div k$]. In the absence of
7 additional data with which to estimate first-order half-lives for triclopyr residues on fruit, the
8 half-lives of 26.9 (16.5 to 73.1) days are used to estimate the longer-term doses associated with
9 the consumption of contaminated fruit (Worksheet D04a).

10
11 As also discussed in Section 3.2.3.4.3 (Gleams-Driver Modeling) and summarized in Tables 1
12 and 2, the foliar dissipation of triclopyr BEE—i.e., foliar half-lives of 4.1 (1.1 to 15)—is
13 somewhat more rapid than that of triclopyr acid. The more rapid dissipation of triclopyr BEE on
14 vegetation is consistent with and would be expected from the higher volatility of triclopyr BEE
15 relative to triclopyr. Conversely, the impact of the higher volatility of triclopyr BEE could be
16 offset by the more rapid penetration of triclopyr BEE into fruit. More importantly, in terms of
17 potential risks to both humans and nontarget species, levels of triclopyr BEE versus levels of
18 triclopyr acid in the fruit do not matter because the toxicity values for humans and terrestrial
19 animals are identical (Sections 3.3 and 4.3). Consequently and in the absence of a study on the
20 dissipation of triclopyr BEE on fruit with respect to total triclopyr residues, the half-lives on
21 cowberries from the study by Siltanen et al. (1981) are applied to triclopyr BEE.

22
23 In a survey of herbicide residues on plants that are important to Native Americans, Segawa et al.
24 (1997, Table 1, p. 559) report maximum triclopyr residues on some plants of up to 0.7 mg/kg.
25 These residue rates are substantially below the residue rates derived from Fletcher et al. (1994)
26 which range from 3.2 to 240 mg/kg.

27 **3.2.3.8. Oral Exposure from Contaminated Vegetation (TCP)**

28 The assessment of potential exposures to TCP in contaminated fruit or vegetation is necessary.
29 As detailed in Section 3.2.3, HQs associated with the consumption of fruit or vegetation
30 contaminated with triclopyr are above the level of concern. Because TCP is a major metabolite
31 of triclopyr and is more toxic than triclopyr, the hazards associated with the consumption of fruit
32 or vegetation contaminated with TCP cannot be dismissed.

33
34 A major limitation in assessing potential exposures to TCP in fruit or vegetation, however, is the
35 lack of information on the kinetics of the formation and degradation/dissipation of TCP in plant
36 tissue. In the extensive and detailed review of the environmental fate of TCP by Knuteson
37 (1999), no information is presented on the rates of formation and degradation of TCP in plants
38 following applications of triclopyr. Similarly, no information on these rates has been
39 encountered in the assessments by U.S. EPA/OPP or open literature reviews (as cited in
40 Section 1).

41
42 Norris et al. (1987) monitored the concentrations of TCP in grass following the application of
43 triclopyr (formulation not specified). While triclopyr concentration in grass followed a biphasic
44 model, the concentrations of TCP in grass over a 365 day period are log-linear. As part of the
45 conduct of the current Forest Service risk assessment, the natural logarithm of the concentrations
46 of TCP in grass from Table 1 in Norris et al. (1987) were regressed against time. The data fit the

1 first-order decay model extremely well ($r^2=0.944$, $p=0.000156$) and yielded and estimated half-
2 life of 71.7 days. A plot of the data from Norris et al. (1987) is given in Figure 6 along with the
3 corresponding data from Siltanen et al. (1981). As discussed in Section 3.2.3.7, Siltanen et al.
4 (1981) estimated half-lives of 26.9 (16.5 to 73.1) days for triclopyr on fruit.

5
6 Ganapathy (1997) summarizes an unpublished study by Dixon-White (1990) from DowElanco
7 (currently Dow AgroSciences) that apparently was submitted to the California Department of
8 Pesticide Regulation. This study is cited as MRID 41961001 but is not discussed in U.S.
9 EPA/OPP (1998a). In this study, triclopyr BEE was applied at application rates of 2.0, 4.0 or 6.0
10 lb a.i./acre to grass. The reported residues of triclopyr in grass at 30 days after treatment are
11 highly variable: 16-88 ppm at 2 lb/acre, 9.6-173 ppm at 4 lb/acre, 34-389 ppm at 6 lb/acre.
12 Taking the average of the reported ranges, the triclopyr residues correspond to 52, 182, and 211
13 ppm. After about the same period of time after application (28 days), residues of TCP in grass
14 were 43 ppm at 2 lb/acre, 88 ppm at 4 lb/acre, and 134 ppm at 6 lb/acre. Thus, the ratio of TCP
15 to triclopyr for the three application rates was about 0.82 [43 ppm \div 52 ppm], 0.48 [88 ppm \div
16 182 ppm], and 0.63 [134 ppm \div 211 ppm].

17
18 Other studies summarized in Ganapathy (1997) report no or very low residues of TCP following
19 applications of triclopyr. In the absence of more detailed information on these studies (e.g.,
20 monitoring schedule), however, these other studies cannot be fully interpreted, except to note
21 that they report low concentrations of TCP in vegetation. As discussed below, low TCP
22 concentrations in vegetation would be expected in some cases, depending on the degradation of
23 kinetics for triclopyr and TCP under the conditions of a particular study.

24
25 As summarized in Table 2 and detailed further in Table 1, TCP is somewhat more persistent than
26 triclopyr in soil but less persistent than triclopyr in water. The only reported half-life for TCP in
27 vegetation is 10 days, a value reported in the review by Ganapathy (1997) from an unpublished
28 study (DowElanco Data package 51566-006) on residues in grass (Ganapathy 1997, pp. 12-13).
29 This half-life does not differ substantially from the reported half-lives for triclopyr in plants. In
30 the absence of more detailed data on the kinetics of TCP in plants, this risk assessment assumes
31 that TCP half-times are comparable to those of triclopyr—i.e., 6.2 (2.6 – 15) days in vegetation
32 and 27 (16.5-73) days in fruit. As with the corresponding analysis of triclopyr given in Section
33 3.2.3.7, the term *vegetation* is used here to designate broadleaf vegetation, the plant group with
34 the highest residue rate of those considered by U.S. EPA/OPP (Table 27).

35
36 The use of equivalent kinetic parameters for triclopyr and TCP is not the most conservative
37 approach that could be taken. The confidence limit on the residues in grass from the study by
38 Norris et al. (1987) are about 57 to 96 days and a case could be made for assuming that the half-
39 life of TCP on contaminated vegetation is comparable to the half-life of triclopyr on
40 contaminated fruit. While this more conservative approach would modestly increase exposure
41 and the subsequent assessment of risk, the risks associated with TCP residues on contaminated
42 vegetation are evident, as detailed further in Section 3.4.3 (risk characterization for members of
43 the general public). In addition, taking the more conservative approach would disregard the
44 information from Ganapathy (1997). The Ganapathy (1997) review is from the Environmental
45 Monitoring and Pest Management Branch of the Department of Pesticide Regulation/California

1 EPA. This is a highly credible organization and it would not be appropriate for the current risk
2 assessment to disregard the review from Ganapathy (1997).

3
4 Under the assumption that the half-lives (T_{50}) of triclopyr and TCP are equal, the degradation
5 rates (k) for the degradation of triclopyr to TCP and the degradation of TCP to other metabolites
6 will also be equal [$k = \ln(2) \div T_{50}$]. The solution to these two simultaneous processes is well
7 characterized (e.g., Goldstein et al. 1974, p. 333-336). Modifying the equations from Goldstein
8 et al. (1974) to estimate mass rather than molar concentrations, the concentration of the
9 metabolite at time t (X_t) is:

$$X_t = k \times \frac{MW_M}{MW_P} \times C_0 \times t \times e^{-kt}$$

10 **Equation 5**

11
12 where C_0 is the initial concentration of the parent compound, MW_M is the molecular weight of
13 the metabolite and MW_P is the molecular weight of the parent. Equation 4 is applied to
14 calculating the time-course of TCP concentrations in fruit and vegetation using the input values
15 summarized in Table 28, and the results of the analysis are illustrated in Figure 7 (fruit) and
16 Figure 8 (vegetation). Details of the calculations are given in Worksheet B05aMet (fruit) and
17 Worksheet B05bMet (vegetation) of Attachment 5.

18
19 The estimated residues of TCP in fruit (Figure 7) are substantially below those on vegetation
20 (Figure 8), which directly reflects the higher initial residues for vegetation, relative to those for
21 fruit (Table 27). Under the assumption of equal rates for the degradation of triclopyr and TCP,
22 the maximum residue of TCP (X_{\max}) is directly proportional to the initial concentration of
23 triclopyr (C_0),

$$X_{\max} = C_0 \times e^{-1} \times \frac{MW_M}{MW_P},$$

24
25 **Equation 6**

26
27 where e^{-1} is the reciprocal of the base of nature logarithms (≈ 0.368) and the ratio of molecular
28 weights —i.e., metabolite (TCP) \div parent (triclopyr) ≈ 0.77 —is again used to convert from molar
29 to mass concentrations. Equation 5 is used in Worksheets B05aMet and B05bMet
30 (Attachment 5) to estimate the peak TCP residues in fruit and vegetation, respectively. Note that
31 the residue rates for triclopyr are identical to those used in the worksheets for estimating acute
32 oral exposures to triclopyr from the consumption of contaminated fruit (Worksheet B05a) and
33 contaminated vegetation (B05b) in Attachments 1 and 2. By dividing both sides of Equation 5
34 by C_0 , the estimated maximum concentration of TCP in vegetation as a fraction of the initial
35 concentration of triclopyr in vegetation can be estimated at about 0.3 [$0.368 \times 0.77 \approx 0.283$].

36
37 As also illustrated in Figures 7 and 8, the times to the peak concentration of TCP differ
38 substantially ranging from about 4 days (lower bound residues on vegetation) to over 3 months
39 (upper bound residues in fruit). These times (t_{\max}) to the formation of the maximum
40 concentration of the metabolite are inversely related to the degradation rates:
41
42

$$t_{\max} = \frac{1}{k}$$

While the time to peak residues are easy to estimate, the longer-term exposure scenarios are concerned with maximum time-weighted average concentrations. In the longer-term exposure scenarios for triclopyr involving the consumption of contaminated fruit (Worksheet B05a) and contaminated vegetation (B05b), the maximum 90-day time-weighted average concentration is calculated simply as the integral of the concentrations from Day 0 to Day 90 divided by the period of 90 days. As illustrated in Figure 7 (fruit) and Figure 8 (vegetation), this approach could be used for most but not all of the time-course data on TCP. This approach, however, cannot be taken for upper bound residues of TCP in fruit. As illustrated in Figure 7, the upper bound of the peak concentration of TCP in fruit will not occur until after 100 days. This delayed peak is due to the long half-life and correspondingly low degradation rate (k) for residues of TCP in fruit.

Analytical solutions to estimating the peak time-weighted average for a metabolite are available but they are somewhat involved. As an alternative to using the analytical solutions, the maximum 90-day time-weighted averages of TCP in fruit and vegetation are numerically calculated in Worksheets B05aMet (fruit) and B05bMet (vegetation) in Attachment 5. The peak 1-day and maximum 90-day time-weighted averages are linked to the appropriate worksheets for the acute and longer-term consumption of fruit and vegetations – i.e., Worksheets D04a-d in Attachment 5. Apart from the calculation of the 90-day average concentrations, all other assumptions and calculations in these worksheets are identical to the corresponding worksheets for triclopyr TEA (Attachment 1) and triclopyr BEE (Attachment 2).

As discussed above, the ratios of TCP to triclopyr at Day 28, taken from the unpublished data summarized by Ganapathy (1997), are in the range of 0.48-0.63. The data on which Figure 8 is based can be used to estimate TCP residues in broadleaf vegetation on Day 28 at about 4.7 (0.05 to 37) mg/kg. The corresponding Day 28 residues for triclopyr, estimated by adjusting Worksheet D04b in Attachment 1 to 28 rather than 90 days, are about 2 (0.009 to 37). The ratios of the concentrations of TCP to triclopyr based on these estimates are about 2.5 (5.5 to 1). Ignoring the lower bound estimates, which are likely to be unstable, the modeled peak concentrations may overestimate the values reported by Ganapathy (1997) by factors of about 1.6 [$1 \div 0.63$] to 5 [$2.5 \div 0.48 \approx 5.2$]. Given the limited details in the data presented by Ganapathy (1997), these comparisons should not be overly interpreted. Nonetheless, Ganapathy (1997) provides information on concurrent residues of triclopyr and TCP in plants and the comparisons to the data summarized in Ganapathy (1997) suggest that the exposure assessment for TCP in vegetation may be somewhat conservative, but not grossly so.

Additional studies on concurrent residues of triclopyr and TCP in vegetation over a sufficient period of time to quantify the kinetics of TCP formation and degradation would be useful in refining and improving the exposure assessments dealing with TCP in contaminated vegetation.

3.3. DOSE-RESPONSE ASSESSMENT

3.3.1. Overview

An overview of the dose-response assessment for potential human health effects is given in Table 29. The U.S. EPA/OPP has derived acute and chronic RfDs for both triclopyr and 3,5,6-trichloro-2-pyridinol (TCP), and these RfDs are adopted without modification. As a general practice in Forest Service risk assessments, the RfDs derived by the U.S. EPA/OPP are used because they generally provide a level of analysis, review, and resources that far exceed those that are or can be conducted in the support of most Forest Service risk assessments. In addition, it is desirable for different agencies and organizations within the federal government to use concordant risk assessment values.

The acute and chronic RfDs for triclopyr are 1 and 0.05 mg/kg bw/day, respectively. Both RfDs are based on NOAELs in rats, and both use an uncertainty factor of 100. The acute RfD is based on a developmental study in which no effects were noted at 100 mg/kg bw/day but severe maternal toxicity was noted at 300 mg/kg bw/day. The chronic RfD is based on a two-generation reproduction study in rats in which no adverse effects were noted at 5 mg/kg bw/day but effects on the kidney were noted at 25 mg/kg bw/day. Because of concerns for the reproductive and developmental toxicity of triclopyr, the acute RfD is not used to assess risks to women of childbearing age. For this group, the chronic RfD is used to assess the risks associated with both acute and longer-term exposures.

The acute and chronic RfDs for TCP are lower than those for triclopyr. For TCP, the acute RfD is 0.025 mg/kg bw/day and the chronic RfD is 0.012 mg/kg bw/day. The acute RfD is based on a developmental study in rabbits in which birth defects were noted at a dose of 100 mg/kg bw/day but no adverse effects were observed at 25 mg/kg bw/day. An uncertainty factor of 1000 is applied to the NOAEL to derive the acute RfD. Unlike the case with triclopyr, however, the acute RfD is applied only to women of childbearing age. The chronic RfD is based on a chronic study in dogs in which the NOAEL was 12 mg/kg bw/day. As with the acute RfD, the chronic RfD is derived using an uncertainty factor of 1000.

3.3.2. Triclopyr

3.3.2.1. Acute RfD

The RED on triclopyr (U.S. EPA/OPP 1998a) does not specifically refer to an acute RfD but does use an “acute NOEL” of 30 mg/kg. The NOEL is based on the study by Bryson (1994a) in which New Zealand white female rabbits were given gavage doses of triclopyr BEE at 0, 10, 30, or 100 mg/kg/day on days 6-18 of gestation. No effects were noted at 30 mg/kg/day. At 100 mg/kg/day, effects included parental mortality as well as decreased numbers of live fetuses, increased numbers of fetal deaths, and increased numbers of fetal and/or litter incidence of skeletal anomalies and variants. Essentially identical results—a NOAEL of 30 mg/kg/day and a LOAEL of 100 mg/kg/day—are also reported for triclopyr BEE in a rabbits study (Breslin and Billington (1995) and for triclopyr TEA in a rat study (Breslin et al. 1996).

While the results of developmental studies often suggest that rabbits are more sensitive than rats, this does not appear to be the case with triclopyr. As summarized in Table 10, several

1 developmental studies (Breslin et al. 1996; Carney et al. 2007; Jones 1995) in rats report
2 NOAELs in the range of 3.6-5 mg/kg bw/day and LOAELs in the range of 22-30 mg/kg bw/day.

3
4 In the most recent pesticide tolerance for triclopyr (U.S. EPA/OPP 2002a), U.S. EPA/OPP
5 recommends an explicit acute RfD of 1 mg/kg/day for the general population. This RfD is based
6 on the NOAEL of 100 mg/kg/day in the study by Jones (1995) in which rats were administered
7 gavage doses of triclopyr BEE at 0, 30, 100, or 300 mg/kg/day on days 6 through 15 of gestation.
8 At 300 mg/kg/day, toxic responses included signs of marked maternal toxicity including four
9 deaths, overt clinical signs in a few dams, mean body weight loss and decreased mean body
10 weight gain, decreased mean feed consumption, increased mean water consumption, and
11 increased mean liver and kidney weights. In addition, fetal effects included both skeletal and
12 soft-tissue malformations. The NOAEL for fetal toxicity, however, was 100 mg/kg bw/day. The
13 EPA uses this NOAEL to derive an acute RfD of 1 mg/kg bw/day. Furthermore, the EPA
14 indicates that the acute RfD is not applicable to females between the ages of 13-50 years—i.e., of
15 child bearing age. The basis for this recommendation appears to be signs of maternal toxicity
16 observed at 30 mg/kg bw/day with the NOAEL of 5 mg/kg bw/day. As discussed below, the
17 chronic RfD for triclopyr is 0.05 mg/kg bw/day, based on a NOAEL of 5 mg/kg bw/day. Thus,
18 for women of childbearing age, the U.S. EPA/OPP (2002) recommends an acute RfD of 0.05
19 mg/kg/day, equivalent to the chronic RfD.

20 **3.3.2.2. Chronic RfD**

21 In the RED on triclopyr (U.S. EPA/OPP 1998a), the U.S. EPA recommends a chronic RfD of
22 0.05 mg/kg/day. As discussed in Section 3.1.9, this chronic RfD is based on the two-generation
23 reproduction study in rats by Vedula et al. (1995) in which degeneration of renal proximal
24 tubules were noted in adult animals at a dose of 25 mg/kg/day but not at 5 mg/kg/day. The 5
25 mg/kg/day NOAEL dose was divided by 100, a factor of 10 to account for uncertainties in
26 species-to-species extrapolation and another factor of 10 to encompass sensitive individuals in
27 the population. Thus, the resulting RfD is 0.05 mg/kg/day. U.S. EPA/OPP (2002) maintains
28 this RfD in the most current pesticide tolerances and applies it to several intermediate exposure
29 scenarios—i.e., exposure periods of 1-6 months.

30
31 As discussed above, the acute RfD for females is also 0.05 mg/kg bw/day, equivalent to the
32 chronic RfD but based on a NOAEL for maternal toxicity from the Jones (1995) study. Under
33 the Food Quality Protection Act (FQPA), the U.S. EPA is required to evaluate whether or not an
34 additional uncertainty factor is required for the protection of children. Because of the
35 concordance of the acute NOAEL based on a developmental study and the chronic NOAEL
36 based on the reproduction study, the EPA determined that no additional FQPA uncertainty factor
37 is required (U.S. EPA/OPP 1998a, 2002).

38 **3.3.3. TCP**

39 While the U.S. EPA has not derived formal acute or chronic RfDs for TCP, the RED on triclopyr
40 (U.S. EPA/OPP 1998a, pp. 31 ff) uses a chronic value of 0.03 mg/kg/day for the risk
41 characterization for TCP. In the more recent pesticide tolerances for triclopyr (U.S. EPA/OPP
42 2002, pp. 58722), a somewhat lower value is used for the risk characterization of TCP: a dose of
43 0.012 mg TCP/kg/day derived using an uncertainty factor of 1000 and data from a chronic study
44 in dogs in which changes in clinical chemistry were observed at the 48 mg/kg/day (LOAEL) and
45 no effects were observed at the 12 mg/kg/day dose (NOAEL).

1
2 For acute effects, the pesticide tolerances for triclopyr (U.S. EPA/OPP 2002, pp. 58722) use a
3 dose of 0.025 mg/kg/day from a developmental toxicity study in rabbits in which the NOAEL of
4 25 mg/kg/day and corresponding LOAEL of 100 mg/kg/day are based on an increased incidence
5 of hydrocephaly and dilated ventricles.

6
7 For both acute and chronic exposures the uncertainty factor for TCP is set at 1000: 10 to account
8 for uncertainties in species-to-species extrapolation and another factor of 10 to encompass
9 sensitive individuals in the population as well as an additional factor of 10 for the potentially
10 higher sensitivity of children—i.e., the FQPA uncertainty factor.

11
12 For the current risk assessment, the values used for risk characterization are identical to the most
13 recent and conservative values proposed by U.S. EPA/OPP: 0.025 mg/kg/day for acute
14 exposures and 0.012 mg/kg/day for chronic exposures.
15

16 **3.3.4. Dose-Severity Relationships**

17 Many Forest Service risk assessments consider dose-severity relationships for pesticides when
18 some of the HQs exceed the level of concern. As discussed further in Section 3.4, several
19 exposure scenarios for workers and members of the general public are substantially above the
20 level of concern. Nonetheless, a formal assessment of dose-severity relationships is not
21 conducted in this dose-response assessment because no detailed studies are available on dose-
22 severity relationships in humans. Consequently, a consideration of dose-severity relationships is
23 limited to studies on experimental mammals. The discussion of these relationships is
24 incorporated into the risk characterization (Section 3.4).

3.4. RISK CHARACTERIZATION

3.4.1. Overview

Aquatic applications of triclopyr do not present apparent or identifiable risks to humans. Details of the HQs for aquatic applications of triclopyr are given in Attachments 3 and 4.

The risk characterization for terrestrial applications of triclopyr is more complex and requires greater discussion. Overviews of the risk characterization associated with terrestrial applications of triclopyr TEA and triclopyr BEE formulations are presented in Table 30 for workers and Table 31 for members of the general public. These tables are discussed in detail in the following subsections.

For workers involved in terrestrial applications of triclopyr, the risk characterization is qualitatively similar to the previous Forest Service risk assessment on triclopyr. At the typical application rate of 1 lb a.e./acre, the central estimates of the HQs indicate that workers will not be subject to hazardous levels of triclopyr during applications of triclopyr TEA. For triclopyr BEE, the central estimates of the HQs based on the chronic RfD range from 0.7 to 1.2. At the upper bounds of the estimated exposures for all application methods, the HQs for both triclopyr TEA (HQs = 1.6 to 3) and triclopyr BEE formulations (HQs = 6 to 12) exceed the level of concern based on the chronic RfD. Based on the acute RfD, no HQs substantially exceed the level of concern. The HQs based on the acute RfD, however, would only apply to male workers. All HQs for workers will increase linearly with the application rate.

For members of the general public, the only non-accidental exposure scenarios of concern involve the consumption of contaminated fruit or vegetation with consequent exposures to triclopyr and 3,5,6-trichloro-2-pyridinol (TCP), the primary metabolite of triclopyr. At an application rate of 1 lb a.e./acre, the upper bound HQ of 27 for triclopyr in the acute exposure scenario for the consumption of contaminated vegetation by a young woman exceeds the upper bound HQs for occupational exposures. In addition, some of the central estimates of exposure to triclopyr or TCP involving a young woman consuming contaminated vegetation or fruit also exceed the level of concern. Relative to the risks associated with the consumption of contaminated fruit or vegetation, risks associated with other exposure scenarios are marginal.

Because triclopyr has been shown to cause adverse developmental effects in mammals, the high HQs associated with terrestrial applications are of particular concern in terms of the potential for adverse reproductive outcomes in females. Adverse developmental effects in experimental mammals have been observed, however, only at doses that cause frank signs of maternal toxicity. No epidemiology studies or case reports have been encountered that associate human exposures to triclopyr with either frank signs of toxicity or developmental effects. In addition, the available toxicity studies suggest that overt and severe toxicity would not be associated with any of the upper bound HQs. This diminishes concern for reproductive effects in females. Conversely, an epidemiology study on Forest Service personnel conducted by OSHA noted a marginally significant increase in the odds ratios for miscarriages among women in the Forest Service who reported using herbicides. While this analysis does not implicate triclopyr or any other herbicide as a causative agent in miscarriages, the lack of epidemiology studies focused on females of

1 reproductive age with documented exposures to triclopyr adds uncertainty to the risk
2 characterization for terrestrial applications of triclopyr.

3 3.4.2. Workers

4 In Table 30 and the corresponding E02 worksheets for terrestrial applications (Attachments 1
5 and 2), two sets of HQs are presented for general exposures of workers —i.e., HQs based on the
6 total exposure that a worker might receive during directed foliar, broadcast ground, and aerial
7 applications —at an application rate of 1 lb/acre. One set of HQs is based on the chronic RfD of
8 0.05 mg/kg/day and the other set of HQs is based on the acute RfD of 1 mg/kg bw/day. The use
9 of the acute RfD for risk characterizations of general worker exposures is intended only to
10 illustrate the consequences of applying triclopyr sporadically as part of other activities. As
11 discussed in Section 3.3, the acute RfD of 1 mg/kg bw applies only to male workers. Risks to
12 women of childbearing age are assessed using the chronic RfD of 0.05 mg/kg bw/day regardless
13 of the duration of exposure.

14
15 As detailed in the RED, U.S. EPA/OPP (1998a), the EPA elected not to conduct quantitative risk
16 characterizations for workers according to the following rationale:

17
18 *No short- or intermediate-term risk assessment was required for handler*
19 *exposures to triclopyr because no toxicological endpoints of concern were*
20 *identified in a 21 day dermal toxicity study in rabbits at the highest dose*
21 *(1000 mg/kg/day) indicating very low toxicity via the dermal route of*
22 *exposure. ... At this time, no chronic risk assessment is required for*
23 *handler exposures to triclopyr, since none of the current handler exposure*
24 *scenarios is likely to result in chronic exposure.*

25 – U.S. EPA/OPP (1998a, p. 28).
26

27 The basis for asserting that *chronic exposure* is unlikely is not clear and may somewhat depend
28 on the definition of the term *chronic*. Clearly, occupational exposures will not occur over a full
29 lifetime. Nonetheless, as discussed in Section 3.3.2, U.S. EPA/OPP (2002) applies the *chronic*
30 RfD to exposure durations of from 1 to 6 six months up to lifetime exposures. All Forest Service
31 risk assessments consider chronic exposures for workers, and this approach is taken in the
32 current risk assessment on triclopyr.

33
34 The EPA statement that no toxicological endpoints for dermal exposure have been identified
35 appears to refer to MRID 42212701. As discussed in Section 3.1.12 and summarized in
36 Appendix 2, Table A2-2, there were no significant toxic effects in rabbits after subchronic
37 exposure to triclopyr acid at the limit dose of 1000 mg a.e./kg bw/day. U.S. EPA/OPP (1998a)
38 does not cite the subchronic dermal toxicity studies in rats using triclopyr TEA and triclopyr
39 BEE. As discussed in Section 3.1.12, triclopyr BEE appears to be about equitoxic in subchronic
40 oral and dermal studies conducted with rats. In addition, U.S. EPA/OPP (2002) appears to
41 characterize risk based on the oral RfD and assumptions concerning dermal exposure. This
42 approach, which is similar to the one taken in other Forest Service risk assessments, is taken in
43 the current risk assessment on triclopyr.

44
45 While systemic toxicity is a focus of the quantitative risk characterization for triclopyr,
46 formulations of 44.1% triclopyr TEA, such as Garlon 3A, are severe eye irritants (Appendix 1).

1 All formulations of triclopyr TEA require the use of protective eyewear. Some triclopyr BEE
2 formulations are moderate eye irritants. From a practical perspective, eye irritation is probably
3 the mostly likely effect that workers will experience during the application of triclopyr
4 formulations; furthermore, eye irritation is the only adverse effect associated with triclopyr
5 exposure in humans (Section 3.1.11). As with all pesticide applications, potential ocular and
6 dermal effects can and should be minimized or avoided by prudent industrial hygiene practices
7 during and after the application of triclopyr formulations.

8 **3.4.2.1. Triclopyr TEA Formulations**

9 **3.4.2.1.1. Terrestrial Applications**

10 The risk characterization for workers involved in terrestrial applications of triclopyr TEA
11 formulations is essentially identical, at least quantitatively, to the risk characterization given in
12 the previous Forest Service risk assessment on triclopyr (SERA 2003). As indicated in Table 30,
13 central estimates of the hazard quotient based on the chronic RfD are below the level of concern
14 (HQ=1) for all application methods at an application rate of 1 lb a.e./acre. As discussed in
15 Section 2.4, an application rate of 1 lb a.e./acre is the typical application rate used in Forest
16 Service Programs.

17
18 At the upper bounds, however, all general exposures result in HQs that are above unity—i.e.,
19 HQs ranging from 1.6 to 3. As detailed in Section 3.3.2, the RfD is based on a NOAEL of 5
20 mg/kg/day. The associated LOAEL is a factor of 5 above the NOAEL (25 mg/kg/day) and, at
21 the LOAEL, kidney damage was noted—i.e., degeneration of renal proximal tubules. Thus, the
22 projected upper bound HQs are in a region above the adjusted NOAEL (i.e., the RfD) but below
23 the corresponding LOAEL.

24
25 The above HQs apply only to the typical application rate of 1 lb a.e./acre. As discussed in
26 Section 2.4.1 and summarized in Table 5, the highest labeled application rate considered in this
27 risk assessment is 9 lbs a.e./acre, although Forest Service programs will seldom use application
28 rates in excess of 6 lbs a.e./acre. At an application rate of 9 lbs a.e./acre, all of the HQs for
29 general exposures summarized in Table 30 would be multiplied by a factor of about 9. The
30 factor of 9 is an approximation due to the rounding methods used—i.e., all rounding is done only
31 in the calculation of the final HQs. In any event, if an application rate of 9 lbs a.e./acre is used in
32 Attachment 1, the central estimates of the HQs would be in the range of 2 to 4 and the upper
33 bounds of the HQs would be in the range of 14 to 27. Since the ratio of the LOAEL to NOAEL
34 in the study used to derive the chronic RfD is a factor of 5, HQs that approach or exceed a factor
35 of 5 could be regarded as clearly unacceptable and possibly hazardous. While frank signs of
36 toxicity might not be detectable in workers, the exposures associated with the maximum
37 application rate of 9 lbs a.e./acre are nonetheless undesirable. At an application rate of 6 lbs
38 a.e./acre, the highest rate that would typically be used in Forest Service programs, the central
39 estimates of the HQs would be in the range of 1.6 to 3 and the upper bounds of the HQs would
40 be in the range of 10 to 18.

41
42 The verbal interpretation of these HQs for general exposures is somewhat ambiguous. Under
43 typical conditions of application and at the typical application rate of 1 lb/acre, there is no
44 indication that workers will be subject to hazardous levels of triclopyr at the central estimates of
45 exposure. Nonetheless, at the upper range of exposures, all application methods exceed the level

1 of concern, based on the chronic RfD. At higher application rates, particularly rates that
2 approach the maximum application rate of 9 lbs a.e./acre, concerns would be substantially
3 greater.

4
5 All of the above discussion applies to longer-term exposures using the chronic RfD. Based on
6 the acute RfD, none of the HQs for general exposures reach or approach a level of concern. At
7 the highest application rate of 9 lbs a.e./acre, only the HQ for ground broadcast applications
8 exceeds the level of concern with an HQ of 1.4. This is a relatively modest exceedance. HQs
9 are often simply rounded to one significant place, and the HQ of 1.4 would round to an HQ of
10 1—i.e., at the level of concern. The verbal interpretation of the risk characterization for
11 infrequent applications of triclopyr TEA formulations is unambiguous: There is no indication
12 that the infrequent application of triclopyr TEA formulations will be associated with identifiable
13 risks to male workers. As noted in Section 3.3, however, the acute RfD is not applied to women
14 of child bearing age, and the chronic RfD of 0.05 mg/kg/day is used.

15
16 Accidental exposures of workers to formulations containing triclopyr TEA do not lead to HQs
17 that exceed a level of concern, based on the acute RfD of 1 mg/kg/day. Using the chronic RfD
18 of 0.05 mg/kg bw for women, none of the HQs for accidental scenarios for triclopyr TEA
19 formulations exceed a level of concern at an application rate of 1 lb a.e./acre. The highest HQ at
20 1 lb a.e./acre is 0.02, which is associated with wearing contaminated gloves for 1 hour. Thus, if
21 a female worker were to wear contaminated gloves for 2½ hours, the HQ would reach the level
22 of concern. At the application rate of 9 lbs a.e./acre, none of the accidental HQs reach a level of
23 concern for male workers. The accidental scenarios for wearing contaminated gloves for 1 hour
24 as well as 1-hour exposures resulting from spills onto the lower legs reach upper bound HQs of
25 0.1, using the acute RfD of 1 mg a.e./kg bw/day. Using the RfD of 0.05 mg/kg bw/day for
26 female workers would correspond to an HQ of 2.

27 **3.4.2.1.2. Aquatic Applications**

28 Two EXCEL workbooks are provided for aquatic applications of triclopyr TEA formulations,
29 application to emergent vegetation (Attachment 3) and applications to submergent vegetation
30 (Attachment 4). The two workbooks are provided largely as a convenience for Forest Service
31 personnel who may wish to modify or otherwise use these workbooks directly. The exposures
32 and consequent risks are essentially identical for both types of applications with only marginally
33 higher HQs for applications to submergent vegetation. Thus, as a matter of economy and
34 simplicity, only Attachment 4 is discussed.

35
36 At the target concentration of 1 ppm (1 mg a.e./L), all of the accidental exposure scenarios are
37 substantially below the level of concern. The highest accidental HQ is 0.04, below the level of
38 concern by a factor of 20. At that highest labeled target concentration of 2.5 ppm, the highest
39 accidental HQ would be 0.1, below the level of concern by a factor of 10.

40
41 Based general exposures, the upper bound HQ is 0.5. As discussed in Section 3.2.2.1.2, there is
42 substantial uncertainty regarding the exposure assessment for workers involved in aquatic
43 applications of triclopyr because of the scant amount of useful data with which to estimate
44 worker exposure levels. Nonetheless, based on the information that is available, there is no basis
45 for asserting that workers involved in applications of triclopyr TEA at a target application rate of
46 1 ppm will be at risk. At the maximum target application rate of 2.5 ppm, the upper bound HQ

1 would be 1.3, a modest excursion above the level of concern (HQ=1). Like the HQs based on
2 the acute RfD for terrestrial applications of triclopyr TEA, the HQ of 1.3 is not of substantial
3 concern and could be viewed as an HQ of 1 when rounded to the nearest integer.

4
5 While the absence of risk cannot be proven, and efforts should always be made to minimize
6 exposures, there is no apparent basis for asserting that workers involved in aquatic applications
7 of triclopyr TEA will be at any identifiable risk, even if applications are made over a prolonged
8 period of time. While not quantitatively considered, HQs based on the acute RfD and applied to
9 male workers involved in short-term and infrequent aquatic applications would be far below a
10 level of concern.

11 **3.4.2.2. Triclopyr BEE Formulations**

12 As discussed in the previous subsection, the HQs for workers applying triclopyr TEA
13 formulations are identical to those given in the previous Forest Service risk assessment (SERA
14 2003). In the 2003 risk assessment, the HQs for workers applying triclopyr BEE formulations
15 were identical to those of workers applying triclopyr TEA formulation. As discussed in Section
16 3.2.2.1.1 and summarized in Table 18, the current risk assessment uses higher worker exposure
17 rates for applications of triclopyr BEE formulations, based on a consideration of the worker
18 exposure studies sponsored by the Forest Service on directed foliar backpack applications of
19 triclopyr BEE (Middendorf 1992b and 1992b; Krieger et al. 2005; Spencer et al. 2000). The use
20 of higher worker exposure rates for triclopyr BEE leads to upper bound estimates of exposures
21 and consequent upper bound estimates of the HQs for triclopyr BEE which are higher than those
22 for triclopyr TEA. Based on the chronic RfD and the typical application rate, the upper bound
23 HQs of 6 to 12 require little elaboration. Based on the acute RfD, the upper bound HQs of 0.3 to
24 0.6 are of below the level of concern at an application rate of 1 lb a.e./acre. For higher
25 application rates, however, concern would increase linearly.

26
27 For many pesticides, the risk characterization for workers is often associated with substantial
28 uncertainty due to the exposure assessment methods used. As discussed in Section 3.4.2.1, such
29 is clearly the case for workers involved in applications of triclopyr TEA. For triclopyr BEE,
30 however, there is little uncertainty in the exposure assessment, as illustrated in Figure 9. In this
31 figure, the doses (not dose-rates) based on biomonitoring from the studies by Middendorf
32 (1992b) and Spenser et al. (2000) are divided by the acute RfD (upper plot in Figure 9) and
33 chronic RfD (lower plot in Figure 9), and the resulting HQs are shown as relative frequency
34 histograms as well as the fit of the data to a lognormal distribution. The combined data from
35 these studies offers a good fit to the lognormal distribution using either the chi-square ($p>0.33$)
36 or Kolmogorov-Smirnov ($p>0.83$) tests.

37
38 The studies by Middendorf (1992b) and Spenser et al. (2000) are directly applicable to the
39 current risk assessment. These studies were funded by the Forest Service and intended to assess
40 realistic worker exposures during backpack foliar applications typical of those used in Forest
41 Service programs. As illustrated in the upper plot based on the acute RfD, there is no indication
42 that male workers involved in the frequent application of triclopyr BEE are at risk. Based on the
43 fit to the lognormal distribution, the probability of a male worker being exposed to levels that
44 exceed the acute RfD is remarkably low ($p<0.000181$ or about 1 in over 5000). Based on the
45 chronic RfD, 12 of the 46 workers (26%) had doses that exceeded the RfD (HQ>1). Based on

1 the fit to the lognormal distribution, HQs for 5% of workers would be expected to be greater than
2 2.66. Based on the worker data, HQs for 2 of 46 workers (4.3%) were greater than 2.66.

3
4 Qualitatively, the risk characterization for workers applying triclopyr BEE at the typical
5 application rate of 1 lb a.e./acre is similar to those for applications of triclopyr TEA. The upper
6 bounds of the HQs based on the chronic RfD exceed the level of concern for all application
7 methods that are considered, including direct foliar backpack, ground broadcast foliar, and aerial
8 applications. The major difference between the risk characterization for triclopyr BEE and TEA,
9 however, involves the interpretation of the upper bound HQs. As noted in Section 3.4.2.1.1 and
10 summarized in Table 29, the chronic RfD is based on a study in which the LOAEL is a factor of
11 5 above the NOAEL. For triclopyr TEA, the projected upper bound HQs are in a region above
12 the adjusted NOAEL (i.e., the RfD) but below the corresponding adjusted LOAEL. For triclopyr
13 TEA the consequent interpretation of the HQs is ambiguous. For triclopyr BEE, the upper bound
14 HQs are in the range of 6 to 12 and the interpretation is unambiguous. Some workers applying
15 triclopyr BEE at the typical application rate of 1 lb a.e./acre will be subject to exposures that
16 exceed the chronic RfD by a substantial margin. By analogy to the dose-response data from the
17 study on which the RfD is based, the development of subclinical adverse effects could not be
18 ruled out.

19
20 Overt toxic effects in workers do not appear to be likely. As summarized in Section 3.1, there
21 are no epidemiology studies or case reports which suggest that systemic toxic effects are
22 associated with occupational or even accidental exposures to any form of triclopyr; furthermore,
23 no poisoning reports involving any form of triclopyr are documented in the reasonably
24 comprehensive summary of human case reports on pesticide exposures by Hayes (1982).

25 **3.4.3. General Public**

26 An overview of the HQs for members of the general public associated with terrestrial
27 applications of triclopyr is given in Table 31. This table summarizes the HQs that approach or
28 exceed the level of concern (HQ=1) as well as a summary of HQs for scenarios that are
29 substantially below the level of concern. A full set of HQs for the general public are given in
30 Worksheet E04 in the EXCEL Workbooks (Attachments 1 through 5) that accompany this risk
31 assessment.

32
33 As with workers, risk is characterized quantitatively as the HQ, the estimated exposure divided
34 by the appropriate RfD. Also as with workers, all HQs for longer-term exposure are based on
35 the chronic RfD of 0.05 mg/kg/day. For acute exposures involving a child or man, the HQs are
36 based on the acute RfD of 1 mg/kg/day for the general population. This acute RfD is not used
37 for women of childbearing age, and all HQs for acute exposure involving a woman are based on
38 the chronic RfD of 0.05 mg/kg/day. As discussed in Section 3.3, U.S. EPA/OPP (2002)
39 recommends this approach for women of childbearing age.

40
41 The risk characterization for aquatic applications of triclopyr TEA formulations is uneventful
42 and not detailed further except to note that the only accidental exposure scenario that exceeds a
43 level of concern is upper bound HQ for the accidental spill of the formulation into a small pond.
44 The highest upper bound HQ for non-accidental exposure scenarios is 0.04, below the level of
45 concern by a factor of 25. For applications of triclopyr TEA to control submergent vegetation,

1 the upper bound HQ for a swimmer modestly exceeds the level of concern (i.e., HQ=1.9,
2 Attachment 7, Worksheet E04).

3
4 As summarized in Table 31, risks associated with terrestrial applications of triclopyr TEA and
5 triclopyr BEE are identical for many exposure scenarios. For exposure scenarios involving
6 dermal absorption, the risks associated with triclopyr BEE formulations are only modestly
7 greater than those for triclopyr TEA formulations.

8
9 The only exposure scenarios of substantial concern involve the consumption of contaminated
10 vegetation, and these risks do not differ between TEA and BEE formulations of triclopyr.
11 Scenarios of concern involving exposures to 3,5,6-trichloro-2-pyridinol (TCP) are also limited to
12 the consumption of contaminated vegetation.

13
14 The upper bound of the acute exposure scenario for the consumption of contaminated vegetation
15 by a young woman is 27, exceeding the corresponding upper bounds for general exposures in
16 workers applying triclopyr BEE based on chronic RfD—i.e., HQs of 11 to 22 as summarized in
17 Table 30.

18
19 Potential exposures to the 3,5,6-trichloro-2-pyridinol (TCP) metabolite of triclopyr also exceed
20 the level of concern at the upper bound of the HQs for both the acute and longer-term
21 consumption of contaminated vegetation and fruit. For TCP, the upper bound of HQs for acute
22 exposures is less than the upper bound of the HQs for longer-term exposures. For the central
23 estimates and the lower bounds, the opposite pattern is apparent. While this may seem
24 incongruous, the calculations are correct and reflect the interplay of the lower chronic RfD and
25 the different half-lives used to estimate the longer-term time-weighted average doses. As
26 indicated in Worksheet E03 of Attachment 5 (the EXCEL workbook for TCP), the 90-day time-
27 weighted average doses for TCP are below the estimated acute doses of TCP.

28
29 The qualitative interpretation of the HQs for TCP is similar to that of the HQs for triclopyr. For
30 TCP, the LOAEL associated with the acute RfD is a factor of 4 higher than the NOAEL on
31 which the RfD is based. As with the discussion of the reproductive NOAELs and LOAELs for
32 triclopyr, this ratio does not indicate that adverse reproductive effects would be predicted in
33 humans at an acute HQ of 4; however, the relationship of the NOAELs to LOAELs in the animal
34 studies does enhance concern for HQs in the range of 4. For TCP, the upper bound acute HQs
35 range from 2 to 15.

36
37 The above discussion focuses on the upper bounds of the HQs. The upper bound HQs are based
38 on very conservative exposure assumptions including the upper bound estimates of food
39 consumption and upper bound estimates of residue rates. For TCP, the conservative nature of
40 the upper bound estimates is compounded by the use of upper bound half-lives. The use of
41 several *worst-case* or at least very conservative assumptions in multiplicative models leads to
42 assessments in which risks may be unrealistically magnified. As discussed in Section 3.2.3.1.1
43 (Likelihood and Magnitude of Exposure), the conservative nature of the upper bound
44 assessments is intentional and intended to encompass risks to the *Most Exposed Individual*.

1 As also discussed in Section 3.2.3.1.1, Forest Service risk assessments use an Extreme Value
2 approach which also estimates the central estimates and lower bounds of exposure and risk. The
3 central estimates of HQs are intended to reflect exposures that are expected using typical values
4 for consumption rates and other inputs. The central estimates of the HQs for the consumption of
5 contaminated vegetation exceed or reach the level of concern (HQ=1) for acute exposures to
6 triclopyr (HQ=3) acute exposures to TCP (HQ=1.8), and chronic exposures to TCP (HQ=1). All
7 of these HQs pertain to the typical application rate of 1 lb a.e./acre and would increase linearly as
8 the application rate increases.

9
10 Finally, as discussed in Section 3.2.3.1.1, lower bounds of exposures are used as *best case*
11 estimates and are generally intended to represent the feasibility of risk mitigation. At an
12 application rate of 1 lb a.e./acre, the lower bound of the HQ for the exposure scenario involving a
13 young woman consuming vegetation contaminated with triclopyr is 0.2. Like all the other HQs,
14 this one will scale linearly with the application rate. Thus, the lower bound HQ for this exposure
15 scenario would reach a level of concern at an application rate of 5 lbs a.e./acre. At the maximum
16 application rate of 9 lbs a.e./acre, the HQ would be 1.8.

17 **3.4.4. Sensitive Subgroups**

18 **3.4.4.1. Women of Childbearing Age**

19 Triclopyr is associated with adverse reproductive effects in experimental mammals (Table 10).
20 While there are no epidemiology studies supporting a link between exposure to triclopyr and
21 adverse reproductive outcomes in humans, reproductive toxicity is an endpoint of particular
22 concern in Forest Service risk assessments. At the request of the Forest Service, the National
23 Institute of Occupational Safety and Health conducted a health hazard evaluation of Forest
24 Service personnel (Driscoll et al. 1998). The study by Driscoll et al. (1998) was designed
25 primarily to assess risks adverse reproductive effects associated with the use of tree-marking
26 paints. Other possible risk factors, including herbicide use, were considered in the study.
27 Driscoll et al. (1998) noted a marginally significant increase in the odds ratios for miscarriages
28 among women in the Forest Service who reported using herbicides— i.e., an odds ratio adjusted
29 for other competing risks of 1.82 with 95% confidence intervals of 1.00 to 3.32 (Driscoll et al.
30 1998, Table 4, p. 45). While self-reporting bias is a general concern in epidemiology studies, no
31 indication of self-reporting bias in women concerning exposures to chemical agents following a
32 miscarriage was noted in the study by Farrow et al. (1996). Nonetheless, the analysis by Driscoll
33 et al. (1998) does not implicate triclopyr or any other herbicide as a causative agent in
34 miscarriages. No epidemiology studies specifically focused on assessing an association of
35 exposure to triclopyr with adverse reproductive outcomes have been identified.

36
37 Given some of the very high chronic HQs, particularly for workers applying triclopyr BEE and
38 members of the general public who might consume contaminated vegetation, the potential for
39 adverse reproductive outcomes in females is an obvious concern. As summarized in Table 10,
40 there are many developmental studies on triclopyr, and these studies consistently indicate that
41 adverse effects on the developing fetus occur only at doses that cause frank signs of maternal
42 toxicity. Triclopyr has been used as a herbicide for more than 30 years (Section 2.2), and
43 continues to be used extensively by the Forest Service (Section 2.4). As discussed in Section
44 3.4.2.2.1, no reports of frank adverse effects in workers (male or female) applying any triclopyr
45 formulation are included in the available literature. In the occupational exposure studies on

1 terrestrial applications of triclopyr (Section 3.2.2.1.1), the highest reported dose for a worker
2 applying triclopyr is about 50.04 mg a.e. — i.e., Worker 5 in the study by Gosselin et al. (2005,
3 Table 4, p. 420). Assuming a body weight of 70 kg, this corresponds to a dose of about 0.7 mg
4 a.e./kg bw. This in turn corresponds to an HQ of about 14. No signs of even mild toxicity in
5 workers are reported in the study by Gosselin et al. (2005) or in any of the other worker exposure
6 studies. All of these studies involve male rather than female workers; however, there is no basis
7 for asserting that frank adverse effects in female workers would be expected at doses
8 substantially below those which might be associated with frank adverse effects in male workers.
9

10 The most reasonable interpretation of the risk characterization for female workers applying
11 triclopyr as well as females in the general public who might consume food items contaminated
12 with triclopyr or TCP is that the exposure assessments clearly indicate that some females could
13 be exposed to triclopyr or TCP levels that are clearly of concern — i.e., above the RfD. Based on
14 the available developmental studies on triclopyr as well as human experience with triclopyr, it is
15 far less certain that adverse reproductive outcomes due to the toxicity of triclopyr would occur.
16 Epidemiology studies on women of childbearing age with documented exposures to triclopyr
17 could be useful in better assessing the potential risks of adverse reproductive outcomes.

18 **3.4.4.2. Other Subgroups**

19 Triclopyr is excreted primarily by the kidney (Section 3.1.2). Individuals with kidney disease
20 could have an impaired ability to excrete triclopyr. No reports, however, linking triclopyr
21 exposures with adverse effects in individuals with kidney disease were identified in the available
22 literature.
23

24 Some individuals report a high degree of sensitivity to multiple chemicals, resulting in a broad-
25 spectrum of effects, many of which are similar to allergic reactions. This condition is generally
26 referred to as *Multiple Chemical Sensitivity* (e.g., ATSDR 1995). There are no reports in the
27 literature associating exposures to triclopyr with adverse effects in individuals who report having
28 Multiple Chemical Sensitivity.

29 **3.4.5. Connected Actions**

30 The Council on Environmental Quality (CEQ), which provides the framework for implementing
31 NEPA, defines connected actions (40 CFR 1508.25) as actions which occur in close association
32 with the action of concern; in this case, pesticide use. Actions are considered to be connected if
33 they: (i) Automatically trigger other actions which may require environmental impact statements;
34 (ii) Cannot or will not proceed unless other actions are taken previously or simultaneously, and
35 (iii) Are interdependent parts of a larger action and depend on the larger action for their
36 justification. Within the context of this risk assessment, “connected actions” include actions or
37 the use of other chemicals which are necessary and occur in close association with use of
38 triclopyr.
39

40 As discussed in detail in 3.1.15 (Impurities and Metabolites), triclopyr will be metabolized to
41 3,5,6-trichloro-2-pyridinol (TCP). Exposures to TCP will be associated with any use of triclopyr
42 in Forest Service programs. The impact of TCP is considered quantitatively in the human health
43 risk assessment and is considered further in the ecological risk assessment.
44

1 The U.S. EPA (1998a, 2002) has conducted extensive analyses of dietary exposure to TCP from
2 the use of triclopyr as well as the aggregate risks from exposure to TCP from the use of both
3 triclopyr and chlorpyrifos. While the dietary exposures estimated by the EPA are substantially
4 below a level of concern, the EPA risk assessment does not consider the types of oral exposures
5 routinely considered in Forest Service risk assessments.

6
7 Adjuvants, as discussed in Section 3.1.14, are a much more difficult issue to address, and it is
8 beyond the scope current risk assessment to address adjuvants in detail. This is a general issue in
9 all Forest Service risk assessments. Nonetheless, many formulations of triclopyr TEA require
10 the use of surfactants, and some surfactants may be hazardous.

11 **3.4.6. Cumulative Effects**

12 Similar to the issues involved in assessing the use of adjuvants, it is beyond the scope of the
13 current risk assessment to identify and consider all agents that might interact with, or cause
14 cumulative effects with triclopyr, and to do so quantitatively would require a complete set of risk
15 assessments on each of the other agents to be considered.

16
17 Notwithstanding the above statement, triclopyr is a relatively typical weak-acid auxin herbicide.
18 Herbicides such as aminopyralid, clopyralid, and picloram are similar with respect to their
19 structure, pharmacokinetics, and toxicity. It is reasonable to anticipate that exposure to triclopyr
20 and other weak acid herbicides would result in essentially additive risks.

21
22 The effect of repeated exposures to triclopyr for both workers and members of the general public
23 is considered explicitly in the current Forest Service risk assessment. Accordingly, the risk
24 characterizations presented in this risk assessment specifically address and encompass the
25 potential impact of the cumulative effects of repeated exposures to triclopyr.

4. ECOLOGICAL RISK ASSESSMENT

4.1. HAZARD IDENTIFICATION

4.1.1. Overview

As in the human health risk assessments, the hazard identification for nontarget organisms is concerned with triclopyr acid, triclopyr TEA, and triclopyr BEE, in addition to the 3,5,6-trichloro-2-pyridinol (TCP) a metabolite of triclopyr. In terrestrial animals, triclopyr TEA and triclopyr BEE appear to be bioequivalent to triclopyr. For terrestrial plants and most groups of aquatic organisms, however, triclopyr BEE is much more toxic than triclopyr TEA or triclopyr acid. The only exception to this generalization involves aquatic macrophytes. In this group of organism, triclopyr TEA appears to be more toxic than triclopyr BEE. TCP is a concern in the ecological risk assessment because it is more toxic than triclopyr (including triclopyr BEE, triclopyr TEA, and triclopyr acid) to most groups of nontarget organisms.

The ecological risk assessment is also concerned with differences in species sensitivity. Triclopyr is an effective herbicide. Triclopyr TEA, however, is more toxic to dicots than monocots. This is the case with both terrestrial and aquatic plants. Differences in the toxicity of triclopyr BEE to dicots and monocots is less pronounced. In terms of toxicity to animals, few systematic differences in species sensitivity are apparent. A clear exception, however, involves mammals. Based on very clear and consistent patterns in both subchronic and chronic studies involving dietary exposures, allometric relationships indicate that sensitivity to triclopyr is greater in larger mammals. The only other apparent species difference involves the toxicity of triclopyr BEE to aquatic invertebrates. Based on acute bioassays, daphnids appear to be more sensitive than aquatic insects with other aquatic arthropods displaying intermediate sensitivity. While there is substantial variability in the results of acute bioassays in other groups of organisms, this variability does not appear to reflect systematic differences among species.

4.1.2. Terrestrial Organisms

4.1.2.1. Mammals

4.1.2.1.1. Triclopyr

As summarized in the human health risk assessment (Section 3.1), several standard toxicity studies in experimental mammals were conducted and submitted to the EPA as part of the registration process for triclopyr. In addition, toxicity studies involving the exposure of mammals to triclopyr were conducted and are published in the available literature. All of these studies, which are used in the human health risk assessment to identify the potential toxic hazards associated with exposures to triclopyr, can also be used to identify potential toxic effects in wildlife mammalian species.

As summarized in Section 3.1.2, in experimental mammals exposed to triclopyr, the kidney appears to be the primary target tissue. In the absence of data on most wildlife species, it seems reasonable to assume that the kidney will also be the primary target organ in mammalian wildlife. As detailed in Section 3.1.9, reproductive effects associated with exposure to triclopyr are investigated in numerous toxicity studies. Although triclopyr causes developmental effects

1 only at doses that cause maternal toxicity, reproductive effects are obviously an endpoint of
2 concern to both the human health and ecological risk assessments.

3
4 The acute oral LD₅₀ values in rats (Appendix 2, Table A2-1) for the different forms of
5 triclopyr—i.e., triclopyr acid, triclopyr TEA and triclopyr BEE—fall within overlapping ranges,
6 indicating no apparent difference in toxicity. Moreover, all of the oral LD₅₀ values span a
7 narrow range from 594 to 828 mg a.e./kg bw.

8
9 The identification of sensitive species and systematic differences in sensitivity among species of
10 mammalian wildlife is an important concern in the hazard identification. The acute toxicity data
11 are not sufficient to identify patterns of sensitivity among wildlife species. Other than the oral
12 LD₅₀ values in rats, the only acute oral toxicity data are reported in the study by Osweiler (1983)
13 in which 4 daily gavage doses at 60 mg a.e./kg bw did not cause signs of toxicity in ponies, while
14 4 daily doses of 300 mg/kg bw/day caused death in two of six ponies. Although designed to
15 assess acute toxicity, this study is not directly comparable to the single gavage dose studies in
16 rats.

17
18 As discussed in Section 3.1.2, the impaired ability of dogs to excrete weak acids makes them
19 more sensitive than other mammals to some weak acids and, perhaps, to triclopyr. Although the
20 acute oral LD₅₀ data do not support this supposition because acute oral LD₅₀ studies are not
21 typically conducted in dogs and no such studies are available on triclopyr, there are useful
22 subchronic and chronic toxicity studies available in mice, rats, and dogs. Details of these studies
23 are given in Table A2-8 of Appendix 2 and an overview of these studies is presented in Table 32
24 of this risk assessment. Table 32 summarizes both NOAEL and LOAEL values from the
25 subchronic and chronic studies. Based on a casual observation of these values, dogs appear to be
26 more sensitive to triclopyr (i.e., evidence adverse effects at lower doses) than rats, and rats
27 appear to be more sensitive than mice.

28
29 Allometric relationships, which are used extensively in the exposure assessment for triclopyr, are
30 sometimes apparent for sensitivity among species. In the biological sciences, allometry is the
31 study of the relationship of body size or mass to various anatomical, physiological, or
32 pharmacological parameters (e.g., Boxenbaum and D'Souza 1990). Allometric relationships take
33 the general form:

Equation 8

$$Y = \alpha W^\beta$$

34
35
36
37 *W* is the weight of the animal, *Y* is the variable to be examined, and the model parameters are
38 designated by alpha (α) and beta (β). If *Y* decreases with body weight, β is negative. If *Y*
39 increases with body weight, β is positive. If there is no relationship of *Y* to body weight, β is
40 near to or at least not significantly different from zero.

41
42 Both the NOAELs and LOAELs in Table 32 suggest that these values decrease with increasing
43 body weight—i.e., larger animals are more sensitive than small animals, and β is negative.
44 NOAELs and LOAELs, however, are not well-suited to statistical analyses. As an alternative,
45 the geometric means of the NOAELs and LOAELs are used. This compositing, sometimes
46 referred to a Maximum Acceptable Tolerance Concentration (MATC) or Maximum Tolerated

1 Dose (MTD), is an estimate of a dose that may represent a threshold for a response. To examine
2 this possibility, typical body weights for mice, rats, and dogs are taken from U.S. EPA/ORD
3 (1988), which provides representative average body weights for mice, rats, and dogs in both
4 subchronic and chronic studies. These body weights along with the geometric means of the
5 NOAELs and LOAELs were fit to the general allometric equation given above.

6
7 As summarized in Table 32 and illustrated in the upper graph in Figure 10, the fit to the
8 allometric function is statistically significant for both the subchronic ($p=0.032$) and chronic
9 ($p=0.037$) data. In these analyses, the means for males and females are generally combined so
10 that the significance of the relationship between body weights and the geometric means is not
11 artificially inflated. The only exception is with the NOAELs and LOAELs for male and female
12 rats from the Barna-Lloyd et al. (1992) study in which the NOAELs and LOAELs for female rats
13 are substantially higher than those for males. In the chronic data set, the data on female mice
14 from Tsuda et al. (1987) is omitted because the dose which could be designated as a LOAEL,
15 135 mg/kg bw/day, caused only a modest increase in kidney weight with no corresponding
16 pathology. The omission of these data has no impact on the analysis.

17
18 The statistically significant fit of the chronic data to the allometric equation is somewhat
19 remarkable in that only three data points are available. In addition, each of the three points
20 reflects the same response in mice, rats, and dogs—i.e., kidney pathology. The subchronic data
21 are somewhat problematic. As indicated in Table 32, the fit to the allometric function is
22 statistically significant only if the data on the inhibition of phenolsulfonphthalein (PSP) excretion
23 by dogs are included. As discussed in Section 3.1.2, the excretion of phenolsulfonphthalein
24 (a.k.a. phenol red) is a classic test for kidney function and can indicate kidney damage in
25 humans. Dogs, however, have a limited ability to excrete weak acids through saturable active
26 transport processes in the kidney. Following the administration of a weak acid, such as triclopyr,
27 a reduction in PSP excretion may reflect a transient saturation of active transport rather than a
28 direct toxic effect on the kidney. Whether this competition alone and in the absence of kidney
29 pathology should be regarded as an adverse toxic effect on the kidney is arguable (e.g., Timchalk
30 et al. 1997). In terms of the allometric relationship, the exclusion of the two points involving
31 PSP excretion in dogs (Quast et al. 1976, 1977) together with the scatter in the subchronic data
32 on rats results in a lack of significant fit to the allometric model ($p=0.28$).

33
34 As discussed in Section 3.1.3.3, the available pharmacokinetic data on triclopyr applied to the
35 plateau principle (Eq. 3) indicate that triclopyr will not accumulate in mammals on repeated
36 dosing. This, in turn, suggests that the subchronic and chronic toxicities of triclopyr should not
37 differ substantially in the absence of a mechanism indicating cumulative damage with slow or
38 negligible rates of repair. Based on the subchronic and chronic toxicity data summarized in
39 Table 32, the lack of a strong temporal relationship seems apparent, particularly since the lower
40 chronic toxicity values, relative to subchronic toxicity values in dogs could be explained by
41 differences in body weight. This supposition is supported by a multiple regression analysis using
42 the natural logarithm of the duration of exposure as an additional explanatory variable. In this
43 reanalysis, duration is not significant ($p=0.774$). Consequently, the subchronic and chronic data
44 were combined, eliminating the data on PSP inhibition in dogs, and refit to the allometric
45 function.

1 As indicated in the Table 32 and illustrated in the bottom graph of Figure 10, the subchronic data
2 (excluding the PSP endpoints) combined with the chronic data illustrate a clear allometric
3 relationship ($p=0.013$) suggesting that larger mammals are more sensitive than smaller mammals
4 ($\beta\sim 0.5$) to triclopyr. A major uncertainty with this analysis, however, is that the allometric
5 relationship includes and is substantially dependent upon the from studies in dogs (i.e., Quast et
6 al. 1976,1977, and 1988). As noted above, have a limited ability to excrete weak acids and it is
7 not clear that the allometric relationships would be significant is data were available on larger
8 non-canid species. Nonetheless, in the absence of data on larger non-canid species, it seems
9 appropriate and conservative to assume that larger mammals are more sensitive to triclopyr than
10 smaller mammals. Consequently, as discussed further in Section 4.3.2.1.1, separate dose-
11 response relationships are derived for small and large mammals.

12
13 As summarized in Appendix 2, Table A2-10, the available literature includes several field
14 studies regarding the impact of triclopyr applications on mammalian wildlife populations. These
15 studies note secondary effects on mammals due to changes in vegetation composition (e.g.,
16 McMurry et al 1993a,b, 1994; Miller et al. 1999). The available studies on mammalian wildlife,
17 however, do not report adverse effects which might be attributed to the toxicity of triclopyr.

18 **4.1.2.1.2. TCP**

19 Like the human health risk assessment (Section 3), the ecological risk assessment is concerned
20 with the toxicity of 3,5,6-trichloro-2-pyridinol (TCP), a major metabolite of triclopyr. Since the
21 data on the toxicity of TCP to mammals is less extensive than that on triclopyr, the current risk
22 assessment relies heavily on the EPA review of unpublished studies on TCP (U.S. EPA/OPP
23 2002b). These data are discussed further in the dose-response assessment for TCP (Section
24 4.3.2.1.2).

25 **4.1.2.2. Birds**

26 **4.1.2.2.1. Triclopyr**

27 Information on the toxicity of triclopyr to birds is summarized in Appendix 3. The most relevant
28 data for this risk assessment are the standard acute dietary and bird reproduction studies as well
29 as the acute oral LD₅₀ studies, all of which are required for pesticide registration.

30 **4.1.2.2.1.1. Acute Gavage**

31
32 In avian toxicity studies, the acute LD₅₀ values for gavage administration of triclopyr range from
33 529 to 1698 mg a.e./kg (Appendix 3, Table A3-1). The lower bound of this range is very similar
34 to the lower bound of the range of LD₅₀ values in rats—i.e., 594 mg a.e./kg bw; while, the upper
35 bound of the range is modestly higher than the upper bound of the corresponding range in
36 mammals—i.e., 828 mg a.e./kg bw.

37
38 With mallard ducks, as with mammals, the acute oral toxicity of triclopyr acid (LD₅₀=1698 mg
39 a.e./L) and triclopyr TEA (LD₅₀≈1418 mg a.e./L) are not substantially different. On the other
40 hand, with bobwhite quail, the gavage LD₅₀ values for triclopyr BEE are lower by a factor of
41 about 2.5—i.e., 611 mg a.e./kg bw for Garlon 4 and 529 mg a.e./kg bw for technical grade
42 triclopyr BEE. The similarities in gavage oral LD₅₀ values in bobwhites for triclopyr BEE and
43 Garlon 4 suggest that the inerts in Garlon 4 do not have a substantial impact on the acute oral

1 toxicity of triclopyr BEE to birds. Based on the gavage oral LD₅₀ values available on the various
2 forms of triclopyr, the U.S. EPA/OPP (1998a) classifies triclopyr as being slightly toxic to birds.

3 4 **4.1.2.2.1.2. Acute Dietary**

5 As also summarized in Appendix 3 (Table A3-2), several acute dietary studies have been
6 conducted on triclopyr acid, triclopyr TEA, and triclopyr BEE (Garlon 4). These are standard
7 studies in which birds are fed the test agent for 5 days followed by an 8-day recovery period.
8 The results of these studies are typically reported as definitive LD₅₀ values, if sufficient mortality
9 occurs; otherwise, the results are reported as non-definitive LD₅₀ values—i.e., a greater than
10 concentration. Most of these acute dietary studies were conducted in either northern bobwhite
11 quail or mallards; one acute dietary study was conducted in Japanese quail (Norris 1973). The
12 only other related study is Holmes et al. (1994), published in the open literature, in which
13 triclopyr BEE was administered in the diet of zebra finches for 8-28-days.

14
15 As with bioassays in other terrestrial species, triclopyr acid, TEA, and BEE appear to be
16 bioequivalent. The reported LC₅₀ values for triclopyr acid (three assays reporting LC₅₀ values
17 ranging from 2934 to 5620 ppm a.e.) are similar to those reported for triclopyr TEA (2 assays
18 reporting LC₅₀ values of 3000 and >4465 ppm a.e.) and triclopyr BEE (four assays reporting
19 LC₅₀ values ranging from about 3885 to >6889 ppm a.e.). The reported LC₅₀ values for
20 triclopyr acid and triclopyr TEA suggest that mallards may be somewhat less sensitive than
21 quail—i.e., LC₅₀ values in mallards of 5620 and >4465 ppm a.e. and LC₅₀ values in quail of
22 2934 to 5189 ppm a.e. Nonetheless, the lack of definitive LC₅₀ values for the mallard in the
23 assay on triclopyr TEA makes the comparison tenuous.

24
25 The data on triclopyr BEE, however, offer a somewhat clearer comparison. Both of the acute
26 LC₅₀ values in mallards are non-definitive (i.e., >3385 and >6689 ppm a.e). Conversely, both of
27 the LC₅₀ values in quail are definitive (i.e., 3885 and 6495 ppm a.e) and clearly suggest that
28 mallards are less sensitive than quail. The dietary study on triclopyr BEE in zebra finches
29 suggests that this species is more sensitive than either mallards or quail to triclopyr BEE. As
30 detailed in Appendix 3 (Table A3-2), Holmes et al. (1994) reports a dietary LC₅₀ of about 1383
31 ppm a.e. in zebra finch.

32
33 The higher sensitivity of finch to triclopyr BEE does not appear to be an artifact of food
34 consumption. Holmes et al. (1994) do not report food consumption values, but according to the
35 study (Table 2, p. 322), the control birds weighed about 13.5 g, the finch consuming the
36 approximate LC₅₀ dose (i.e., the 1800 ppm a.i. exposure group) weighed approximately 14% less
37 than control birds after an 8-day exposure—i.e., 13.64 g vs 11.77 g. Using the food consumption
38 algorithm from U.S. EPA/OPP (1993)—as detailed in Appendix 3, Table A3-2—the food
39 consumption for the control birds can be estimated at about 3.6 g/day or about 27% of body
40 weight/day. Because the birds at the LC₅₀ dose lost weight, and Holmes et al. (1994) report that
41 this weight loss was associated with an unspecified reduction in food consumption, a reasonable
42 estimate of food consumption relative to body in these birds is about 0.23 [0.27 x (1-0.14) ≈
43 0.2322]. Using this approximation, the LC₅₀ of 1383 ppm a.e. corresponds to a dose of about
44 318 mg a.e./kg bw [1383 mg a.e./kg food x 0.23 kg food/kg bw ≈ 318.09 mg a.e./kg bw]. This
45 LD₅₀ is about 5 times lower than the estimated LD₅₀ in quail [1708 mg a.e./kg bw ÷ 318 mg
46 a.e./kg bw ≈ 5.37]. Passerines may be more sensitive than game birds or waterfowl to triclopyr;

1 however, in the absence of a systematic allometric relationship among species and other
2 confirming studies, the apparently higher sensitivity of zebra finch to triclopyr BEE could be due
3 to random variability or other differences in methods used in the open literature publication
4 versus the standard protocols used in the acute dietary studies. The potential sensitivity of
5 passerines is discussed further in the dose-response assessment for birds (Section 4.3.2.2.1).
6

7 Based on the studies in mallards and quail, U.S. EPA/OPP (1998a) classifies triclopyr acid as
8 being *practically non-toxic* to *slightly toxic* to birds and triclopyr TEA and triclopyr BEE (Garlon
9 4) as *practically non-toxic to birds*. Although passerines may be more sensitive than the
10 standard test species, the classification scheme used by U.S. EPA/OPP results in a designation of
11 *slightly toxic*, based on the dietary LC₅₀ of 1383 ppm a.e.
12

13 **4.1.2.2.1.2.3. Reproduction Studies**

14 U.S. EPA/OPP generally requires two avian reproduction studies for pesticide registration. As
15 summarized in Appendix 3 (Table A3-3), reproduction studies on mallards (Beavers et al. 1980)
16 and quail (Beavers et al. 1979b) were submitted to U.S. EPA/OPP. In addition, Dow
17 AgroSciences recently completed another reproduction study in mallards (Temple et al. 2007), a
18 copy of which was provided by Dow AgroSciences during the preparation of this risk
19 assessment.
20

21 In both of the earlier reproductions studies in mallards and quail, the NOEC for triclopyr was
22 100 ppm with a corresponding LOAEC of 200 ppm. Based on body weight and food
23 consumption data reported in these earlier studies, the NOAECs were equivalent to doses of
24 about 7.5 mg/kg bw/day in quail and 10 mg/kg bw/day in mallards.
25

26 The more recent study by Temple et al. (2007) reports an NOAEC of 400 ppm in mallards
27 equivalent to a dose of about 56 mg a.e./kg bw/day, based on reported food consumption and
28 body weights. The study by Temple et al. (2007), like the earlier avian reproduction studies, was
29 conducted at Wildlife International. The primary investigator from the earlier studies, Dr. Joann
30 Beavers, is currently the director of mammalian toxicity at Wildlife International, and is listed as
31 a coauthor of the Temple et al. (2007) study. While the study by Temple et al. (2007) is well
32 reported and appears to have followed standard protocols, it does not provide a discussion of the
33 higher NOAECs and LOAECs reported in the 2007 study, relative to the values reported earlier
34 in Beavers et al. (1980). The magnitude of the difference, which is a factor of about 5 in terms
35 of mg/kg bw/day doses, is not insubstantial but could be due to random variation or other
36 unidentified factors.
37

38 The lowest reported NOAEL of 7.5 mg/kg bw/day for reproductive effects in quail (Beavers et
39 al. 1979b), is virtually identical to the reproduction NOAEL of 5 mg/kg bw/day in rats, on which
40 the RfD for humans is based (Table 29).
41

42 **4.1.2.2.1.2.4. Field Studies**

43 As summarized in Appendix 3, Table A3-4, There are two field studies, Boren et al. (1993) and
44 Schulz et al. (1992a), which involve triclopyr applications in the range of application rates that
45 may be used in Forest Service programs. Neither study indicates that the triclopyr applications
46 caused adverse effects in birds; what is more, the study by Schulz et al. (1992b) suggests that

1 some bird species benefited from the applications due to changes in vegetation. These types of
2 observations of population effects secondary to changes in habitat are common in field studies
3 involving herbicide applications.

4 **4.1.2.2.2. TCP**

5 As summarized in Appendix 3, Table 1, the acute gavage LD₅₀ for TCP in bobwhite quail is
6 >2000 mg/kg bw (Campbell et al. 1990). Based on this index of toxicity, TCP would be
7 regarded as less toxic than triclopyr acid, triclopyr TEA or triclopyr BEE. As summarized in
8 Appendix 3, Table 1, Campbell et al. (1990) note a NOAEC of 125 mg TCP/kg bw based on
9 weight loss that was noted at 250 mg/kg bw. This NOAEL is virtually identical to the NOAEL
10 of \cong 126 mg a.e./kg bw for triclopyr BEE based on signs of toxicity (Campbell and Lynn 1991a).

11
12 The only other study on the toxicity of TCP to birds is the standard 5-day dietary study in
13 mallards by Long et al. (1990). In this study, the dietary LC₅₀ for TCP was >5620 ppm.
14 Nonetheless, a NOAEC was not determined with reduced body weight gain noted at 562 ppm.
15 Based on the reported food consumption and body weights in the study by Long et al. (1990), the
16 dietary concentration of 562 ppm corresponded to a dose of about 116 mg/kg bw based on Day 5
17 body weights and food consumption.

18 **4.1.2.3. Reptiles and Amphibians (Terrestrial Phase)**

19 The toxicity of triclopyr or TCP to reptiles or terrestrial phase amphibians is not addressed in the
20 available literature. Information about the toxicity of triclopyr to terrestrial phase amphibians is
21 not available the open literature or in the studies submitted to the U.S. EPA. More specifically,
22 toxicity data involving the exposure of terrestrial phase amphibians to triclopyr are not included
23 in either the recent EPA ecological risk assessment on triclopyr (U.S. EPA/OPP 2009a) or in the
24 database on amphibian and reptile toxicity data maintained by the Canadian National Wildlife
25 Research Centre (Pauli et al. 2000).

26 **4.1.2.4. Terrestrial Invertebrates**

27 The honey bee is the standard test organism for assessing the potential effects of pesticides on
28 terrestrial invertebrates. Acute contact toxicity studies in honey bees are available on triclopyr
29 acid and triclopyr TEA (U.S. EPA/OPP 1998a). In both bioassays, the LD₅₀ values were greater
30 than 100 μ g/bee. Based on these results, U.S. EPA/OPP (1998a) classifies triclopyr as
31 practically non-toxic to bees. U.S. EPA/OPP (2009a) summarizes a more recent study on the
32 toxicity of triclopyr BEE to honey bees in which the contact LD₅₀ is reported as >72 μ g/bee.

33
34 Several acute (14 day) toxicity studies are available in earthworms. These studies include
35 bioassays on triclopyr acid (Mallett and Hayward 2000b), triclopyr TEA (McCormac 2010),
36 triclopyr BEE (Mallett and Hayward 2000a), and Garlon 4 (Mallett and Hayward 2000c). Of
37 these materials, triclopyr acid is the least toxic to earthworms with an NOAEL of about 790 ppm
38 a.e. – i.e., 790 mg a.e./kg soil dry weight – and an LC₅₀ of 1110 ppm a.e. The most toxic
39 material is triclopyr TEA with an LC₅₀ of about 146 ppm a.e and an LOAEC of 134 ppm a.e.
40 based on a significant increase in mortality (35% relative to 0 % in the control groups) as well a
41 significant decrease in body weight (17%) relative to the control group (McCormac 2010).
42 While the study by McCormac (2010) suggests that triclopyr TEA may be moderately toxic to
43 earthworm relative to triclopyr acid, the toxic concentrations are far higher than soil
44 concentrations of triclopyr that will occur in the environment.

1
2 One chronic bioassay is available in earthworms (Hayward 2000). In this study, earthworms
3 were exposed to Garlon 4 at concentrations of about 1.4 ppm a.e. or 6.9 ppm a.e. for a total of 56
4 days – i.e., a 28 day exposure for adults followed by a 28 day exposure for juveniles. No adverse
5 effects were noted on reproduction or growth at either concentration. These results are
6 consistent with the study by Potter et al. (1990) which assayed for the impact of triclopyr (Garlon
7 3A) to earthworms and other invertebrates at an application rate of 0.56 kg a.i./ha (\approx 0.36 lb
8 a.e./acre) to turf plots. There was no significant reduction in mixed earthworm populations,
9 mites, springtails, or ants in turf and soil core samples.

10
11 The only other information on the potential effect of triclopyr on terrestrial invertebrates comes
12 from a series of field studies, all of which suggest that the most likely effects on terrestrial
13 invertebrates will be secondary to changes in vegetation cover. Secondary effects were noted in
14 beetles (Asteraki et al. 1992; Duchesne et al. 1999; Lindgren et al. 1998), butterflies (Bramble et
15 al. 1997), and spiders (Asteraki et al. 1992). In these studies, the effects in invertebrates were
16 attributable to changes in vegetation rather than any potential toxic effect of triclopyr, because
17 similar changes in invertebrate populations were observed with other methods (e.g., mechanical)
18 of vegetation management.

19 **4.1.2.5. Terrestrial Plants**

20 Triclopyr and other pyridinecarboxylic acid herbicides, such as picloram, mimic indole auxin
21 plant growth hormones and cause uncontrolled growth in plants. These herbicides behave
22 similarly to the chlorophenoxy acid herbicides, such as 2,4-D. At sufficiently high levels of
23 exposure, the abnormal growth is so severe that vital functions cannot be maintained and the
24 plant dies (Bovey and Meyer 1981; Coffman et al. 1993; Extoxnet 1996; Hatterman-Valenti et al.
25 1995). Triclopyr is absorbed by foliage and translocated to roots (Gorrell et al. 1988;
26 Hutchinson et al. 2010). As discussed below, triclopyr is effective in the control of dicots and
27 relatively ineffective in controlling monocots (Lautenschlager et al. 1998), at least in terms of
28 foliar application. A similar pattern is apparent in the effects of triclopyr on aquatic macrophytes
29 (Section 4.1.3.4.2). Pine, an important group of nontarget plant species, tends to be tolerant to
30 triclopyr exposures after fall dormancy but more sensitive to triclopyr during the spring and
31 summer (Radosevich et al. 1977).

32
33 As noted in Section 2, triclopyr has been used as a herbicide for more than 30 years, and there
34 are numerous studies regarding its efficacy. While some of these studies (e.g., Balneaves and
35 Davenhill 1990; Bovey et al. 1979; Clay 1987; Delanoy and Archibold 2007; Holt et al. 1985;
36 Hutchinson et al. 2010; Katovich et al. 1996; Ottis et al. 2005; Powers and Ferrell 1996;
37 Radosevich et al. 1977; Strizke et al 1991) were reviewed in the process of conducting this risk
38 assessment, efficacy studies are not discussed in this risk assessment in detail unless they include
39 effects on nontarget species.

40
41 Standard toxicity studies in non-target plants are summarized in Appendix 4. The U.S. EPA
42 requires studies of seedling emergence and vegetative vigor in non-target plants for herbicides.
43 Vegetative vigor studies, which involve direct foliar applications to young plants, are
44 summarized in Appendix 4, Table A4-1. While the dose-response assessment for terrestrial
45 plants focuses on NOAECs, the hazard identification focuses on EC₂₅ values because they
46 provide a better measure of relative potency. With terrestrial plants, as with terrestrial animals,

1 triclopyr TEA and triclopyr BEE appear to be bioequivalent. The sunflower (a dicot) is the most
2 sensitive species for both TEA ($EC_{25} = 0.005$ lb a.e./acre) and triclopyr BEE ($EC_{25} = 10$ g a.i./ha)
3 (≈ 0.0064 lb a.e./acre). For both triclopyr TEA and BEE, the monocots, like wheat and oats, are
4 much more tolerant with EC_{25} values in excess of about 0.3 lb a.e./acre.

5
6 In seedling emergence studies (Appendix 4, Table A4-2), the two forms of triclopyr are not
7 bioequivalent. Triclopyr BEE is much more toxic than triclopyr TEA, at least in some species.
8 For example, the EC_{25} for triclopyr BEE in alfalfa is 40 g a.i./ha (≈ 0.02 lb a.e./acre). For
9 triclopyr TEA, the EC_{25} values for all species are in excess of 0.23 lb a.e./acre. Also unlike
10 foliar applications, triclopyr BEE is about equally effective against dicots and at least some
11 monocots. The higher toxicity of triclopyr BEE in the seedling emergence assay may relate to
12 the more rapid absorption of the BEE form, relative to the TEA form. This difference has been
13 demonstrated quantitatively in chickweed, wheat, and barley (Lewer and Owen 1990), and is
14 likely to be true for most other plant species.

15
16 Variations in species sensitivity to triclopyr BEE appear to be related directly to the rate of
17 metabolic ester hydrolysis by the plant (Lewer and Owen 1990). As with 2,4-D and 2,4,5-T, arid
18 conditions do not affect the rate of triclopyr absorption but do inhibit translocation and thus
19 efficacy (Bollig et al. 1995; Seiler et al. 1993).

20
21 The study by Newmaster et al. (1999) suggests that some bryophytes and lichens may be
22 sensitive to long-term effects after triclopyr exposure. The EC_{50} for a decrease in relative
23 abundance 6 months after application is about 1 kg/ha or 0.89 lbs/acre (Newmaster et al. 1999,
24 Figure 3, p. 1105). Also, changes in relative abundance were apparent at 6 weeks after
25 application (Newmaster et al. 1999, Figure 7, p. 1108). The statistical analyses provided by
26 Newmaster et al. (1999) involve the use of a non-threshold polynomial model. While this may
27 be a reasonable method for quantifying effects among the two herbicides studied (glyphosate and
28 triclopyr), this may be less appropriate for risk assessment. Nonetheless, this study does appear
29 to present a plausible basis for concern that exposure to substantial triclopyr drift may have long-
30 term impacts on bryophyte and lichen communities.

31
32 As summarized in Table 1, triclopyr BEE is also much more volatile than triclopyr TEA. While
33 not specifically discussing triclopyr BEE, Saunders et al. (1985) and Bacci et al. (1990) note that
34 some of the more volatile herbicides can cause damage to nontarget plants through vapor
35 transport. None of the field studies involving triclopyr BEE document damage to nontarget plant
36 species through volatilization. Nonetheless, anecdotal reports from the Forest Service suggest
37 that volatilization of triclopyr may damage nontarget plants if triclopyr BEE is applied under a
38 poorly ventilated canopy and high temperatures.

39 **4.1.2.6. Terrestrial Microorganisms**

40 Several diverse studies are available on the toxicity of triclopyr to terrestrial microorganisms.
41 None of these studies suggests that triclopyr is likely to have an impact on soil microorganisms.

42
43 Estok et al. (1989) examined the effects of Garlon 4 at concentrations of 1, 10, 100, 1000, 5000,
44 or 10,000 ppm a.i. in growth medium (agar) over 26-to 48-day growth periods on three species
45 of fungi. The results indicate a significant reduction of radial growth in each species at
46 concentrations ≥ 1000 ppm. Total growth inhibition was observed at ≥ 5000 ppm. *Cenococcum*

1 *geophilum*, the slowest growing fungus, was least sensitive to the effects of triclopyr. In a
2 similar study, Chakravarty and Sidhu (1987) studied the inhibitory effects of triclopyr (specified
3 only as a Garlon formulation with 48% a.i.) over a 30-day growth period in five fungal species:
4 *Hebeloma crustuliniforme*, *Laccaria laccata*, *Thelophora americana*, *Thelophora terrestris*, and
5 *Suillus tomentosus*. The most sensitive species was *Thelophora americana* for which a slight
6 growth inhibition (93.75% of controls) based on dry weight was reported to be statistically
7 significant at 0.1 ppm. In other species, statistically significant decreases in growth were
8 observed between 1 and 10 ppm. In a series of soil assays, Remde (1995) noted no effect on soil
9 respiration at a concentration of 9.6 mg a.e./L.

10
11 Hallborn and Bergman (1979) noted no effect of triclopyr on nitrogen fixation of lichen at 100
12 ppm; however, the reporting units are not clear. Similarly, Pell et al. (1998) conducted a general
13 screening study on the impact of numerous compounds on ammonium oxidation in soil. At a
14 concentration of 100 ppm, triclopyr caused a slight (12%) but significant decrease in ammonium
15 oxidation activity. Last, Houston et al. (1998) notes that triclopyr TEA had no impact on soil
16 microbial function or community structure at an application rate of 1.9 kg a.i./ha (\approx 1.2 lb
17 a.e./acre).

18 **4.1.3. Aquatic Organisms**

19 Like hazard identification for nontarget terrestrial species, the hazard identification for aquatic
20 species is concerned with identifying patterns of toxicity for the various forms of triclopyr under
21 review (i.e., acid, TEA, and BEE) as well as the 3,5,6-trichloro-2-pyridinol (TCP) metabolite of
22 triclopyr. Also like the hazard identification for nontarget terrestrial species, the ecological
23 hazard identification is concerned with identifying differences in species sensitivity as well as
24 differences in the toxicity of the various forms of triclopyr and TCP to various groups of aquatic
25 organisms, including, fish, amphibians, invertebrates, aquatic macrophytes and algae.

26
27 In addressing the above issues, the hazard identification for aquatic organisms uses cumulative
28 frequency distributions of LC₅₀ or EC₅₀ values, an example of which is given in Figure 11 for
29 toxicity data in fish.

30
31 In Figure 11, the x-axis is the LC₅₀ value and the y-axis is the cumulative frequency of the LC₅₀
32 values for the various forms of triclopyr as well as for TCP. The individual values for the
33 cumulative frequency are based on the following equation:

$$34 \quad \text{Freq}_i = \frac{1-0.5}{N}$$

35 **Equation 9**

36
37 where Freq_i is the cumulative frequency for the i^{th} value and N is the number of values in the
38 data set. For example, the data on triclopyr TEA consists of 14 LC₅₀ values for fish. The lowest
39 value is an LC₅₀ of 40 mg a.e./L. Thus, the frequency for the first point ($i=1$) is calculated as
40 $(1-0.5) \div 14$ or 0.037. Similarly, the second lowest LC₅₀ value ($i=2$) is 65.1 mg a.e./L, which is
41 assigned a frequency of $(2-0.5) \div 14$ or 0.107. Note that the x-axis in Figure 11 represents the
42 LC₅₀ values, which are given on a logarithmic scale, under the standard assumption that LC₅₀
43 and EC₅₀ values for different chemicals or different groups of organisms will be distributed
44 lognormally. While the dose-response assessment for aquatic species is focused on NOAECs,
45

1 the comparisons of toxicity in the hazard identification uses LC₅₀ or EC₅₀ values, because they
2 estimate population means and are more amenable to comparisons, relative to NOAELs which
3 are simply exposure concentrations used in experiments. Each figure illustrating a distribution of
4 LC₅₀ or EC₅₀ values is accompanied by a table giving the LC₅₀ or EC₅₀ data used in the
5 corresponding plot. When available, chronic NOAECs are plotted separately on the x-axis. The
6 same symbol is used to plot the chronic NOAECs and the acute data, except that the chronic
7 NOAECs are not connected by lines to the corresponding acute LC₅₀ or EC₅₀ values.

8
9 The cumulative frequency distributions of toxicity values are related to figures often referred to
10 as *species sensitivity distributions* (e.g., Awkerman et al. 2008; Posthuma et al. 2002). As
11 discussed by Posthuma et al. (2002), species sensitivity distributions can be used quantitatively
12 as tools in probabilistic risk assessment. Probabilistic methods are not routinely used in Forest
13 Service risk assessments. Nonetheless, cumulative distribution plots, like those in Figure 11, are
14 useful for illustrating differences in and among different agents or groups of organisms. The
15 cumulative frequency distributions used in this risk assessment, however, differ from species
16 sensitivity distributions in that species sensitivity distributions typically provide only one data
17 point for each species with various methods used to composite multiple studies on the same
18 species. As discussed in the following subsections, the frequency distributions do not do
19 composite multiple studies on the same species. To the contrary, one of the uses of these plots
20 and the corresponding data tables is to illustrate the variability in data, including variability in
21 reported toxicity values for the same species.

22 **4.1.3.1. Fish**

23 The acute lethal potency of triclopyr TEA and triclopyr BEE as well as TCP is relatively well
24 characterized, and details of the available studies are summarized in Appendix 5, Tables A5-1
25 through A5-4. As illustrated in Figure 11, clear differences are apparent in the toxicity of each
26 of these agents. Triclopyr TEA is much less toxic to fish than either triclopyr BEE or TCP. As
27 summarized in Table 33, the geometric mean (i.e., the median) of the LC₅₀ values for triclopyr
28 TEA is about 131 mg a.e./L. The geometric mean for corresponding values of TCP is 3.19
29 mg/L—i.e., TCP is more toxic than triclopyr TEA by a factor of about 40 [131 mg a.e./L ÷ 3.19
30 mg/L ≈ 41.07]. Relative to triclopyr BEE, triclopyr TEA is less toxic by a factor of about 240
31 [130.7 mg a.e./L ÷ 0.539 mg a.e./L ≈ 242.48] and TCP is less toxic by a factor of about 6 [3.19
32 mg/L ÷ 0.539 mg a.e./L ≈ 5.92]

33
34 In discussing the relative toxicity of TCP to various forms of triclopyr, U.S. EPA/OPP (2009a)
35 expresses the toxicity of TCP in units of “acid equivalents.” In other words, the molecular
36 weight of triclopyr acid is 256.47 g/mole and the molecular weight of TCP is 198.43 g/mole.
37 Thus, in comparing the LC₅₀ and other toxicity values, U.S. EPA/OPP (2009a) converts the
38 toxicity value for TCP from units of mg TCP/L to units of “*acid equivalents*” by multiplying it
39 by 1.292 [256.47 g triclopyr acid/mole ÷ 198.43 g TCP/mole ≈ 1.292_{a.e./TCP}]. The method of
40 assessing relative toxicity is equivalent to comparing molar concentrations. Using molar
41 comparisons, the median LC₅₀ value for triclopyr TEA of 131 mg a.e./L is equivalent to about
42 0.511 mMole/L [131 mg a.e./L ÷ 256.47 mg/mMole] and the median LC₅₀ value for TCP is 3.19
43 mg/L is equivalent to about 0.0161 mMole/L [3.19 mg/L ÷ 198.43 mg TCP/mMole]. Thus, the
44 molar potency of TCP relative to triclopyr TEA is about 31.7[0.511 mMole/L ÷ 0.0161
45 mMole/L]. Because the differences in the molecular weights of triclopyr and TCP are small
46 relative to the substantial differences in toxicity, the use of molar concentrations does not have a

1 substantial impact on assessments of relative potencies and relative molar potencies are not used
2 further in this risk assessment.

3
4 As also illustrated in Figure 11 and detailed in Appendix 5 (Table A5-5), chronic studies are
5 available on both triclopyr TEA and TCP. The NOAEC for triclopyr TEA is about 32.4 mg
6 a.e./L (Mayes 1983), and the reported NOAECs for TCP are 0.0808 mg/L (Marino et al. 1999)
7 and 0.178 mg/L (Marino et al. 2003). As noted in Appendix 5, U.S. EPA/OPP classifies the
8 earlier study by Marino et al. (1999) as invalid because of issues associated with the solvent
9 control (Mossler et al. 2000). Deferring to the EPA on this classification and using the higher
10 NOAEC for TCP of 0.178 mg/L, TCP is more toxic than triclopyr TEA, based on chronic
11 effects, by a factor of about 180 [32.4 mg a.e./L ÷ 0.178 mg/L ≈ 182.02].

12
13 In the U.S. EPA/OPP (2009a) hazard identification for fish, the EPA appears to dismiss potential
14 risks associated with exposures to TCP:

15
16 *Triclopyr acid forms the degradation products; 3,5,6-trichloro-2-pyridinal*
17 *(TCP) and 3,5,6-trichloro-2-methoxy pyridine (TMP) as a result of microbial*
18 *degradation in aerobic soil. TMP is considered a minor degradate and TCP,*
19 *although a major degradate, is not of toxicological concern since (in terms*
20 *of acid equivalency) it is not more sensitive than the lowest triclopyr*
21 *endpoints. As a result, neither TCP nor TMP will be further evaluated in*
22 *this assessment.*

23 U.S. EPA/OPP 2009a, p. 20

24
25 With respect to triclopyr TEA or triclopyr acid, the basis for the above statement is not apparent.
26 As discussed, TCP is more toxic than triclopyr TEA by a factor of about 40, based on acute
27 toxicity, and by a factor of 180, based on chronic toxicity.

28
29 The above statement by U.S. EPA/OPP may refer to the toxicity of triclopyr BEE. As illustrated
30 in Figure 11, technical grade triclopyr BEE as well as the Garlon 4 formulation of triclopyr BEE
31 is more toxic than TCP. As also illustrated in Figure 11, several studies are available on
32 triclopyr BEE and Garlon 4. Based on these studies, the toxicity of triclopyr BEE and Garlon 4
33 are essentially identical, with median LC₅₀ values of 0.539 and 0.588 mg a.e./L, respectively. As
34 discussed in Section 3.1.14 and summarized in Table 4, Garlon 4 contains several potentially
35 toxic inerts, including kerosene, ethylene glycol, and petroleum hydrocarbons. Nonetheless,
36 based on the relatively abundant and well-documented studies on both triclopyr BEE and Garlon
37 4, the inerts in Garlon 4 do not appear to contribute to the formulation's acute toxicity to fish.
38 Consequently, the LC₅₀ values of 0.539 and 0.588 mg a.e./L for these two agents can be
39 composited, using the median, to an estimated LC₅₀ of 0.563 mg a.e./L. Based on this LC₅₀,
40 triclopyr BEE is more acutely toxic than TCP to fish by a factor of about 6 [3.19 mg/L ÷ 0.563
41 mg a.e./L ≈ 5.666].

42
43 Chronic toxicity data in fish are available on triclopyr BEE. As summarized in Appendix 5,
44 Table A5-5, Weinberg et al. (1994d) conducted a standard egg-to-fry study in trout using a flow-
45 through system in which constant concentrations of triclopyr BEE could be maintained in the test
46 chambers. Based on this study, the NOAEC for triclopyr BEE is 0.017 mg a.e./L. Thus, based

1 on chronic exposures, triclopyr BEE is more toxic than TCP to fish by a factor of about 10
2 [0.178 mg/L ÷ 0.017 mg a.e./L ≈10,47].

3
4 Notwithstanding the greater toxicity of triclopyr BEE relative to TCP, the current risk assessment
5 is concerned with applications of either triclopyr TEA or triclopyr BEE (Section 2). In
6 applications of triclopyr TEA, the 3,5,6-trichloro-2-pyridinol (TCP) is clearly an agent of
7 concern. As discussed further in Section 4.2.3 (exposure assessment for aquatic organisms),
8 concern for TCP is diminished by the lower concentrations of TCP relative to triclopyr;
9 nevertheless, the potential hazards of TCP relative to triclopyr cannot be dismissed, based on the
10 available toxicity data on fish. Thus, for fish exposures, TCP is identified as an agent of
11 concern, and risks associated with these exposures are assessed quantitatively.

12
13 The toxicity of TCP is also potentially relevant to longer-term exposure to triclopyr BEE. While
14 the acute toxicity of triclopyr BEE is an obvious concern in terms of exposures which occur
15 shortly after triclopyr BEE formulation is applied, particularly when there is significant drift to
16 surface water, the practical significance of the chronic toxicity data on triclopyr BEE to fish is
17 marginal because longer-term concentrations of triclopyr BEE in surface water will be extremely
18 low. These relationships of toxic potency to exposure are considered further in the risk
19 characterization for fish (Section 4.4.3.1).

20
21 As summarized in Table 33, there are no remarkable differences among species in terms of
22 sensitivity to the various agents covered in this risk assessment. Note that each of the sections in
23 Table 33—i.e., for the different forms of triclopyr—are sorted by lowest to highest LC₅₀. If
24 there were substantial and significant differences in toxicity among different species of fish, a
25 pattern of toxicity would emerge in the list of species in the first column of Table 33. This is not
26 the case. For example, of the 17 LC₅₀ values listed for triclopyr TEA, four are for fathead
27 minnows; however, they are scattered from positions of 4 to 17. Similarly, the two LC₅₀ values
28 for bluegills occupy positions 2 and 14.

29
30 As illustrated in Figure 11, the frequency distributions for triclopyr TEA, triclopyr BEE, and
31 Garlon 4 follow a reasonably smooth pattern. This pattern is particularly apparent for triclopyr
32 TEA, and a similar pattern is seen if the data on triclopyr BEE and Garlon 4 are combined.
33 These relatively smooth frequency distributions and the overlapping order of species within the
34 distributions (Table 33) suggest that the variability in the LC₅₀ values reflect little more than
35 random variability. Based on the geometric means of the available LC₅₀s in fish expressed in
36 units of mg a.e./L, Garlon 4 appears to be about 30% less toxic than triclopyr BEE [0.716 mg
37 a.e./L ÷ 0.539 mg a.e./L ≈ 1.3284]. As discussed in Section 3.1.14, Garlon 4 contains a number
38 of inerts that are toxic. Nonetheless, the data on the toxicity to fish of technical grade triclopyr
39 BEE and Garlon 4 indicate that the toxicity of Garlon 4 can be attributed solely to triclopyr BEE
40 – i.e., there is no indication that the inerts in Garlon 4 impact the toxicity of Garlon 4 to fish.

41
42 The frequency distributions for the LC₅₀s in fish of TCP and triclopyr acid are not smooth
43 (Figure 11). As summarized in Table 33, six of the eight LC₅₀ values for TCP are from the study
44 by Wan et al. (1987), and these values are smoothly distributed. The two high LC₅₀ values are
45 taken from U.S. EPA/OPP (2009a). Citations to these MRID studies are not given in U.S.
46 EPA/OPP (2009a), but the similarities in the MRID numbers (i.e., MRID 41829003 and

1 41829004) suggest that these studies were conducted by the same investigators and probably at
2 about the same time. It is not uncommon for bioassays from only two studies to yield apparently
3 disjointed results. While somewhat speculative, it seems reasonable to suggest that the LC₅₀
4 values for TCP would follow a smoother distribution, if more studies were available from a
5 greater number of investigators.

6
7 The disjointed frequency distribution for triclopyr acid is also associated with the study by Wan
8 et al. (1987). As with TCP, the data from the Wan study account for six of the eight LC₅₀ values
9 and these six LC₅₀ values are substantially lower than the other two toxicity values which come
10 from another MRID study – i.e., Batchelder (1973). The most peculiar aspect of the data,
11 however, are the very low LC₅₀s reported by Wan et al. (1987) for triclopyr acid – i.e., 6.3 mg
12 a.e./L to 9.7 mg a.e./L. As illustrated in Figure 11, the LC₅₀s from Wan et al. (1989) appear to be
13 outliers not only with respect to the data from Batchelder (1973) but also with respect to all of
14 the 17 LC₅₀s on Garlon 3A. These results from Wan et al. (1989) on triclopyr acid cannot be
15 attributed to experimental factors or methods. Note that 5 of the 17 LC₅₀s on triclopyr TEA
16 (Garlon 3A) and six of the 13 LC₅₀s on triclopyr BEE (Garlon 4) are from the Wan et al. (1989)
17 study and these LC₅₀s are reasonably consistent with all of the other LC₅₀s on triclopyr TEA and
18 triclopyr BEE reported in the literature. Wan et al. (1987) indicate that pH was measured but do
19 not report specifically the pH values for the test solutions.

20
21 While the U.S. EPA/OPP (2009a) cites the Wan et al. (1989) data on TCP, the Agency risk
22 assessment does not cite or use the Wan et al. (1989) data on triclopyr acid. The current risk
23 assessment is concerned primarily with triclopyr TEA and triclopyr BEE. Nonetheless, both of
24 these will degrade to triclopyr acid. While the bioassay by Wan et al. (1989) cannot be
25 dismissed as irrelevant, the results cannot be explained. Given the large number of studies on
26 triclopyr TEA indicating that triclopyr TEA is far less toxic than the bioassays by Wan et al.
27 (1989) on triclopyr acid would suggest, the LC₅₀s by Wan et al. (1989) on triclopyr acid are not
28 used in the dose-response assessment for fish (Section 4.3.3.2).

29
30 The sublethal effects of Garlon 4 on salmonid (rainbow trout) were investigated by Johansen and
31 Geen (1990) using flow-through systems. At concentrations of 0.32-0.43 mg/L, about a factor of
32 2 below the 96-hour LC₅₀ determined by these investigators, fish were lethargic. At levels ≤0.1
33 mg/L, fish were hypersensitive over 4-day periods of exposure. This is reasonably consistent
34 with the threshold for behavioral changes in rainbow trout for Garlon 4 of 0.6 mg/L (Morgan et
35 al. 1991). The corresponding threshold for behavioral changes to Garlon 3A was 200 mg/L
36 (Morgan et al. 1991), which is consistent with the relative acute lethal potencies of these two
37 agents.

38
39 A recent study by Xie et al. (2005) suggests that triclopyr TEA may induce vitellogenin in
40 juvenile rainbow trout. In laboratory studies, however, these effects were noted only at a
41 concentration of 1 mg/L and only when triclopyr exposure was accompanied by exposure to a
42 surfactant, Target Prospreader Activator. In a field simulation phase of this study, Xie et al.
43 (2005) note:

44
45 *When trout were exposed to water collected from a site where triclopyr was*
46 *used in combination with TPA, a concentration dependent increase in Vtg*

1 [vitellogenin] expression was observed. Measured values of 4-NP [4-
2 nonylphenol] were 3.7 µg/L, and triclopyr concentrations were below
3 detection limit (5 ng/L).

4 Xie et al. 2005, p. 391.

5
6 As noted in the Xie et al. (2005) publication, exposures to 4-nonylphenol have been shown to
7 have estrogenic effects in trout.

8
9 Based on the results presented in Xie et al. (2005), there does not appear to be a plausible basis
10 for asserting that <5 nanogram/L concentrations of triclopyr were responsible for the effects in
11 trout. Nonetheless, this publication has been cited in the literature to suggest that triclopyr may
12 have estrogenic effects (Kortenkamp 2007). As noted in Section 3.1.8, the substantial toxicity
13 data on triclopyr in mammals do not support this supposition. Finally, several aspects of the Xie
14 et al. (2005) study have been critiqued in the open literature (Kramer et al. 2008), and this
15 critique raises issues of merit, particularly concerning the small number of fish used in the Xie et
16 al. (2005) study and the uncertainties that this raises in trout studies that do not identify the
17 gender of the trout in assays for vitellogenin. Responses to the criticisms raised by Kramer et al.
18 (2008) have not been encountered in the open literature.

19
20 As summarized in Table 26, the concentration of 1 mg a.e./L used in the study by Xie et al.
21 (2005) is much higher than would be expected in terrestrial applications of triclopyr in Forest
22 Service programs. Given the lack of any studies confirming estrogenic effects in trout as well as
23 the concerns with the Xie et al. (2005) study raised by Kramer et al. (2008), the usefulness of the
24 Xie et al. (2005) study is questionable. In the recent U.S. EPA ecological risk assessment of
25 triclopyr (U.S. EPA/OPP 2009a), the paper by Xie et al. (2005) is cited but is not discussed or
26 otherwise used.

27 **4.1.3.2. Amphibians**

28 Studies on the toxicity of triclopyr TEA and triclopyr BEE to aquatic phase amphibians are
29 detailed in Appendix 6. The LC₅₀ values are given in Table 34, and these data are illustrated in
30 Figure 12. Data on the toxicity of TCP to aquatic phase amphibians were not identified in the
31 conduct of the current risk assessment. Specifically, the relatively comprehensive summary by
32 Pauli et al. (2000) on the toxicity literature relating to amphibians contains no information on
33 TCP.

34
35 Compared to the data on fish, the acute toxicity data on aquatic phase amphibians are sparse.
36 The only acute toxicity value for triclopyr TEA is the 96-hour LC₅₀ of 84 mg a.e./L in *Xenopus*
37 *laevis* exposed to Garlon 3A in the study by Perkins (1997). This LC₅₀ is modestly lower than
38 the median LC₅₀ in fish (≈130 mg a.e./L) but well within the range of LC₅₀ values for triclopyr
39 TEA in fish—i.e., ≈40 to 420 mg a.e./L.

40
41 All of the LC₅₀ values in amphibians for triclopyr BEE involve triclopyr formulations, either
42 Release or Garlon 4. No data are available on the toxicity of unformulated triclopyr BEE in
43 amphibians. Based on the detailed study by Edington et al. (2005) with triclopyr BEE and
44 supported by additional data from Perkins (1997) and Wojtaszek et al. (2005), tadpoles are more
45 sensitive than embryos. While the ranges in the LC₅₀ values overlap somewhat (Figure 12), the
46 difference in sensitivity between tadpoles and embryos spans about an order of magnitude – i.e.,

1 median LC₅₀ values of about 2 mg a.e./L in tadpoles and 20 mg a.e./L in embryos. While
2 modestly speculative, this difference in sensitivity probably reflects the rapid uptake of triclopyr
3 BEE through the gills of tadpoles, relative to passive uptake by amphibian embryos.

4
5 While tadpoles are more sensitive to formulated triclopyr BEE than amphibian embryos, the
6 LC₅₀ values in tadpoles are somewhat higher – i.e., about 2.3 mg/L – than the corresponding
7 LC₅₀ values in fish—i.e., a median LC₅₀ of about 0.5 mg a.e./L. Thus, based on the most
8 sensitive stage, amphibians appear to be less sensitive than fish by a factor of about 4.

9
10 As illustrated in Figure 12, the cumulative frequency distribution for tadpoles is somewhat
11 uneven with the upper most point apparently right-shifted. This point is the LC₅₀ of 11.50 in
12 *Rana clamitans* from the study by Edington et al. (2005). As summarized in Table 34, *Rana*
13 *clamitans* is the least sensitive species, and the LC₅₀ in this species is a factor of about 15 higher
14 than the most sensitive species—i.e., *Rana pipiens* with an LC₅₀ of 0.79 mg a.e./L also from the
15 study by Edington et al. (2005) [$11.50 \div 0.79 \text{ mg/L} \approx 14.56$]. When the frequency data on
16 tadpoles are converted to units of standard deviations—e.g., using the NORMSINV function in
17 EXCEL— and regressed against the natural logarithm of the concentrations, the correlation is
18 highly significant (adjusted $r^2=0.02$, $p=0.000461$). The significance of this correlation indicates
19 that the distribution of the LC₅₀ values for amphibian tadpoles is consistent with a log-normal
20 distribution and that the LC₅₀ in *Rana clamitans* is not an outlier. While this correlation might in
21 turn suggest that *Rana clamitans* is a tolerant species, the study by Wojtaszek et al. (2005), also
22 summarized in Table 34, notes very little difference in the LC₅₀ values for *Rana pipiens* and
23 *Rana clamitans* —i.e., LC₅₀ values of 3.01 and 3.39 mg a.e./L, respectively. Thus, in the absence
24 of additional data demonstrating any clear patterns in species sensitivity, the differences in LC₅₀
25 values for amphibians may reflect simple random variability, similar to the LC₅₀ values for fish
26 discussed in Section 4.1.3.1.

27
28 The observation of hind limb deformities in free-living amphibians substantially increases
29 concern for the effects of xenobiotics on amphibian populations (e.g., Sparling et al. 2000).
30 Garlon 3A and Garlon 4 were specifically tested for malformations in the frog embryo
31 teratogenesis assay (Perkins et al. 2000). In this assay, frog (*Xenopus laevis*) embryos are
32 exposed to the test solution in Petri dishes for 96 hours. No hind limb abnormalities were
33 reported in this study. The only abnormalities specified in the publication include uncoiling of
34 the gut, edema, blistering, abnormal pigmentation, and axial twisting in control embryos. No
35 statistically significant increases in abnormalities were seen in any groups exposed to Garlon 3A
36 or Garlon 4 at levels that were not lethal. The precise number and nature of abnormalities in the
37 groups exposed to lethal concentrations of the triclopyr formulations are, however, not specified.
38 Nonetheless, this report is consistent with the much larger body of literature on reproductive
39 toxicity in mammals (Section 3.1.9) indicating that the triclopyr is not likely to cause
40 reproductive or teratogenic effects at sublethal concentrations.

41
42 Berrill et al. (1994) also assayed the toxicity of Garlon 4 using embryos and tadpoles of *Rana*
43 *pipiens* (leopard frog), *Rana clamitans* (green frog), and *Rana catesbeiana* (bullfrog) in a static
44 assay with aeration, which was conducted in darkness to prevent hydrolysis of triclopyr BEE.
45 Exposures to 0.6, 1.2, and 4.6 mg a.e./L had no effect on hatching success, malformations, or
46 subsequent avoidance behavior of embryos. Newly hatched tadpoles died or became immobile

1 after exposure to the two higher concentrations. The approximate EC₅₀ values for response to
2 prodding were between 1.2 and 4.6 mg a.e./L after a 24-hour exposure period. As summarized
3 in Table 34, these EC₅₀ values for response to stimuli are very close to the LC₅₀ values for frog
4 larvae and probably reflect signs of nearly lethal exposures rather than sublethal effects on
5 behavior.

6 **4.1.3.3. Aquatic Invertebrates**

7 Studies on the toxicity of triclopyr acid, triclopyr TEA, triclopyr BEE, and TCP to aquatic
8 invertebrates are detailed in Appendix 7. The LC₅₀ values are given in Table 35, and these data
9 are illustrated in Figure 13.

10
11 As with groups of aquatic organisms, triclopyr BEE is clearly more toxic than triclopyr TEA.
12 Based on the median 48-hour LC₅₀ values, the triclopyr BEE is more toxic than triclopyr TEA to
13 aquatic invertebrates by a factor of about 140 [401 mg a.e./L ÷ 2.9 mg a.e./L ≈ 138.27]. This
14 difference is somewhat less than the well-documented difference in fish—i.e., triclopyr BEE is
15 more toxic than triclopyr TEA to fish by factor of about 240. The difference in sensitivity
16 between fish and aquatic invertebrates to triclopyr BEE, relative to the triclopyr TEA, is due
17 almost entirely to the greater tolerance of aquatic invertebrates to triclopyr TEA. Relative to
18 fish, aquatic invertebrates are more tolerant to triclopyr TEA by a factor of about 3 [402 ÷ 131
19 mg a.e./L ≈ 3.069]. In terms of sensitivity to triclopyr BEE, aquatic invertebrates are less
20 sensitive than fish by about a factor of 5 [2.9 mg a.e./L ÷ 0.54 mg a.e./L ≈ 5.37].

21
22 The above discussion excludes aquatic bivalves. As illustrated in Figure 13 and summarized in
23 Table 35, the toxicity values for aquatic bivalves are lower than those for other aquatic
24 invertebrates by a factor of about 20 for triclopyr TEA and 10 for triclopyr BEE. The difference
25 is an artifact of the types of bioassays conducted on bivalves. With the exception of the study by
26 Heitmuller (1975), all bivalve studies are standard assays for shell deposition using the Eastern
27 oyster (*Crassostrea virginica*). These studies are conducted for a period of 96 rather than 48
28 hours and do not involve mortality as an endpoint. Thus, these toxicity values are not directly
29 comparable to the 48-hour LC₅₀ values reported for most other invertebrates. One study with
30 two species of freshwater snail using triclopyr acid (Neuderfer 2009) suggests snails may be
31 somewhat more tolerant than aquatic arthropods are to triclopyr.

32
33 Only one study is available on the toxicity of TCP to aquatic invertebrates. This is a standard
34 48-hour LC₅₀ determination in *Daphnia magna*, which is summarized in U.S. EPA/OPP (2009a,
35 MRID 41829003). As with other organisms, TCP appears to be more toxic than triclopyr TEA
36 but less toxic than triclopyr BEE (Figure 13).

37
38 The distributions of LC₅₀ values for aquatic invertebrates (Figure 13) are reasonably smooth as is
39 true for other groups of aquatic organisms (Figures 11 and 12). As with other groups of aquatic
40 organisms, the ordering of species in the data for triclopyr TEA, however, do not suggest any
41 clear pattern in species sensitivity. Of the nine available LC₅₀ values for triclopyr TEA, the
42 ranking order of the values for daphnids range from of 2 to 8 (Table 35). For triclopyr BEE,
43 however, differences in sensitivity among the various groups of aquatic invertebrates do appear
44 to be systematic. These differences are illustrated in Figure 14 in which the Y-axis is plotted as
45 standard deviations from the mean (a frequency of 0.5) rather than as frequency proportions.
46 Except for not compositing species, Figure 14 may be viewed as species sensitivity distribution.

1 As illustrated in this figure, daphnids appear to be more sensitive than insects with other
2 arthropods (grass shrimp and crayfish) displaying intermediate sensitivity. The line in Figure 14
3 is based on a regression of the standard deviations against the logarithm of the dose.
4 Nonetheless, the overall distribution of the LC₅₀ values fits the assumption of a lognormal
5 distribution of tolerances extremely well ($r^2=0.98$, $p=1.7 \times 10^{-10}$).

6
7 As discussed further in Section 4.2 (exposure assessment), standard accidental exposure
8 scenarios considered in this risk assessment include a spill of the pesticide into a small pond as
9 well as accidental direct spray of a pond and stream. The accidental spray of a stream is likely to
10 involve very short-term pulse exposures. Kreutzweiser et al. (1992) conducted a series of 1-hour
11 bioassays of triclopyr BEE in several species of stream invertebrates. Based on these bioassays
12 (Kreutzweiser et al. 1992, Table 4), LC₅₀ values for these aquatic invertebrates were greater than
13 290 mg/L (≈ 200 mg a.e./L). These LC₅₀s are higher than the standard 48-hour LC₅₀s for
14 triclopyr BEE by about 2 orders of magnitude. While 1-hour LC₅₀ values are not typically
15 available and are not routinely used in Forest Service risk assessments, these data from
16 Kreutzweiser et al. (1992) are considered further in the risk characterization for aquatic
17 invertebrates (Section 4.4.3.4).

18 **4.1.3.4. Aquatic Plants**

19 **4.1.3.4.1. Algae**

20 Studies on the toxicity of triclopyr TEA, triclopyr BEE, and TCP to aquatic algae are detailed in
21 Appendix 8, Table A8-1. The EC₅₀ values for growth inhibition are given in Table 36, and these
22 data are illustrated in Figure 15.

23
24 While data are available on relatively few species of algae, six species have been assayed with
25 triclopyr TEA. While there is some scatter in terms of the sensitivity of *Chlorella* species
26 (spherical algae), the available data suggest that filamentous or rod shaped algae – e.g., species
27 of *Ankistrodesmus*, *Anabaena*, and *Skeletonema* – may be somewhat more sensitive to triclopyr
28 than more spherical species of algae such as *Chlorella* species.

29
30 As with aquatic animals, triclopyr BEE is more toxic than triclopyr TEA to algae by about a
31 factor of 10. Because triclopyr is an effective herbicide, it might be expected that triclopyr
32 would be more toxic to algae than to aquatic animals. While this is the case with triclopyr TEA,
33 triclopyr BEE appears to be as toxic if not slightly more toxic to fish (with a median LC₅₀ of
34 about 0.5 mg a.e./L) than to algae.

35
36 Only two bioassays are available on the toxicity of TCP to algae. Both of these report EC₅₀s of
37 1.8 mg/L. Thus, the line for TCP in Figure 15 is vertical. Nonetheless, based on these
38 admittedly limited data, TCP appears to be more toxic to algae than triclopyr TCP.

39
40 In addition to the standard toxicity bioassays summarized in Table 36, Peterson et al. (1994)
41 examined the effects of triclopyr on carbon fixation in several algal species. The investigators
42 noted no or relatively little inhibition in carbon fixation at concentrations of 2.6 mg/L triclopyr
43 acid.

1 **4.1.3.4.2. Aquatic Macrophytes**

2 Studies on the toxicity of triclopyr TEA and triclopyr BEE to aquatic macrophytes are detailed in
3 Appendix 8, Table A8-2. The 7- to 14-day EC₅₀ values for damage to aquatic macrophytes are
4 given in Table 37, and these data are illustrated in Figure 16. Data are not available on the
5 toxicity of TCP to aquatic macrophytes. While TCP is typically viewed as not being phytotoxic,
6 at least to terrestrial plants, the data on algae discussed in the previous section suggest that TCP
7 may be as phytotoxic as triclopyr BEE to aquatic plants.

8
9 The data illustrated in Figure 16 are segregated by the effects of both triclopyr TEA and triclopyr
10 BEE to both monocots and dicots. As discussed in Section 4.1.2.5, triclopyr TEA is more toxic
11 to dicots than to monocots, and this pattern is clearly true for aquatic macrophytes as well.
12 Aquatic dicots represent the only group of organisms to which triclopyr TEA appears to be
13 clearly more toxic than triclopyr BEE. While only four EC₅₀ values support this assertion, they
14 are based on data from three separate studies. As summarized in Table 37, Roshon et al. (1999)
15 report a much higher EC₅₀ for triclopyr TEA than the EC₅₀ values reported by Perkins (1997) and
16 Poovey et al. (2007); furthermore, this EC₅₀ from Roshon et al. (1999) accounts for the highly
17 irregular pattern in Figure 16 for the effect of triclopyr TEA on dicots. The reason for the
18 substantial difference in the study by Roshon et al. (1999) is not apparent.

19
20 Also as with terrestrial plants (Section 4.1.2.5), the differences in the efficacy to dicots and
21 monocots for triclopyr BEE is much less pronounced than that for triclopyr TEA. Based on the
22 limited data that are available, triclopyr BEE appears to be about equally toxic to both monocots
23 and dicots. Numerous efficacy studies are available on the use of Garlon 3A to control unwanted
24 aquatic vegetation (Appendix 8, Table A8-3).

4.2. EXPOSURE ASSESSMENT

4.2.1. Overview

For terrestrial applications, a standard set of exposure assessments is given for broadcast foliar application methods of triclopyr TEA (Attachment 1) and triclopyr BEE (Attachment 2). A subset of the standard exposure scenarios is provided for aquatic application to emergent vegetation (Attachment 3) and submerged vegetation (Attachment 4). All workbooks use a unit application rate of 1 lb a.e./acre, except for the workbook for submergent applications which uses a target concentration of 1 mg a.e./L. The use of other application rates is discussed in the risk characterization. Exposure assessments are also conducted for TCP for terrestrial applications (Attachment 5), emergent aquatic applications (Attachment 6), and submergent aquatic applications (Attachment 7). As in the human health risk assessment, three general types of exposure scenarios are considered: accidental, acute non-accidental, and longer-term.

Exposure assessments for mammals and birds are summarized in Worksheet G01 of the EXCEL workbooks that accompany this risk assessment. The highest exposures are associated with the consumption of contaminated grasses, and the lowest exposures are associated with the consumption of contaminated water. This is a common pattern for pesticides applied to vegetation. The exposure assessment for mammals is somewhat more detailed to encompass more diverse body weights. This approach is taken because the toxicity data (Section 4.3.2) indicate that larger mammals are more sensitive than smaller mammals to triclopyr.

For terrestrial plants, five exposure scenarios are considered quantitatively: direct spray, spray drift, runoff, wind erosion, and the use of contaminated irrigation water. Unintended direct spray is expressed simply as the application rate. As with terrestrial animals, all exposure assessments used in the workbooks accompanying this risk assessment are based on a unit application rate of 1 lb a.e./acre. The consequences of using other application rates are discussed in the risk characterization. Exposures of aquatic plants and animals to triclopyr are based on essentially the same information used to assess the exposure to terrestrial species from contaminated water.

Exposures to aquatic organisms are based on the same concentrations of triclopyr and TCP in surface water used in the human health risk assessment.

4.2.2. Terrestrial Vertebrates

All exposure scenarios for terrestrial animals are summarized in Worksheet G01 in the EXCEL workbooks accompanying this risk assessment (Attachments 1 to 3 for terrestrial applications, Attachments 3 and 4 for aquatic applications, and Attachments 5 to 7 for TCP).

For terrestrial applications of triclopyr, mammals and birds might be exposed to any applied pesticide from direct spray, the ingestion of contaminated media (e.g., vegetation, prey species, or water), grooming activities, or indirect contact with contaminated vegetation. In the exposure assessments for the ecological risk assessment, estimates of oral exposure to mammals and birds are expressed in the same units as the available toxicity data. As in the human health risk assessment, these units are usually expressed as mg of agent per kg of body weight and abbreviated as mg/kg for terrestrial animals. Unless otherwise specified, all exposure estimates for triclopyr are expressed as mg a.e. (acid equivalents).

1 An overview of the mammalian and avian receptors considered in the current risk assessment is
2 given in Table 38. These data are discussed in the following subsections. Because of the
3 relationship of body weight to surface area as well as to the consumption of food and water,
4 small animals will generally receive a higher dose, in terms of mg/kg body weight, relative to
5 large animals, for a given type of exposure. Thus, most Forest Service risk assessments focus on
6 the small mammal. As discussed in Section 4.1.2.1.1, however, a complication with triclopyr is
7 that larger mammals appear to be substantially more sensitive than smaller mammals to triclopyr
8 (i.e., evidence adverse effects at lower doses). In order to more fully consider the offsetting
9 factors of exposure and sensitivity in large and small mammals, the exposure assessment for
10 mammals is elaborated to consider five nontarget mammals: small (20 g) and medium (400 g)
11 sized omnivores, a 5 kg canid, a 70 kg herbivore, and a 70 kg carnivore. No remarkable
12 differences in sensitivities among birds are apparent (Section 4.3.2.2). Consequently, only four
13 standard avian receptors are considered: a 10 g passerine, a 640 g predatory bird, a 2.4 kg
14 piscivorous bird, and a 4 kg herbivorous bird.

15
16 No toxicity data are available on terrestrial phase amphibians (Section 4.1.2.3). Consequently,
17 exposure assessments for these terrestrial vertebrates are not developed.

18
19 For aquatic applications, the exposure assessments for terrestrial animals are a subset of those
20 included for terrestrial applications. In aquatic applications, triclopyr will be applied directly to
21 or under the surface water; consequently exposure scenarios concerning the consumption of
22 contaminated vegetation or fruit, the direct spray of a small mammal, and the consumption of a
23 sprayed small mammal by a predator are not included for aquatic applications.

24 **4.2.2.1. Direct Spray**

25 The unintentional direct spray of wildlife during broadcast applications of a pesticide is a
26 credible exposure scenario, similar to the accidental exposure scenarios for the general public
27 discussed in Section 3.2.3.2. In a scenario involving exposure to direct spray, the amount of
28 pesticide absorbed depends on the application rate, the surface area of the organism, and the rate
29 of absorption.

30
31 For this risk assessment, two direct spray or broadcast exposure assessments are conducted for
32 terrestrial applications. The first spray scenario (Worksheet F01) concerns the direct spray of
33 half of the body surface of a 20 g mammal during pesticide application. This exposure
34 assessment assumes first-order dermal absorption. The second exposure assessment (Worksheet
35 F02) assumes complete absorption over Day 1 of exposure. This assessment is included in an
36 effort to encompass the increased exposure due to grooming.

37
38 Although larger mammals appear to be more sensitive than smaller mammals, exposure
39 assessments for the direct spray of a large mammal are not developed. As discussed further in
40 Section 4.4.2.1, the direct spray scenarios lead to HQs far below the level of concern, and an
41 elaboration for body size would have no impact on the risk assessment.

42 **4.2.2.2. Dermal Contact with Contaminated Vegetation**

43 As discussed in the human health risk assessment (Section 3.2.3.3), the only approach for
44 estimating the potential significance of dermal contact with contaminated vegetation is to assume
45 a relationship between the application rate and dislodgeable foliar residue. Unlike the human

1 health risk assessment, in which estimates of transfer rates are available, there are no transfer
2 rates available for wildlife species. Wildlife species are more likely than humans to spend long
3 periods of time in contact with contaminated vegetation. It is reasonable to assume that for
4 prolonged exposures, equilibrium may be reached between pesticide levels on the skin, rates of
5 dermal absorption, and pesticide levels on contaminated vegetation. Since data regarding the
6 kinetics of this process are not available, a quantitative assessment for this exposure scenario
7 cannot be made in the ecological risk assessment.
8

9 For triclopyr, as well as most other herbicides and insecticides applied in broadcast applications,
10 the failure to quantify exposures associated with dermal contact adds relatively little uncertainty
11 to the risk assessment, because the dominant route of exposure will be the consumption of
12 contaminated vegetation.

13 ***4.2.2.3. Ingestion of Contaminated Vegetation or Prey***

14 In foliar applications, the consumption of contaminated vegetation is an obvious concern.
15 Exposure assessments for the consumption of contaminated vegetation are developed for all
16 mammals and birds listed in Table 38, except for the large carnivorous mammal and the
17 predatory bird. Both acute and chronic exposure scenarios are developed for the consumption of
18 contaminated fruit (Worksheets F04a-e for acute and Worksheets F10a-e for chronic) and the
19 consumption of short grass (Worksheets F05a-e for acute and Worksheets F11a-e for chronic).
20

21 As summarized in Table 27, fruit and short grass are the food items that comprise the
22 commodities with the lowest residue rates (fruit) and the highest residue rates (short grass).
23 These food items are not necessarily intended to be interpreted literally; instead, they are
24 intended to encompass the range of triclopyr and TCP concentrations in food items likely to be
25 consumed by a variety of mammals and birds.
26

27 For both the acute and chronic exposure scenarios, the assumption is made that 100% of the diet
28 is contaminated. This may not be a realistic assumption for some acute exposures and will
29 probably be a rare event in chronic exposures—i.e., animals may move in and out of the treated
30 areas. While estimates of the proportion of the diet that is contaminated could be incorporated
31 into the exposure assessment, the estimates would be an essentially arbitrary set of adjustments.
32 Because the proportion of the diet that is contaminated is linearly related to the resulting HQs,
33 the impact of variations in the proportion of the diet that consists of contaminated food is
34 discussed further in the risk characterization (Section 4.4.2.1).
35

36 The initial concentrations of triclopyr in the food items is based on the U.S. EPA/OPP (2001)
37 adaptation of the residue rates from Fletcher et al. (1997), as summarized in Table 27. The
38 methods of estimating the peak and time-weighted average concentrations of triclopyr and TCP
39 are identical to those used in the human health risk assessment (Section 3.2.3.7 for triclopyr and
40 Section 3.2.3.8 for TCP).
41

42 The estimated food consumption rates by various species of mammals and birds are based on
43 field metabolic rates (kcal/day), which, in turn, are based on the adaptation of estimates from
44 Nagy (1987) by the U.S. EPA/OPP (1993). These allometric relationships account for much of
45 the variability in food consumption among mammals and birds. There is, however, residual
46 variability, which is remarkably constant among different groups of organism (Nagy 1987, Table

1 3). As discussed further by Nagy (2005), the estimates from the allometric relationships may
2 differ from actual field metabolic rates by about $\pm 70\%$. Consequently, in all worksheets
3 involving the use of the allometric equations for field metabolic rates, the lower bound is taken
4 as 30% of the estimate and the upper bound is taken as 170% of the estimate.

5
6 The estimates of field metabolic rates are used to calculate food consumption based on the
7 caloric value (kcal/day dry weight) of the food items considered in this risk assessment and
8 estimates of the water content of the various foods. Estimates of caloric content are summarized
9 in Table 39. Most of the specific values in Table 39 are taken from Nagy (1988) and U.S.
10 EPA/ORD (1993).

11
12 Along with the exposure scenarios for the consumption of contaminated vegetation, similar sets
13 of exposure scenarios are provided for the consumption of small mammals by either a predatory
14 mammal (Worksheet F08a) or a predatory bird (Worksheet F08b) as well as the consumption of
15 contaminated insects by a small mammal, a 400 g mammal, and a small bird (Worksheets
16 F07a-c).

17 **4.2.2.4. Ingestion of Contaminated Water**

18 The methods for estimating triclopyr and TCP concentrations in water are identical to those used
19 in the human health risk assessment (Section 3.2.3.4). The only major differences in the
20 estimates of exposure involve the weight of the animal and the amount of water consumed. As
21 with the estimates of food consumption, water consumption rates are well characterized in
22 terrestrial vertebrates. The water consumption rates are based on allometric relationships in
23 mammals and birds, as summarized in Table 38. Based on these estimates, exposure scenarios
24 involving the consumption of contaminated water are developed for mammals and birds for
25 accidental spills (Worksheets F02a-e), expected peak expected concentrations (Worksheets
26 F06a-e), and expected longer-term concentrations (Worksheets F12a-e).

27
28 As with food consumption, water consumption in birds and mammals will vary substantially
29 with diet, season, and many other factors; however, there are no well-documented quantitative
30 estimates regarding the variability of water consumption by birds and mammals in the available
31 literature. Accordingly, the variability in water consumption rates of birds and mammals is not
32 considered in the exposure assessments. As summarized in Table 26, however, the upper and
33 lower bounds of the estimated concentrations of both triclopyr and TCP in surface water vary by
34 several orders of magnitude. Given this variability in the concentrations of triclopyr and TCP in
35 surface water, it seems likely that a quantitative consideration of the variability in water
36 consumption rates of birds and mammals would have a no substantial impact on the risk
37 characterization.

38 **4.2.2.5. Ingestion of Contaminated Fish**

39 In addition to the consumption of contaminated vegetation, insects, and other terrestrial prey
40 (Section 4.2.2.3), exposure pathways for triclopyr and TCP may be associated with the
41 consumption of contaminated fish. Thus, sets of scenarios are developed for an accidental spill
42 (Worksheets F03a-b), expected peak exposures (Worksheets F09a-c), and estimated longer-term
43 concentrations (Worksheets F13a-c). These exposure pathways are applied to 5 and 70 kg
44 carnivores as well as a piscivorous bird.

1 The 70 kg carnivorous mammal would be typical of a black bear (which does not actively hunt
2 fish) but could be representative of a small or immature Great Plains Grizzly Bear (*Ursus arctos*
3 *horribilis*) which is an endangered species and does actively feed on fish (Reid 2006). While a
4 larger body weight could be used for a grizzly bear, this is not done in order to avoid an
5 unreasonable extrapolation of toxicity values. As discussed further in Section 4.3.2.1.1, the
6 human RfD for triclopyr is somewhat supportive of the extrapolation of the allometric
7 relationships for toxicity up to a body weight of 70 kg. Extrapolations to the body weight of a
8 large grizzly bear (≈ 950 kg, Reid 2006, p. 451) would result in a lower toxicity value (≈ 0.46
9 mg/kg bw) than is used for the 70 kg bear (≈ 1.8 mg/kg bw); however, the degree of extrapolation
10 would be far beyond the body weights on which toxicity data are available (≈ 10 kg).

11 **4.2.3. Terrestrial Invertebrates**

12 **4.2.3.1. Direct Spray and Drift**

13 Estimated levels of exposure associated with broadcast terrestrial applications of triclopyr are
14 detailed in Worksheet G02b of Attachment 1 (terrestrial applications of triclopyr TEA) and
15 Attachment 2 (terrestrial applications of triclopyr BEE). These are custom worksheets which
16 include aerial, ground broadcast (high boom and low boom), and backpack applications.

17
18 Honeybees are used as a surrogate for other terrestrial insects, and honeybee exposure levels
19 associated with broadcast applications are modeled as a simple physical process based on the
20 application rate and surface area of the bee. The surface area of the honeybee (1.42 cm²) is
21 based on the algorithms suggested by Humphrey and Dykes (2008) for a bee with a body length
22 of 1.44 cm.

23
24 The amount of a pesticide deposited on a bee during or shortly after application depends on how
25 close the bee is to the application site as well as foliar interception of the spray prior to
26 deposition on the bee. The estimated proportions of the nominal application rate at various
27 distances downwind given in G02b are based on Tier 1 aerial estimates from AgDrift (Teske et
28 al. 2002) for distances of 0 (direct spray) to 900 feet downwind of the treated site.

29
30 In addition to drift, foliar interception of a pesticide may occur. The impact of foliar interception
31 would vary depending on the nature of the canopy above the bee. For example, in studies
32 investigating the deposition rate of diflufenzuron in various forest canopies, Wimmer et al.
33 (1993) report that deposition in the lower canopy, relative to the upper canopy, generally ranged
34 from about 10% (90% foliar interception in the upper canopy) to 90% (10% foliar interception by
35 the upper canopy). In Worksheet G02b, foliar interception rates of 0% (no interception), 50%,
36 and 90% are used.

37
38 During broadcast applications of a pesticide, it is likely that terrestrial invertebrates other than
39 bees will be subject to direct spray. As discussed in further detail in Section 4.3.2.3 (dose-
40 response assessment for terrestrial invertebrates), the available toxicity data on terrestrial
41 invertebrates do not support the derivation of separate toxicity values for different groups of
42 terrestrial insects. Thus, the honeybee is used as a surrogate for other insect species.

1 **4.2.3.2. Ingestion of Contaminated Vegetation or Prey**

2 Like terrestrial mammals and birds, terrestrial invertebrates may be exposed to triclopyr through
3 the consumption of contaminated vegetation or contaminated prey. For broadcast foliar
4 applications, estimates of residues on contaminated vegetation or prey are based on estimated
5 residue rates (i.e., mg/kg residues per lb a.i. applied) from Fletcher et al. (1994), as summarized
6 in Table 27.

7
8 An estimate of food consumption is necessary to calculate a dose level for a foraging
9 herbivorous insect. Insect food consumption varies greatly, depending on the caloric
10 requirements in a given life stage or activity of the insect and the caloric value of the food to be
11 consumed. The derivation of consumption values for specific species, life stages, activities, and
12 food items is beyond the scope of the current analysis. Nevertheless, general food consumption
13 values, based on estimated food consumption per unit body weight, are readily available.

14
15 Reichle et al. (1973) studied the food consumption patterns of insect herbivores in a forest
16 canopy and estimated that insect herbivores may consume vegetation at a rate of about 0.6 of
17 their body weight per day (Reichle et al. 1973, pp. 1082 to 1083). Higher values (i.e., 1.28-2.22
18 in terms of fresh weight) are provided by Waldbauer (1968) for the consumption of various types
19 of vegetation by the tobacco hornworm (Waldbauer 1968, Table II, p. 247). The current risk
20 assessment uses food consumption factors of 1.3 (0.6 to 2.2) kg food /kg bw. The lower bound
21 of 0.6 is taken from Reichle et al. (1973), and the central estimate and upper bound are taken
22 from the range of values provided by Waldbauer (1968).

23
24 Details concerning estimated exposure levels for the consumption of contaminated vegetation by
25 herbivorous insects are provided in Worksheets G07a, G07b, G07c, and G07d. These levels
26 pertain to the four food items included in the standard residue rates provided by Fletcher et al.
27 (1994). The exposure estimates are included in the EXCEL workbooks only for terrestrial foliar
28 applications (Attachments 1 and 2).

29 **4.2.3.3. Foraging Honeybees**

30 If data are available on the concentration of a pesticide in pollen or nectar, exposure assessments
31 may be conducted for honeybees (e.g., Alix and Vergnet 2007; Halm et al. 2006; Rortais et al.
32 2005). No such data are available for triclopyr; consequently, an exposure assessment is not
33 developed for foraging honeybees.

34 **4.2.4. Terrestrial Plants**

35 Generally, the primary hazard to nontarget terrestrial plants associated with the application of
36 most herbicides is unintended direct deposition or spray drift. In addition, herbicides may be
37 transported off-site by percolation or runoff or by wind erosion of soil. As noted in Section
38 4.1.2.5 (Hazard Identification for Terrestrial Plants) and discussed further in Section 4.3.2.5
39 (Dose-Response Assessment for Terrestrial Plants), the toxicity data on triclopyr are sufficient to
40 interpret risks associated with these exposure scenarios. Consequently, exposure assessments are
41 developed for each of these exposure scenarios, as detailed in the following subsections. These
42 exposure assessments are detailed in Worksheet G04 (runoff), Worksheet G05 (direct spray and
43 drift), Worksheet G06a (contaminated irrigation water), and Worksheet G06b (wind erosion) of
44 the attachments for broadcast foliar applications—i.e., Attachment 1 for triclopyr TEA and
45 Attachment 2 for triclopyr BEE.

1 **4.2.4.1. Direct Spray**

2 Unintended direct spray will result in an exposure level equivalent to the application rate. For
3 many types of herbicide applications, it is plausible that some nontarget plants immediately
4 adjacent to the application site could be sprayed directly. This type of scenario is modeled in the
5 worksheets that assess off-site drift (see below).

6 **4.2.4.2. Off-Site Drift**

7 Because off-site drift is more or less a physical process that depends primarily on droplet size
8 and meteorological conditions rather than specific properties of the compound being sprayed,
9 estimates of off-site drift can be modeled using AgDrift. These estimates are summarized in
10 Worksheet G05 of the EXCEL workbook for terrestrial applications (Attachments 1). This
11 custom worksheet includes estimates of drift for aerial, ground broadcast, and backpack
12 applications.

13
14 The drift estimates used in the current risk assessment are based on AgDRIFT (Teske et al. 2002)
15 using Tier 1 analyses for aerial and ground broadcast applications. The term *Tier 1* is used to
16 designate relatively generic and simple assessments that may be viewed as plausible upper limits
17 of drift. Aerial drift estimates are based on Tier 1 using ASAE Fine to Medium drop size
18 distributions. Tier 1 estimates of drift for ground broadcast applications are modeled using both
19 low boom and high boom options in AgDRIFT. For both types of applications, the values are
20 based on Very Fine to Fine drop size distributions and the 90th percentile values from AgDrift.

21
22 Drift associated with backpack applications (directed foliar applications) is likely to be much less
23 than drift from ground broadcast applications. Few studies, however, are available for
24 quantitatively assessing drift after backpack applications. For the current risk assessment,
25 estimates of drift from backpack applications are based on an AgDRIFT Tier 1 run of a low
26 boom ground application using Fine to Medium/Coarse drop size distributions (rather than very
27 fine to fine) as well as 50th percentile estimates of drift (rather than the 90th percentile used for
28 ground broadcast applications).

29
30 The values for drift used in the current risk assessment should be regarded as little more than
31 generic estimates similar to the water concentrations modeled using GLEAMS (Section
32 3.2.3.4.3). Actual drift will vary according to a number of conditions—e.g., the topography,
33 soils, weather, and the pesticide formulation. All of these factors cannot be considered in this
34 general risk assessment.

35 **4.2.4.3. Runoff and Sediment Loss**

36 Exposures to terrestrial plants associated with runoff and sediment losses from the treated site to
37 an adjacent untreated site are summarized in Worksheet G04 of the EXCEL workbook for
38 terrestrial applications (Attachments 1).

39
40 Any pesticide can be transported from the soil at the application site by runoff, sediment loss, or
41 percolation. Runoff, sediment loss, and percolation are considered in estimating contamination
42 of ambient water. Only runoff and sediment loss are considered in assessing off-site soil
43 contamination. This approach is reasonable because off-site runoff and sediment transport will
44 contaminate the off-site soil surface and could impact non-target plants. Percolation, on the
45 other hand, represents the amount of the herbicide that is transported below the root zone and

1 thus may impact water quality but should not affect off-site vegetation. The GLEAMS modeling
2 used to estimate concentrations in water provides data on loss by runoff. As with the estimates
3 of triclopyr in surface water, runoff estimates are modeled for clay, loam, and sand at nine sites
4 that are representative of different temperatures and rainfall patterns (Table 21).

5
6 For triclopyr, the results of the standard GLEAMS modeling of runoff and sediment losses are
7 summarized in Appendix 9, Table A9-1 (triclopyr BEE) and Appendix 12, Table A12-1
8 (triclopyr TEA). Note that the proportion of runoff as a fraction of the application rate will vary
9 substantially with different types of soils as well as climates—i.e., temperature and rainfall.

10
11 The runoff for triclopyr TEA as a proportion of the application rate is taken as 0.00266 (0.00001
12 to 0.108) rounded to 0.0027 to 0.11. The central estimate and upper bound is taken directly from
13 the Gleams-Driver modeling—i.e., the median and empirical upper 95% bound. The lower limit
14 is the approximate lower bound for clay soils in areas with moderate to heavy rain. Although
15 lower loss rates of 1×10^{-6} to 1×10^{-8} are plausible, they have no impact on the risk
16 characterization. For triclopyr BEE, the rates, which are similarly derived, are much lower due
17 to the binding of triclopyr BEE to soil—i.e., rates of 0.0006 (2×10^{-7} to 0.046).

18
19 The amount of pesticide not washed off in runoff or sediment will penetrate into the soil column,
20 and the depth of penetration will depend on the properties of the chemical, the properties of the
21 soil, and the amount of rainfall. The GLEAMS model provides estimates of pesticide
22 concentrations in soil layers of varying depths. These concentrations are output by GLEAMS in
23 mg pesticide/kg soil (ppm). The minimum non-zero value that GLEAMS will output is
24 0.000001 mg/kg, equivalent to 1 nanogram/kg soil or 1 part per trillion (ppt).

25
26 The deepest penetration of triclopyr TEA in clay, loam, and sand modeled using GLEAMS is
27 summarized in Table A12-4 of Appendix 12. Based on GLEAMS modeling, the maximum
28 penetration of triclopyr into all three soil textures is 36 inches, the depth of the root zone used in
29 the Gleams-Driver modeling. As would be expected, lower penetration will occur in arid areas,
30 relative to soil penetration in areas with moderate to heavy rainfall.

31
32 Triclopyr BEE is much less likely to penetrate into the soil column. As indicated in Appendix 9,
33 Table A9-4, the maximum penetration is 24 inches, and this occurs only in sandy soils, cool
34 temperatures, and heavy rainfall. In relatively arid locations, the maximum penetration is
35 estimated at 4-8 inches.

36 ***4.2.4.4. Contaminated Irrigation Water***

37 Unintentional direct exposure of nontarget plants is possible from the use of contaminated
38 ambient water for irrigation, as observed by Bhandary et al. (1991) for certain herbicides. The
39 levels of exposure associated with this scenario will depend on the pesticide concentration in the
40 ambient water used for irrigation and the amount of irrigation water used. Concentrations in
41 ambient water are based on the peak concentrations modeled in the human health risk assessment
42 (Section 3.2.3.4). The amount of irrigation used will depend on the climate, soil type,
43 topography, and plant species under cultivation. Thus, the selection of an irrigation rate is
44 somewhat arbitrary.

1 In the absence of any general approach for determining and expressing the variability of
2 irrigation rates, the application of 1 inch of irrigation water is used in this risk assessment.
3 Details of the calculations used to estimate the functional application rates based on irrigation
4 using contaminated surface water are provided in Worksheet G06a (Attachments 1 and 2).
5

6 At a unit application rate of 1 lb a.e./acre, the functional application rate associated with the use
7 of contaminated surface water for irrigation after applications of triclopyr TEA is about 0.000068
8 (5.6×10^{-8} to 0.11) lb a.e./acre. For triclopyr BEE, the functional application rates are somewhat
9 lower—i.e., 9×10^{-5} (8.4×10^{-9} to 0.013) lb a.e./acre. The central and lower bounds of these
10 functional application rates are below the level of concern. The upper bound rates, however,
11 exceed the level of concern for sensitive species, and this matter is considered further in the risk
12 characterization.

13 **4.2.4.5. Wind Erosion**

14 Wind erosion can be a major transport mechanism for soil (e.g., Winegardner 1996), and wind
15 erosion is also associated with the environmental transport of herbicides (Buser 1990). Wind
16 erosion leading to off-site movement of pesticides is likely to be highly site-specific. The
17 amount of triclopyr that might be transported by wind erosion depends on several factors,
18 including application rate, depth of incorporation into the soil, persistence in the soil, wind
19 speed, and topographical and surface conditions of the soil. Under desirable conditions—e.g.,
20 relatively deep (10 cm) soil incorporation, low wind speed, and surface conditions which inhibit
21 wind erosion—it is likely that an insubstantial amount of triclopyr will be transported by wind.
22

23 For this risk assessment, the potential effects of wind erosion are estimated in Worksheet G06b.
24 In this worksheet, it is assumed that triclopyr is incorporated into the top 1 cm of soil, which is
25 identical to the depth of incorporation used in GLEAMS modeling. Average soil losses are
26 estimated to range from 1 to 10 metric tons/ha/year with a typical value of 5 metric tons/ha/year.
27 These estimates are based on the results of agricultural field studies which found that wind
28 erosion may account for annual soil losses ranging from 2 to 6.5 metric tons/ha (Allen and
29 Fryrear 1977).
30

31 As noted in Worksheet G07b, offsite losses are estimated to reach as much as 0.014% of the
32 application rate. Larney et al. (1999), however, report that wind erosion of other herbicides
33 could be associated with losses up to 1.5% of the nominal application rate following soil
34 incorporation or 4.5% following surface application. This difference appears to be at least
35 partially due to the much higher soil losses noted by Larney et al. (1999)—i.e., up to 56.6 metric
36 tons/ha from a fallow field. The losses reflected in Worksheet G06b may be somewhat more
37 realistic for forest or rangeland applications, because herbicide applications are rarely made to
38 fallow areas. In any event, the higher offsite losses reported by Larney et al. (1999) are
39 comparable to exposures associated with offsite drift at distances of about 50 feet from the
40 application site following low boom (0.017) and high boom (0.05) ground broadcast applications
41 (Worksheet G05). All of the estimates for wind erosion and offsite drift are likely to vary
42 dramatically according to site conditions and weather conditions.

1 **4.2.5. Aquatic Organisms**

2 The plausibility of effects on aquatic species is assessed based on estimated concentrations of
3 triclopyr and TCP in water which are identical to those used in the human health risk assessment.
4 These values are summarized in Table 26 and discussed in Section 3.2.3.4.6 for both terrestrial
5 and aquatic applications of triclopyr.

4.3. DOSE-RESPONSE ASSESSMENT

4.3.1. Overview

Like other major sections of this risk assessment, the dose-response assessment addresses triclopyr acid, triclopyr BEE, and the 3,5,6-trichloro-2-pyridinol (TCP) metabolite of triclopyr. Overviews of the toxicity values for terrestrial organisms are given in Table 40 for triclopyr acid, Table 41 for triclopyr BEE, and Table 42 for TCP. Similar overviews of the toxicity values for aquatic organisms are given in Table 43 for triclopyr acid, Table 44 for triclopyr BEE, and Table 45 for TCP. In the summary tables and in the following subsections, doses or exposures to triclopyr TEA and triclopyr BEE are expressed in units of mg a.e./kg bw or lb a.e./acre for plants. All toxicity values for TCP are expressed as mg/kg bw (i.e., mg TCP/kg bw) or mg/L (i.e., mg TCP/L).

For the most part, the toxicity values are either experimental NOAELs (mg/kg bw) or NOAECs (mg/L). The only major exceptions are longer-term toxicity values for triclopyr BEE in aquatic invertebrates. As detailed in Section 4.3.3.2, NOAELs for triclopyr BEE in aquatic invertebrates cannot be estimated reasonably. A minor exception to the use of NOAELs is the EC₁₀ used as a toxicity value for triclopyr BEE in sensitive species of aquatic phase amphibians (Section 4.3.3.2.2). Another minor exception involves the use of an indeterminate LD₅₀ for honeybees. Honeybees are not sensitive to triclopyr, and the use of an indeterminate LD₅₀ for honeybees has no impact on the characterization of risk (Section 4.4).

Data on triclopyr TEA are typically included in the dose-response assessment for triclopyr acid because these two forms of triclopyr appear to be bioequivalent in most groups of organisms. Data on triclopyr BEE and formulations of triclopyr BEE are discussed separately for some groups of organisms, primarily because the toxicity of triclopyr BEE formulations, expressed in units of triclopyr a.e., and technical grade triclopyr BEE, also expressed in units of triclopyr a.e., appears to be the same. In other words, the inerts used in the triclopyr BEE formulations do not have an obvious impact on the toxicity of the triclopyr BEE formulations on which data are available (primarily Garlon 4).

The dose-response assessments for triclopyr acid and triclopyr BEE in terrestrial animals are relatively standard and uncomplicated, except for mammals. The available toxicity data on triclopyr indicate that larger mammals are substantially more sensitive than smaller mammals, and this relationship can be characterized quantitatively. Most Forest Service risk assessments consider only small mammals and canids; however, the dose-response assessment for mammalian wildlife is elaborated to include a large herbivorous mammal, such as a deer. The dose-response assessments in terrestrial plants are also relatively standard for triclopyr acid and the triclopyr ester. For TCP, the available data limit the dose-response assessment for terrestrial organisms to mammals.

The dose-response assessment for aquatic species is somewhat detailed, because triclopyr acid and triclopyr BEE are not bioequivalent in aquatic organisms. With the exception of aquatic dicots, triclopyr BEE is much more toxic than triclopyr acid or triclopyr TEA. Within most groups of aquatic organisms, the toxicity values differ substantially for both triclopyr TEA and triclopyr BEE. Typically, this high variability reflects differences among bioassays conducted

1 by different investigators at different times rather than true underlying differences in species
2 sensitivity. A possible exception involves the toxicity of triclopyr BEE to aquatic arthropods.
3 Within this group, cladocerans appear to be more sensitive than aquatic insects to triclopyr BEE.
4

5 The toxicity values for TCP span much narrower ranges than the toxicity values for triclopyr.
6 This difference is almost certainly due to the fewer number of studies available on TCP.

7 **4.3.2. Toxicity to Terrestrial Organisms**

8 **4.3.2.1. Mammals**

9 **4.3.2.1.1. Triclopyr**

10 Typically, the dose-response assessment for mammalian wildlife is simple, and the acute and
11 chronic NOAELs used to derive the acute and chronic RfDs in the human health risk assessment
12 are used to characterize risks to mammalian wildlife. As discussed in Section 3.3.2 and
13 summarized in Table 29, the acute NOAEL is 100 mg/kg bw and the chronic NOAEL is 5 mg/kg
14 bw/day. Both of these NOAELs are based on studies in rats. Because triclopyr and triclopyr
15 BEE appear to be bioequivalent in mammals, the acute and chronic RfDs are applied to both
16 triclopyr acid and triclopyr BEE.
17

18 For triclopyr, however, the dose-response assessment for mammalian wildlife is more elaborate.
19 As discussed in Section 4.1.2.1.1, a clear allometric relationship is apparent in mammals for
20 subchronic and chronic toxicity. These relationships are summarized in Table 32 and illustrated
21 in Figure 10. Based on pooled subchronic and chronic NOAELs and LOAELs, the allometric
22 relationship for the geometric mean of the NOAELs and LOAELs (Y) is defined by the
23 following equation:
24

$$Y = 451 W^{-0.5}$$

Equation 10

25
26
27 where W is body weight in grams.
28

29 The above equation can be used to estimate the geometric mean of the NOAELs and LOAELs,
30 and these estimates can be used to define the relative sensitivity of different size mammals to
31 triclopyr. Using representative body weights from Table 32, Y would about 100 mg/kg bw for a
32 20 gram mouse, 22.5 mg/kg bw/day for a 400 g rat, 4.5 mg/kg bw for a 10 kg dog, and 1.7 mg/kg
33 bw/day for a 70 kg mammal such as a deer.
34

35 As noted above, the acute and chronic RfDs are based on the rat. Thus, in terms of sensitivities
36 relative to the rats, a 10 kg canid would be more sensitive by a factor of about 5 [$22.5 \div 4.5 = 5$]
37 and a 70 kg mammal would be more sensitive by a factor of about 13 [$22.5 \div 1.7 \approx 13.23$].
38 Similarly, a 20 g mouse would be less sensitive than the rat by a factor of about 4.4 [100 mg/kg
39 $\text{bw} \div 22.5 \text{ mg/kg bw} \approx 4.4444$]. Thus, the acute NOAEL of 100 mg/kg bw for the rat and the
40 chronic NOAEL of 5 mg/kg bw/day for the rat, can be used to estimate acute and chronic
41 toxicity values for other mammalian species sensitivities, relative to rats.
42

43 The only substantial reservation with the approach outlined above involves the developmental
44 studies in mammals. As discussed in Section 3.1.9.1 and summarized in Table 10, the results of

1 a series of developmental studies involving gavage exposure suggests that rats are somewhat
2 more sensitive than rabbits to triclopyr. Although this finding clearly contradicts the allometric
3 relationship cited above, mammalian wildlife exposures to triclopyr will typically be more
4 consistent with those described in dietary studies on which the above allometric relationship is
5 based. Accordingly, the consistent allometric pattern observed in the dietary studies (Section
6 4.1.2.1.1) is used in the dose-response assessment for mammalian wildlife.

7
8 This allometric relationship can be used to estimate an RfD consistent with the EPA/OPP
9 (1998a) RfD of 0.05 mg/kg. As noted above, the geometric mean of the NOAEL and LOAEL
10 for a 70 kg mammal is about 1.7 mg/kg bw/day. Adjusting this dose by a factor of $10^{0.5}$ —i.e.,
11 the mid-point of the standard uncertainty factor of 10 to estimate a NOAEL for a LOAEL —
12 leads to an estimated NOAEL of about 0.54 mg/kg bw/day. The uncertainty factor applied to
13 this NOAEL would be 10 (for sensitive individuals within the human population) rather than 100
14 (an added factor of 10 for species extrapolation), because the allometric relationship accounts for
15 species extrapolation. The resulting RfD based on the allometric relationships in mammals,
16 rounded to one significant place, would be 0.05 mg/kg bw/day [$0.54 \text{ mg/kg bw/day} \div 10 = 0.054$
17 mg/kg bw/day], identical to the RfD derived by the U.S. EPA/OPP based on reproduction studies
18 (Section 3.3.2.2). Thus, the allometric relationship used in the dose-response assessment for
19 mammalian wildlife is consistent with the dose-response relationship used in the human health
20 risk assessment.

21
22 The remaining issue involves the applicability of the allometric relationship to acute exposures.
23 As discussed in Section 4.1.2.1.1, the acute LD_{50} values in mammals are limited to rats, and there
24 are no other data that can be used to estimate differences in species sensitivity for acute
25 exposures. In the absence of data, the allometric relationship derived from subchronic and
26 chronic toxicity data is applied to acute exposures.

27
28 Mammalian receptors used in Forest Service risk assessments are typically limited to a 20 g
29 mammal and 10 kg canid. The current risk assessment is elaborated to include a 400 g mammal,
30 typical of many species of squirrels, and a 70 kg herbivorous mammal, typical of many species
31 of deer (Reid 2006). For the 400 g mammal, the acute and chronic NOAELs for the rat are used
32 directly. As discussed above, the 20 g mammal is assumed to be less sensitive than the rat by a
33 factor of 4.4, the 10 kg canid is more sensitive than the rat by a factor of 5, and the 70 kg
34 herbivore is more sensitive than the rat by a factor of 13.

35
36 Using the above estimates, the NOAELs for the 20 g mammal are estimated as 440 mg/kg bw for
37 acute exposures [$100 \text{ mg/kg bw} \times 4.4$] and 22 mg/kg bw/day for chronic exposures [5×4.4
38 mg/kg bw]. For the canid, the toxicity values are taken as 20 mg/kg bw/day for acute exposures
39 [$100 \text{ mg/kg bw} \div 5$] and 1 mg/kg bw for chronic exposures [$5 \text{ mg/kg bw} \div 5$]. For the 70 kg
40 mammal, the toxicity values are taken as 8 mg/kg bw/day for acute exposures [$100 \text{ mg/kg bw} \div$
41 $13 \approx 7.69$] and 0.4 mg/kg bw/day for chronic exposures [$5 \text{ mg/kg bw} \div 13 \approx 0.38$]. As in the
42 human health risk assessment and for the same reasons, these estimated NOAELs are used for
43 both triclopyr acid and triclopyr BEE.

44 **4.3.2.1.2. TCP**

45 As noted in Section 4.1.2.1.2, the current risk assessment relies on the EPA review of the toxicity
46 of TCP (U.S. EPA/OPP 2002b). Neither the data in the EPA review nor the data found in the

1 open literature permits an assessment of species sensitivity to TCP for mammals. Consequently,
2 the NOAELs of 25 mg/kg bw for acute exposures and 12 mg/kg bw for longer-term term
3 exposures are used to characterize risks to all mammalian receptors associated with exposures to
4 TCP.

5 **4.3.2.2. Birds**

6 **4.3.2.2.1. Triclopry**

7 As discussed in Section 4.1.2.2.1, there is no remarkable difference in the toxicity of triclopry
8 acid, triclopry TEA, and triclopry BEE to birds. Similarly, the toxicity data, available only on a
9 few avian species, do not indicate substantial or systematic differences in species sensitivities to
10 triclopry.

11
12 The lowest acute NOAEL for signs of toxicity is 126 mg a.e./kg bw/day in Northern bobwhite
13 quail after gavage exposure to triclopry BEE (Campbell and Lynn 1991a). The Holmes et al.
14 (1994) study suggests that zebra finches (small passerines) may be somewhat more sensitive than
15 game birds or waterfowl. Based on the 8-day dietary study in zebra finches, body weight was
16 reduced at a dietary concentration of about 575 ppm a.e., corresponding to an estimated dose of
17 about 155 mg a.e./kg bw. The decrease in body weight, however, was associated with a decrease
18 in food consumption. Overt signs of toxicity were not observed. Thus, the NOAEL of 126 mg
19 a.e./kg bw in quail is used as a NOAEL for acute exposures in all birds. This is an admittedly
20 conservative approach because gavage dosing – i.e., the route of the 126 mg a.e./kg bw/day dose
21 – typically leads to severe effects than equivalent dietary dosing.

22
23 Based on standard reproduction studies in mallards and quail (Appendix 3, Table A3-3), the
24 estimated NOAELs range from about 7.5 mg a.e./kg bw/day in bobwhites to 54.7 mg a.e./kg
25 bw/day in mallards. The NOAEL in bobwhites is supported by the longer-term NOAEL of 9.7
26 mg a.e./kg bw in zebra finches reported by Holmes et al. (1994). The reproductive NOAEL of
27 7.5 mg/kg bw/day is applied to all species of birds to assess the consequences of longer-term
28 exposures to triclopry.

29
30 While some field studies investigate the impact of triclopry on bird populations (Appendix 3,
31 Table A3-4), they involve secondary effects of exposure and cannot be used to evaluate the acute
32 or chronic NOAELs discussed above.

33 **4.3.2.2.2. TCP**

34 As discussed in Section 4.1.2.2.2, relatively little information is available on the toxicity of TCP
35 to birds. No chronic toxicity studies are available and thus no dose-response assessment for
36 chronic effects in birds can be proposed. A single dose gavage study in quail reports an NOAEL
37 of 125 mg/kg bw (Campbell et al. 1990) but an acute 5-day dietary study reports a LOAEL based
38 on decreased body weight gain and food consumption of about 116 mg/kg bw/day (Long et al.
39 1990).

40
41 In the study by Long et al. (1990) no overt signs of toxicity were noted. The food consumption
42 in the 116 mg/kg bw/day dose group was about 65% of that in the control groups and the
43 decrease in body weight gain was about 79% of that in the control groups. Thus, while the 116
44 mg/kg bw/day dose group may be viewed as a LOAEL based on decreased food consumption

1 and body weight gain, the decrease in body weight gain could simply reflect taste aversion.
2 Given these relationships, the LOAEL of 116 mg/kg bw/day may have little toxicologic
3 significance and dividing the LOAEL by a factor of 10 to approximate a NOAEL does not seem
4 appropriate. As an alternative, the LOAEL of 116 mg/kg bw/day will be used directly and the
5 interpretation of the resulting HQs is discussed in the risk characterization (Section 4.4.2.2.2).

6 **4.3.2.3. Reptiles and Amphibians (Terrestrial Phase)**

7 Dose-response assessments for triclopyr and TCP in reptiles and terrestrial phase amphibians are
8 not proposed because no toxicity data are available on reptiles or terrestrial phase amphibians.

9 **4.3.2.4. Terrestrial Invertebrates**

10 Most ecological risk assessments conducted by the U.S. EPA/OPP use the honeybee as a
11 surrogate for other terrestrial insects. U.S. EPA/OPP (2009a, Table 4-3, p. 76) uses an indefinite
12 LD₅₀ of >72 µg a.e./bee for the honeybee. Typical body weights for worker bees range from 81
13 to 151 mg (Winston 1987, p. 54). Taking 116 mg as an average body weight, a dose of 72
14 µg/bee corresponds to about 620 mg/kg bw [$0.072 \text{ mg} \div 0.000116 \text{ kg} \approx 620.68 \text{ mg/kg bw}$]. In
15 the absence of additional data on other species of insects, the dose of 620 mg/kg bw is used to
16 derive hazard quotients for exposure scenarios associated with the direct spray of or drift onto a
17 bee (Section 4.2.3.1) as well as for the consumption of contaminated vegetation by herbivorous
18 insects (Section 4.2.3.2).

19
20 The use of an indefinite LD₅₀ rather than a well-documented NOAEC for the calculation of
21 hazard quotients is not a typical practice in Forest Service risk assessments. As discussed in
22 Section 4.1.2.4, there is a reasonably extensive group of field studies indicating that effects on
23 terrestrial invertebrates are likely to be based on secondary effects on vegetation rather than
24 toxicity. While hazard quotients (HQs) are derived for insects using the toxicity value of 620
25 mg/kg bw, the risk characterization for insects is based primarily on the field studies rather than
26 the HQs (Section 4.4.2.4).

27
28 A dose-response assessment of the toxicity of TCP to terrestrial invertebrates cannot be proposed
29 due to the lack of pertinent data.

30 **4.3.2.5. Terrestrial Plants (Macrophytes)**

31 For both triclopyr TEA and triclopyr BEE formulations, as for most herbicides, there are
32 adequate data from which to derive toxicity values for sensitive and tolerant species of terrestrial
33 plants. The available studies are discussed in Section 3.1.2.5 and summarized in Appendix 4.
34 The available studies include assays for both foliar spray, which are used to assess effects
35 associated with direct spray, wind erosion, or drift, as well as seedling emergence assays, which
36 are used to assess soil exposures associated with herbicide runoff to an untreated field.

37
38 Assays involving foliar spray are summarized in Appendix 4, Table A4-1. As discussed in
39 Section 3.1.2.5, foliar studies do not suggest any remarkable differences in potency between
40 triclopyr TEA and triclopyr BEE formulations; what's more, dicots are more sensitive than
41 monocots to both formulations. For both types of formulations, the lowest NOAEC is 0.0028 lb
42 a.e./acre in sunflowers, which is used to characterize risks to sensitive species of terrestrial
43 plants. Monocots are much more tolerant to both types of formulations. The highest reported

1 NOAEC is about 2 lb a.e./acre (2.242 g/ha) in oats and is used to assess risks to tolerant species
2 of terrestrial plants.

3
4 The EC₂₅ values reported in seedling emergence studies (Appendix 4, Table A4-2) indicate that
5 triclopyr BEE formulations are somewhat more toxic than triclopyr TEA formulations. In terms
6 of NOAECs, the differences are less substantial. For triclopyr BEE, the NOAECs range from 35
7 g a.i./ha (\approx 0.022 lb a.e./acre) to >2242 g a.i./ha (\approx 2 lb a.e./acre). For triclopyr TEA, relatively
8 few NOAECs are available, and the lower and upper bounds of the range of NOAECs are used
9 for sensitive species (NOAEC = 0.0028 lb a.e./acre) and tolerant species (NOAEC = 0.23 lb
10 a.e./acre).

11
12 A dose-response assessment of the phytotoxicity of TCP is not proposed because no data are
13 available on the toxicity of TCP to terrestrial plants.

14 **4.3.2.6. Terrestrial Microorganisms**

15 No formal dose-response assessment is developed for terrestrial microorganisms. As discussed
16 further in Section 4.4.2.6, the available field studies on triclopyr are used to qualitatively
17 characterize risks in this group of organisms.

18 **4.3.3. Aquatic Organisms**

19 In the hazard identification (Section 4.1.3), LC₅₀ and EC₅₀ values are used to identify the
20 toxicities of triclopyr acid, triclopyr BEE, and TCP to aquatic organisms as well as to identify
21 differences in species sensitivity among these organisms. The Forest Service prefers, however,
22 to use NOAECs and not LC₅₀ and EC₅₀ values to characterize risk for aquatic organisms exposed
23 to triclopyr. Thus, whenever possible, NOAECs are identified and selected as the basis of the
24 dose-response assessment for aquatic organisms, in the following subsections. For all groups of
25 organisms covered in the following subsections, an attempt is made to identify NOAECs for both
26 sensitive and tolerant species.

27
28 If NOAECs are not available, LC₅₀ or EC₅₀ values may be multiplied by 0.05 to approximate an
29 NOAEC. This procedure is based on the U.S. EPA/OPP general approach of using LC₅₀ or EC₅₀
30 values with levels of concern (LOC) of 0.05 for the ratio of exposure to the LC₅₀ or EC₅₀ for
31 endangered species (e.g., U.S. EPA/OPP 2009a, Appendix C). It should be noted that this is a
32 very conservative approach, equivalent to treating all aquatic species as endangered species.

33
34 As noted in several instances below, an intermediate approach can be taken to estimate NOAECs
35 for sensitive and tolerant species. When there is not an NOAEC for the most sensitive or most
36 tolerant species within a group of organisms, but there is either an LC₅₀ or EC₅₀ with a
37 corresponding NOAEC for one or more other species in the group, the ratio of the available
38 NOAEC to the available LC₅₀ or EC₅₀ can be used to estimate an NOAEC for the most sensitive
39 or tolerant species.

40
41 Few chronic NOAECs are available for any group of aquatic organisms. For some groups (e.g.,
42 algae), the lack of a chronic NOAEC is not a concern, because chronic is not meaningful in the
43 context of exposure for organisms with very short lifespans. For fish and invertebrates, however,
44 attempts are made to incorporate the very well-documented variability in acute data into the
45 chronic dose-response assessment. Consequently, acute-to-chronic ratios are developed for the

1 species on which both acute and chronic toxicity data are available; furthermore, these ratios are
2 used to estimate chronic NOAECs for sensitive and tolerant species. As detailed below, this
3 approach is used only when it appears to be sensible given the available species-specific data.

4 **4.3.3.1. Fish**

5 **4.3.3.1.1. Triclopyr Acid**

6 Acute LC₅₀ values for triclopyr TEA range from 40.1 to 422.8 mg a.e./L (Table 33) and
7 encompass the more limited number of LC₅₀ values available on triclopyr acid (Appendix 5,
8 Table A5-1). The acute sublethal toxicity of triclopyr acid and triclopyr TEA is not well
9 documented either in standard acute toxicity studies or field studies (Appendix 5, Table A5-6).
10 Morgan et al. (1991) reports a NOAEL of 63.6 mg a.e./L for behavioral changes in rainbow
11 trout; however, this NOAEL is not directly useful because it is close to the LC₅₀ of 79.2 mg
12 a.e./L in trout reported by Batchelder (1973). In a standard acute bioassay in silversides, Ward
13 and Boeri (1989) report an NOAEC of 44 mg a.e./L with a corresponding LC₅₀ of 93 mg
14 a.e./L—i.e., the ratio of the NOAEC to the LC₅₀ is about 0.5 [44 mg a.e./L ÷ 93 mg a.e./L ≈
15 0.47]. This ratio is identical to the LOC of 0.5 used in U.S. EPA/OPP (2009a, Appendix C) for
16 acute risk.

17
18 For triclopyr acid, the factor of 0.5 can be used to estimate NOAECs based on the range of LC₅₀
19 values—i.e., 40.1 to 422.8 mg a.e./L x 0.5. Thus, the NOAECs range from 20 mg a.e./L
20 (sensitive species) to 210 mg a.e./L (tolerant species).

21
22 The only chronic data available on triclopyr acid is the 32.2 mg a.e./L NOAEC in the egg-to-fry
23 study by Mayes (1983), as summarized in Appendix 5, Table A-5. Two acute bioassays in
24 fathead minnows, also by Mayes (1984, 1990c), report acute LC₅₀ values of about 86 mg a.e./L.
25 Thus, for this species, the acute-to-chronic ratio for NOAECs is about 0.37 [32.2 mg a.e./L ÷ 86
26 mg a.e./L ≈ 0.3744]. In the absence of additional chronic data, this acute-to-chronic ratio is used
27 to estimate chronic NOAECs of about 7.4 mg a.e./L for sensitive species [20 mg a.e./L x 0.37 =
28 7.4 mg a.e./L] and 78 mg a.e./L for tolerant species [210 mg a.e./L x 0.37 ≈ 77.7 mg a.e./L].

29 **4.3.3.1.2. Triclopyr BEE**

30 There are more toxicity data for triclopyr BEE than for triclopyr TEA, including more acute
31 toxicity studies, many of which report both LC₅₀ values and NOAECs. As with triclopyr TEA,
32 there is only one chronic study available.

33
34 Acute LC₅₀ values for triclopyr BEE range from 0.2 to 1.5 mg a.e./L (Table 33). The lowest
35 LC₅₀ is 0.2 mg a.e./L in bluegills with a corresponding NOEC of 0.13 mg a.i./L (Woodburn et al.
36 1993c). Correcting for compound purity and converting from a.i. to a.e., the NOAEC is about
37 0.091 mg a.e./L. This NOAEC is not contradicted by any of the available field studies—i.e., the
38 NOAEC of 0.11 mg a.e./L Garlon 4 in minnows (Fontaine 1990) and the possible LOAEL of
39 0.25 mg a.e./L, based on growth inhibition in trout (Kreutzweiser et al. 1995). Thus, the
40 NOAEC of 0.091 mg a.e./L is used as the NOAEC for potentially sensitive species of fish.

41
42 The highest LC₅₀ is 1.5 mg a.e./L in fathead minnows with a corresponding NOAEL of about
43 0.97 mg a.e./L for signs of toxicity (Milazzo and Batchelder 1981a). While a full copy of
44 Milazzo and Batchelder (1981a) was available for the current risk assessment, and the

1 information in the document is reported in some detail, the frequency of the examination of the
2 fish for signs of toxicity is not clear. As a modestly conservative approach, the LC₅₀ of 1.5 mg
3 a.e./L is multiplied by 0.5—i.e., the U.S. EPA/OPP LOC for acute risk—and the NOAEC is
4 estimated at 0.75 mg a.e./L. This NOAEC does not seem to be overly conservative and
5 maintains a level of consistency with U.S. EPA/OPP.

6
7 The one egg-to-fry study on triclopyr BEE was conducted in rainbow trout (Weinberg et al.
8 1994d). This study reports an NOAEC of 0.017 mg a.e./L. It appears the EPA reanalyzed this
9 study and derived a modestly higher NOAEC of 0.019 mg a.e./L (U.S. EPA/OPP 2009a). These
10 two concentrations are not substantially different. To maintain consistency with the EPA
11 analysis, the current risk assessment uses the NOAEC of 0.019 mg a.e./L. As detailed in
12 Table 33 under the entries for Garlon 4, the reported LC₅₀ values in rainbow trout span nearly the
13 entire range of acute LC₅₀ values for Garlon 4 in fish—i.e., of the seven bioassays, rainbow trout
14 have positions (most to least sensitive) of 2, 3, and 7. For both technical grade triclopyr BEE
15 and Garlon 4, the most sensitive species is bluegill sunfish. Like trout, bluegills span a range of
16 positions for bioassays using triclopyr BEE—i.e., positions 1, 5, and 6 in the eight bioassays.

17
18 While it is conceptually desirable to express uncertainties, species sensitivities, and data
19 variability in the dose-response assessment, an objective and sensible method for reflecting these
20 factors in the chronic NOAEC for fish is not apparent. As summarized in Table 26, longer-term
21 exposures to triclopyr BEE are far below the chronic NOAEC of 0.019 mg a.e./L, and any
22 plausible adjustments to the chronic NOAEC would have no impact on the risk characterization.
23 Consequently, the chronic NOAEC of 0.019 mg a.e./L is applied to both sensitive and tolerant
24 species of fish.

25 **4.3.3.1.2. TCP**

26 Data on the acute toxicity of TCP to fish come from the open literature study by Wan et al.
27 (1987) and two MRID submissions, all of which are summarized in Table 33. The six LC₅₀
28 values reported by Wan et al. (1987) range from 1.5 to 2.7 mg/L and the MRID submissions
29 report much higher LC₅₀ values of 12.5 and 12.6 mg/L. LC₅₀ values for rainbow trout are
30 reported by both Wan et al. (1987)—i.e., 1.5 mg/L—and one of the MRID studies—i.e., 12.6
31 mg/L. These two sets of studies are obviously inconsistent and reflect experimental variability
32 or other unidentified factors rather than any differences in species sensitivity.

33
34 None of the studies on TCP report NOAECs. Consequently, the lower and upper bounds of the
35 range of LC₅₀ values for TCP are multiplied by 0.05 to estimate an NOAEC of 0.075 mg/L for
36 sensitive species and a NOAEC of 0.63 mg/L for tolerant species. Both of these estimated
37 NOAECs are based on bioassays using rainbow trout. Thus, the terms *sensitive species* and
38 *tolerant species* should be interpreted very loosely to indicate sensitivities that could occur by
39 chance or under different conditions of exposure.

40
41 Two egg-to-fry studies are available on TCP. In the earlier study by Marino et al. (1999) acetone
42 is used as a vehicle and the lowest NOAEC (fry weight and length) is 0.0808 mg/L. The U.S.
43 EPA/OPP classifies this study as invalid because of concerns with the acetone control group
44 (Mossler et al. 2000). The classification of the study as invalid indicates that the EPA deems the
45 study unsuitable for quantitative use in a risk assessment. A subsequent study by Marino et al.
46 (2003) used dimethylformamide as a vehicle. A full copy of this study was provided for the

1 conduct of the current risk assessment, but a review of this study by the U.S. EPA/OPP has not
2 been identified. Nonetheless, no issues are apparent with the solvent control or any other aspects
3 of the Marino et al. (2003) study. This study reports an NOAEC for fry weight and growth of
4 0.178 mg/L—i.e., about a factor of 2.4 higher than the NOAEC from Marino et al. (1999).

5
6 With some reservation, the current risk assessment defers to the U.S. EPA/OPP and does not use
7 the lower NOAEC of 0.0808 mg/L from the earlier study by Marino et al. (1999). The
8 reservations with discarding the earlier study by Marino et al. (1999) concern the responses in
9 the negative control (no solvent) and groups exposed to triclopyr BEE in the study by Marino et
10 al. (1999). While highly variable responses were observed in the solvent control group, the
11 negative controls and the groups exposed to triclopyr BEE (with solvent) evidence a clear and
12 consistent dose-response relationship.

13
14 Deferring to U.S. EPA/OPP, the chronic NOAEC 0.178 mg/L from Marino et al. (2003) is
15 rounded to 0.18 mg/L and used in the current risk assessment to characterize risks to fish of
16 longer-term exposures to triclopyr BEE.

17
18 Because this chronic NOAEC is higher than the estimated acute NOAEC of 0.075 mg a.e./L
19 based on the lower bound LC_{50} , the lower bound acute NOAEC is adjusted upward to 0.18 mg/L.
20 Since both the upper and lower bounds of the acute NOAECs as well as the longer-term NOAEC
21 are based on studies using rainbow trout, there is no sensible approach to proposing different
22 chronic NOAECs for potentially sensitive and tolerant species of fish.

23 **4.3.3.2. Amphibians**

24 Information on the toxicity of triclopyr to amphibians is much less abundant than the information
25 on fish. Since there are no chronic bioassays involving amphibian exposure to triclopyr, explicit
26 longer-term NOAECs are not developed. Nonetheless, the field study by Wojtaszek et al. (2005)
27 involving longer-term observations of amphibian populations following forestry applications of
28 triclopyr BEE is highly relevant to the current risk assessment. This study is used below in the
29 development of acute NOAECs and is discussed further in the risk characterization for
30 amphibians (Section 4.4.3.2).

31
32 A dose-response assessment of the toxicity of TCP to amphibians is not proposed because no
33 data are available on the toxicity of TCP to aquatic phase amphibians.

34 **4.3.3.2.1. Triclopyr Acid**

35 The only toxicity study involving the exposure of amphibians to triclopyr acid reports a
36 substantially higher LC_{50} of 750 mg a.e./L with an NOAEC for growth of 125 mg a.e./L. The
37 ratio of the NOAEC to the LC_{50} is about 0.17, which is very close to the median (0.16) of the
38 LOCs of 0.5 for acute risk and 0.05 for endangered species used by the U.S. EPA/OPP. In the
39 absence of any studies on triclopyr TEA and any additional studies on triclopyr acid, the acute
40 NOAEC of 125 mg a.e./L is applied to both sensitive and tolerant species of amphibians.

41 **4.3.3.2.2. Triclopyr BEE**

42 As summarized in Table 34 and illustrated in Figure 12, the five reported LC_{50} values for
43 amphibian embryos exposed to triclopyr BEE span a very narrow range: 13.7 to 24.6 mg a.e./L.
44 Perkins (1997) reports an LC_{50} of 15 mg a.e./L and a corresponding NOAEC of 2.5 mg a.e./L for

1 growth inhibition. The ratio of this NOAEC to the LC₅₀ is about 0.17, which is identical to the
2 corresponding ratio for triclopyr acid discussed in previous subsection. Applying the ratio of
3 0.17 to the range of LC₅₀ values for triclopyr BEE results in estimated acute NOAECs ranging
4 from 2.3 to 4.2 mg a.e./L.

5
6 The reported LC₅₀ values for amphibian larvae range from 0.79 to 11.5 mg a.e./L, substantially
7 lower than the LC₅₀ values for amphibian embryos. As discussed in Section 4.1.3.2, the upper
8 bound LC₅₀ of 11.5 mg a.e./L does not appear to be a statistical outlier, in that the data on
9 amphibian larvae are well-fit to the lognormal distribution. While NOAECs in amphibian larvae
10 are not reported, Wojtaszek et al. (2005) report sublethal EC₁₀ values for abnormal avoidance
11 response, the lowest of which is 0.1 mg a.e./L.

12
13 Chen et al. (2008) report concentration-related decreases in survival in *Rana pipiens* tadpoles
14 (Gosner stage 25) at concentrations as low as 0.25 mg a.e./L. Based on an examination of
15 Figure 4 in the Chen et al. (2008) publication, the concentration of 0.25 mg a.e./L is clearly a
16 LOAEL. The study by Chen et al. (2008) did not assay lower concentrations and did not define a
17 NOAEL.

18
19 The sublethal NOAEL of 0.1 mg a.e./L from Wojtaszek et al. (2005) is below the LOAEL of
20 0.25 mg a.e./L from the study by Chen et al. (2008). For the current risk assessment, the acute
21 EC₁₀ of 0.1 mg a.e./L is used as a surrogate acute NOAEC for sensitive species and sensitive
22 life-stages (larvae) of amphibians. For tolerant species and life stages, the NOAEL is taken as
23 4.2 mg a.e./L, based on the upper bound LC₅₀ in amphibian embryos.

24 **4.3.3.3. Aquatic Invertebrates**

25 Information on the toxicity of triclopyr acid, triclopyr TEA, triclopyr BEE, and TCP to aquatic
26 invertebrates is summarized in Table 35 and illustrated in Figure 13. In terms of sensitive and
27 tolerant organisms, the assays in eastern oysters and aquatic arthropods clearly differ. This
28 difference, however, is due to different endpoints, shell deposition in the eastern oyster and
29 lethality or immobilization in arthropods.

30
31 As discussed in 4.1.3.3, there is one bioassay on triclopyr acid, which involves acute lethality a
32 freshwater snail, *Physella gyrina* (Neuderfer 2009). The sensitivity of this snail is similar to that
33 of aquatic arthropods. Another snail assayed in the same study, the European ambersnail
34 (*Succinea putris*), survived exposure to concentrations of up 400 mg a.e./L, and this NOEC for
35 mortality is consistent with very high LC₅₀ values reported for triclopyr acid and triclopyr TEA
36 in the arthropod bioassays discussed below. Based on this admittedly scant data, there is no
37 apparent basis for asserting that non-arthropod aquatic invertebrates are substantially different
38 from aquatic arthropods in their sensitivity to triclopyr.

39
40 For the dose-response assessment, the data on eastern oysters are not specifically considered.
41 These data, however, are discussed in the risk characterization.

42 **4.3.3.3.1. Triclopyr Acid**

43 LC₅₀ values for triclopyr acid and triclopyr TEA range from about 100 to about 6400 mg a.e./L,
44 spanning a factor of 64. This is not an exceptional range in LC₅₀ values for a well-studied
45 chemical, and a similar range is apparent for triclopyr BEE, as discussed further below.

1
2 NOAECs are not available for triclopyr acid or triclopyr TEA. Consequently, the factor of 0.05
3 is used to estimate acute NOAECs ranging from 5 to 320 mg a.e./L. However, as discussed
4 below, the actual experimental chronic NOAEC for daphnids is 25 mg a.e./L. Thus, the
5 estimated acute NOAEC of 5 mg a.e./L is adjusted upward to 25 mg a.e./L. While the use of the
6 0.05 factor may be viewed as an extremely conservative approach, it has no impact on the risk
7 characterization because the estimated NOAECs are far above estimated levels of exposure
8 including the upper bound concentrations associated with an accidental spill.
9

10 Two chronic daphnid studies are available which report similar NOAECs. The study by Gersich
11 et al. (1985a), however, does not clearly indicate whether the units for exposure are a.e., a.i., or
12 formulation. The earlier study, Gersich et al. (1982), clearly indicates the units, and the reported
13 NOAEC is about 25.95 mg a.e./L. U.S. EPA/OPP (2009a) reports an inconsequentially lower
14 NOAEC of 25 mg a.e./L, which is used in the current risk Forest Service assessment.
15

16 As summarized in Table 35, the sensitivity ranks of daphnids in the nine available bioassays are
17 2, and 5 through 8. In other words, daphnids cannot be classified as sensitive, tolerant, or
18 intermediate, in terms of species sensitivity to triclopyr. Consequently, the chronic NOAEC of
19 25 mg a.e./L is used for both potentially sensitive and potentially tolerant species.

20 **4.3.3.3.2. Triclopyr BEE**

21 LC₅₀ values for triclopyr BEE range from about 0.25 to 20 mg a.e./L, spanning a factor of 80
22 (Table 35). This range is only modestly greater than the range of 64 for triclopyr acid.
23

24 Several of the acute toxicity studies with triclopyr BEE report both NOAECs and LC₅₀ values,
25 and the ratios of NOAEC to LC₅₀ values range from 0.11 to 0.63—i.e., ratios of 0.37 (Gorzinski
26 and Barron 1996), 0.25 (Milazzo and Batchelder 1981b), 0.55 (Ward and Boeri 1991b), 0.11
27 (Ward and Boeri 1991e), and 0.63 (Weinberg et al. 1994c) from the data given in Appendix 7,
28 Table A7-3. The mean and 90% confidence intervals on these ratios are 0.38 (0.18 to 0.58). The
29 lower bound of 0.18 is used to estimate the NOAECs from the LC₅₀ values. Based on the LC₅₀
30 range of 0.25 to 20 mg a.e./L, the acute NOAECs are estimated as ranging from 0.045 to 3.6 mg
31 a.e./L.
32

33 As detailed in Appendix 7, Table A-7, estimated NOAECs from field studies are 3.2 mg/L
34 (Kreutzweiser and Capell 1992) and 2.7 mg/L (Kreutzweiser et al. 1998). The observations from
35 Kreutzweiser et al. (1998) involve populations of stonefly and caddisfly. As discussed in Section
36 4.1.3.3, summarized in Table 35, and illustrated in Figure 14, stonefly and caddisfly are part of
37 the group of aquatic insects that appear to be tolerant to triclopyr BEE. Transient increases in
38 drift without other effects such as changes in stream invertebrate abundance have been noted at
39 concentrations ranging from about 0.6 to 0.95 mg/L (Kreutzweiser et al. 1995; Thompson et al.
40 1993). These relatively mild effects, however, occur at concentrations substantially above the
41 lower bound acute NOAEC of 0.045 mg a.e./L and above the geometric mean (0.40 mg a.e./L) of
42 the range of estimated acute NOAECs. In other words, the available field observations involving
43 applications of triclopyr BEE appear to be at least somewhat supportive of the estimated
44 NOAECs for aquatic invertebrates.
45

1 One chronic study is available on triclopyr BEE —i.e., the reproduction study by Chen et al.
2 (2008) which noted concentration-related decreases in reproduction in *Simocephalus vetulus* at
3 concentrations of 0.25 and 0.5 mg /L. These concentrations appear to be reported in units of mg
4 a.e./L. *Simocephalus vetulus* is a cladoceran similar to *Daphnia magna*. While no acute LC₅₀
5 values for triclopyr BEE are available on *Simocephalus vetulus*, it seems reasonable to suggest
6 that this cladoceran represents a sensitive species. As illustrated in Figure 13, a concentration of
7 0.25 mg a.e./L is identical to the lowest reported 48-hour LC₅₀ in *Daphnia magna*.
8 Consequently, it seems reasonable to assume that that longer-term exposure to a concentration of
9 0.25 mg a.e./L triclopyr BEE would cause adverse effects in cladocerans.

10
11 No objective method for estimating an NOAEC from the study by Chen et al. (2008) is apparent.
12 An analogy to fish does not seem biologically plausible. In addition, the chronic NOAEC in fish
13 is on rainbow trout and the acute toxicity data in trout are highly variable, complicating the
14 development of a reasonable acute-to-chronic ratio (Section 4.3.3.1.2).

15
16 In the absence of a reasonable approach to estimating a longer-term NOAEL, the LOAEC of
17 0.25 mg a.e./L in *Simocephalus vetulus* is used directly in the dose-response assessment for
18 sensitive species of aquatic invertebrates. Although this approach is unusual, the LOAEC for
19 sensitive species of aquatic arthropods is useful in the risk characterization.

20
21 As noted above, the LC₅₀ values for tolerant species of aquatic arthropods are a factor of up to 80
22 higher than the LC₅₀ for the most sensitive species of aquatic arthropod. The chronic LOAEC of
23 0.25 mg a.e./L is multiplied by a factor of 80 to estimate a chronic LOAEC of 20 mg a.e./L for
24 tolerant species of aquatic invertebrates. Again, this is not a typical approach ; however, the
25 estimated chronic LOAEC of 20 mg a.e./L is useful in the risk characterization (Section 4.4.3.4).

26 **4.3.3.3.3. TCP**

27 The data on the toxicity of TCP to aquatic arthropods consists of a single acute LC₅₀ of 10.9
28 mg/L and a single chronic NOAEC of 0.058 mg/L. Both of these values are for *Daphnia magna*.
29 The acute LC₅₀ is multiplied by 0.05 to estimate a NOAEC of 0.55 mg/L. The estimated acute
30 NOAEC and the chronic NOAEC are applied to both sensitive and tolerant species.

31 **4.3.3.4. Aquatic Plants**

32 **4.3.3.4.1. Algae**

33 The EC₅₀ data for algae are summarized in Table 36 and illustrated in Figure 15. The
34 information on toxicity to algae is discussed in Section 4.1.3.4.1. For this group of organisms,
35 data are available on triclopyr acid, triclopyr BEE, and TCP.

36 37 **4.3.3.4.1.1. Triclopyr Acid**

38 One study (MRID 41633705) is not included in the dose response assessment for triclopyr TEA,
39 because the available summary of this study appears to contain an error (see Appendix 8,
40 Table A8-1 for details). This exclusion has no substantial impact on the dose response
41 assessment for triclopyr TEA.

1 The EC₅₀ values for growth inhibition in algae range from about 0.49 to 80 mg a.e./L. This range
2 of concentrations spans a factor of about 160, greater than the variability noted in either fish or
3 invertebrates.

4
5 The lower bound EC₅₀ of 0.49 mg a.e./L is from the study by Gardner et al. (1997) in a species of
6 *Ankistrodesmus*. Gardner et al. (1997) report a NOAEC of about 0.23 mg a.e./L, which is used
7 directly for sensitive species of algae.

8
9 The upper bound EC₅₀ of 80 mg a.e./L for *Chlorella pyrenoidosa* is from the study by Baarschers
10 et al. (1988), which does not report an NOAEC. As summarized in Appendix 8, Table A8-1, the
11 reported ratios of the NOAECs to EC₅₀ values for triclopyr TEA range from 0.46 (Gardner et al.
12 1997) to 0.06 (MRID 41633707 as summarized in U.S. EPA/OPP 1998a). Typically, the
13 application of the standard 0.05 adjustment factor to algae EC₅₀ values can be viewed as
14 unreasonably conservative, because no species of algae are listed as threatened or endangered
15 (<http://www.fws.gov/endangered/>). Nonetheless, the ratio of 0.06 is very close to the standard
16 factor of 0.05. The raw data from MRID 41633707 are given in the summary of this study by
17 Mayes (1991d). A review of the raw data for Day 4 of the study indicates an exponential decline
18 in both cell count and cell number. Thus, for tolerant species the standard adjustment of 0.05 is
19 used and the upper bound NOAEC for tolerant species of algae is taken as 4 mg a.e./L.

20 21 **4.3.3.4.1.2. Triclopyr BEE**

22 As with most other groups of aquatic organisms, algae are more sensitive to triclopyr BEE than
23 to triclopyr TEA. Based on median EC₅₀ values (Table 36), triclopyr BEE is more toxic than
24 triclopyr TEA by a factor of 10. For triclopyr BEE, the EC₅₀ values for growth inhibition in
25 algae range from about 0.073 to 5.9 mg a.e./L. This range of concentrations spans a factor of
26 about 80, virtually identical to the range of EC₅₀ values for triclopyr BEE in aquatic invertebrates
27 (Section 4.3.3.3).

28 29 **4.3.3.4.1.2.1. NOAEC for Sensitive Species**

30 The most sensitive species is *Navicula pelliculosa* from the study by Hughes and Alexander
31 (1993c). As noted in Appendix 8, Table A8-1, there are substantial inconsistencies in the
32 toxicity values reported for this study. In the study itself, Hughes and Alexander (1993c) report
33 an EC₅₀ equivalent to 0.193 mg a.i./L with a NOAEC of 0.104 mg a.i./L. Apparently, the EPA
34 conducted a reanalysis of the data and U.S. EPA/OPP (1998a) reports the EC₅₀ as 0.1 mg a.i./L
35 with an NOAEC of 0.002 mg a.i./L. The much lower NOAEC is also reported in U.S. EPA/OPP
36 (2009a). The substantial discrepancies between these estimates appear to be related to
37 differences in statistical methods.

38
39 A careful consideration of the dose-response assessment for sensitive species of algae is
40 important to the current risk assessment, because effects in sensitive species of algae could have
41 secondary effects on higher trophic levels. Given the rapid degradation of triclopyr BEE in
42 water, effects on sensitive species of algae could be the only direct effect reasonably attributable
43 to triclopyr BEE.

44
45 The concentration-response data from the study by Hughes and Alexander (1993c) are illustrated
46 in Figure 17 based on mean cells counts at Day 5. Figure 17 gives three views of the data:

1 (A) the untransformed data, (B) a conversion of the dose to logarithmic units, and (C) a
2 conversion of the response to logarithmic units. The fourth graph in Figure 17, labeled D, is a
3 reanalysis of the data discussed below.

4
5 The untransformed data (Figure 17A) clearly indicate that triclopyr BEE is very toxic to this
6 species of algae and low concentrations cause a precipitous decrease in cell count. While the
7 untransformed data can be analyzed directly, such analyses are awkward and unnecessary.

8
9 Hughes and Alexander (1993c) elected to analyze the data using a log-transformation on dose
10 (Figure 17B). While these investigators do not include detailed output of the statistical analyses,
11 a visual examination of Figure 17B might suggest that the reported NOAEL of 0.104 mg a.i./L is
12 a more reasonable estimate of the NOAEC than the 50-fold lower NOAEC 0.002 mg a.i./L
13 indicated in U.S. EPA/OPP (1998a).

14
15 While U.S. EPA/OPP (1998a) does not discuss the reanalysis of this study and the DER for this
16 study was not obtained for the current risk assessment, it does seem obvious that the EPA used
17 an analysis different from that of Hughes and Alexander (1993c). One sensible alternative
18 approach that could be used is the logarithmic transformation of response rather than dose, as
19 illustrated in Figure 17C. This view of the data suggests that algal cell count decreases at a
20 constant proportion as the concentration of triclopyr BEE increases. As illustrated in Figure
21 15C, the only aberrant data point appears to be the response at 0.5 mg a.i./L, which indicates
22 substantially greater inhibition ($\approx 99.3\%$), relative to the next lower concentration—i.e., 0.258 mg
23 a.i./L with 69% inhibition—and somewhat greater inhibition relative to the next higher
24 concentration —i.e., 1.03 mg a.i./L with 96% inhibition.

25
26 The log-response/linear-concentration plot in Figure 17-D corresponds to the exponential dose-
27 or concentration-response model:

Equation 11

$$Y = \alpha e^{-\beta c}$$

28
29
30
31 where Y is the response, c is the concentration (or dose), and α and β are model parameters.
32 Mathematically, the exponential dose-response model is identical to the first-order excretion
33 model (Section 3.1.3.2.1) as well as the first-order dissipation model (Section 3.2.3.7).

34
35 In a reanalysis of the mean responses conducted as part of the current risk assessment, the
36 exponential model offered an adequate fit to the concentration-response data from Hughes and
37 Alexander (1993c) ($p \approx 0.004$, $r^2 = 0.67$). When the aberrant response at 0.5 mg a.i./L was
38 censored (illustrated as an open circle in Figure 17-D), the fit was improved ($p = 0.000001$,
39 $r^2 = 0.97$).

40
41 Note that the exponential model is a non-threshold model—i.e., the concept of a true NOAEC
42 does not apply. The EPA selection of 0.002 mg a.i./L as an NOAEC in U.S. EPA/OPP (1998a,
43 2009a) is probably based on a comparison to the solvent control (for which the 0.002 mg a.i./L
44 group evidenced about a 6% inhibition) rather than to the untreated control (19% inhibition).

1 Based on the above analysis, the NOAEC of 0.002 mg a.i./L (≈ 0.0014 mg a.e./L) appears to be a
2 reasonable estimate of the NOAEC, and 0.0014 mg a.e./L is used as the NOAEC for sensitive
3 species of algae.
4

5 **4.3.3.4.1.2.2. NOAEC for Tolerant Species**

6 Based on the available data, the algal species least sensitive to triclopyr BEE is *Skeletonema*
7 *costatum*. As indicated in Table 36, this species has a rank order of 2 and 5 in the five available
8 algal bioassays on triclopyr BEE. Thus, the designation of *Skeletonema costatum* as a tolerant
9 species appears to reflect variability in the available bioassays rather than a true species
10 tolerance. The study indicating that *Skeletonema costatum* is tolerant to triclopyr BEE is from
11 the MRID study by Cowgill et al. (1989b).
12

13 As with the bioassay on *Navicula pelliculosa* discussed in the previous subsection, there are
14 discrepancies between the analysis of the data reported in the study by Cowgill et al. (1989b) and
15 reanalysis of the data presented in U.S. EPA/OPP (1998a, 2009a). These differences, detailed in
16 Appendix 8 (Table A8-1), are relatively minor and do not have a substantial impact on the
17 current risk assessment. Based on the analysis provided in U.S. EPA/OPP (1989a), the NOAEC
18 is reported as 1.65 mg a.e./L. Cowgill et al. (1989b) report a slightly lower NOAEC of 1.0 mg
19 a.e./L for both cell count and cell volume. The somewhat lower NOAEC of 1.0 mg a.e./L is used
20 as the NOAEC for tolerant species of algae in the current risk assessment.
21

22 **4.3.3.4.1.3. TCP**

23 Only two bioassays are available on the toxicity of TCP to algae, and both report an EC₅₀ of 1.8
24 mg/L (Table 36). The NOAECs for these studies, however, do differ slightly from one
25 another—i.e., 0.36 mg/L in *Anabaena flos-aquae* and 0.65 mg/L in *Kirchneria subcapitata*. By
26 analogy to the more extensive data on triclopyr in algae as well as other species, this range of
27 NOAECs is not likely to encompass the range of sensitivities that would be expected if data were
28 available on more species.
29

30 For example, under the assumption that approximate NOAECs may be lognormally distributed,
31 the 90% confidence intervals of the NOAECs based on the two reported NOAECs of 0.36 and
32 0.65 mg a.e./L would be about 0.074- 3.2 mg/L. Note that the range in the confidence interval
33 spans a factor of about 40, which is reasonably close to the variability in macrophyte EC₅₀
34 values for triclopyr TEA (a factor of 160) and triclopyr BEE (a factor of about 80). This
35 relatively simple and somewhat simple probabilistic elaboration would have no impact on the
36 risk characterization for terrestrial applications, because exposures to TCP will be far below the
37 lower bound of the confidence interval (0.074 mg/L). Consequently, the reported NOAECs of
38 0.36 and 0.65 mg/L are used directly to characterize risks to sensitive and tolerant species of
39 algae.

40 **4.3.3.4.2. Macrophytes**

41 The EC₅₀ data for aquatic macrophytes are summarized in Table 37 and illustrated in Figure 16.
42 The information on the toxicity of triclopyr to these species is discussed in Section 4.1.3.4.2. A
43 dose-response assessment of the toxicity of TCP to macrophytes is not proposed because no data
44 are available on the toxicity of TCP to aquatic macrophytes.
45

1 **4.3.3.4.2.1. Triclopyr TEA**

2 As illustrated in Figure 16, the relative sensitivity of aquatic macrophytes to triclopyr TEA is
3 assessed based on an analogy to differences in the sensitivity of monocots and dicots, with dicots
4 comprising the sensitive species and monocots comprising the tolerant species. Within both
5 groups, the available standard toxicity bioassays are limited to very few species—i.e., only two
6 *Lemna* species for the monocots and only watermilfoil (*Myriophyllum*) for the dicots. Dicots are
7 the only group of aquatic organisms in which triclopyr TEA is substantially more toxic than
8 triclopyr BEE.

9
10 Based on the standard bioassay, the lowest EC₅₀ is 0.04 mg a.e./L in Eurasian watermilfoil
11 (*Myriophyllum spicatum*), and identical values are reported for this species using either triclopyr
12 TEA (Poovey et al. 2007) or triclopyr acid (Perkins 1997). Based on a visual inspection of
13 Figure 2 in Poovey et al. (2007), the NOAEC for effects on shoot length is 0.01 mg a.e./L.
14 Perkins (1997) does not provide information that can be used to estimate an NOAEC.

15
16 As summarized in Appendix 8, Table A8-2, several other studies provide information on
17 sublethal toxicity in dicots (Lembi and Chand-Goyal 1994; Glomski and Nelson 2008; Madsen
18 et al. 2008; Netherland and Getsinger 1992, 1993; Poovey and Getsinger 2007). Most of these
19 studies report NOAEC or LOAEC values consistent with the NOAEC of 0.01 mg a.e./L from
20 Poovey et al. (2007). The one exception is the study by Lembi and Chand-Goyal (1994) on
21 Eurasian watermilfoil. In this study, a concentration of 0.0005 mg/L is associated with a
22 decrease in photosynthesis and chlorophyll and a concentration of 0.001 mg/L is associated with
23 a decrease in stem length and abnormal root development. Reasons for the different results
24 reported in the two studies are not apparent. As noted frequently in previous subsections on
25 other groups of organisms, differences of this order of magnitude are common between similar
26 studies conducted at different times by different (or sometimes the same) investigators.
27 Consequently, the lower concentration of 0.0005 mg a.e./L from Lembi and Chand-Goyal (1994)
28 is used as a marginal NOAEL—i.e., a concentration associated with a biochemical indicator of
29 an adverse effect but no overt toxic effect.

30
31 Note that watermilfoil is a target species for triclopyr. The use of data on a sensitive target
32 species is a conservative assumption, based on the possibility that some unidentified (and
33 untested) nontarget species may be equally sensitive.

34
35 The data on monocots do not vary substantially with EC₅₀ values spanning a very narrow range
36 from about 6 to 16 mg a.e./L. All of these EC₅₀ values involve species of *Lemna*. The highest
37 EC₅₀ of 15.8 mg a.e./L in *Lemna minor* is from the MRID study by Cowgill et al. (1988), which
38 is also published in the open literature as Cowgill et al. (1989a). Based on the data tables in
39 Cowgill et al. (1988) as well as Table 6 in Cowgill et al. (1989a), the lowest NOAEL in the
40 strains of *Lemna* assayed is 7.8 mg a.i./L or about 5.6 mg a.e./L. The NOAEL of 5.6 mg a.e./L is
41 used to characterize risks to tolerant species of aquatic macrophytes.

42 **4.3.3.4.2.2. Triclopyr BEE**

43
44 There is not a substantial difference in the toxicity of triclopyr BEE to monocots and dicots
45 (Table 37, Figure 16), as there is with triclopyr TEA. Both the lowest and highest EC₅₀ values,
46 0.86 and 6.25 mg a.e./L, are for *Lemna gibba* (a monocot).

1
2 The only NOAEC reported for triclopyr BEE in macrophytes is the NOAEC of 0.14 mg a.e./L
3 with a corresponding EC₅₀ of 1.7 mg a.e./L in *Lemna gibba* reported in the study by Milazzo et
4 al. (1993). As with other studies discussed above, the EPA appears to have conducted a
5 reanalysis of the Milazzo et al. (1993) data and reports the EC₅₀ as 0.86 mg a.e./L with an
6 NOAEC of <0.111 mg a.e./L in U.S. EPA/OPP (2009a). In terms of plausible exposures to
7 triclopyr BEE, this difference in analyses by the EPA and the study authors has no impact on this
8 risk assessment. Consequently, the EC₅₀ values of 0.86 and 6.25 mg a.e./L are multiplied by the
9 factor of 0.05 to estimate a NOAEC of 0.043 mg a.e./L for sensitive species and a NOAEC of
10 0.31 mg a.e./L for tolerant species of aquatic macrophytes.

4.4. RISK CHARACTERIZATION

4.4.1. Overview

An overview of the quantitative risk characterization for nontarget species is given in Table 46. This table summarizes the upper bounds of the acute and chronic HQs for sensitive species in several groups of organisms covered in the current risk assessment. Some groups for which HQs are very low (e.g., terrestrial invertebrates) are omitted. In Table 46, separate sets of HQs are given for triclopyr TEA and triclopyr BEE. Detailed summaries of HQs for different groups of organisms are given in the attachments that accompany this risk assessment, as discussed in the following subsections.

Qualitatively, the risk characterization for ecological receptors is parallel in many respects to the risk characterization for human health effects. HQs exceed the level of concern (HQ=1) for exposures involving the consumption of contaminated vegetation. With the exception of aquatic plants, risks associated with the contamination of surface water are low relative to risks associated with contaminated vegetation.

Based on the HQs resulting from extreme value exposure assessments, it appears that large mammals consuming contaminated vegetation are the nontarget organisms at greatest risk. The magnitude of the HQs for these exposure scenarios is similar to the magnitude of the exposure scenario involving the consumption of contaminated vegetation by a young woman, as described in the human health risk assessment (Section 3.4). As with the human health risk assessment, the high HQs suggest the potential for adverse effects in large mammals. This assessment based on HQs is consistent with the recent EPA risk assessment, U.S. EPA/OPP (2009a). The available field studies neither support nor substantially refute concerns for adverse effects in large mammals. The lack of detailed field studies involving longer-term observations in populations of large mammals following applications of triclopyr adds substantial uncertainty to the risk characterization for mammalian wildlife.

Some upper bound HQs exceed the level of concern for exposure scenarios in which smaller mammals or birds consume contaminated vegetation or insects. The magnitude of these HQs, however, is much lower than the magnitude of HQs for large mammals, particularly at the upper bounds. Based on the findings of available field studies, triclopyr is not likely to cause frank adverse effects in small mammals and birds. These observations are not contradicted by the relatively moderate exceedances above the level of concern (HQ=1) in the central estimates of the HQs for small mammals and birds.

Neither terrestrial nor aquatic applications of triclopyr TEA pose substantial risks to aquatic animals across the range of labeled application rates. Triclopyr BEE, however, is much more toxic than triclopyr TEA to aquatic animals. At application rates in excess of about 3 lb a.e./acre, peak concentrations of triclopyr BEE in surface water could pose acute risks to sensitive species of fish and aquatic phase amphibians. Similarly, acute risks to sensitive species of aquatic invertebrates could occur if application rates exceed about 1.5 lb a.e./acre. The likelihood of acute risks to aquatic animals depends very much on site-specific conditions. In areas with low rates of rainfall, acute risks to aquatic animals would be negligible, so long as drift to surface water were minimal. In areas with high rates of rainfall, the surface water

1 contamination is more likely. Because triclopyr BEE is not persistent in soil or surface water,
2 longer-term risks to aquatic animals after terrestrial applications of triclopyr BEE appear to be
3 negligible.

4
5 Since triclopyr is an effective herbicide, damage to terrestrial vegetation is to be expected in the
6 event of direct spray, substantial drift, and substantial runoff from the application site.
7 Substantial runoff from the treated site would depend on the same site-specific factors that
8 determine contamination of surface water. Damage to aquatic plants, particularly macrophytes,
9 may result from terrestrial applications of triclopyr. Triclopyr is an effective aquatic herbicide
10 and damage to sensitive species of aquatic macrophytes following effective aquatic applications
11 is certain.

12
13 The application of any effective herbicide is likely to alter vegetation, the secondary effects of
14 which may include changes to food availability and quality of habitat for both terrestrial and
15 aquatic organisms. These secondary effects are likely to vary over time and vary among
16 different species of mammals. For aquatic applications of triclopyr or in cases of gross
17 contamination of surface water with triclopyr, damage to aquatic vegetation could be substantial,
18 and this could result in temporary but severe oxygen depletion. In such cases, substantial
19 mortalities in fish and other certain other aquatic organisms would be expected.

20 **4.4.2. Terrestrial Organisms**

21 **4.4.2.1. Mammals**

22 The risk characterization for mammals is summarized in Worksheet G02a of the workbooks for
23 terrestrial applications of triclopyr TEA (Attachment 1), triclopyr BEE (Attachment 2),
24 Worksheet G02 for aquatic applications (Attachments 3 and 4), and TCP (Attachments 5 to 7).

25 **4.4.2.1.1. Contaminated Vegetation**

26 **4.4.2.1.1.1. Triclopyr**

27 The risk characterization for mammals is dominated by exposure scenarios associated with the
28 consumption of contaminated vegetation or insects. As a convenience, a summary of the HQs
29 associated with the consumption of contaminated vegetation by mammals and birds (discussed in
30 the following subsection) is given in Table 47. As with other similar tables given in this risk
31 assessment, these HQs apply to the unit application rate of 1 lb a.e./acre and will scale
32 proportionately to the application rate.

33
34 The HQs for mammals increase as body weight increases. While small mammals may consume
35 more than larger animals, the higher sensitivity of larger mammals to triclopyr suggest they are
36 at greater risk. At the unit application rate of 1 lb a.e./acre, the acute HQs for a large (70 kg)
37 mammal consuming contaminated short grass are 2 (0.2 to 11). The corresponding chronic HQs
38 are 5 (0.2 to 53). For the small (20 g) mammal, the corresponding HQs are much lower—i.e.,
39 0.3 (0.02 to 1.6) for acute exposures and 0.7 (0.03 to 7) for longer-term exposures. As
40 summarized in Table 32 and discussed in Section 4.3.2.1.1, the relationship of NOAELs to
41 LOAELs suggests that HQs of about 4 might be associated with subclinical adverse effects,
42 although overt signs of toxicity might not be evident.

1 The very high HQs for mammals consuming contaminated vegetation suggest that triclopyr
 2 applications may cause adverse effects in mammalian wildlife populations. This HQ-based risk
 3 characterization for mammals is similar to the EPA’s RQ-based risk characterization in U.S.
 4 EPA/OPP (2009a, Table 5-9, p. 101):

5
 6 *Acute and chronic-dose based and chronic dietary-based RQs exceed the*
 7 *Agency’s acute and chronic endangered species LOC (0.1 acute and 1.0*
 8 *chronic) for all foliar application uses of triclopyr (Table 5-9). The*
 9 *recommended mitigated maximum foliar application rate of 9 lbs ae/A*
 10 *would still result in exceedances of the Agency’s acute and chronic LOC*
 11 *of 0.1 and 1.0 respectively (Table 5-9).*

12 U.S. EPA/OPP 2009a, p. 100

13
 14 The term RQ used in the above quotation refers to *Risk Quotients*. RQs are similar to HQs,
 15 except that acute RQs are based on LC₅₀ values rather than NOAELs. Thus, the EPA uses a
 16 level of concern (LOC) of 0.1 rather than 1 for acute exposures.

17
 18 In assessments of forestry applications, the EPA derives RQs of 0.8 to 4.3 for small mammals,
 19 based on acute exposures and RQs of 10.6 to 489.2 for chronic exposures (U.S. EPA/OPP 2009a,
 20 Table 5-9, pp. 101-102). Because the exposure and dose-response assessments developed in the
 21 current risk assessment are different from those developed by the EPA, the RQs derived in the
 22 EPA assessment are not directly comparable to the HQs derived in the current risk assessment.
 23 Nonetheless, quantitative comparisons can be made, at least crudely, by adjusting the RQs
 24 derived by EPA to reflect the HQ method used in the current risk assessment.

25
 26 Take for example, the acute RQ of 0.8 for a small mammal exposed to triclopyr applications to
 27 Douglas fir (U.S. EPA/OPP 2009a, Table 5-9, p. 102). Adjusting the LOC to 1 (comparable to
 28 the current risk assessment), the acute RQ of 0.8 corresponds to an HQ of 8. The acute RQ,
 29 however, is based on 17 sequential applications of 1.5 lb a.e./acre at 21-day intervals.
 30 Normalizing the application rate used by EPA to 1 lb a.e./acre, the HQ of 8, which is based on 17
 31 applications, corresponds to an HQ of 5.3 for a single application, as elaborated in the next
 32 paragraph.

33
 34 The correction for the number of applications and the application interval can be made using the
 35 plateau principle (Eq. 2, Section 3.1.3). The EPA uses a default foliar half life of 35 days,
 36 corresponding to a first-order rate coefficient (*k*) of about 0.02 day⁻¹ [$\ln(2)/35 \text{ days} \approx 0.0198$
 37 days^{-1}]. Using this rate coefficient with 17 applications at 21-day intervals, the increase in
 38 concentration from a single application rate would be about 2.9:

39
 40 **Equation 12**

$$41 \quad \frac{X_{N \text{ Apps}}}{X_{1 \text{ App}}} = \frac{(1 - (e^{-kt^*})^N)}{1 - e^{-kt^*}} = \frac{(1 - (e^{-0.02 \times 21})^{17})}{1 - e^{-0.02 \times 21}} = \frac{1 - 0.000792}{1 - 0.657} \approx 2.913$$

42
 43 where *N* is the number of applications, *t** is the application interval, and *k* is the first-order
 44 dissipation rate coefficient. Thus, the HQ of 5.3 for 17 applications corresponds to an HQ for a
 45 single application of about 1.8 [$5.3 \div 2.9 \approx 1.828$]. As summarized in Table 46, the current risk

1 assessment derives upper bound acute HQs of 0.1 to 1.6 for a small mammal. The upper bound
2 HQ of 1.6 from the current risk assessment is virtually identical to the adjusted HQ of 1.8 based
3 on the EPA's RQ of 0.8.

4
5 A similar and somewhat more direct comparison can be made based on chronic toxicity. The
6 EPA gives two chronic RQs for the Douglas-fir application: 91.7, based on estimated doses, and
7 10.6, based on dietary concentrations. As discussed in Section 4.2.2.3, the current risk
8 assessment uses dose-based HQs. Thus, only the RQ of 91.7 is comparable to the HQs presented
9 in the current risk assessment. In that both the chronic RQ as well as the chronic HQ are based
10 on NOAELs, only the application rate and the number of applications must be adjusted.
11 Adjusting the RQ as in the acute comparison discussed above, the chronic RQ of 91.7
12 corresponds to a chronic HQ of about 21 [$91.7 \div (1.5 \times 2.9) \approx 21.08$]. As summarized in
13 Table 46, the current risk assessment derives upper bound chronic HQs of 1.8 to 7 for a small
14 mammal. Given the substantial differences between the methods used in the two risk
15 assessments, a difference of a factor of 3 [$21 \div 7$] is insubstantial.

16
17 The rather elaborate comparison of the current Forest Service risk assessment with the recent
18 EPA ecological risk assessment (U.S. EPA/OPP 2009a) is justified by the interpretation of the
19 HQs as well as the EPA's RQs. The different methods used in Forest Service risk assessments
20 and EPA risk assessments both suggest that adverse effects on mammalian wildlife could be
21 expected at triclopyr application rates typically used in Forest Service programs.

22
23 The reasonable approximation of the numeric risk characterizations in the current risk with those
24 in U.S. EPA/OPP (2009a) is not necessarily predictive of the actual effects of field applications
25 of triclopyr. In other words, the quantitative risk characterization must be tempered by
26 information from field applications of triclopyr.

27
28 As reviewed in U.S. EPA/OPP (2009a, p. 82 ff) and detailed in Appendix O of the EPA risk
29 assessment, the U.S. EPA/OPP maintains a database of ecological incidents associated with
30 pesticide applications. A total of 63 incidents regarding triclopyr applications were reported to
31 the EPA. None of these incidents reported adverse effects in mammals. As summarized in
32 Appendix 2, Table A2-10, of the current risk assessment, none of the available field studies
33 associate adverse effects in mammals with the direct toxicity of triclopyr.

34
35 Two general factors may contribute to the apparent discrepancy between the high HQs (as well
36 as the high RQs) and the lack of reported adverse effects in field studies or incident reports. Like
37 the human health risk assessment, the ecological risk assessment uses the extreme value
38 approach. The upper bound HQs represent multiple worst case exposure assumptions that may
39 not occur frequently in the field. Also, the field study by Leslie et al. (1996) suggests that some
40 mammals, such as deer, may avoid treated areas. As discussed in the exposure assessment, the
41 scenarios for the consumption of contaminated vegetation assume that 100% of the diet is
42 contaminated. If larger mammals avoid treated areas, the proportion of the contaminated diet
43 could be much less than 100%. As the proportion of the diet that is contaminated decreases, the
44 consequent HQs will also decrease.

1 Several field studies suggest that triclopyr applications in the range of about 2 lbs a.e./acre are
2 not likely to cause adverse effects in small mammals and that changes in mammalian populations
3 are likely to result from secondary effects on vegetation (Boggs et al. 1991a; Lautenschlager et
4 al. 1997, 1998; Lochmiller et al. 1995; McMurry et al. 1993a,b; Nolte and Fulbright 1997).
5 These studies are consistent with HQs for smaller mammals consuming contaminated grasses—
6 i.e., 0.3 (0.02 to 1.6) at 1 lb a.e./acre. At an application rate of 2 lb a.e./acre, these HQs would
7 double, but the central estimate of the HQ would remain below the level of concern (HQ=0.6)
8 and the upper bound HQ would only modestly exceed the level of concern (HQ=3.2).

9
10 While it is beyond the scope of the current effort to conduct a full probabilistic risk assessment
11 on mammals or other groups of organisms, a very simple and conservative probabilistic
12 assessment may help to explain the apparent discrepancies between the HQ analysis and the field
13 studies. The 70 kg deer is used as an example. As noted above, there are no incident reports
14 associating adverse effects on deer with triclopyr applications. Given that triclopyr has been
15 used extensively in forestry as well as agriculture for many years this may call into question the
16 HQ analysis. As a preliminary probabilistic assessment, the assumptions are that the NOAEL of
17 8 mg/kg bw (Table 40) is a reliable estimate, that the residue rates from Fletcher (Table 27) are
18 appropriate, and that the algorithms developed by Nagy (Table 38) and the estimates of the
19 caloric content of food items (Table 39) are reasonable. As detailed in Worksheet F05c
20 (Attachment 1 and 2), the extreme value approach uses central estimates and ranges for residue
21 rates for grass—i.e., 85 (30 to 240) mg/kg food—and the amount of food consumed by a 70 kg
22 mammal grazing on vegetation with a water content of 85%—i.e., 15.4 (4.6 to 26) kg/day.

23
24 For the refined assessment, the following changes are made. Deer will not consume large
25 amounts of grass and prefer to feed on forbs. Thus, for the probabilistic assessment, the residue
26 rates for forage plants are used—i.e., 45 (15 to 135) mg/kg food from Table 27. As discussed in
27 Section 4.2.2.3, all exposure assessments for contaminated vegetation are based on the
28 assumption that 100% of the diet is contaminated. As discussed above, the study by Leslie et al.
29 (1996) suggests that deer may avoid feeding in treated areas. Thus, for the refined assessment,
30 the assumption is that the contaminated proportion of the diet is 30% with a range of 10 to 100%.
31 Note that the only impact of these changes on the upper bound HQ of 53 for a large mammal is a
32 reduction based on the upper bound of residues—i.e., decreasing the upper bound from 240 to
33 135 mg/kg bw/day. Thus, the maximum HQ using the extreme value method would be about 21
34 for the deer [$53 \times 135 \div 240 \approx 21$].

35
36 The probabilistic assessment uses a triangular distribution to describe the variability in residues,
37 food consumption, and the proportion of the contaminated diet. This distribution is extremely
38 conservative because, unlike the normal and lognormal distributions, the triangular distribution
39 does tail off at the extremes. At least for food consumption, the publications by Nagy (1987,
40 2005) clearly indicate that field metabolic rates will be lognormally distributed. For residue
41 rates, Fletcher et al. (1994) do not address the distribution of values. Nonetheless, most of
42 monitoring studies indicate that values such as residues in food have a lognormal distribution
43 (e.g., Gilbert 1987). Thus, in the example involving the 70 kg deer, the use of the triangular
44 distribution generally overestimates the probability of high HQs.

1 The results of the simulation for the 70 kg deer are illustrated in Figure 18. Based on 1000
2 simulations, the maximum HQ from the simulation is about 5.4, which is a factor of about 4
3 below the upper bound extreme value HQ of 21. As would be expected from the standard HQ
4 method, the probability of exceeding an HQ of 1 is relatively high—i.e., about 44%. The
5 probability of exceeding an HQ of 3, however, is much lower, about 3.4%, and the probability of
6 exceeding an HQ of 4 is only about 1.1%. As summarized in Table 32, the average ratio of the
7 LOAEL to the NOAEL in mammals is about 4. Thus, at an HQ of 4, it might be expected that a
8 deer would be adversely affected, although overt signs of toxicity would not be likely.
9 Consequently, it seems reasonable that some field studies might not detect adverse effects in deer
10 following the typical application rate of 1 lb/acre. At higher application rates, the probability of
11 observing adverse effects would increase; however, unlike the HQ, the increase in the probability
12 of observing an adverse effect would not be directly proportional to the change in application
13 rate.

14
15 Considering all of the above factors, the risk characterization for terrestrial mammals based on
16 the HQ method does not appear to be unreasonable. Based on relatively standard methods used
17 to estimate risks to mammals from well-conducted toxicity studies as well as reasonably well-
18 documented estimates of exposure, it is likely that mammals will be exposed to triclopyr at doses
19 that exceed the level of concern (HQ=1). In extreme cases, adverse effects could be anticipated
20 in some mammals, particularly larger mammals, at application rates as low as 1 lb a.e./acre.
21 These effects, however, might not involve overt signs of toxicity that would be observed in field
22 studies.

23
24 The chronic HQs for mammals are substantially higher than the acute HQs. This matter suggests
25 that while overt signs of toxicity might not be evident shortly after triclopyr applications, longer-
26 term adverse effects on mammalian populations, possibly involving changes in reproductive
27 rates, could occur. While these effects are not reported or otherwise noted in field studies, it is
28 the case that the available field studies focus on small mammals, and the available literature does
29 not include longer-term studies on populations of larger mammals (carnivores or herbivores). As
30 with the risk characterization for human health (Section 3.4.4.1), well-designed field studies
31 (comparable to epidemiology studies) on populations of large mammalian carnivores and
32 herbivores with documented exposures to triclopyr could be useful in better assessing the
33 potential risks to mammalian wildlife.

34 35 **4.4.2.1.1.2. TCP**

36 As with triclopyr, the only exposure scenarios for mammals that approach or exceed the level of
37 concern involve the consumption of contaminated vegetation. The HQs for these exposure
38 scenarios are summarized in Table 48. A full set of HQs covering all exposure scenarios is given
39 in Worksheet G02 of Attachment 3.

40
41 Unlike the case with triclopyr, the HQs associated with exposure to TCP are highest for smaller
42 mammals, relative to large mammals, and this reflects the greater food consumption rate for
43 smaller mammals as well as the use of the same NOAEL for all mammals (Table 42). The HQs
44 are higher for contaminated grasses than contaminated fruit because of the higher estimated
45 residue rates in short grass relative to fruit (Table 27).

1 The most extreme scenario involves the longer-term consumption of contaminated grasses by a
2 small mammal. The HQs for this scenario are 0.9 (0.04 to 10) at the unit application rate of 1 lb
3 a.e./acre. The HQs for the less extreme scenario for the consumption of contaminated fruits are
4 0.3 (0.02 to 1.3). As with triclopyr, the relationship of the NOAEL to the LOAEL suggests that
5 HQs of about 4 could be associated with adverse effects which could range from subclinical
6 changes in blood chemistry to birth defects (Table 29).

7
8 As discussed in the previous subsection, field studies on forestry applications of triclopyr do not
9 support the assertion that triclopyr applications in the range of about 2 lb a.e./acre will cause
10 detectable adverse effects in populations of small mammals. These field observations are
11 consistent with the above HQs. At the central estimate of the exposure assumptions for an
12 application rate of 2 lb a.e./acre, the HQs would be in the range of about 0.6 to 2. The modest
13 excursion above the level of concern (HQ=1) would not necessarily result in detectable effects
14 on populations of mammals. The upper bound HQs would mostly likely reflect extreme
15 exposures which might occur only rarely.

16 **4.4.2.1.1. Other Routes of Exposure**

17 Exposure scenarios not involving the consumption of contaminated vegetation—i.e., direct spray
18 and the consumption of contaminated water and fish—lead to HQs for triclopyr and TCP that are
19 far below the level of concern. The consumption of contaminated vegetation in aquatic
20 applications is not explicitly estimated. Consequently, risks to mammals associated with aquatic
21 applications of triclopyr, either submergent or emergent, are minimal and require little
22 discussion. The highest non-accidental HQ for aquatic applications is 0.02—i.e., the
23 consumption of contaminated fish by a large carnivore after submergent applications at a target
24 concentration of 1 mg a.e./L. At the maximum target concentration of 2.5 mg a.e./L, the HQ
25 would be about 0.05, below the level of concern by a factor of 20. The highest accidental
26 exposure scenario for any aquatic application is 0.9. Again, this is the upper bound for a large
27 mammalian carnivore consuming contaminated fish.

28
29 The only residual concern with mammals following aquatic applications of triclopyr involves the
30 treatment of emergent vegetation. It seems reasonable that mammals could feed on treated
31 emergent vegetation shortly after triclopyr was applied. Methods to estimate doses from this
32 type of exposure are not available. By analogy to the consumption of terrestrial vegetation by
33 mammals, mammals consuming treated emergent aquatic vegetation could be exposed to
34 triclopyr at levels which might exceed the level of concern.

35 **4.4.2.2. Birds**

36 **4.4.2.2.1. Triclopyr**

37 The risk characterization for birds is essentially identical to that for mammals except for
38 differences in the impact of body size on apparent risk. For birds, there is no clear indication of
39 systematic differences in sensitivity with body size. Thus, smaller birds have somewhat higher
40 HQs than larger birds, because smaller birds will consume more food per unit body weight than
41 will larger birds.

42
43 As with mammals, the upper bound HQs for exposure scenarios associated with the consumption
44 of contaminated vegetation are substantial at an application rate of 1 lb a.e./acre and will increase

1 linearly with the application rate. Based on the HQs, adverse effects in birds could be
2 anticipated. The avian field studies (Appendix 3, Table A3-4) are not as numerous or as detailed
3 as those involving mammals and neither confirm nor substantially refute concerns based on the
4 HQs.

5
6 Also as with mammals, exposures associated with the consumption of contaminated water and
7 contaminated fish are negligible. In the accidental spill scenarios, the highest HQ for birds is
8 0.04, the upper bound of the HQ for a small bird consuming contaminated water. The exposure
9 scenario for a small bird consuming contaminated insects does lead to a modest excursion above
10 the level of concern at the upper bound—i.e., HQs of 0.3 (0.02 to 1.8). As summarized in
11 Table 27, this exposure scenario is based on residue rates from Fletcher et al. (1997) which are
12 equivalent to the rates used for broadleaf vegetation. These rates are intermediate between those
13 for short grass and fruit.

14 **4.4.2.2.2. TCP**

15 Because no chronic data are available on the toxicity of TCP to birds, risks associated with
16 chronic exposure to TCP residues cannot be characterized quantitatively. For acute exposures,
17 risks are characterized based on a LOAEL of 116 mg/kg bw rather than a NOAEL. As discussed
18 in Section 4.3.2.2.2, the LOAEL of 116 mg/kg bw is based only on decreases in body weight
19 gain and food consumption in which no overt signs of toxicity were observed (Long et al. 1990)
20 and the toxicologic significance of this LOAEL is questionable.

21
22 As with mammals, the only HQs for birds that approach or exceed a level of concern are the
23 upper bound HQs associated with the acute consumption of contaminated vegetation.

24 **4.4.2.3. Reptiles and Amphibians (Terrestrial Phase)**

25 No toxicity data are available for triclopyr or TCP in reptiles or terrestrial phase amphibians.
26 Consequently, risks to these groups of organisms are not characterized directly.

27
28 In the absence of data, the U.S. EPA/OPP will typically characterize risks to amphibians based
29 on the risk characterization for birds. In the recent EPA risk assessment on the California red-
30 legged frog, U.S. EPA/OPP (2009a, p. 75) uses toxicity studies on birds, identical to those used
31 in the current risk assessment, to derive RQs ranging from 0.01 to about 5, based on acute
32 exposures, and from about 1 to 134, based on chronic exposures.

33 **4.4.2.4. Terrestrial Invertebrates**

34 The quantitative risk characterization for terrestrial invertebrates is limited by the available
35 toxicity data (Section 4.3.2.4.). The toxicity value used to develop HQs is an indeterminate LD₅₀
36 of >620 mg a.e./kg bw. This dose is used to develop HQs for direct spray (Worksheet G02b)
37 and the consumption of contaminated vegetation (G08b). All HQs are below the level of
38 concern at the unit application rate of 1 lb a.e./acre. Similar to the risk characterization for
39 mammals, the direct spray scenarios lead to HQs far below the level of concern, although the
40 dietary HQs approach a level of concern based on the consumption of all standard food
41 commodities (Table 27) except fruit.

42
43 While HQs are not typically derived for soil invertebrates, there is little indication that
44 concentrations of triclopyr in soil are likely to adversely affect soil invertebrates. As

1 summarized in Appendices 10 and 12, the peak concentrations of triclopyr that are likely to
2 occur in the upper 12 inches of soil following applications of triclopyr are about 0.24 ppm a.e.
3 following an application of 1 lb a.e./acre. At the maximum application rate of 9 lb a.e./acre, the
4 maximum expected concentrations would be about 2.2 ppm a.e. This maximum concentration is
5 a factor of about 3 below the chronic NOAEC for earthworms in the study by (Hayward 2000)
6 [6.9 ppm a.e. ÷ 2.2 ppm a.e. \cong 3.13].
7

8 As noted in Section 4.1.2.4, there are numerous field studies suggesting that effects on terrestrial
9 invertebrates are most likely to be associated with changes in habitat and food availability rather
10 than direct toxic effects. The risk characterization based on the HQ method is consistent with
11 these suppositions.

12 **4.4.2.5. Terrestrial Plants**

13 Quantitative risk characterizations for terrestrial plants are given for several types of exposures:
14 runoff (Worksheet G04), direct spray and drift (Worksheet G05), the use of contaminated
15 irrigation water (Worksheet G06a), and exposures to contaminated soils via wind erosion
16 (Worksheet G06b). These worksheets are included in the EXCEL workbooks for terrestrial
17 applications of triclopyr TEA (Attachment 1) and triclopyr BEE (Attachment 2).
18

19 As with all effective herbicides, a direct spray of triclopyr will adversely affect a sensitive plant.
20 As indicated in Worksheet G05, the HQ for this scenario is 357, and HQs of this magnitude do
21 not require further elaboration. Tolerant species of plants, however, might not be killed or even
22 adversely affected—i.e., the HQ for the direct spray of a tolerant plant is 0.5. For sensitive plant
23 species, drift will be an issue, and the hazards associated with drift will vary with the application
24 method, being greatest for aerial application and least for backpack application. As noted in
25 Section 4.2.4.2, the drift estimates used in the current risk assessment are generic, while actual
26 drift during a field application could vary substantially from these estimates, based on a number
27 of site-specific conditions.
28

29 The offsite transport of triclopyr through runoff and sediment loss differs between the TEA and
30 BEE formulations. For triclopyr TEA, the HQs for sensitive plants are 1 (0.0004 to 4). For
31 triclopyr BEE the HQs are 0.03 (0.00001 to 2). The remarkably broad range of HQs reflects the
32 range of site conditions used in the Gleams-Driver modeling (Section 3.2.3.4.3). In many
33 locations, runoff and sediment losses will be insubstantial. In other areas, sensitive species of
34 plants could be damaged. If triclopyr is applied at a site that may be conducive to runoff or
35 sediment loss, refined estimates of offsite transport should be considered based either on the
36 range of values given in Appendix 9 (triclopyr BEE) or Appendix 12 (triclopyr TEA). If this
37 refinement suggests that offsite losses could be a concern, site-specific Gleams-Driver modeling
38 could be considered.
39

40 The HQs for the use of contaminated surface water for irrigation are given in Worksheet G06a.
41 For tolerant plant species, the HQs are far below a level of concern. For sensitive species, the
42 HQs are 0.03 (3×10^{-6} to 5). As discussed in Section 4.2.34.4, the generic estimates of exposure
43 on which these HQs are based may not represent all site-specific conditions. A major factor
44 influencing the site-specific HQs would be the extent of irrigation as well as the site-specific
45 concentrations of triclopyr in surface water. Again, these factors may need to be considered in

1 site-specific applications of triclopyr by modifying the inputs in Worksheet G06a to reflect site-
2 specific conditions.

3
4 For the exposure of nontarget plants to contaminated soil transported by wind, the HQs are
5 substantially below the level of concern. For sensitive plant species, the HQs are 0.02 (0.005 to
6 0.05). It seems reasonable that the only times when soil erosion by wind might pose a risk to
7 nontarget plant species would be when triclopyr is applied to bare ground. Even then, impacts
8 on nontarget plants could vary substantially with site-specific conditions.

9 **4.4.2.6. Terrestrial Microorganisms**

10 The potential for substantial effects on soil microorganisms appears to be low. As summarized
11 in Section 4.1.2.6, laboratory bioassays conducted in artificial growth media suggest a very high
12 degree of variability in the response of soil bacteria and fungi to triclopyr with NOAELs of up to
13 1000 ppm in some species and growth inhibition at concentrations as low as 0.1 ppm in other
14 species. For triclopyr BEE, concentrations of triclopyr in the top 12 to 36 inches of soil range
15 from about 0.04 to 0.1 ppm (Appendix 4, Table A4-2 and A4-4). The corresponding values for
16 triclopyr TEA are essentially identical. If the laboratory bioassays were used to characterize
17 risks to terrestrial microorganisms, transient inhibition in the growth of some bacteria or fungi
18 might be expected. This inhibition could result in a shift in the population structure of microbial
19 soil communities, but substantial impacts on soil, including gross changes in capacity of soil to
20 support vegetation, do not seem plausible. This assessment is consistent with the field
21 experience involving the use of triclopyr to manage vegetation.

22 **4.4.3. Aquatic Organisms**

23 **4.4.3.1. Fish**

24 For terrestrial applications of triclopyr TEA formulations (Attachment 1, Worksheet G03), no
25 risks to fish are identified, based on expected peak concentrations or longer-term concentrations
26 of triclopyr acid in surface water. The highest HQ is 0.01, the upper bound of HQs associated
27 with expected peak concentrations of triclopyr acid in surface water. Thus, at the highest
28 application rate of 9 lb a.e./acre, the upper bound HQ would be 0.09, substantially below the
29 level of concern. For the accidental spill scenario, the upper bound HQ is 0.9. Aquatic
30 applications of triclopyr TEA do not lead to HQs that exceed the level of concern for either
31 emergent applications (Attachment 3) or submergent applications (Attachment 4).

32
33 As discussed in Section 4.3.3.1, triclopyr BEE is much more toxic than triclopyr acid to fish, as
34 reflected in the risk characterization. For terrestrial applications of triclopyr BEE formulations
35 (Attachment 2, Worksheet G03), the HQs for expected peak concentrations of triclopyr BEE (not
36 triclopyr acid) in water are 0.004 (2×10^{-6} to 0.3). As with all other HQs associated with
37 concentrations of triclopyr BEE or triclopyr acid in surface water, the wide range of HQs reflects
38 the diverse conditions used in the Gleams-Driver modeling. Thus, the upper bound HQ of 0.3
39 may be regarded as a *worst-case* assessment for an application rate of 1 lb a.e./acre. This upper
40 bound HQ would reach a level of concern at an application rate of about 3 lb a.e./acre.
41 Consequently, there is no basis for asserting that application rates of triclopyr BEE at up to 3 lbs
42 a.e./acre are likely to pose risks to fish. For higher application rates, consideration of local site-
43 conditions would be required to assess the possibility of risks to fish.

1 Triclopyr BEE will not persist in surface water; accordingly, the HQs associated with chronic
2 exposures to triclopyr BEE are negligible—i.e., 0.0001 (1×10^{-9} to 0.004). While risks to
3 sensitive and tolerant species of fish cannot be differentiated due to limitations in the available
4 toxicity data, the very low longer-term HQs for triclopyr BEE suggest that no species of fish are
5 likely to be at risk from longer-term exposures to triclopyr BEE.

6
7 The above risk characterization for triclopyr BEE is not consistent with U.S. EPA/OPP (2009a).
8 The U.S. EPA/OPP (2009a, Table 5-1, p. 89) gives chronic RQs for forestry applications of
9 triclopyr ranging from about 2 to 22. These RQs are based on the chronic NOAEC of 0.019 mg
10 a.e./L in trout for triclopyr BEE (Weinberg et al. 1994d), the same chronic NOAEC for fish used
11 in the current risk assessment. Based on the discussion of the input values used in the Agency's
12 exposure assessment (U.S. EPA/OPP 2009a, p. 60 ff), the exposure estimates used by the
13 Agency for chronic effects in fish appear to be based on modeling of triclopyr acid rather than
14 triclopyr BEE. As discussed below in the risk characterization for aquatic macrophytes (Section
15 4.4.3.4.2), it seems sensible to use toxicity and exposure data for triclopyr acid as the basis for
16 the risk characterization for the effects of triclopyr BEE on aquatic macrophytes. The rationale
17 for using exposure data on triclopyr acid and toxicity data on triclopyr BEE for the risk
18 characterization of fish is not clear. The very low chronic HQs for triclopyr BEE in fish given in
19 the current risk assessment are based on estimated longer-term exposures to triclopyr BEE as
20 well as chronic toxicity data on triclopyr BEE. The very low chronic HQs in fish for triclopyr
21 acid are based on chronic toxicity data on triclopyr acid in fish as well as estimated longer-term
22 exposures of fish to triclopyr acid.

23
24 The accidental spill scenario for triclopyr BEE does lead to very high HQs—i.e., 20 (2 to 200)
25 for sensitive species of fish and 2 (0.3 to 24) for tolerant species of fish. These high HQs require
26 little elaboration. If a large amount of triclopyr BEE is spilled into a small body of water,
27 adverse effects on fish, probably involving substantial fish kills, could be expected.

28
29 Terrestrial applications of both triclopyr BEE and triclopyr TEA formulations will result in the
30 contamination of surface water with TCP (Attachment 3, Worksheet G03). At an application
31 rate of 1 lb a.e./acre of either triclopyr TEA or triclopyr BEE, the upper bound of the HQ for
32 sensitive species of fish, based on peak concentrations of TCP, is 0.2. Thus, at application rates
33 of up to 5 lb a.e./acre, TCP is not likely to pose a risk to sensitive species of fish. Longer-term
34 concentrations of TCP are far below the level of concern—i.e., HQs of 0.0003 (2×10^{-11} to 0.01).
35

36 **4.4.3.2. Amphibians**

37 The risk characterization for amphibians is essentially identical to that for fish. Triclopyr BEE is
38 much more toxic than triclopyr acid to amphibians. Acute risks to amphibians following
39 applications of triclopyr BEE would reach a level of concern at an application rate of about 3 lbs
40 a.e./acre, based on potential peak exposures to triclopyr BEE. A formal quantitative risk
41 characterization for longer-term exposures of amphibians to triclopyr BEE is not developed
42 because of the lack of adequate chronic toxicity studies on amphibians. Given the very low
43 longer-term concentrations of triclopyr BEE in surface water, however, the lack of chronic
44 toxicity data on triclopyr BEE in amphibians is not a major limitation in the risk characterization.
45

1 The field study by Wojtaszek et al. (2005) involving longer-term observations of amphibian
2 populations following forestry applications of triclopyr BEE offers the following informal risk
3 characterization for forestry applications of triclopyr BEE:

4
5 *Given the risk to only a small proportion of equivalently exposed native*
6 *amphibian larvae, the low frequency of silvicultural herbicide application*
7 *in a given area (e.g., 1-2 applications over an 50-year forestry rotation)*
8 *and the overall limited use of this herbicide product in Canadian forestry,*
9 *ecological risk to native amphibian populations under current use*
10 *scenarios would be considered negligible.*

11 Wojtaszek et al. (2005), p. 2543

12
13 None of the available information identified during the conduct of the current risk assessment
14 disputes the above assessment by Wojtaszek et al. (2005).

15
16 Risks associated with the potential impact of TCP on amphibians are not assessed because of the
17 lack of data on the toxicity of TCP to amphibians.

18
19 The risk characterization for amphibians associated with aquatic applications of triclopyr TEA
20 does not lead to HQs that exceed the level of concern for either emergent applications
21 (Attachment 3) or submergent applications (Attachment 4).

22 **4.4.3.4. Aquatic Invertebrates**

23 The risk characterization for aquatic invertebrates is very similar to that for fish. No risks
24 associated with exposures to triclopyr acid are apparent. For triclopyr BEE, the estimated
25 NOAEC for sensitive species of aquatic invertebrates is somewhat lower than that for sensitive
26 species of fish. At an application rate of 1 lb a.e./acre, the HQs associated with peak exposures
27 of sensitive species of aquatic invertebrates are 0.009 (3×10^{-6} to 0.7). Thus, based on the upper
28 bound exposure, the HQ would reach a level of concern at an application rate of about 1.5 lbs
29 a.e./acre, somewhat lower than the rate of 3 lbs a.e./acre for fish and amphibians. Consequently,
30 some consideration of site-specific conditions would be warranted for a more refined assessment
31 of acute risks to aquatic invertebrates at application rates of triclopyr BEE that exceed 1.5 lbs
32 a.e./acre. Longer-term risks to aquatic invertebrates are based on a LOAEL rather than a
33 NOAEL. The upper bound HQ for longer-term exposures, however, is 0.0004, which suggests
34 that the very low longer-term concentrations of triclopyr BEE in surface water will not pose a
35 risk to aquatic invertebrates. Risks associated with exposures of aquatic invertebrates to TCP
36 following terrestrial applications are far below the level of concern.

37
38 As with the risk characterization for both fish and amphibians, the risk characterization for
39 aquatic invertebrates involving aquatic applications of triclopyr TEA do not lead to HQs that
40 exceed the level of concern for either emergent applications (Attachment 3) or submergent
41 applications (Attachment 4).

1 **4.4.3.4. Aquatic Plants**

2 **4.4.3.4.1. Algae**

3 As is true for aquatic animals, triclopyr BEE is much more toxic than triclopyr acid to algae.
4 Following terrestrial applications of triclopyr TEA at the unit application rate of 1 lb a.e./acre,
5 the HQs for sensitive species of algae reach but do not exceed the level of concern—i.e., HQs of
6 0.01 (4×10^{-6} to 1.0). Thus, for application rates in excess of 1 lb a.e./acre, refinements to the
7 exposure assessment for surface could be warranted in areas in which off-site transport might
8 occur. For triclopyr BEE, however, the corresponding HQs for sensitive species of algae are 0.3
9 (0.0001 to 21). Consequently, any application of triclopyr BEE could result in adverse effects in
10 algae in an area where substantial drift or offsite movement in runoff is likely. As detailed in the
11 Gleams-Driver appendices —i.e., Appendix 9 for triclopyr BEE and Appendix 12 for triclopyr
12 TEA—the likelihood of significant surface water contamination due to runoff is remote in arid
13 areas. As rainfall rates increase, so does the potential for substantial runoff which might have an
14 impact on algae.

15 **4.4.3.4.2. Macrophytes**

16 The risk characterization for aquatic macrophytes is similar to that for algae, except that the
17 apparent relative hazards associated with triclopyr TEA and triclopyr BEE are reversed.
18 Triclopyr acid is much more toxic than triclopyr BEE to macrophytes. For triclopyr TEA
19 applications (Attachment 1, Worksheet G03), the acute HQs are 6 (0.003 to 480) and the longer-
20 term HQs are 2 (4×10^{-7} to 120).

21
22 The HQs for aquatic macrophytes following terrestrial applications of triclopyr BEE are much
23 lower than those for triclopyr TEA. The assessment of likely effects on aquatic macrophytes,
24 however, is one example where the use of toxicity values and exposure estimates for triclopyr
25 BEE to develop HQs is probably not justified. As discussed in Section 3.2.3.4.3, triclopyr BEE
26 will rapidly degrade to triclopyr acid. Consequently, for the risk characterization of aquatic
27 macrophytes, the HQs for triclopyr TEA applications should be applied to the assessment of
28 triclopyr BEE applications, since triclopyr TEA is also rapidly hydrolyzed to triclopyr acid.
29 Thus, for both triclopyr TEA and triclopyr BEE terrestrial applications, risks to aquatic
30 macrophytes are substantial. As with algae, these risks will be much less in arid areas, so long as
31 drift to surface water is avoided. If substantial drift occurs, damage to aquatic macrophytes
32 following applications of either triclopyr TEA or triclopyr BEE could occur.

33
34 Depending on site-specific conditions, damage to aquatic macrophytes could be evident over a
35 prolonged period of time. As noted above, the longer-term HQs for sensitive species of aquatic
36 macrophytes are 2 (4×10^{-7} to 120), and these HQs are based on estimates of average
37 concentrations of triclopyr in water over a 1-year period.

38
39 Risks to aquatic macrophytes associated with aquatic applications of triclopyr TEA formulations
40 are given in Worksheet G03 of Attachment 3 (emergent applications) and Attachment 4
41 (submergent applications). At a unit application rate of 1 lb a.e./acre, HQs for sensitive species
42 of aquatic macrophytes are 12 (1.5 to 47). At a target concentration of 1 mg a.e./L, HQs for
43 sensitive species of aquatic macrophytes are 64 (16 to 128). These HQs require little elaboration
44 or consideration of application rates. If triclopyr TEA is applied to water at an effective
45 application rate, substantial damage to sensitive species of aquatic macrophytes is certain. For

- 1 both emergent and submergent applications, however, risks to tolerant species of aquatic
- 2 macrophytes are substantially below the level of concern and would remain below the level of
- 3 concern over the range of labeled aquatic application rates and target concentrations.

5. REFERENCES

NOTE: The initial entry for each reference in braces {} simply specifies how the reference is cited in the text. The final entry for each reference in brackets [] indicates the source for identifying the reference.

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FOIA01	Initial FOIA to EPA.
Internet	References obtained from various sites on the Internet.
TriArch1	Archived papers from previous Forest Service risk assessments.
MRID03	CBI studies available for and summarized in the 2003 Forest Service risk assessment on triclopyr (SERA 2003).
MRID03r	CBI studies available for and summarized in the 2003 Forest Service risk assessment on triclopyr (SERA 2003) and requested again for 2010 risk assessment.
MRID10	Registrant studies provided by Dow AgroSciences in 2010.
MRID11	Registrant studies provided by Dow AgroSciences in 2011.
MRID11a	Registrant studies provided by Dow AgroSciences after peer review.
MCS	Papers on Multiple Chemical Sensitivity
Sec	Studies taken from secondary sources
SET00	Papers from preliminary scoping and other communications.
SET01	Initial Update: a:Toxline; b 2003 MIA; c ECOTOX; d Rescreen of Toxline; e Late Additions
SET02	Some additional papers based on Internet screen.
Std	Standard references used in most Forest Service risk assessments.
Tric03	From 2003 Forest Service risk assessment.

Note: The MRID studies are cited consistent with U.S. EPA/OPP (1998a). Some of the full studies give authors in more detail and sometimes with a different order. Reference list is current as of February 23, 2011.

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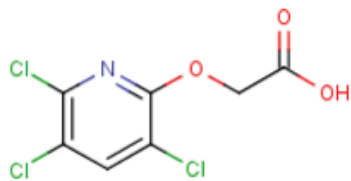
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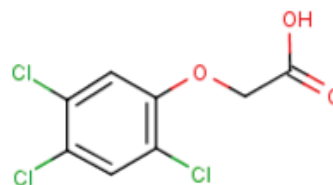
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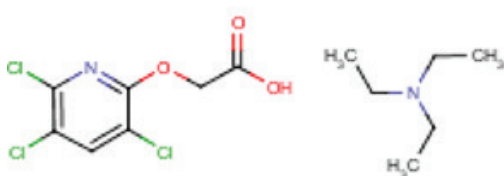
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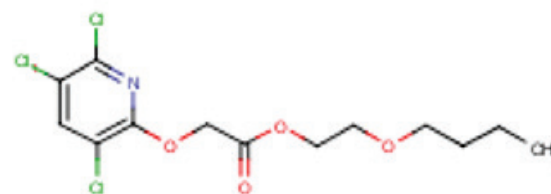
Triclopyr Acid



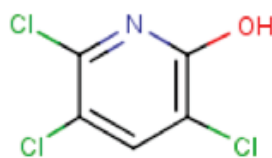
2,4,5-T



Triclopyr TEA



Triclopyr BEE



TCP
(3,5,6-Trichloro-2-pyridinol)

Figure 1: Structure of Triclopyr and Related Compounds

Structures taken from ChemIDplus (<http://chem.sis.nlm.nih.gov/chemidplus/>).
See Section 2.2 for discussion.

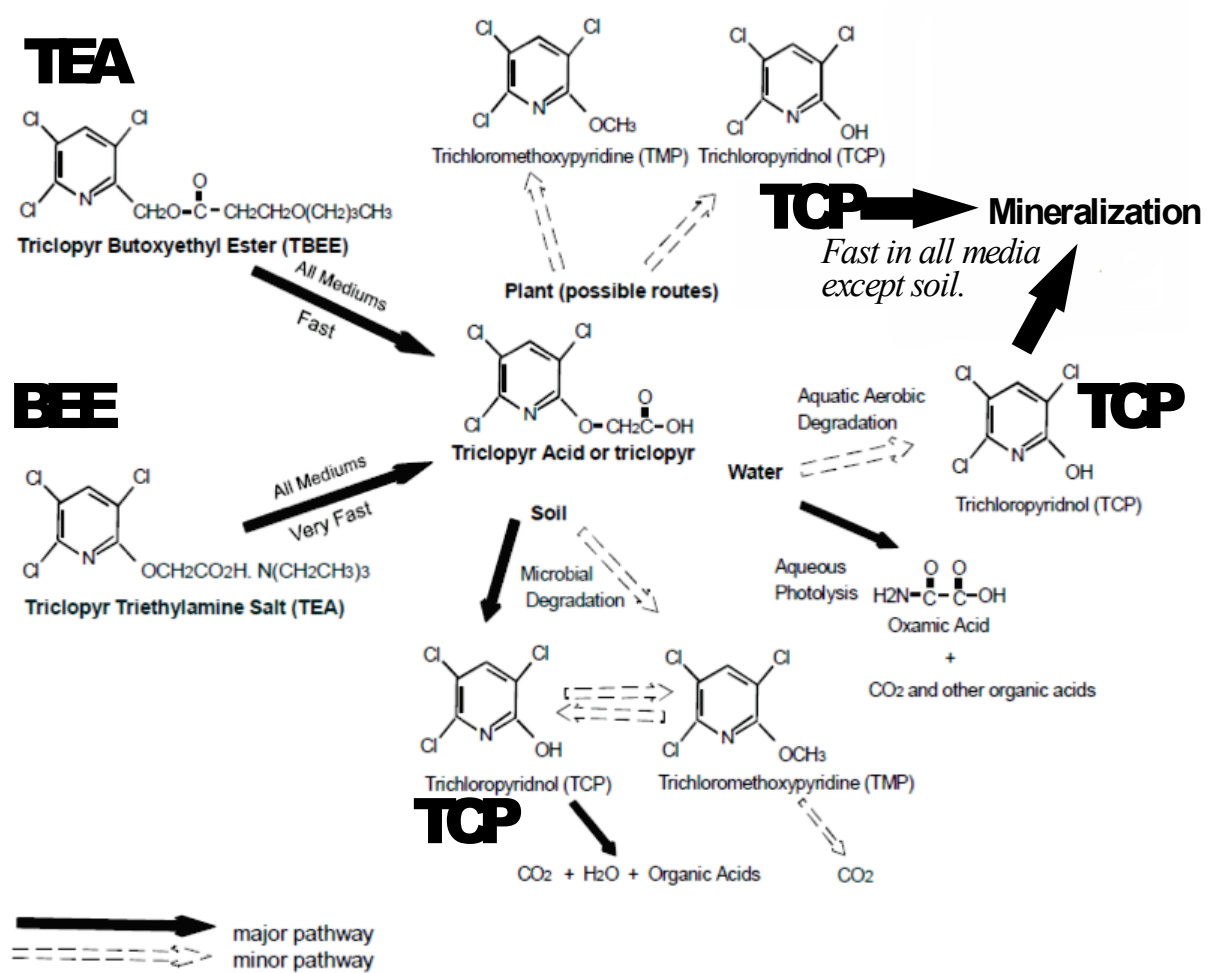


Figure 2: Pathways for the Degradation of Triclopyr and Related Compounds

Source: Modified from Ganapathy (1997)
See Section 2.2. for discussion.

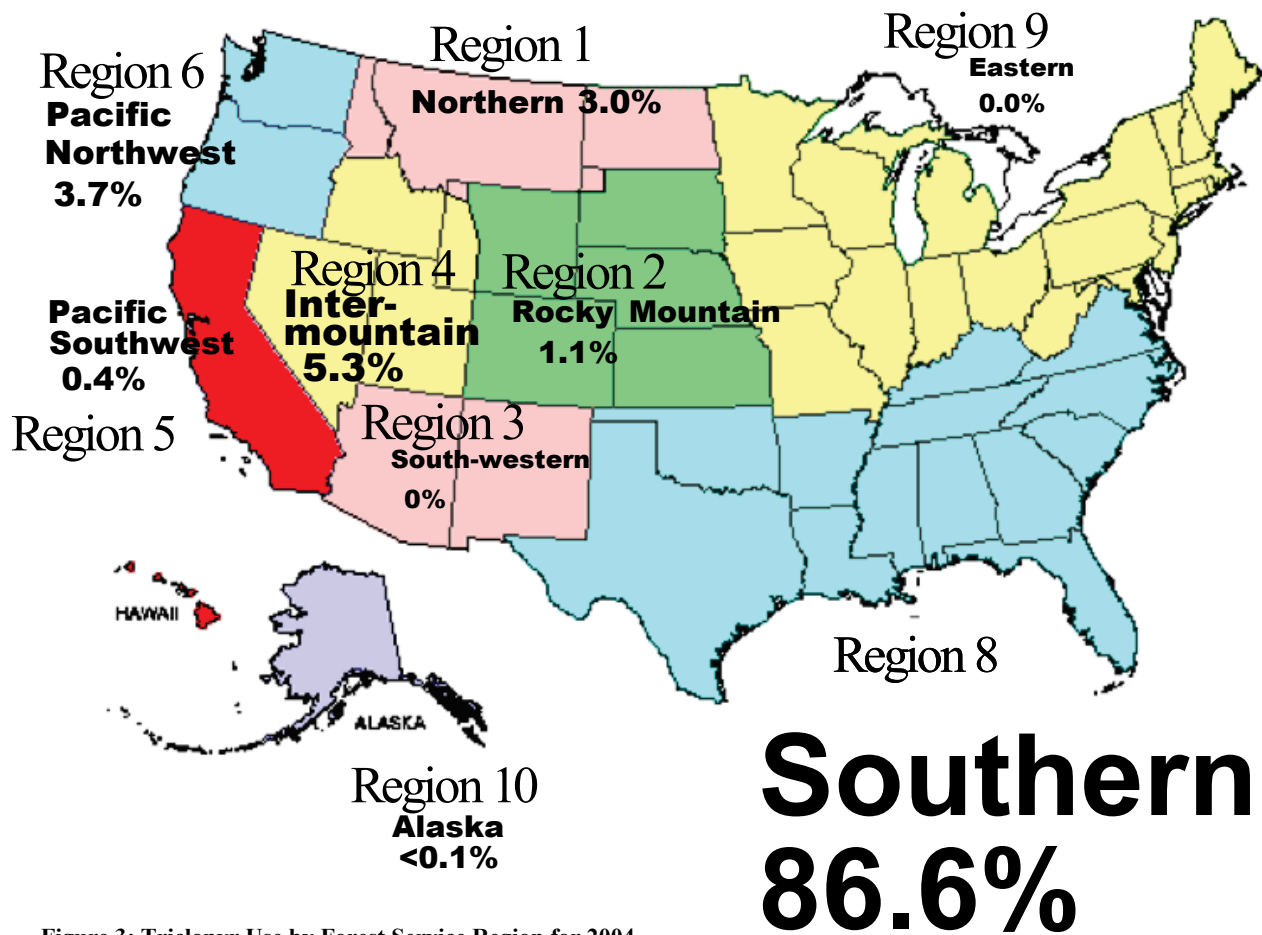


Figure 3: Triclopyr Use by Forest Service Region for 2004

TRICLOPYR - herbicide
 2002 estimated annual agricultural use

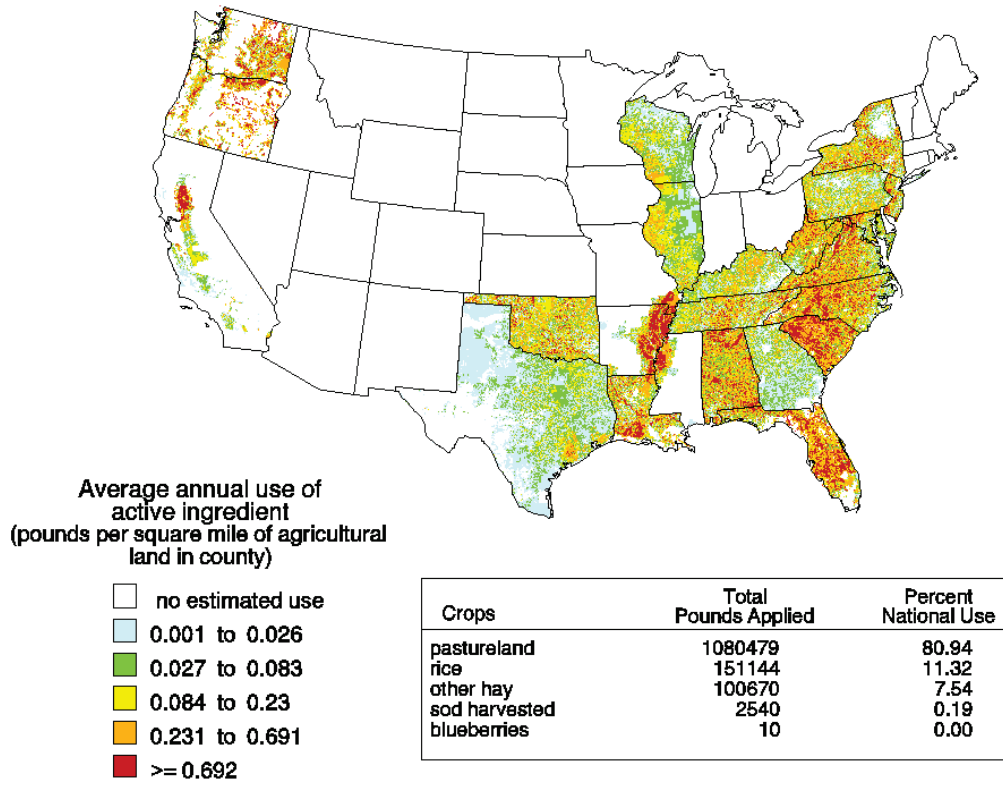
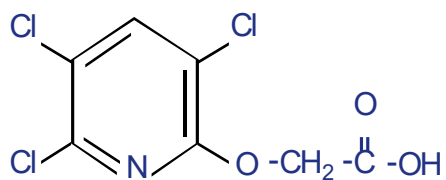


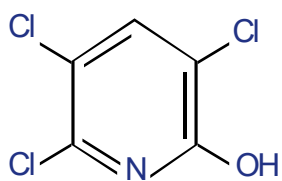
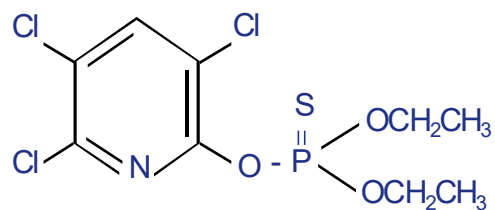
Figure 4: Agricultural Use of Triclopyr in 2002

Source: USGS 2003a

Triclopyr acid



Chlorpyrifos



TCP

Figure 5: Chemical Structures of Triclopyr, Chlorpyrifos, and TCP

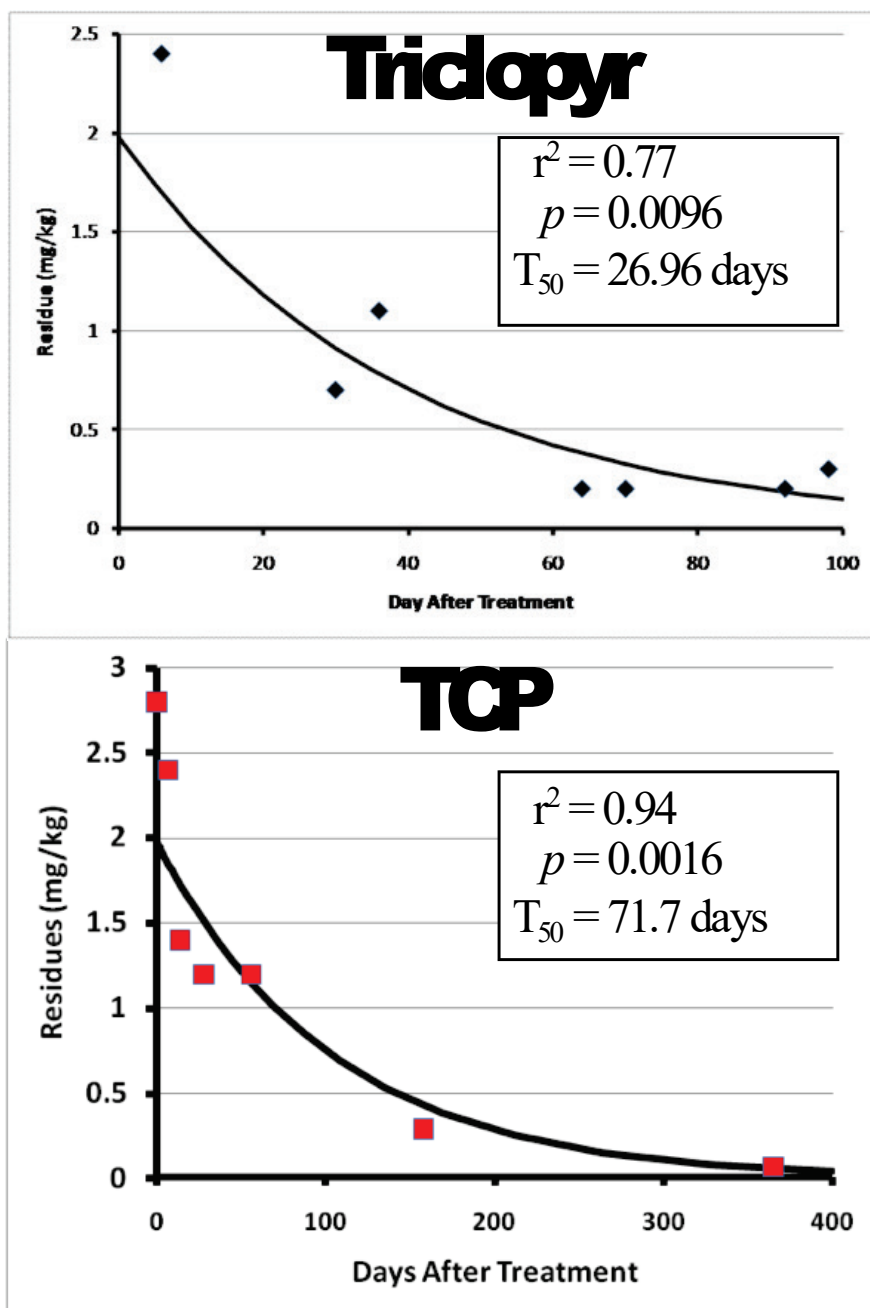


Figure 6: Degradation of Triclopyr and TCP in Plants

Triclopyr Source: Siltanen et al. 1981, Table 1, p. 733.

See Section 3.2.3.7 for discussion.

TCP Source: Norris et al. 1987, Table 1, p. 136.

See Section 3.2.3.8 for discussion.

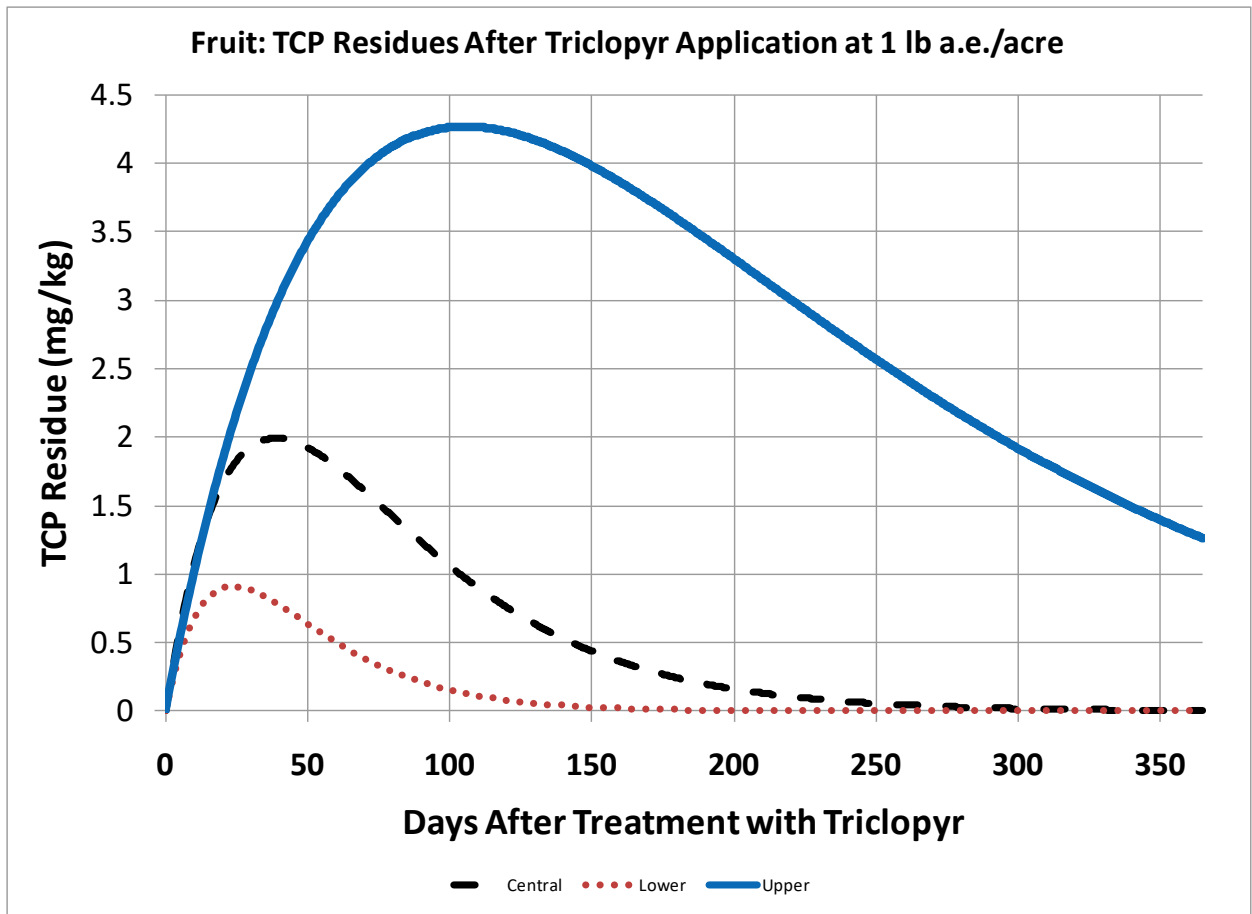


Figure 7: Estimated Residues of TCP in Fruit

See Table 27 for a summary of the estimates.

See Section 3.2.3.8 for discussion.

Details of calculations are given in Worksheet B07a of Attachment 1.

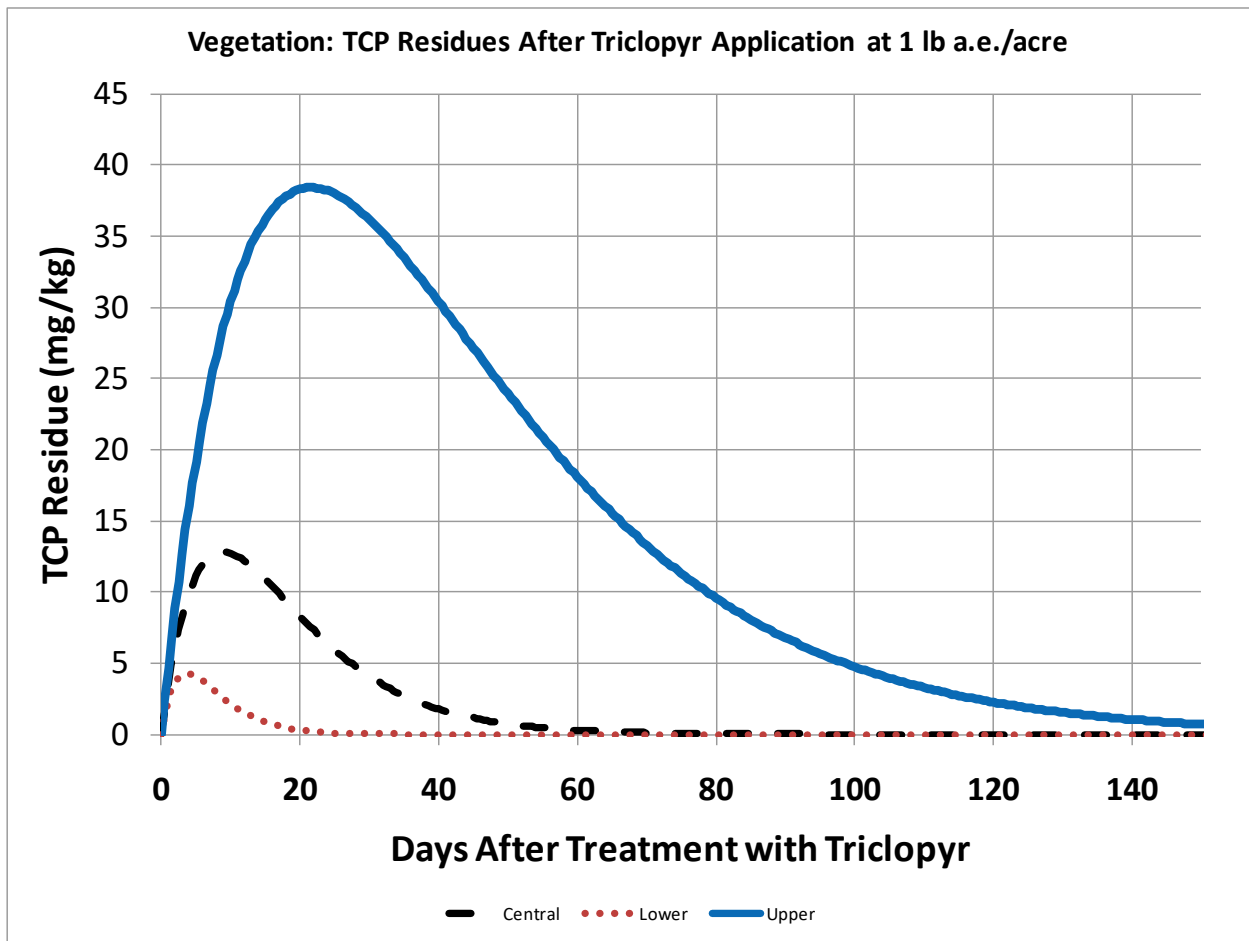


Figure 8: Estimated Residues of TCP in Vegetation

See Table 27 for a summary of the estimates.

See Section 3.2.3.8 for discussion.

Details of calculations are given in Worksheet B07a of Attachment 1.

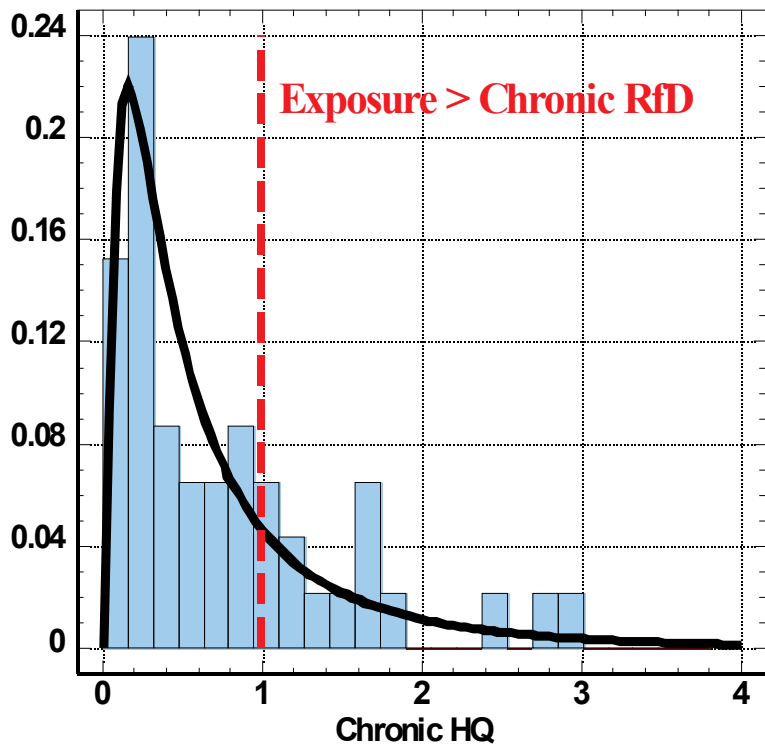
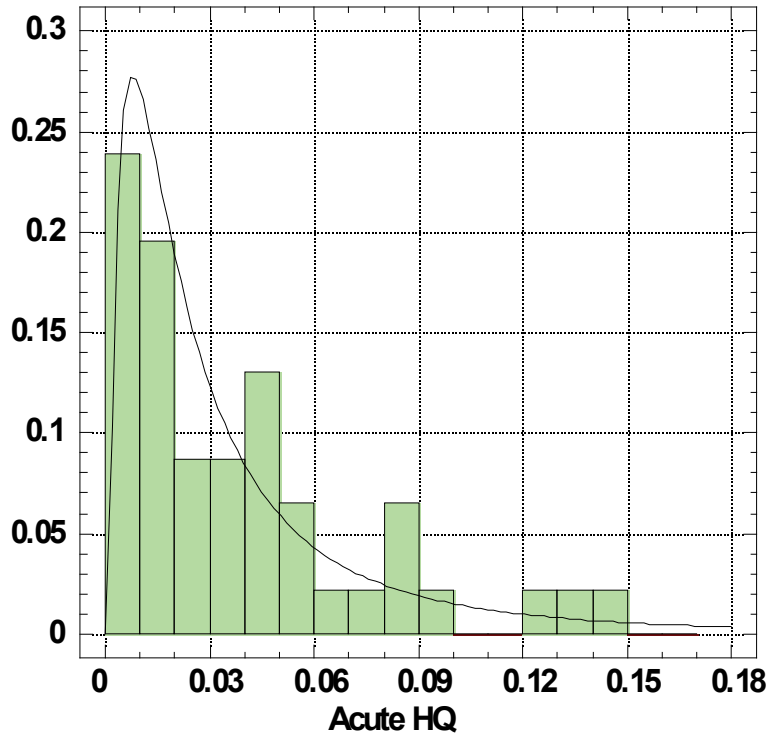


Figure 9: HQs for Male Workers in Backpack Foliar Applications of Triclopyr BEE

NOTE: The chronic HQs illustrated above should be treated as acute HQs for females.

Worker exposure data from Middendorf (1992b) and Spenser et al. (2000).

See Section 3.4.2.2 for discussion.

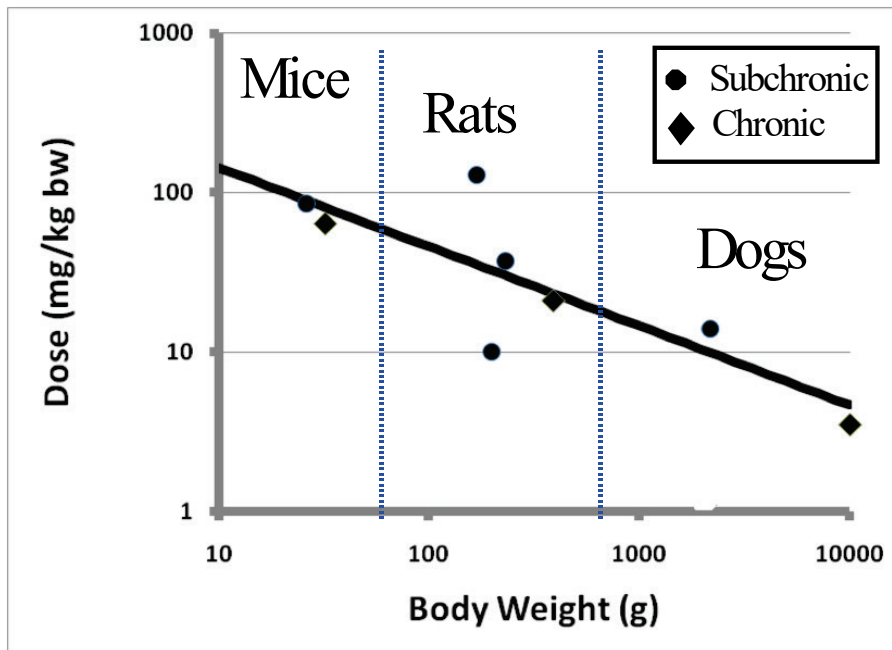
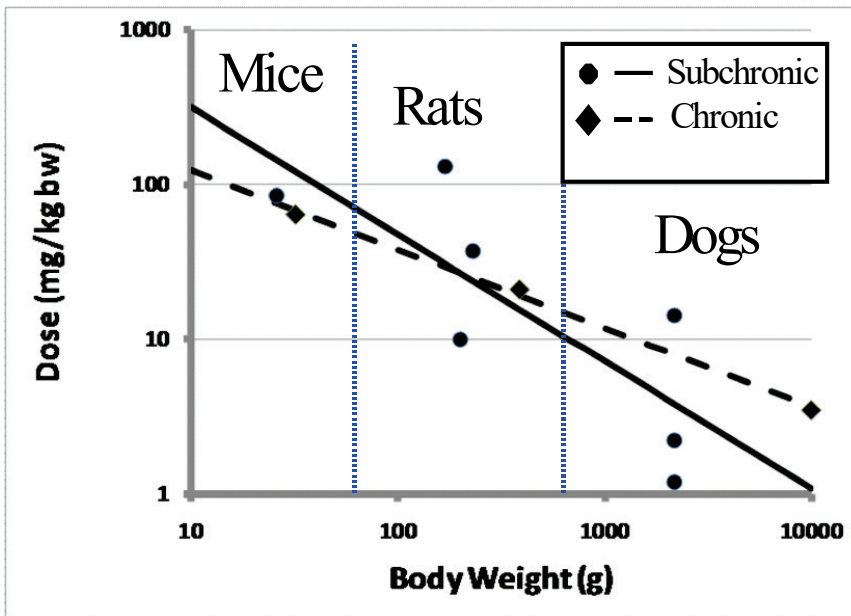


Figure 10: Allometric Relationships for Toxicity of Triclopyr in Mammals

See Table 32 for data.
See Section 4.1.2.1.1 for discussion.

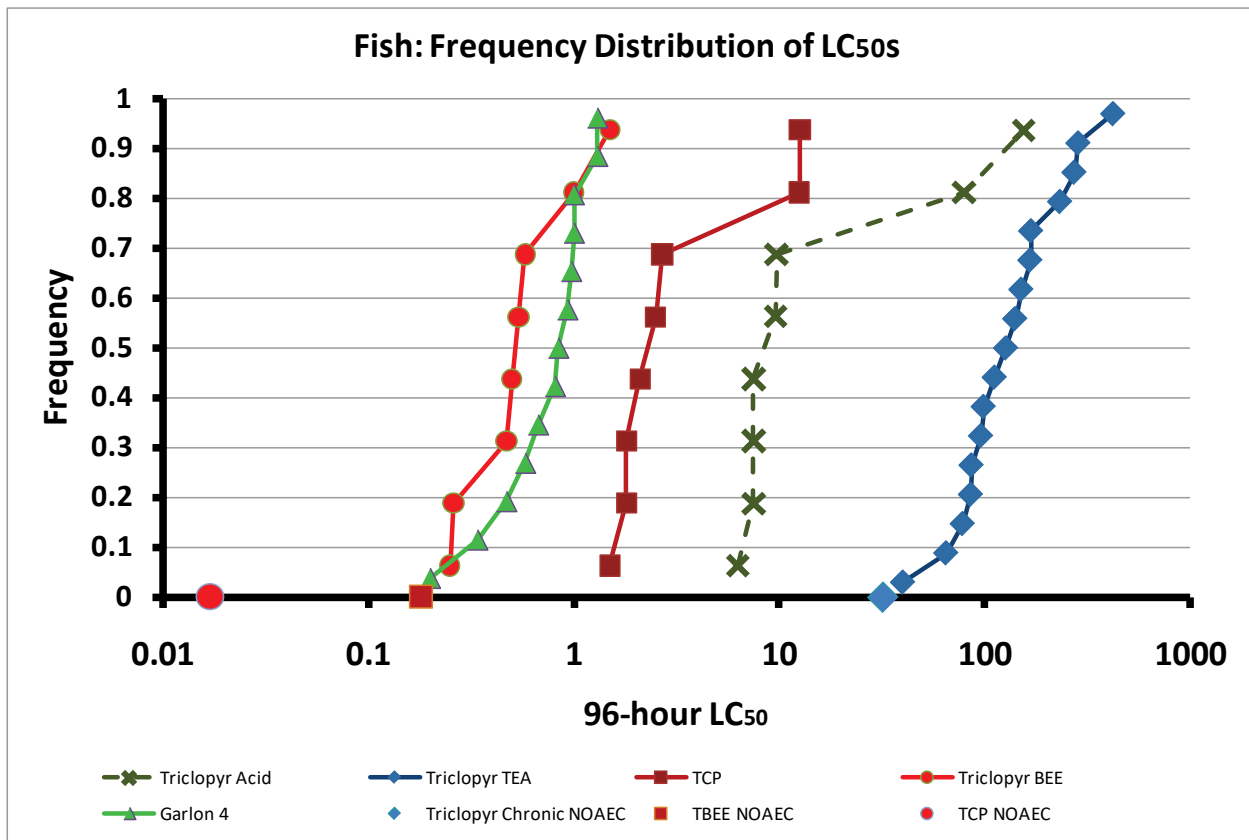


Figure 11: Fish, Frequency Distributions of LC₅₀ Values

See Section 4.1.3 for general discussion of plot.
 See Table 33 for summary of data and Appendix 6 for details of data.
 See Section 4.1.3.1 for discussion.

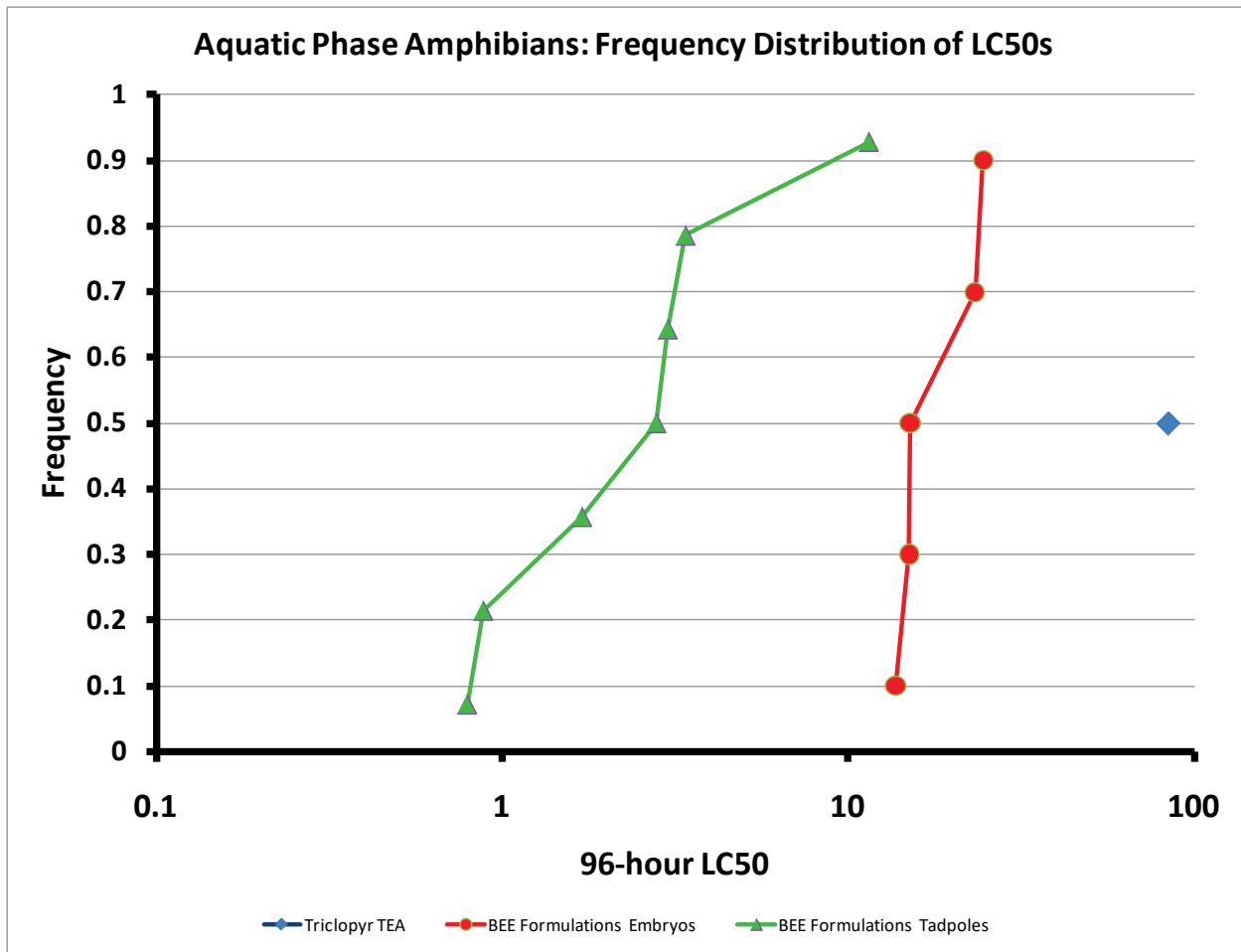


Figure 12: Aquatic Phase Amphibians, Frequency Distributions of LC₅₀ Values

See Section 4.1.3 for general discussion of plot.
 See Table 34 for summary of data and Appendix 7 for details of data.
 See Section 4.1.3.2 for discussion.

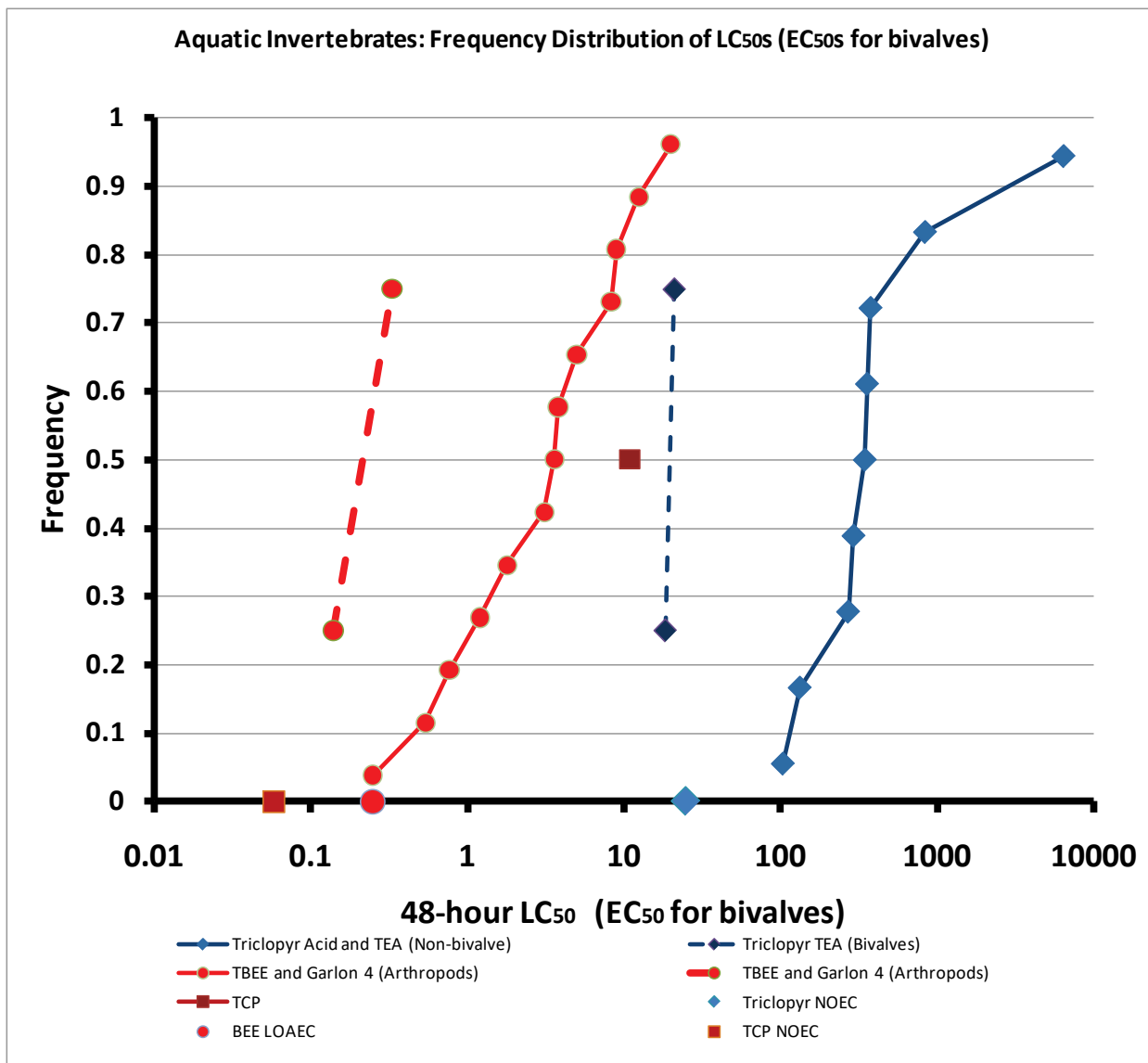


Figure 13: Aquatic Invertebrates, Frequency Distributions of LC₅₀ Values

See Section 4.1.4 for general discussion of plot.
 See Table 35 for summary of data and Appendix 8 for details of data.
 See Section 4.1.3.3 for discussion.

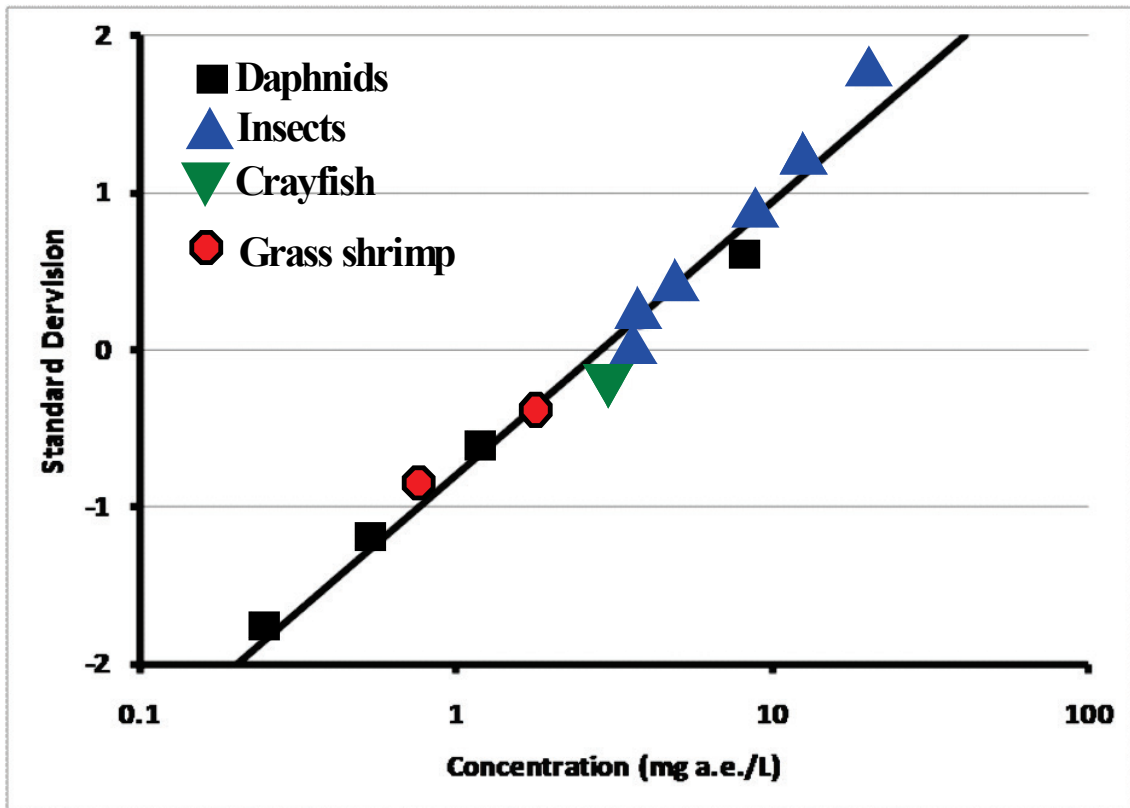


Figure 14: Aquatic Invertebrates, Species Sensitivity Distribution for Triclopyr BEE

See Table 35 for summary of data and Appendix 8 for details of data.
 See Section 4.1.3.3 for discussion of plot and data.

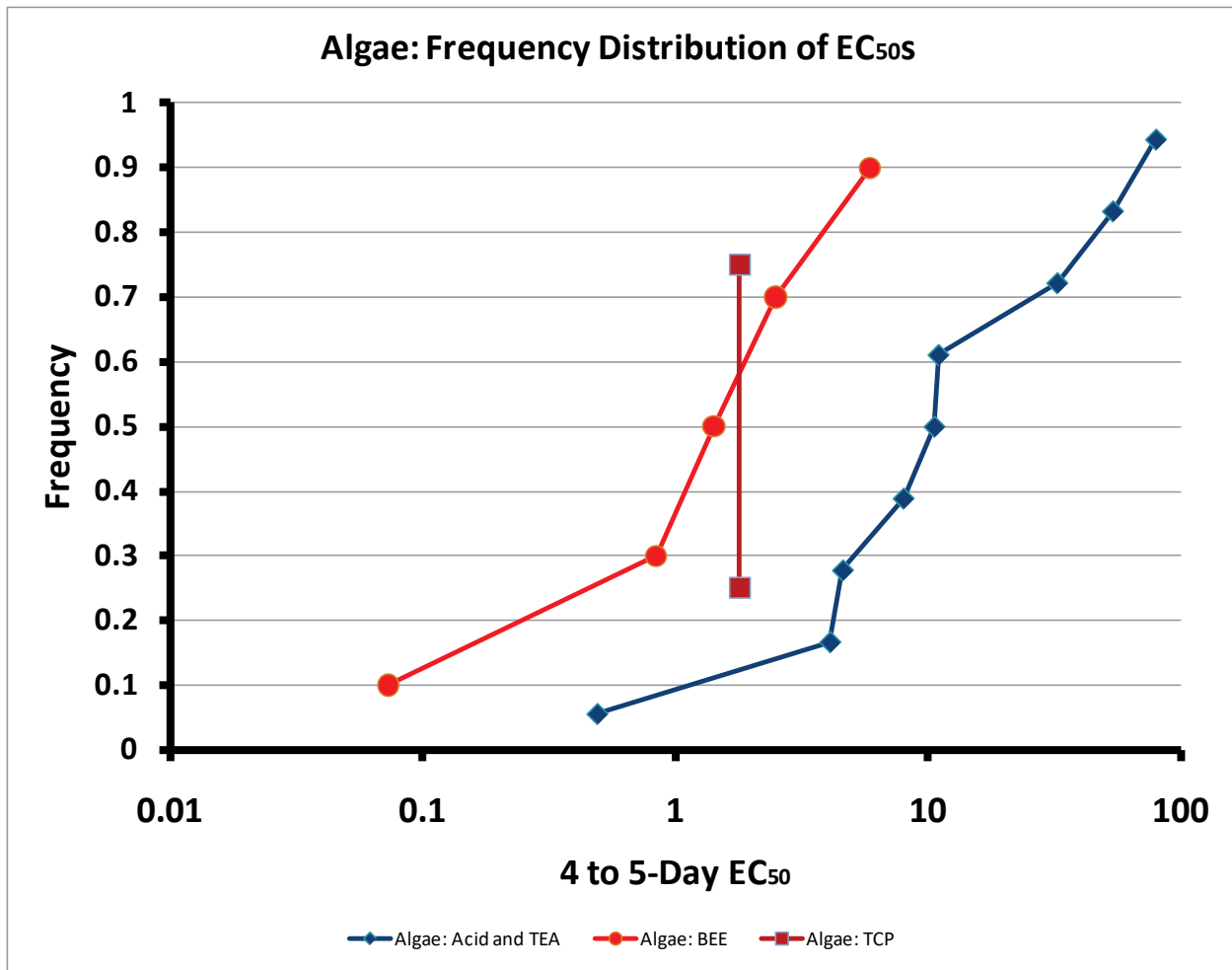


Figure 15: Aquatic Algae, Frequency Distributions of EC₅₀ Values

See Section 4.1.4 for general discussion of plot.
 See Table 36 for summary of data and Appendix 8, Table A8-1 for details of data.
 See Section 4.1.3.4.1 for discussion.

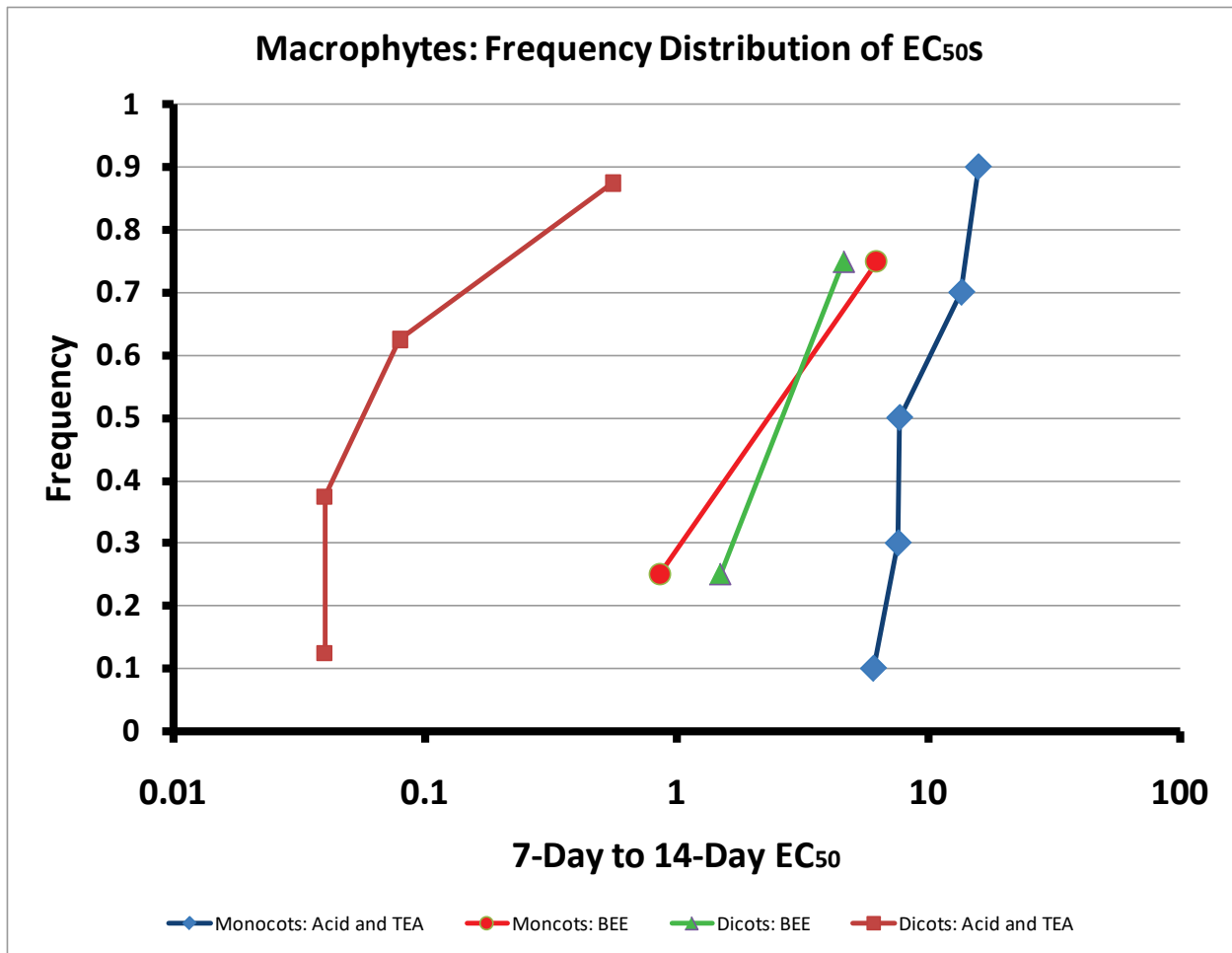
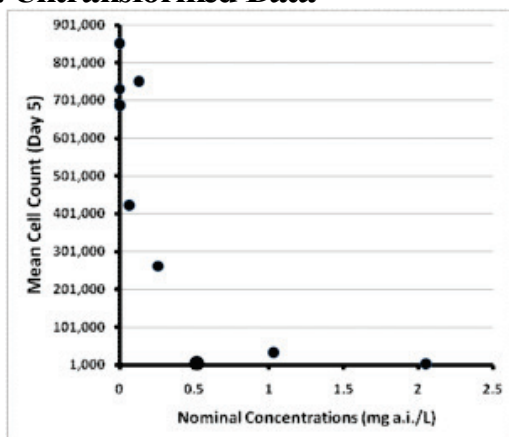


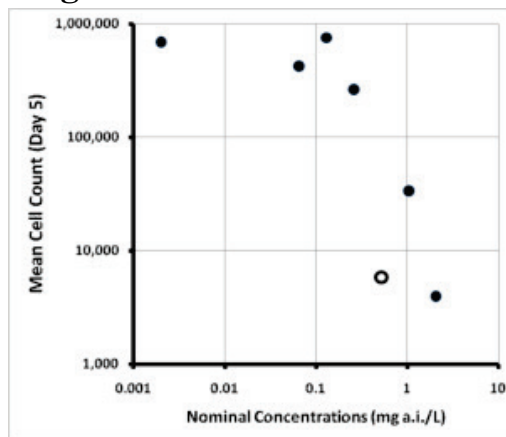
Figure 16: Aquatic Macrophytes, Frequency Distributions of EC₅₀ Values

See Section 4.1.4 for general discussion of plot.
 See Table 37 for summary of data and Appendix 8, Table A8-2 for details of data.
 See Section 4.1.3.4.1 for discussion.

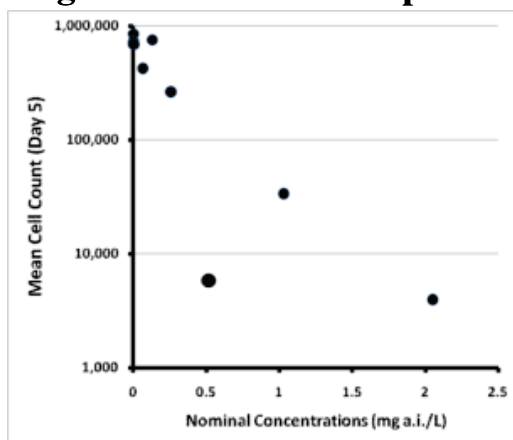
A. Untransformed Data



B. Log transformation of Dose



C. Log transformation of Response



D. Exponential Model

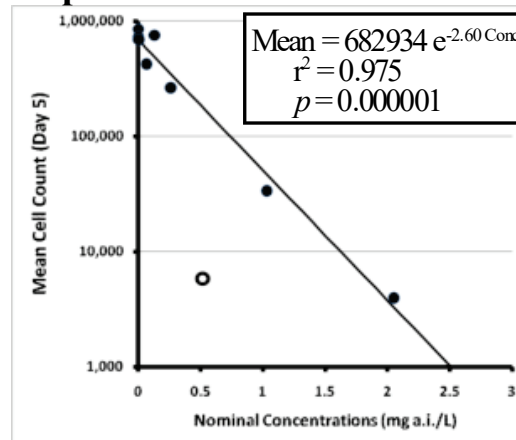


Figure 17: Concentration-Response Relationship for Triclopyr BEE in *Navicula pelliculosa*

Source: Hughes and Alexander 1993c, Table 5
See Section 4.3.3.4.1.2 for discussion.

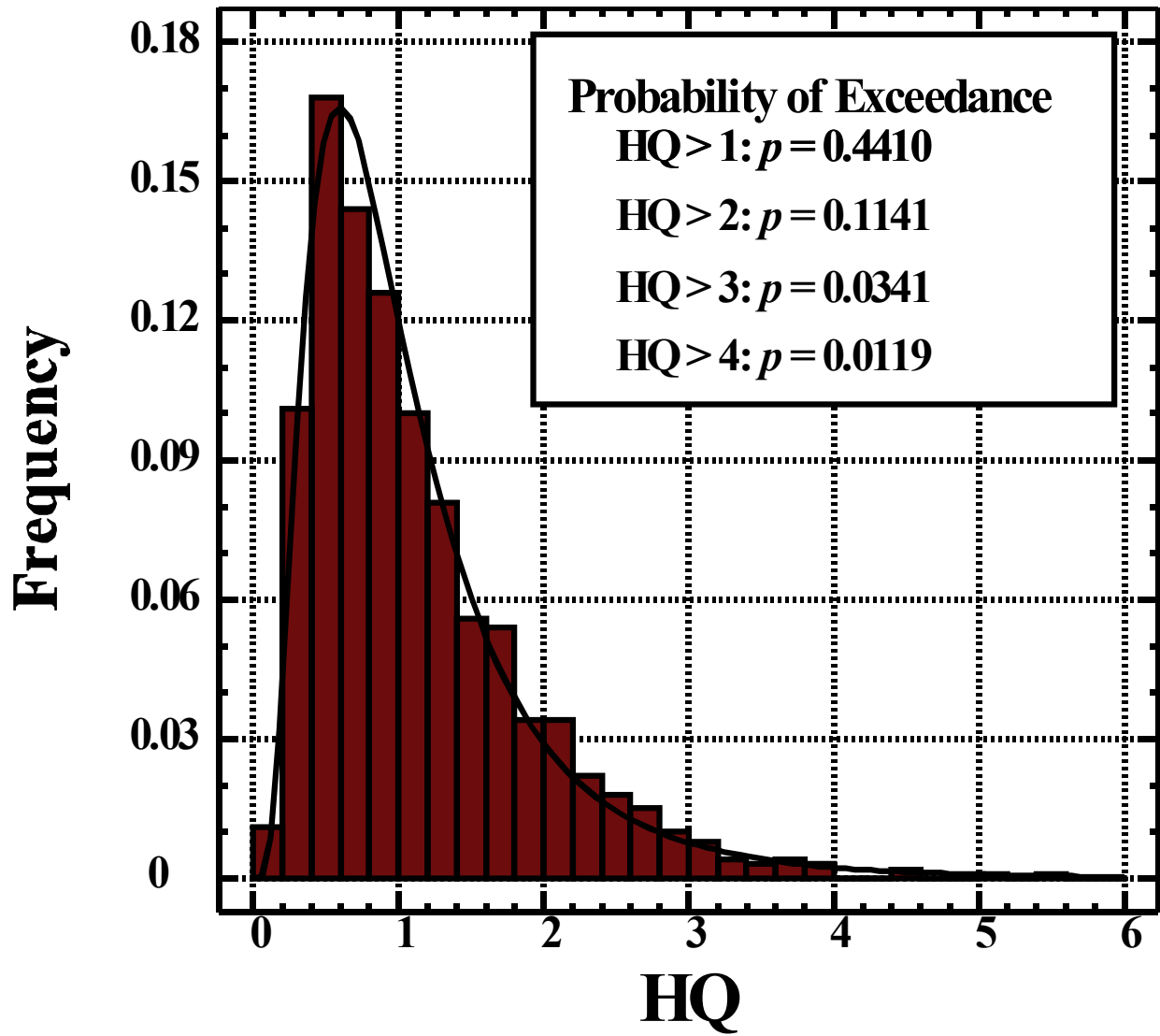


Figure 18: Grazing Deer, Probability of Exceeding HQ=1 at 1 lb a.e./acre

Table 1: Physical and Chemical Properties Triclopyr (various forms) and TCP		
Item	Value	Reference
	Identifiers^[1]	
Common name:	Triclopyr	Tomlin 2004
CAS Name	[(3,5,6-trichloro-2-pyridyl)oxy]acetic acid	Tomlin 2004
IUPAC Name	3,5,6-trichloro-2-pyridyloxyacetic acid	Tomlin 2004
CAS No.	Form (Abbrev)	CAS No.
	Acid	55335-06-3
	Butoxyethyl ester (BEE)	64700-56-7
	Triethylamine salt (TEA)	57213-69-1
	3,5,6-Trichloro-2-pyridinol (TCP)	6515-38-4
	Tomlin 2004 (triclopyr) http://www.chemblink.com (TCP)	
CAS No.	Triclopyr BEE: 64470-88-8	NLM TOXLINE ChemDplus Advanced
Molecular formula	C ₇ H ₄ Cl ₃ NO ₃ [Acid] C ₁₃ H ₁₆ Cl ₃ NO ₄ [BEE] C ₁₃ H ₁₉ Cl ₃ N ₂ O ₃ [TEA]	Tomlin 2004
	Chemical Properties⁽¹⁾	
Henry's Law Constant (air-water partition coefficient)	Form (Abbrev)	Pa m ³ mol ⁻¹
	Acid	9.77 x 10 ⁻⁵
	Butoxyethyl ester (BEE)	5.2 x 10 ⁻²
	Triethylamine salt (TEA)	1.16 x 10 ⁻⁹
	9.66 x 10 ⁻⁷ atm m ³ mol ⁻¹ (acid)	U.S. EPA/OPP 2009a, Table 2-1
	Form (Abbrev)	Atm m ³ mol ⁻¹
	Acid	9.66 x 10 ⁻⁶
	Butoxyethyl ester (BEE)	2.47 x 10 ⁻⁷
	Triethylamine salt (TEA)	1.15 x 10 ⁻¹⁴
	<	(Table 3-2 for acid; Appendix P for TEA and BEE)
Hydrolysis	Stable	Tomlin 2004
	Stable at pH 5,7,9 (MRID 41879601)	U.S. EPA/OPP 2009a, Table 2-1
	TEA: N.A. BEE Sterile Buffered Solutions: pH 5 = 84 days pH 7 = 8.7 days pH 9 = 0.3 days Natural Water: pH 6.7 = 0.5 days	U.S. EPA/OPP 2009a, Appendix P, MRID 134174 BEE data also published in McCall et al. 1988
	BEE: 0.3 to 1.2 days	Milazzo et al. 1993
	TCP: stable	Knutson 1999
Kow	Form not specified, presumably acid pH 5: 2.63 [Log Kow = 0.42] pH 7: 0.35 [Low Kow = -0.45] pH 9: 0.11 [Low Kow = -0.96]	Tomlin 2004
	Acid: 2.95 [Log Kow = 0.47] Ester: ≈123,000 [Log Kow = 5.09]	Brudenell et al. 1995 citing unpublished data from Kenaga and Goring (Dow Chemical)
	TEA: 1.23 Ester: 15,589	Ganapathy 1997 (citing personal communication from Vanelle Carrithers, Dow Elanco October 15, 1996.

Item	Value	Reference	
	BEE: $\approx 12,300$ [Log Kow = 4.09] to $\approx 30,900$ [Log Kow = 4.49]; geometric mean $\approx 19,500$	BEE: Garlon 4 MSDS	
	Acid: ≈ 337 [Log Kow = 2.5281] TEA: ≈ 31.7 [Log Kow = 1.5011] BEE: $\approx 10,311$ [Log Kow = 4.0133] TCP: ≈ 1020 [Log Kow = 3.01]	U.S. EPA/OPPTS 2011 (QSAR from EPI Suite)	
Molecular weight (g/mole)	Form (Abbrev)	MW (g/mole)	U.S. EPA/OPP 2009a (Table 3-2 for acid; Appendix P for TEA and BEE) http://www.chemblink.com (TCP)
	Acid	256.47	
	Butoxyethyl ester (BEE)	356.63	
	Triethylamine salt (TEA)	358.67	
	3,5,6-Trichloro-2-pyridinol (TCP)	198.43	
a.i. to a.e. conversion	Form (Abbrev)	Factor	See Molecular Weight data in previous entry.
	Butoxyethyl ester (BEE)	0.719	
	Triethylamine salt (TEA)	0.715	
	3,5,6-Trichloro-2-pyridinol (TCP)	1.292	
	a.i. to a.e. calculated as MW of acid \div MW of BEE, TEA, or TCP.		
Melting point	150.5 °C	Tomlin 2004	
	148 -150 °C	USDA/ARS 1995	
pKa	3.97	Tomlin 2004	
	2.7	Brudenell et al. 1995; McCall and Gavit 1986	
	2.93	Woodburn et al. 1993a; USDA/ARS 1995	
	2.68	Weber 1994	
	Photolysis (aqueous)	Acid: 8-9 hours (natural light), degrades to 5-Chloro-3,6-dihydroxy-2-pyridinyloxyacetic acid and oxamic acid (combined = 48%) MRIDs 41732201 and 42411804	U.S. EPA/OPP 2009a, Table 2-1
Photolysis (aqueous)	Acid: 0.375 days (Assuming 24 hours of daylight); 0.75 days (Adjusted for 12 hours of daylight)	U.S. EPA/OPP 2009a, Table 3-2; MRIDs 41732201 and 42411804 <small>[Note: This is just a transformation of the above entry.]</small>	
Photolysis (aqueous)	Acid: 1.7 days (In river water), Oxamic acid (16%), MRID 41732201 and 42411804	U.S. EPA/OPP 2009a, Table 2-1	
Photolysis (aqueous)	BEE: 6.6 days in Sterile pH 5 aqueous buffer solution	U.S. EPA/OPP 2009a, Appendix P, MRIDs 41732201 and 42411804	
Photolysis (aqueous)	TEA: Sterile solution: 0.6 days in natural light, pH7 0.36 days in artificial light, pH7	U.S. EPA/OPP 2009a, Appendix P, MRIDs 41732201 and 42411804	
Photolysis (aqueous)	TEA: River water 1.7 days in natural light 0.7 days in artificial light	U.S. EPA/OPP 2009a, Appendix P, MRIDs 41732201 and 42411804	
Photolysis (aqueous)	Acid: 0.71-1.86 days	Woodburn et al. 1993a	
Photolysis (aqueous)	TCP: 1 day (as conservative estimate). <2 hours based on MRID 00095241 as summarized in Knuteson (1999).	Knuteson 1999	

Table 1: Physical and Chemical Properties Triclopyr (various forms) and TCP				
Item	Value			Reference
Photolysis (aqueous)	TCP: 2 hours (0.08 day)			Cessna et al. 2002
Specific gravity	1.85 (21 °C)			Tomlin 2004
Vapor pressure	0.2 mPa (25 °C)			Tomlin 2004
	torr			U.S. EPA/OPP 2009a, Tables 2-1 and 3-2
	Form	VP (torr)	Source	U.S. EPA/OPP 2009a (Table 3-2 for acid; Appendix P for TEA and BEE)
	Acid	1.26 x 10 ⁻⁶	http://toxnet.nlm.nih.gov	
	BEE	3.6x10 ⁻⁶	MRIDs 40557003; 42443402	
	TEA	<1 x 10 ⁻⁸	MRID 41219104	
Water solubility	Acid: 7,690 mg/L (pH 5, 20 °C) 8,100 mg/L (pH 7, 20 °C) 8,220 mg/L (pH 9, 20 °C)			Tomlin 2004
	Acid: 440 mg/L			U.S. EPA/OPP 2009a, Tables 2-1 and 3-2.
	BEE: 7.4 mg/L at 25 °C			U.S. EPA/OPP 2009a, Appendix P
	BEE: 6.8 mg/L			U.S. EPA/OPP 1998a
	BEE: 8.6 mg/L			Milazzo and Batchelder 1981, MRID 151965.
	TEA 12,000 mg/L (pH 5, 25 °C) 412,000 mg/L (pH 7, 25 °C) 1,278,000 mg/L (pH 9, 25 °C)			U.S. EPA/OPP 2009a, Appendix P
	TEA: 2,100,000 mg/L BEE: 23 mg/L			Knisel and Davis 2000
	TCP: 49,100 mg/L at pH 7			Knuteson 1999
Environmental Fate Properties				
Foliar washoff fraction	TEA: 0.95 BEE: 0.70			Knisel and Davis 2000
	BEE 0.62: immediately after drying 0.11 to 0.17: after two days			Michael et al. 1992
Foliar half-life	TEA: 15 days BEE: 15 days			Knisel and Davis 2000
	Acid: 2.6 to 5.7 days BEE: 1.1 to 1.4 days			Thompson et al. 1994
	TEA: 1 to 3 days			McCormick and Robb 2000, MRID 45249901
	TCP: 10 days			Ganapathy 1997
	Average 42% decline over 6 days of triclopyr applied to various forest vegetation in northern Idaho – i.e., half-life of ≈7.6 days.			Whisenant and McArthur 1989
Fruit half-life	≈27 (16.5-73) days on cowberry. See Section 3.2.3.7 for discussion.			Siltanen et al. 1981
Kd/Koc	Kd: 0.08 to 0.61; Koc: 11.4 to 84			Johnson et al. 1995a
	Kd: 0.33 to 38.12 increasing with increasing humic acid content and decreasing pH.			Pusino et al. 1994

Table 1: Physical and Chemical Properties Triclopyr (various forms) and TCP				
Item	Value			Reference
Kd/Koc (continued)	Triclopyr Acid			U.S. EPA/OPP 2009a, Table 2-1 MRID 40749801
	Soil (%OC)	Kd	Koc	
	Sand (0.73%)	0.975	134	
	Sandy loam (2.25%)	0.571	25	
	Silty loam (0.67%)	0.165	25	
	Clay loam (1.38%)	0.733	53	
	Average:		59.25	
	Acid: 59 (25 to 134)			Cessna et al. 2002
	Acid: 14 and 66			Bernard et al. 2005
	Acid: 33.4 to 49.3			Raturi et al. 2005
	Acid: 59.25 [average for PRZM/EXAMS input]			U.S. EPA/OPP 2009a, Table 3-2
	BEE: 1038			Michael et al. 1996
	BEE: 1233 (640 to 1650)			Cessna et al. 2002
	TEA: 20 BEE: 780			Knisel and Davis 2000, Michael et al. 1996
	NOS: 20			Diaz-Diaz and Loague 2001
	NOS: 27			Kenaga 1980
	TCP: 14 to 86			Antunes-Kenyon and Kennedy 2004
	TCP: 149 (81 to 242) 4 soils			Cessna et al. 2002
	TCP: 151(136 and 168) (151 median of two assays)			Knuteson 1999
Soil half-life (NOS)	TEA: 46 days; BEE: 46 days (value for BEE appears to reflect degradation of triclopyr moiety).			Knisel and Davis 2000
	NOS: 1.4 and 3.9 days (dissipation)			Bernard et al. 2005
	TEA: 10 days			Deubert and Corte-Real 1986
	NOS: 40 days			McCall and Gavit 1986
	NOS: 45 days (average)			Neary et al. 1993
Soil half-life, aerobic	BEE: MRID 43799101 0.9 hours Hanford Sandy Loam Soil at 25° C 1.4 hours Commerce Silt Loam Soil at 25° C TEA: MRID 43837501 5.6 days Sandy Loam Soil at 25° C 13.7 days Silt Loam Soil at 25° C			U.S. EPA/OPP 2009a, Appendix P BEE study appears to be Yoder 2007.
	Acid: 8 days in silty clay loam soil at 25 °C TCP (26.4%) Acid: 18 days in silt loam soil at 25 °C			U.S. EPA/OPP 2009a, Table 2-1
	Acid: 42 and 130 days			Houtman et al. 1997c
	TCP: 69 (40 to 95) days [15 studies]			Knuteson 1999
	Acid: 28.39 days (for PRZM/EXAMS input)			U.S. EPA/OPP 2009a, Table 3-2, MRID 40346304.
Field dissipation half-life, terrestrial	Forest floor: About 39 to 60 days. Dissipation was not first-order.			Thompson et al. 2000
	BEE: MRID 43837503 1.1 days (degraded to triclopyr acid; depth 0 to 7.5 cm) 10.6 days (dissipation of BEE and triclopyr acid; depth 0-7.5 cm) MRID 42730601 ~2 weeks (dissipation of BEE and triclopyr acid; depth 0 to 6 inches)			U.S. EPA/OPP 2009a, Appendix P

Table 1: Physical and Chemical Properties Triclopyr (various forms) and TCP		
Item	Value	Reference
	TEA: 1.1 days (BEE degraded to triclopyr acid; depth 0 to 7.5 cm) 10.6 days (dissipation of BEE and triclopyr acid; depth 0- 7.5 cm)	U.S. EPA/OPP 2009a, Appendix P, MRID 43837503
	Acid: MRID 43955901 and 43033401 7.6 to 10.6 days)	U.S. EPA/OPP 2009a, Table 2-1
	BEE: 10 days (turf) and 100 days (soil)	Wilcock et al. 1991
Water half-times (NOS)	Acid: 2.8 to 14.1 hours BEE: 16.7 to 83.4 hours	McCall and Gavit 1986
Water, aerobic metabolic half-times	2-butanol: 0.6-3.4 days at 25 °C, MRID 43799106 Triethylamine: 14-18 days, MRID 43837503	U.S. EPA/OPP 2009a, Appendix P
	Acid: 142 days in silty clay soil at 24-26° C, TCP (< 5%) MRID 40479101	U.S. EPA/OPP 2009a, Table 2-1; Houtman et al. 1997c
	Acid: 426 days [for PRZM/EXAMS input, single value x 3] MRID 40479101	U.S. EPA/OPP 2009a, Table 3-2
	TCP: 6 days	Knuteson 1999
	TCP: 4 to 10 days	Petty et al. 2003
Water, anaerobic metabolic half-times	2-butanol: 1.4 days, MRID 43799103 Triethylamine: 2 years, MRID 43837502	U.S. EPA/OPP 2009a, Appendix P
Water, anaerobic metabolic half-times	Acid: Stable (1300 days), TCP (26%), MRID 151967	U.S. EPA/OPP 2009a, Tables 2-1 and 3-2.
Water, field dissipation half-time	6 days in lake following aquatic application	Fox et al. 2002.
	19.4 (14.9-26.4) hours (river in Iowa)	Getsinger et al. 1996
	3.7 to 4.7 days (lake water) 5.4 days (sediment)	Getsinger et al. 1997
	TEA: 0.5 to 3.4 days (lake water)	Green et al. 1989
	Acid: 0.8 to 7.5 days TCP: 0.5 to 10 days	Houtman et al. 1997c
	Triclopyr: 5.9 to 7.5 days (pond) TCP: 4 to 8.8 days TMP: 4 to 10 days	Petty et al. 2001.
	TCP: 3.8 to 13.3 days (sediment)	Petty et al. 2003
	BEE: 3.8-4.3 days	Solomon et al. 1988
	TEA: 6.9 hours or 0.29 days (river)	Turner et al. 1994
	TEA: 0.5 to 3.5 days, MRID 41714304 and 42821301	U.S. EPA/OPP 2009a, Appendix P
	Acid at 30 °C Lake, 3.6 days Aquatic Plants: 3.4 days Crayfish: 11.5 days Clam Tissue: 1.5 days	U.S. EPA/OPP 2009a, Table 2-1, MRID 41714304
	T-BEE: 0.6 day (Site A) and 1 day (Site B). Triclopyr: 9 days (Site A) and 27 days (Site B)	Wojtaszek et al. 2005
	Triclopyr: 0.5-3.6 days TCP: < 1 day	Woodburn et al. 1993b

^[1] All values apply to triclopyr acid unless otherwise specified.

Table 2: Chemical Properties Used Quantitatively in Risk Assessment

Parameter ^[Note 1]	Triclopyr BEE	Triclopyr Acid	TCP	Note/ Reference
Halftimes (days)				
Foliar	4.1 (1.1 – 15)	6.2 (2.6 – 15)	6.2 (2.6 – 15)	Note 2
Fruit	26.9 (16.5-73)	26.9 (16.5-73)	26.9 (16.5-73)	Note 2
Soil	0.2	14 (8 - 28.4)	69 (40 - 95)	Note 3
Water	0.5	426	6	Note 4
Soil K _{o/c} , mL/g	1233 (640 - 1650)	59 (25 to 134)	149 (81 to 242)	Note 5
K _{ow} , unitless	20,000	0.35	1000	Note 6
Water Solubility, mg/L	7.4	440	49,100	Note 7
Molecular weight	356.63	256.47	198.43	
Note 1	These as well as some additional parameters used in Gleams-Driver modeling are summarized in Table 21.			
Note 2	Foliar: For BEE and acid, lower bound is from Thompson and upper bound is from Knisel and Davis (2000). Little data describing the kinetics of TCP in vegetation have been identified. Value for TCP taken as identical to that for triclopyr acid. The central estimate is taken as the geometric mean of the lower and upper bounds. Fruit: Triclopyr, 26.9 (16.5 to 73.1) days from the study in fruit on triclopyr is Siltanen et al. (1981). See Section 3.2.37 for discussion.			
Note 3	Acid from U.S. EPA/OPP (2009a). BEE conservatively set to 0.2. Much more rapid dissipation is reported in U.S. EPA/OPP (2009a). Values for TCP from Knuteson 1999.			
Note 4	BEE: 0.5 days for hydrolysis of T-BEE to acid (U.S. EPA/OPP 2009a, Appendix P, MRID 134174 and McCall et al. 1988); Acid: 426 days (U.S. EPA/OPP 2009a, Table 3-2, PRZM/EXAMS input; TCP: lumped aquatic degradation from Knuteson 1999). This appears to be very conservative given the rapid aquatic photolysis of TCP. See Table 1 of this risk assessment. For aquatic applications, the aquatic field dissipation halftimes of 0.5 to 4 days are used based on MRID 41714304 and 42821301 as reviewed by U.S. EPA/OPP (2009a). See Section 3.2.3.4.5.2 for discussion.			
Note 5	BEE: Cessna et al. 2002. This is consistent with Knisel and Davis (2000). Acid: Cessna et al. 2002. Central estimate is consistent with U.S. EPA/OPP (2009a) PRZM/EXAMS input.			
Note 6	Acid: From Tomlin 2004 for pH 7; BEE and TCP: Taken from U.S. EPA/OPPTS 2011. Reported values for BEE (see Table 1) are variable. The Kow of 20,000 is the geometric mean of the range given on the MSDS for Garlon 4 (rounded to one significant place) and is close to values from Dow cited by Ganapathy (1997) and QSAR estimates from U.S. EPA/OPPTS 2011.			
Note 7	BEE: U.S. EPA/OPP (2009a, Appendix P); Acid: U.S. EPA/OPP (2009a, Tables 2-1 and 3-2); TCP: Knuteson (1999).			

Table 3: Triclopyr Formulations Explicitly Considered in Risk Assessment

Formulation Name ^[1,2]	Supplier	EPA Reg. No.	lb a.e./gal	a.i.	% a.i.	% a.e.	Other
Forestry Garlon	Dow AgroSciences	62719-40	4	BEE	61.6%	44.3%	
Forestry Garlon XRT	Dow AgroSciences	62719-553	6.3	BEE	83.9%	60.3%	
Garlon 3A	Dow AgroSciences	62719-37	3	TEA	44.4%	31.8%	Aquatic, 1.135 g/mL
Garlon 4	Dow AgroSciences	62719-40	4	BEE	61.6%	44.3%	
Garlon 4 Ultra	Dow AgroSciences	62719-527	4	BEE	60.45%	43.46%	
Pathfinder II	Dow AgroSciences	62719-176	0.75	BEE	13.6%	9.81%	
Remedy	Dow AgroSciences	62719-70	4	BEE	61.6%	44.3%	
Remedy RTU	Dow AgroSciences	62719-176	0.75	BEE	13.6%	9.81%	
Remedy Ultra	Dow AgroSciences	62719-552	4	BEE	60.45%	43.46%	
Renovate 3	SePRO	62719-37-67690	3	TEA	44.4%	31.8%	Aquatic, 1.135 g/mL
Renovate OTF granular	SePRO	67690-42	N/A	TEA	14%	10%	Aquatic
Tahoe 3A	Riverdale	228-384	3	TEA	44.4%	31.8%	
Tahoe 4E	Riverdale	228-385	4	BEE	61.6%	44.3%	
Triclopyr 3A	Albaugh	42750-127	3	TEA	44.4%	31.8%	Aquatic, 1.1-1.17 g/mL
Triclopyr 3SL	Makhteshim Agan	66222-152	3	TEA	44.4%	31.8%	Aquatic, 1.14 g/mL
Triclopyr 4 Ester R&P	Micro Flo	51036-377	4	BEE	61.6%	44.3%	
Triclopyr 4E	Albaugh	42750-126	4	BEE	61.6%	44.3%	
Triclopyr R&P	Albaugh	42750-129	4	BEE	61.6%	44.3%	
Triquad	Makhteshim Agan	66222-153	4	BEE	61.6%	44.3%	

^[1] Sources: Specimen labels from www.Greenbook.net and www.CDMS.net.

^[2] Formulations that do not appear to have forestry or related uses (e.g., Grandstand R, Triclopyr Rice, Truflon Ester, and Truflon Ester Ultra) are not included in the above table.

^[3] Specific gravity (g) is given for aquatic formulations. This is used only in WorksheetMaker and is converted for lb/gal [1 g/mL = 8.345 lb/gal].

Table 4: Disclosed Inerts in Triclopyr Formulations

Formulation Name ^[1]	% a.i.	Inert (CAS No. if specified)	Amount
BEE Ester			
Forestry Garlon	61.6%	Kerosene (8008-20-6) NOS	31.0% 7.4%
Forestry Garlon XRT	83.8%	NOS	16.1%
Garlon 4	61.6%	Kerosene (8008-20-6) Ethylene glycol monobutyl ether (111-76-2) Solvent naphtha (petroleum), light aromatic NOS	≥18.6% to ≤ 31% 0.5% 0.2% ≥6.7% to ≤ 19.1%
Garlon 4 Ultra	60.5%	Ethylene glycol monobutyl ether (111-76-2) NOS	0.5% 39.0%
Pathfinder II	13.8%	NOS	86.2%
Remedy	61.6%	Kerosene (8008-20-6) NOS	31% 7%
Remedy RTU	13.6%	Other (including proprietary solvent)	86.4%
Remedy Ultra	60.5%	NOS	39.5%
Tahoe 4E	61.6%	Other (including kerosene and proprietary surfactant)	38.4%
Triclopyr 4 Ester R&P	61.6%	Kerosene (8008-20-6)	>25%
Triclopyr 4E	61.6%	Kerosene (8008-20-6)	>25%
Triclopyr R&P	61.6%	Kerosene (8008-20-6)	>25%
Triquad	61.6%	Kerosene (8008-20-6)	<27.14%
TEA Salt			
Garlon 3A	44.4%	Ethanol (64-17-5) NOS	2.1% 50.5%
Renovate 3	44.4%	Ethanol (64-17-5) NOS	2.1% 50.5%
Renovate OTF (granular)	10 to 30%	Proprietary Fiber Proprietary Clay Proprietary Salt Titanium dioxide (13463-67-7)	30 to 60% 5 to 10% 5 to 10% 0.1 to 1%
Tahoe 3A	44.4%	Other (including ethanol)	55.6%
Triclopyr 3A	44.4%	Ethylenediaminetetraacetic acid [EDTA] (64-02-8) ^[2]	<5.0%
Triclopyr 3SL	44.4%	Ethylenediaminetetraacetic acid [EDTA] (60-00-4) ^[2] Ethylene glycol (107-21-1)	2.5% 1.0%

^[1] Sources: MSDSs from www.Greenbook.net and other sites.

^[2] The CAS No. for EDTA is 60-00-4 (anion). CAS No. 64-02-8 designates the tetrasodium salt of EDTA.

Table 5: Overview of Label Directions for Terrestrial Applications

Formulation(s)	Application Rates and Volumes	Adjuvants
Note: This table presents a cursory overview of label directions. In any specific application, consult and follow the product label for the formulation that is being used.		
BEE		
4 lb a.e./gallon: Forestry Garlon, Garlon 4 Ultra, Triclopyr 4E	1 to 8 lb a.e./acre Max Rate (forestry sites): 6 lb a.e./ac per season. Max Rate (non-grazing): 8 lb a.e./acre. Max Rate (grazing sites): 2 lb a.e./acre Spray Vol.: 10 to 400 gallons/acre	If agricultural surfactant is used, apply the surfactant at rates of 1 to 2 lb/acre. For basal bark applications, mix with diesel fuel, No. 1 or No. 2 fuel oil, kerosene or a commercially available basal oil. Aerial applications: Helicopter only.
4 lb a.e./gallon: Garlon 4, Remedy Ultra, Tahoe 4E, Triclopyr R&P, Triquad	Same as above	As above with the exceptions noted below. Aerial applications: Fixed wing aircraft or helicopter.
4 lb a.e./gallon: Remedy, Triclopyr 4 Ester	Max Rate (grazing): Remedy: 2 lb a.e./acre Triclopyr 4: 1 lb a.e./acre Max Rate: 8 lb a.e./acre (non-grazing) Ground: 10 to 40 gal/acre [The label for Triclopyr 4 Ester recommends 15 to 25 gal/acre] Aerial: >2 gal/acre	Mix with basal oil, diesel fuel, fuel oil, or kerosene plus an emulsifier such as Sponto 712 or Triton X-100. Ground: Use 5-10% oil mix. Aerial: Fixed wing aircraft or helicopter. Use 20% oil/80% water.
6.3 lb a.e./gallon: Forestry Garlon XRT	1 to 8 lb a.e./acre Max Rate (forestry sites): 6 lb a.e./ac per season. Max Rate (ROW): 8 lb a.e./acre. Max Rate (grazing sites): 2 lb a.e./acre Spray Vol.: 10 to 400 gallons/acre	If agricultural surfactant is used, apply the surfactant at rates of 1 to 2 lb/acre. Aerial applications: Helicopter only.
0.75 lb a.e./gallon: Pathfinder II, Remedy RTU	Same as above.	Application methods: Basal bark, cut stump, streamline basal bark (Southern U.S.). Not intended for broadcast applications.
TEA Salt, Terrestrial		
3 lb a.e./gallon: Garlon 3A, Renovate 3, Tahoe 3A, Triclopyr 3A, Triclopyr 3SL	1 to 8 lb a.e./acre Max Rate (forestry sites): 6 lb a.e./ac per season. Max Rate (grazing sites): 2 lb a.e./acre per season Max Rate (other): 9 lb a.e./acre per season 10 to 400 gallons/acre	The use of non-ionic surfactants is recommended for most applications. Aerial applications: Helicopter only (for forestry applications). Fixed wing aircraft may be used on rice.

Table 6: Overview of Label Directions for Aquatic Applications

Formulation(s)	Application Rates and Volumes	Adjuvants
<p>Note: This table presents a cursory overview of label directions. In any specific application, consult and follow the product label for the formulation that is being used.</p>		
<p>3 lb a.e./gallon: Garlon 3A, Triclopyr 3A</p>	<p>Follow directions for forestry and non-cropland sites. Emergent weeds only.</p>	<p>The use of nonionic surfactants is recommended for most applications. Appears to be labeled only for emergent weeds.</p>
<p>3 lb a.e./gallon: Renovate 3, Triclopyr 3SL</p>	<p>Emergent weeds: 0.5 to 6 lb a.e./acre Max Rate: 6 lb a.e./acre per season. Ground: 20 to 200 gallons/acre Aerial: ≥ 10 gal/acre Submerged weeds: 0.75 to 2.5 mg a.e./L Max Rate: 2.5 mg a.e./L per season</p>	<p>Nonionic surfactant recommended for most applications.</p>
<p>Granular, 10% a.e. Renovate OTF</p>	<p>1 to 2.5 mg a.e./L (floating and emersed) 0.5 to 2.5 mg a.e./L (submersed)</p>	<p>Not labeled for terrestrial applications. The use of surfactants is not included in the label directions.</p>

Table 7: Forest Service Use by Region for 2004

Region	Acres	Pounds	Average lbs/acre	Proportion of Total Acres	Proportion of Total Pounds
R1 (Northern)	363	424	0.86	0.034	0.030
R2 (Rocky Mountain)	128	155	0.83	0.012	0.011
R3 (Southwestern)	0	0	N/A	0	0
R4 (Intermountain)	637	546	1.17	0.044	0.053
R5 (Pacific Southwest)	45.3	74.6	0.61	0.006	0.004
R6 (Pacific Northwest)	442	1015	0.44	0.081	0.037
R8 (Southern)	10,410	10,302	1.01	0.823	0.866
R9 (Eastern)	0	0	N/A	0	0
R10 (Alaska)	0	0	N/A	0	0
Total	12,027	12,516	0.96		

Table 8: Forest Service Use by Management Objective for 2004

Objective	Acres	Pounds	Average lbs/acre	Acres, Proportion of Total^[1]	Pounds, Proportion of Total^[1]
Release: Conifer	3869.3	4447.0	0.87	0.36	0.32
Noxious weeds	3275.5	3327.8	0.98	0.27	0.27
Site preparation	2196.8	2017.0	1.09	0.16	0.18
Release: Hardwood & Conifer	1432.6	1403.0	1.02	0.11	0.12
Release: Hardwood	661.6	962.0	0.69	0.077	0.055
Rights-of-way	483.0	251.0	1.92	0.020	0.04
Recreation Improvement	64.0	89.8	0.71	0.0072	0.0053
Wildlife Habitat Improvement	32.0	8.0	4.00	0.0006	0.0027
Facilities maintenance	5.8	4.6	1.27	0.0004	0.0005
Nursery weeds	3.4	4.7	0.72	0.0004	0.0003
Aquatic weeds	3.0	1.7	1.76	0.0001	0.0002
Total:	12,027.0	12,516.5	0.96		

^[1]Note: Due to rounding, the proportion of total acres sum to 1.0057 and the proportion of total pounds sums to 0.994.

Table 9: Dermal Absorption of Triclopyr BEE in Human Volunteers

Experimental Data ^a			
Subject Number	Estimated Absorbed Dose	Estimated k_a	Square of Error
1	2.21%	0.002793	0.000000494
2	0.95%	0.001193	0.000000804
3	1.38%	0.001737	0.000000124
4	3.10%	0.003936	0.000003408
5	0.63%	0.000790	0.000001689
Average		0.002089800	
SSE		0.000006519	
Sample Standard Deviation		0.001276617	
Critical Value of <i>t</i> at 0.025		2.776	
Value of 2.5% Lower Bound		0.0005049248	
Value of 97.5% Upper Bound		0.0036746752	
Triclopyr BEE First-order Dermal Absorption Rate (95% CI) from Study		2.1x10 ⁻³ 5.0x10 ⁻⁴ – 3.7x10 ⁻³	hour ⁻¹
Estimated First-order Dermal Absorption Rates From Structure Activity Relationships ^b			
Triclopyr BEE		3.1x10 ⁻³ 1.2x10 ⁻³ – 8.1x10 ⁻³	hour ⁻¹

^a Data from Carmichael et al. (1989), Table 2, p. 435.

^b Worksheet B06 in Attachment 2 to the current risk assessment.

See Section 3.1.3.2.1 for discussion.

Table 10: Developmental and Reproduction Studies on Triclopyr acid, TEA, and BEE

Effect and No-Effect Doses (mg/kg bw/day)				Reference
Dams		Offspring		
NOAEL	LOAEL	NOAEL	LOAEL	
Rats				
Acid				
	50	100	200	Breslin 1990a
	50	100	200	Hanley et al. 1983
TEA				
	50	100	200	Thompson et al. 1979
22	72	72	216	Carney et al. 2007
30	100	100	300	Breslin et al. 1996
100	300	100	300	Bryson 1994b
BEE				
	22	72	216	Carney et al. 2007, Study I
3.6	22	216		Carney et al. 2007, Study II
5	30	100	300	Breslin et al. 1996
	30	30	100	Jones 1995, Phase I
5	30	100	300	Jones 1995, Phase II
Rabbits				
Acid				
10	25	25		Hanley et al. 1983
	25	100		Smith et al. 1960
25	75	75		Kirk et al. 1989
TEA				
10	30	100		Breslin and Billington 1995
30	100	30	100	Bryson 1994c
BEE				
30	100	30	100	Breslin and Billington 1995
30	100	30	100	Bryson 1994a

^[1] TEA = triethylamine salt of triclopyr. BEE= butoxyethyl ester of triclopyr. See Appendix 1, Table 7 for details. Black cells indicate that a NOAEL or LOAEL was not determined.

See Section 3.1.9.1 for discussion.
See Appendix 2, Table 7 for additional details.

Table 11: Comparison of Worker Exposure Rates

Worker Group	Rate (mg/kg bw/day per lb applied)			Reference
	Central	Lower	Upper	
Standard Rates				
Directed foliar	0.003	0.0003	0.010	SERA 2007a
Broadcast foliar	0.0002	0.00001	0.0009	SERA 2007a
Aerial	0.00003	0.000001	0.0001	SERA 2007a
Triclopyr Studies with Individual Exposure Rates for Backpack Workers				
Basal stem	0.00124	0.00015	0.010	Middendorf 1992a ^[1]
Directed Foliar	0.0058	0.00086	0.039	Middendorf 1992b ^[2]
Release	0.015	0.0042	0.052	Spenser et al. 2000 ^[3]
Foliar	0.0215			Krieger et al. 2005

^[1] Excluding workers H and I (no gloves). See Table 12 for additional details.

^[1] See Table 13 for additional details.

^[3] See Table 14 for additional details.

^[4] Central estimate of average absorbed dose of 0.043 mg/kg bw taken from p.1 of Krieger et al. 2005. Amount handled of 2 lb/day from Table 11, p. 31 of Krieger et al. 2005.

See Section 3.2.2.1. for discussion.

Table 12: Worker Exposure Rates Basal Stem Applications from Middendorf (1992a).

Worker	Amount Handled (lb a.e.) ^a	Body Weight (kg) ^b	Amount Absorbed (mg) ^b	Dose (mg/kg bw)	Exposure Rate (mg/kg bw per lb)
Site 1 (all wore gloves)					
A ^c	4.8	91.2	0.065	0.000713	0.00015
B	4.8	83.3	0.259	0.003109	0.00065
C	4.8	93.2	0.697	0.007479	0.00156
D	4.8	78.3	1.902	0.024291	0.00506
Geometric mean:				0.004479	0.0009
Site 2					
G ^c	4	103	0.561	0.005447	0.00136
H (no gloves)	4	71.9	4.108	0.057135	0.01428
I (no gloves)	4	63.8	3.001	0.047038	0.01176
J (no gloves)	4	85.1	0.831	0.009765	0.00244
K (no gloves)	4	61.5	0.921	0.014976	0.00374
L	4	74.2	1.152	0.015526	0.00388
Geometric mean:				0.017931	0.0045
Site 3					
M ^c (no gloves) ^c	5.6	93.2	1.143	0.012264	0.00219
N (no gloves) ^c	5.6	90.5	2.006	0.022166	0.00396
O	5.6	71.9	1.039	0.014451	0.00258
P	5.6	71.9	0.745	0.010362	0.00185
Q	5.6	91.9	0.647	0.00704	0.00126
R	5.6	105	0.207	0.001971	0.00035
Geometric mean:				0.009092	0.0016
All Workers Combined					
Geometric mean:				0.0098	0.0021
95% Bounds for Observations ^d :				0.0011 to 0.089 (<i>p</i> =0.93)	0.00021 to 0.021 (<i>p</i> =0.87)
Workers with Gloves					
Geometric mean:				0.0061	0.00124
95% Bounds for Observations ^d :				0.00075 to 0.049 (<i>p</i> =0.96)	0.00015 to 0.010 (<i>p</i> =0.79)
Workers without Gloves					
Geometric mean:				0.021	0.0049
95% Bounds for Observations ^d :				0.0052 to 0.09 (<i>p</i> =0.97)	0.0010 to 0.023 (<i>p</i> =0.78)

^a Middendorf (1992a), Table 2, pp. 7-8, Average weight for all workers=83.1 lbs.

^b Middendorf (1992a), Table 3, p. 25

^c Mixer

^d Based on StatGraphics fit to log-normal distribution with *p*-value for Kolmogorov-Smirnov test.

^e Gloves worn during mixing but not during application.

See Section 3.2.2.1. for discussion.

Table 13: Worker Exposure Rates for Foliar Applications from Middendorf (1992b)

Site	Volunteer	Estimated Dose (µg/worker) ^[1]	Amount Applied (lb) ^[2]	Estimated Dose mg/kg bw ^[3]	Exposure Rate (mg/kg bw per lb handled)
Site 1	WT	741	2.1	0.00892	0.004246
	MH	440	2.1	0.00529	0.002521
	JF	183	2.1	0.00220	0.001049
	JDA	676	2.1	0.00813	0.003874
	CWH	1459	2.1	0.01756	0.008361
Geometric Mean for site:				0.00683	0.003252
Site 2	MEC	812	2.2	0.00977	0.004442
	TRH	892	2.2	0.01073	0.004879
	RVA	778	2.2	0.00936	0.004256
	LJK	6693	2.2	0.08054	0.036610
	WHS	374	2.2	0.00450	0.002046
	SGG	2106	2.2	0.02534	0.011520
Geometric Mean for site:				0.01443	0.006559
Site 3	NM [no gloves]	7690	1.4	0.09254	0.066099
	RR	2681	1.4	0.03226	0.023045
	RH [no gloves]	1207	1.4	0.01452	0.010375
	MD	900	1.4	0.01083	0.007736
	JJ [no gloves] ^[5]	6903	1.4	0.08307	0.059335
Geometric Mean for site:				0.03298	0.023557
Site 4	G	603	1.2	0.00726	0.006047
	H	1549	1.35	0.01864	0.013808
	I	1720	1.2	0.02070	0.017248
	J	267	1.47	0.00321	0.002186
	K	474	1.62	0.00570	0.003521
	L	3737	1.41	0.04497	0.031894
Geometric Mean for site:				0.01150	0.008409
All Sites Combined					
Geometric Mean:				0.0138	0.0080
95% Bounds for Observations ^[4] :				0.0018 to 0.10 (<i>p</i> =0.80)	0.00088 to 0.073 (<i>p</i> =0.88)
Excluding Site 3					
Geometric Mean:				0.0107	0.0058
95% Bounds for Observations ^[4] :				0.0017 to 0.066 (<i>p</i> =0.87)	0.00086 to 0.039 (<i>p</i> =0.77)

^[1] From Middendorf (1992b), Table 7, p. 52.

^[2] From Middendorf (1992b), Table 2, pp. 9-10.

^[3] Using the average body weight of 83.1 lbs from Middendorf (1992a). See Table 12 for individual data.

^[4] Based on StatGraphics fit to log-normal distribution with *p*-value for Kolmogorov-Smirnov test.

^[5] Gloves worn during mixing but not during application.

See Section 3.2.2.1 for discussion.

Table 14: Worker Exposure Rates from Spenser et al. (2000)

Worker	Amount Handled (lb) ^[1]	Body Weight (kg) ^[2]	Amount Excreted in Urine (mg) ^[2]	Estimated Dose (mg/kg bw)	Exposure Rate (mg/kg bw per lb handled)
Day 1 (7/10/95)					
1	3.12	85	5.75	0.06765	0.02168
2	3	75	1.96	0.02613	0.00871
3	3.12	63.6	3.53	0.05550	0.01779
4	3.37	77.3	3.12	0.04036	0.01198
5	3.37	79.5	3.57	0.04491	0.01333
6	3.37	75	1.12	0.01493	0.00443
7	3.25	61.4	0.81	0.01319	0.00391
8	3.25	75	9.45	0.12600	0.03877
9	3.5	72.7	4.12	0.05667	0.01619
10	3.25	58.2	2.86	0.04914	0.01512
Day 2 (7/11/95)					
1	3.67	85	6.16	0.07247	0.01975
2	2.66	75	3.89	0.05187	0.01950
3	3.67	63.6	8.81	0.13852	0.03774
4	2.91	77.3	3.81	0.04929	0.01694
5	2.91	79.5	2.49	0.03132	0.01076
6	2.91	75	1.57	0.02093	0.00719
7	3.42	61.4	2.70	0.04397	0.01286
8	3.54	75	11.05	0.14733	0.04162
9	3.16	72.7	2.65	0.03645	0.01153
10	3.16	58.2	4.66	0.08007	0.02534
Both Days Combined					
Geometric mean:				0.048	0.015
95% Bounds for Observations ^[3] :				(0.013 to 0.175) <i>p</i> =0.98	(0.0042 to 0.052) <i>p</i> =0.97
^[1] Table VI in Spenser et al. 2000, p. 21. ^[2] Table IV in Spenser et al. 2000, p. 17. Average = 72.3 kg. ^[3] Table IX in Spenser et al. 2000, p. 25. ^[4] Based on StatGraphics fit to log-normal distribution with <i>p</i> -value for Kolmogorov-Smirnov test.					

See Section 3.2.2.1. for discussion.

Table 15: Comparisons of Estimated Daily Absorbed Doses for Workers

Worker Group	Estimated Absorbed Dose (mg/kg bw/day)			Reference
	Central	Lower	Upper	
	Standard FS Estimated Daily Doses (mg/kg bw/day)			
Directed foliar	0.013	0.00045	0.08	Attach. 2, WS E02
Broadcast foliar	0.022	0.00066	0.15	Attach. 2, WS E02
Aerial	0.015	0.00024	0.08	Attach. 2, WS E02
Estimated Daily Doses from Triclopyr BEE Studies				
Backpack, basal	0.0061	0.00075	0.049	Middendorf 1992a ^[2]
Backpack, foliar	0.0107	0.0017	0.066	Middendorf 1992b ^[3]
Backpack, release	0.048	0.013	0.175	Spenser et al. 2000 ^[4]
Backpack, NOS	0.043			Krieger et al. 2005 ^[5]
Backpack, cut stump	0.115	0.024	0.552	Gosselin et al. 2005 ^[6]
Boom spray, foliar	0.200	0.103	0.339	Gosselin et al. 2005 ^[6]
U.S. EPA/OPP Estimated Daily Doses (mg/kg bw/day)				
Directed foliar	0.0074	0.0014	0.030	WS PHEDBkPk ^[1]
Broadcast Foliar	0.0020	0.00089	0.0049	WS PHEDBoom ^[1]
Aerial broadcast	0.0010	0.00034	0.0031	WS PHEDAerial ^[1]

^[1] These are custom worksheets in Attachment 2 (Triclopyr BEE) that follow Worksheet C01c.

^[2] Basal stem applications censoring workers without gloves. See Table 12 for additional details.

^[3] Directed foliar backpack applications, excluding Site 3. See Table 13 for additional details.

^[4] Directed foliar backpack applications, See Table 14 for additional details.

^[5] Central estimate taken from p. 13 and ranges taken from Table 13 of Krieger et al. 2005.

^[6] See Table 16 for additional details.

See Section 3.2.2.1. for discussion.

Table 16: Doses in Backpack and Boom Spray Workers (Gosselin et al. 2005)

Worker	Body Weight (kg)^[1]	Mean Absorbed Dose (mg)^[2]	Mean Dose mg/kg bw	95% Bounds on Absorbed Dose (mg/kg bw)
Backpack Workers				
1	77	7.77	0.101	
2	77	6.87	0.089	
3	68	12.13	0.178	
4	81	2.99	0.037	
5	75	37.18	0.496	
6	73	3.89	0.053	
7	75	8.7	0.116	
8	91	15.87	0.174	
Geometric Mean ^[3] :			0.115	0.024 to 0.552 <i>p</i> =0.98
Boom Spray Workers				
9	73	11.49	0.157	
10	66	16.78	0.254	
Geometric Mean ^[3] :			0.200	0.103 to 0.339 <i>p</i> =0.999

^[1]From Gosselin et al. 2005, Table 2.

^[2]From Gosselin et al. 2005, Table 5.

^[3] Based on StatGraphics fit to log-normal distribution with *p*-value for Kolmogorov-Smirnov test.
See Section 3.2.2.1. for discussion.

Table 17: Summary of PHED Exposure Rates

Scenario	mg/lb a.i. handled ^[1]			
	No clothing	Single Layer, No gloves	Single layer, Gloves	Inhalation
1. Dry flowable, open mixing and loading	1.1	0.066	0.066	0.00077
2. Granular, open mixing and loading	0.032	0.0084	0.0069	0.0017
3. All liquids, open mixing and loading	3.1	2.9	0.023	0.0012
4. Wettable powder, open mixing and loading	6.7	3.7	0.17	0.04342
5. Wettable powder, water soluble bags	0.039	0.021	0.0098	0.00024
6. All liquids, closed mixing and loading			0.0086	0.000083
7. Aerial-fixed wing, enclosed cockpit/liquid	0.0050	0.0050	0.0022	0.000068
8. Aerial-fixed wing, enclosed cockpit/granular	0.0044	0.0017	0.0017	0.0013
9. Helicopter application, enclosed cockpit		0.0019	0.0019	0.0000018
10. Aerosol application	480	190	81	1.3
11. Airblast application, open cockpit	2.2	0.36	0.24	0.0045
12. Airblast application, enclosed cockpit			0.019	0.00045
13. Groundboom applications, open cab	0.046	0.014	0.014	0.00074
14. Groundboom applications, enclosed cab	0.010	0.0050	0.0051	0.000043
15. Solid broadcast spreader, open cab, AG	0.039	0.0099		0.0012
16. Solid broadcast spreader, enclosed cab, AG	0.0021	0.0021	0.0020	0.00022
17. Granular bait dispersed by hand			71	0.47
18. Low pressure handwand	25	12	7.1	0.94
19. High pressure handwand	13	1.8	0.64	0.079
20. Backpack applications	680			0.33
21. Hand gun (lawn) sprayer			0.34	0.0014
22. Paintbrush applications	260	180		0.280
23. Airless sprayer (exterior house stain)	110	38		0.830
24. Right-of-way sprayer	1.9	1.3	0.39	0.0039
25. Flagger/Liquid	0.053	0.011	0.012	0.00035
26. Flagger/Granular	0.0050			0.00015
27. WP or liquid/open pour/airblast/open cab	26			0.021
28. WP or liquid/open pour/airblast/closed cab	0.88	0.37	0.057	0.0013
29. Liquid or DF /open pour/ground boom/closed cab	0.22	0.089	0.029	0.00035
30. Granule/open pour/belly grinder	210	10	9.3	0.062
31. Push type granular spreader		2.9		0.0063
32. Liquid/open pour/low pressure handwand	110	100	0.43	0.030
33. WP/open pour/low pressure handwand			8.6	1.1
34. Liquid/open pour/backpack			2.5	0.03
35. Liquid/open pour/high pressure handwand			2.5	0.12
36. Liquid/open pour/garden hose end sprayer	34			0.0095
37. Liquid/open pour/termiticide injection			0.36	0.0022

^[1] Note that the above values are in mg a.i./lb handled and not mg a.i./kg bw per lb a.i. handled. Scenarios and values in bold are used in current risk assessment.

Source: Keigwin 1988
See Section 3.2.2.1 for discussion.

Table 18: Worker Exposure Rates Used in this Risk Assessment

Application Method	Worker Rates in mg/kg bw per lb handled			Source/Method
Rates for Triclopyr TEA Formulations				
Directed foliar	0.003	0.0003	0.01	SERA 2007a
Ground boom	0.0002	0.00001	0.0009	SERA 2007a
Aerial	0.00003	0.000001	0.0001	SERA 2007a
Rates for Triclopyr BEE Formulations				
Directed foliar ^[1]	0.0058	0.00086	0.039	Middendorf 1992b
Ground boom	0.00038	0.00003	0.0035	Adjusted ^[1]
Aerial	0.00006	0.000003	0.0004	Adjusted ^[1]

^[1] The ratio of rates from Middendorf (1992b) to standard Forest Service rates for directed foliar spray are approximately 1.9, 2.9, and 3.9 based on the central estimate, lower bound, and upper bound. These ratios are used to adjust rates for ground boom and aerial applications of triclopyr BEE based on the standard rates for these application methods.

See Section 3.2.2.1 for discussion.

Table 19: Concentrations of Triclopyr and TCP in Pond Water and Sediment

Concentration in Water (mg/L)		Water Concentrations of Triclopyr ÷ TCP	Concentrations of in Sediment (mg/kg)		Concentrations in Sediment ÷ Water	
Triclopyr	TCP		Triclopyr	TCP	Triclopyr	TCP
2.087	0.012	173.9	0.68	0.128	0.326	10.667
2.663	0.017	156.6	0.86	0.154	0.323	9.059
2.799	0.011	254.5	0.173	0.071	0.062	6.455
2.345	0.004	586.3	0.08	0.085	0.034	21.250
2.389	0.015	159.3	0.264	0.134	0.111	8.933
2.743	0.02	137.2	0.453	0.159	0.165	7.950
3.039	0.017	178.8	0.621	0.166	0.204	9.765
2.5 (2.1 to 3.0)	0.014 (0.004 to 0.02)	235 (137 to 586)	Approximate Averages and Range		0.175 (0.03 to 0.3)	10.6 (6.5 to 21)

Data from Petty et al. 2003, Table 3, p. 74.
See Section 3.2.3.4.1.2 for discussion.

Table 20: Site Characteristics and Parameters Used in Gleams-Driver Modeling

Field Characteristics		Description		Pond Characteristics		Description			
Type of site and surface	Pine-hardwood		Surface area	1 acre		Drainage area:	10 acres		
Treated and total field areas	10 acres		Initial Depth	2 meters		Minimum Depth	1 meter		
Field width	660 feet		Maximum Depth	3 meters		Sediment Depth	2 centimeters		
Slope	0.1								
Depth of root zone	36 inches								
Cover factor	0.15								
Type of clay	Mixed								
Surface cover	No surface depressions								
Stream Characteristics			Value						
Width		2 meters							
Flow Velocity		6900 meters/day							
Initial Flow Rate		710,000 liters/day							
GLEAMS Crop Cover Parameters ^[3]		Description		Value					
ICROP		Trees, hardwood + conifer		71					
CRPHTX		Maximum height in feet.		20					
BEGGRO		Julian day for starting growth		32					
ENDGRO		Julian day for ending growth		334					
Application, Field, and Soil Specific Factors ^[1]		Code ^[3]		Clay		Loam		Sand	
Proportion applied to soil:		SOLFRC		0.5		0.5		0.5	
Proportion applied to foliage:		FOLFRC		0.5		0.5		0.5	
Percent clay (w/w/):		CLAY		50%		20%		5%	
Percent silt (w/w/):		SILT		30%		35%		5%	
Percent sand (w/w/):		N/A		20%		45%		90%	
Percent Organic Matter:		OM		3.7%		2.9%		1.2%	
Bulk density of soil (g/cc):		BD		1.4		1.6		1.6	
Soil porosity (cc/cc):		POR		0.47		0.4		0.4	
Soil erodibility factor (tons/acre):		KSOIL		0.24		0.3		0.02	
SCS Runoff Curve Number ^[2] :		CN2		83		70		59	
Evaporation constant (mm/d):		CONA		3.5		4.5		3.3	
Saturated conductivity below root zone (in/hr):		RC		0.087		0.212		0.387	
Saturated conductivity in root zone (in/hr)		SATK		0.087		0.212		0.387	
Wilting point (cm/cm):		BR15		0.28		0.11		0.03	
Field capacity (cm/cm):		FC		0.39		0.26		0.16	
^[1] The qualitative descriptors are those used in the QuickRun window of Gleams-Driver. Detailed input values for the soil types are given in the sub-table below which is adapted from SERA (2007b, Tables 2 and 3). All fields are run for about 6 months before the pesticide is applied in early summer.									
^[2] From Knisel and Davis (Table H-4), <i>Clay</i> : Group D, Dirt, upper bound; <i>Loam</i> : Group C, woods, fair condition, central estimate; <i>Sand</i> : Group A, meadow, good condition, central estimate.									
^[3] Codes used in documentation for GLEAMS (Knisel and Davis 2000) and Gleams-Driver (SERA 2007a)									

Table 21: Precipitation, Temperature and Classifications for Standard Test Sites

Location	Precipitation	Temperature	Average Annual Rainfall (inches)	Average Annual Temperature (°F)
HI, Hilo	Wet	Warm	126.06	73.68
WA, Quillayute ¹	Wet	Temperate	95.01	49.14
NH, Mt. Washington²	Wet	Cool	98.49	27.12
FL, Key West	Average	Warm	37.68	77.81
IL, Springfield	Average	Temperate	34.09	52.79
MI, Sault Ste. Marie	Average	Cool	32.94	40.07
AR, Yuma Test Station	Dry	Warm	3.83	73.58
CA, Bishop	Dry	Temperate	5.34	56.02
AK, Barrow	Dry	Cool	4.49	11.81

¹ Based on composite estimation in WEPP using a latitude of 47.94 N and a longitude of -124.54 W. See SERA (2007b) for details.

² This site yielded the maximum concentration of triclopyr in surface water. See text for discussion.

Table 22: Chemical input parameters used in Gleams-Driver modeling

Parameter	Triclopyr BEE	Triclopyr Acid	TCP	Note/Reference
Halftimes (days)				
Aquatic Sediment	1	1300	7.5 (5 to 11.3)	Note 1
Foliar	1.1 - 15	2.6 - 15	2.6 - 15	Note 2
Soil	0.2	14 (8 - 28.4)	69 (40 to 95)	Note 3
Water	0.5	426	6	Note 4
Soil K_{oc} , mL/g	1233 (640 - 1650)	59 (25 to 134)	149 (81 to 242)	Note 5
Sediment K_d , mL/g	12 (0.64-16.5)	0.6 (0.25-1.32)	10.6 (6.5 to 21)	Note 6
Water Solubility, mg/L	7.4	440	49,100	Note 7
Foliar wash-off fraction	0.7	0.95	N/A (0.5)	Note 8
Fraction applied to foliage	0.5	0.5	N/A (0.5)	Note 9
Coefficient of Transformation	1	1	0.774	Note 10
Coefficient of Uptake	0	0	0	
Number of metabolites	2	1	0	
Depth of Soil Incorporation	1 cm			Surface application
Note 1	BEE: Use twice the half-life in water. Acid: Assume no degradation per U.S. EPA/OPP 2009a (Tables 2-1 and 3-2). Based on field studies, this is probably very conservative. TCP: Sediment half-times in ponds from Petty et al. 2003.			
Note 2	For BEE and acid, lower bound is from Thompson and upper bound is from Knisel and Davis (2000). No data for TCP. Value for TCP taken as identical to that for triclopyr acid. Ranges are modeled with a uniform distribution.			
Note 3	Acid from U.S. EPA/OPP (2009a). Triclopyr BEE conservatively set to 0.2. Much more rapid dissipation is reported in U.S. EPA/OPP (2009a). Values for TCP from Knuteson 1999. Central values and ranges are modeled with a triangular distribution.			
Note 4	BEE: 0.5 days for hydrolysis of triclopyr BEE to acid (U.S. EPA/OPP 2009a, Appendix P, MRID 134174 and McCall et al. 1988); Acid: 426 days (U.S. EPA/OPP 2009a, Table 3-2, PRZM/EXAMS input, this is very conservative.); TCP: lumped aquatic degradation from Knuteson (1999). This appears to be very conservative given the rapid aquatic photolysis of TCP. See Table 1 of this risk assessment.			
Note 5	BEE: Cessna et al. 2002. This is consistent with Knisel and Davis (2000). Acid: Cessna et al. 2002. Central estimate is consistent with U.S. EPA/OPP (2009a) PRZM/EXAMS input. TCP: From Cessna et al. 2002 and consistent with Knuteson 1999. Central values and ranges are modeled with a triangular distribution.			
Note 6	BEE and triclopyr: Based on the values for Koc with the assumption of 1% OC in soils. Values for BEE have little impact because of rapid conversion to acid. Estimates for triclopyr are consistent with sediment/water concentrations from Petty et al. (2003). TCP: Based on sediment/water concentrations from Petty et al. (2003). Central values and ranges are modeled with a triangular distribution.			
Note 7	BEE: U.S. EPA/OPP (2009a, Appendix P); Acid: U.S. EPA/OPP (2009a, Tables 2-1 and 3-2); TCP: Knuteson (1999).			
Note 8	Values for BEE and acid from Knisel and Davis (2000). This parameter is not used by GLEAMS for metabolites.			
Note 9	Conservative assumption used in all Forest Service risk assessments for foliar applications. This parameter is not used by GLEAMS for metabolites.			
Note 10	For TCP, the value of 0.774 is the ratio of the molecular weight of TCP to triclopyr acid. Because application rates will be expressed in units of a.e., no adjustment is needed for triclopyr BEE to triclopyr acid.			

Note: As indicated above, the Gleams-Driver runs utilize Monte Carlo methods for some input parameters. These are done using the Full Run capabilities in Gleams-Driver. In the database that is released with Gleams-Driver, only central estimates are entered into the chemical data table and only triclopyr TEA and BEE are included. Also note that the modeling of metabolites can only be done using a Full Run. This is true for all pesticides in Gleams-Driver.

Table 23: Summary of Modeled Concentrations and Monitoring in Surface Water

Scenario	Concentrations (ppb or µg/L)					
	Peak			Long-Term Average		
MODELING FOR THIS RISK ASSESSMENT (1 lb a.i./acre)						
Accidental Spill	1,600 (230 to 18,000)			N/A		
Direct Spray and Spray Drift						
Pond, Direct Spray (Section 3.2.3.4.2) ^[1]	110			N/A		
Pond, drift at 25 feet (Section 3.2.3.4.2) ^[1]	0.93 to 25			N/A		
Stream, Direct Spray (Section 3.2.3.4.2) ^[1]	90			N/A		
Stream, drift at 25 feet (Section 3.2.3.4.2) ^[1]	0.8 to 20			N/A		
Glams-Driver	BEE	Acid	TCP	BEE	Acid	TCP
Garlon 4 (Triclopyr BEE) Application						
Appendix in This Risk Assessment	App. 9	App. 10	App. 11	App. 9	App. 10	App. 11
Pond, Section 3.2.3.4.4	0.047 (0 - 2.94)	3.34 (0-142)	0.42 (0-19.4)	0.00023 (0 - 0.012)	1.37 (0 - 62)	0.041 (0-1.85)
Stream, Section 3.2.3.4.4	0.41 (0-17.1)	2.8 (0-62)	0.88 (0-26.5)	0.00181 (0 - 0.07)	0.037 (0-1.94)	0.0293 (0-1.53)
Garlon 3A (Triclopyr TEA) Application						
Appendix in This Risk Assessment	N/A	App. 12	App. 13	N/A	App. 12	App. 13
Pond, Section 3.2.3.4.4	N/A	4.55 (0 - 221)	0.43 (0 - 14.7)	N/A	1.91 (0 - 93)	0.052 (0 - 1.93)
Stream, Section 3.2.3.4.4	N/A	3.93 (0 - 84)	0.86 (0 - 22.5)	N/A	0.056 (0 - 2.45)	0.036 (0 - 1.57)
Other Modeling						
U.S. EPA	All concentrations are for triclopyr acid unless otherwise specified.					
GENEEC, ground application, WRC, 1 lb/acre ^[2]	30			19 (56-d ave.)		
GENEEC, aerial application, WRC, 1 lb/acre ^[2]	31			19.8 (56-d ave.)		
GENEEC, Triclopyr BEE ^[3]	19					
PRZM EXAMS, 20 lb a.e./acre ^[4]	710 (5.3 to 5800)			586 (4 to 4770)		
PRZM EXAMS, forestry related sites normalized to 1 lb/acre ^[5]	106 (3 to 244)			85 (60-d ave.) (2 to 204)		
Triclopyr (a.e.) Monitoring (Terrestrial Applications)						
Stream adjacent to application site (3.4 kg a.e./ha) (Norris et al. 1987).	Stream Peak: 95 µg/L (WCR: ≈31.3 µg/L per lb/ac.) Pond Delayed Peak: 12 µg/L (WCR: 4.0 µg/L)					
Streams in Maine watershed of forest treated with 1.9 kg a.e./ha (Smith and McCormack 1988).	Peak: 56 µg/L (WCR: 33 µg/L per lb/ac.) Delayed Peak: 11 to 48 µg/L (WCR: ≈6.5 to 28 µg/L per lb/ac.)					
USGS Stream Monitoring, 1992-2001 (Gilliom et al. 2007)	No reported detections at limit of 0.040 µg/L. Extent of monitoring for triclopyr is not clear.					
Red River and tributaries (Rawn et al. 1999).	0.42					
Surface water (Woudneh et al. 2007)	0.00218					
Triclopyr BEE Monitoring (Terrestrial Applications)						
Aerial application of Garlon 4 at 3.67 kg a.e./ha over a forest stream (Thompson et al. 1991)	Max: 230 to 350 µg/L Max WCR: ≈70 to 107 µg/L per lb/ac.					
Monitoring (Aquatic Applications)						
Maximum concentrations in ponds after aquatic applications at a nominal rate of 2,500 ppb (µg/L) (Petty et al. 2003)	Agent	Min.	Max			
	Acid	2100	3039			
	TCP	0.4	1.7			

^[1] Section 3.2.3.4.2 discusses expected concentrations in terms of the nominal application rate of 1 lb a.e./acre. The values for direct spray and drift are taken from Worksheet 10a (direct spray and drift as 25 feet for a pond) and Worksheet 10b (direct spray and drift as 25 feet for a stream).

^[2] U.S. EPA/OPP 1998a, p. Table 32, p. 67. The peak concentration for aerial application is reported as 186 ppb at an application rate of 6 lb/acre.

^[3] U.S. EPA/OPP 2004, Table 6, p. 17, *the worst-case assumption that triclopyr BEE was stable to aerobic soil metabolism.*

^[4] U.S. EPA/OPP 2009a, p. Table 3-3, pp. 62-63. All modeling runs appear to have been conducted for multiple applications (up to 17 with a 21-day application interval) at an application rates up to 20 lb a.e./acre. See Table 23 in current risk assessment for details.

^[5] U.S. EPA/OPP 2009a, Tables 3-2 and 3-3 normalized to 1 lb a.e./acre. **No adjustment for multiple applications.**

^[6] U.S. EPA/OPP 2009a, Appendix K, pp. 1-2, CA Residential scenario. Apparently only one application at 1.68 kg/ha (≈1.5 lb/acre).

Table 24: Summary of PRZM/EXAMS Modeling Conducted by U.S. EPA/OPP (2009a)

Crops Represented	Date ^[1]	Concentrations in ppb (µg/L)		
		Peak	21-day	60-day
Terrestrial Applications				
Douglas-Fir (Forest/Shelterbelt)	1-Jan	44.0	40.4	35.5
Conifer Release	1-Jan	127.7	116.6	107.6
Christmas Tree Plantations, Conifer Release, Forest Trees (All or Unspecified), Forest Tree Management/Forest Pest Management	2-Jan	194.7	176.5	136.9
Christmas Tree Plantations, Forest Trees (All Or Unspecified), Conifer Release	1-Jan	534.6	491.9	426.3
Forest Tree Management/Forest Pest Management, Forest Trees (All Or Unspecified)	2-Jan	337.9	309.8	286.0
ORCHARDS (Non-Food Stump Treatment)	1-Apr	148.4	131.0	109.7
Airports/Landing Fields, Commercial/Institutional/Industrial Premises/Equipment (Outdoor)	2-Jan	3479.0	3141.0	2864.0
Paved Areas (Private Roads/Sidewalks), Drainage Systems, Industrial Areas (Outdoor), Nonagricultural Rights-Of-Way/Fencerows/Hedgerows	2-Jan	1363.0	1242.0	1006.5
Commercial Storages/Warehouses Premises, Paved Areas (Private Roads/Sidewalks), Drainage Systems, Industrial Areas (Outdoor)	2-Jan	5802.0	5244.0	4770.0
Agricultural Rights-Of-Way/Fencerows/Hedgerows	2-Jan	250.1	226.9	190.7
		1319.2	1200.2	1098.5
Nonagricultural Rights-Of-Way/Fencerows/Hedgerows	2-Jan	2929.6	2666.9	2442.3
Ornamental Herbaceous Plants, Ornamental Non-flowering Plants	1-Apr	34.0	30.2	23.9
Ornamental and/or Shade Trees, Ornamental Woody Shrubs and Vines	1-Apr	415.3	376.2	308.5
		382.6	338.4	268.7
Structures/Buildings And Equipment	1-Apr	77.3	70.0	65.0
Agricultural/Farm Premises	1-Apr	103.1	93.7	87.1
Agricultural Fallow/Idle land, Nonagricultural Uncultivated Areas/Soils	1-Apr	87.8	81.2	66.5
Agricultural Fallow/Idle land	1-Apr	64.6	60.6	49.9
Agricultural/Farm Premises	1-Apr	990.2	908.2	793.9
Agricultural/Farm Structures/Buildings And Equipment, Agricultural Uncultivated Areas, Nonagricultural Uncultivated Areas/Soils	1-Apr	990.2	908.2	793.9
Pastures, Rangeland	1-Apr	32.9	30.4	24.9
1-Apr	394.8	354.8	321.9	
Recreation Area Lawns, Residential Lawns	1-Feb	75.0	69.1	61.7
Residential Lawns	1-Feb	415.0	376.3	309.0
Household/Domestic Dwellings Outdoor Premises, Recreation Area Lawns	1-Feb	1499.4	1317.2	1171.6
Ornamental Lawns And Turf	2-Jan	5.3	4.7	4.0
Commercial/Industrial Lawns, Ornamental Lawns And Turf	2-Jan	34.6	31.7	28.6
Ornamental Sod Farm (Turf)	2-Jan	20.8	18.9	15.7
Commercial/Industrial Lawns	2-Jan	124.8	113.1	94.3
Ornamental Sod Farm (Turf)	2-Jan	165.1	154.6	133.8
Golf Course Turf	2-Jan	270.0	245.9	219.5
Rice	NA	763.0	763.0	763.0
	Average:	710.2	645.5	586.2
	Minimum:	5.3	4.7	4.0
	Maximum:	5802.0	5244.0	4770.0
Aquatic Applications				
All	NA	2500.0	2500.0	2500.0

^[1]Forestry uses are highlighted in **bold type**. See Table 24 for details. See Section 3.2.3.4.4 for discussion.

Table 25: Summary of PRZM/EXAMS Modeling for Forestry Sites

Crops Represented	Application Schedule			Water Contamination Rates ¹¹ (µg/L per lb/acre)		
	Rate	# Apps	Interval	Peak	21-day	60-day
Douglas-Fir (Forest/Shelterbelt)	1.5	17	21	29	27	24
Conifer Release	3.2	17	21	85	78	72
Christmas Tree Plantations, Conifer Release, Forest Trees (All or Unspecified), Forest Tree Management/Forest Pest Management	6	17	21	130	118	91
Forest Tree Management/Forest Pest Management, Forest Trees (All Or Unspecified)	6	17	21	225	207	191
ORCHARDS (Non-Food Stump Treatment)	9	17	21	99	87	73
Nonagricultural Rights-Of-Way/Fencerows/Hedgerows	12	1	N/A	244	222	204
Pastures, Rangeland	4.5	1	N/A	3	3	2
Pastures, Rangeland	9	17	21	33	30	27
Arithmetic mean:				106	96	85
Geometric mean:				60	54	47

¹¹ Water Contamination Rates estimated by dividing the modeled concentrations in µg/L by the application rate. No adjustment is made for multiple applications.

Reformatted from U.S. EPA/OPP 2009a, Tables 3-2 and 3-3.
See Section 3.2.3.4.4 for discussion.

Table 26: Concentrations in surface water used in this risk assessment

Water contamination rate in mg a.e./L per lb/acre applied ^[1]

Terrestrial Applications			
		Peak	Longer-term
Triclopyr BEE	Central	0.0004	2 x 10 ⁻⁶
	Lower	1.5 x 10 ⁻⁷	2 x 10 ⁻¹¹
	Upper	0.030	7 x 10 ⁻⁵
		Peak	Longer-term
Triclopyr acid	Central	0.003	0.001
	Lower	1 x 10 ⁻⁶	2x10 ⁻¹⁰
	Upper	0.24	0.06
		Peak	Longer-term
TCP	Central	0.0009	5x10 ⁻⁵
	Lower	1x10 ⁻⁸	3x10 ⁻¹²
	Upper	0.028	0.002
Aquatic Applications			
		Peak	Longer-term
Emergent Vegetation			
Triclopyr	Central	0.18	0.0059
	Lower	0.09	0.00074
	Upper	0.36	0.021
TCP	Central	0.055	0.0050
	Lower	0.026	0.000060
	Upper	0.16	0.045
		Peak	Longer-term
Submergent Vegetation			
Triclopyr	Central	1.0	0.032
	Lower	1.0	0.0080
	Upper	1.0	0.064
TCP	Central	0.30	0.027
	Lower	0.29	0.0065
	Upper	0.44	0.12

^[1] For submergent aquatic applications, application rates in units of lb a.e./acre are not applicable and the concentrations are based on a target concentration of 1 mg a.e./L.

See Section 3.2.3.4.5.1 for discussion of terrestrial applications.

See Section 3.2.3.4.5.2 for discussion of aquatic applications.

Table 27: Estimated residues in food items as ppm per lb applied

Food Item	Concentration in Food Item (ppm per lb/acre)		
	Central ^a	Lower ^b	Upper ^a
Rates adopted from Fletcher et al. 1997			
Short grass	85	30	240
Tall grass	36	12	110
Broadleaf/forage plants and small insects	45	15	135
Fruits, pods, seeds, and large insects	7	3.2	15
^a U.S. EPA/EFED 2001, p. 44 as adopted from Fletcher et al. (1997).			
^b Central values \times (Central Value \div Upper Value).			

Table 28: Estimates of TCP on Vegetation and Fruit

Item	Central	Lower	Upper	Units
Fruit				
Inputs				
Initial Triclopyr Residue ^[1]	7.2	3.2	15	mg/kg
Half-life	26.9	16.5	73.1	days
Estimated Values				
Maximum Residue of TCP	1.99	0.91	4.27	mg/kg
Time to Maximum TCP Residue	38.8	23.8	105	days
90-day TWA Residues of TCP ^[2]	1.63	0.58	4.14	mg/kg
Vegetation				
Inputs				
Initial Triclopyr Residue ^[1]	45	15	135	mg/kg
Half-life	6.2	2.6	15	days
Estimated Values				
Maximum Residue of TCP	12.8	4.26	38.4	mg/kg
Time to Maximum TCP Residue	8.9	3.8	21.6	days
90-day TWA Residues of TCP ^[2]	3.44	0.48	23.1	mg/kg

^[1] See Table 27.

^[2] This is the maximum 90-day time-weighted average.

See Figures 7 and 8 for the time course of residues.

See Section 3.2.3.8 for discussion.

Table 29: Summary of Toxicity Values Used in Human Health Risk Assessment			
Duration	Derivation of RfD	Reference	Comment
Triclopyr			
Acute – single exposure (excluding women of childbearing age)			
NOAEL Dose	100 mg/kg bw/day	Jones 1995 MRID 43675801	Developmental study in rats with triclopyr BEE. Not applicable to females of childbearing age. For women between 13 and 50 years of age, the chronic RfD is used as the acute RfD.
LOAEL Dose	300 mg/kg bw/day		
LOAEL Endpoint(s)	Severe maternal toxicity		
Species, sex	Rats, females	U.S. EPA/OPP 2002a	
Uncertainty Factor	100		
RfD	1 mg/kg bw/day		
Acute for women of childbearing age Chronic for other individuals			
NOAEL Dose	5 mg/kg bw/day	Vedula et al. 1998 MRID 43545701	Two generation dietary reproduction study with triclopyr acid. This RfD is also used by U.S. EPA/OPP (2002) for short-term, intermediate, and longer-term occupational exposures.
LOAEL Dose	25 mg/kg bw/day		
Species, sex	Rats, males and females		
LOAEL Endpoint(s)	Kidney toxicity	U.S. EPA/OPP 1998a	
Uncertainty Factor	100		
RfD	0.05 mg/kg bw/day		
TCP (3,5,6-trichloro-2-pyridinol)			
Acute – single exposure			
NOAEL Dose	25 mg/kg bw/day	U.S. EPA/OPP 2002b	Birth defects included hydrocephaly and dilated ventricles. No acute dietary RfD is derived for members of the general population.
LOAEL Dose	100 mg/kg bw/day		
LOAEL Endpoint	Birth defects		
Species, sex	Rabbits, female		
Uncertainty Factor/MOE	1,000		
Equivalent RfD	0.025 mg/kg bw/day		
Chronic – intermediate to lifetime exposure			
NOAEL Dose	12 mg/kg bw/day	U.S. EPA/OPP 2002b	Standard chronic toxicity study in dogs. The uncertainty factor of 1000 includes an FQPA factor of 10.
LOAEL Dose	48 mg/kg bw/day		
LOAEL Endpoint	Clinical chemistry		
Species, sex	Dogs		
Uncertainty Factor/MOE	1,000		
Equivalent RfD	0.012 mg/kg bw/day		

Table 30: Overview of HQs for Workers, Terrestrial Applications

Triclopyr TEA (Worksheet E02, Attachment 1)

Scenario	Receptor	Hazard Quotients			Toxicity Value
		Central	Lower	Upper	
Accidental/Incidental Exposures					
Contaminated Gloves, 1 min.	Worker	2E-05	7E-06	3E-04	1
Contaminated Gloves, 1 hour	Worker	1E-03	4E-04	2E-02	1
Spill on Hands, 1 hour	Worker	4E-04	9E-05	6E-03	1
Spill on lower legs, 1 hour	Worker	1E-03	2E-04	1E-02	1
General Exposures					
Acute					
Backpack Applications:		1E-02	5E-04	8E-02	1
Ground Broadcast Applications:		2E-02	7E-04	0.2	1
Aerial Applications:		1E-02	2E-04	8E-02	1
Chronic					
Backpack Applications:		0.3	9E-03	1.6	0.05
Ground Broadcast Applications:		0.4	1E-02	3	0.05
Aerial Applications:		0.3	5E-03	1.6	0.05

Triclopyr BEE Formulations (Worksheet E02, Attachment 2)

Scenario	Receptor	Hazard Quotients			Toxicity Value
		Central	Lower	Upper	
Accidental/Incidental Exposures					
Contaminated Gloves, 1 min.	Worker	1E-02	4E-03	0.1	1
Contaminated Gloves, 1 hour	Worker	0.7	0.2	7	1
Spill on Hands, 1 hour	Worker	1E-03	3E-04	2E-02	1
Spill on lower legs, 1 hour	Worker	4E-03	9E-04	5E-02	1
General Exposures					
Chronic Exposures					
Backpack Applications:		0.5	3E-02	6	0.05
Ground Broadcast Applications:		0.9	4E-02	12	0.05
Aerial Applications:		0.6	1E-02	6	0.05
Acute Exposures (Male Workers Only)					
Backpack Applications:		3E-02	3E-02	3E-02	1
Ground Broadcast Applications:		4E-02	4E-02	4E-02	1
Aerial Applications:		3E-02	3E-02	3E-02	1

See Section 3.4.2 for discussion.

Table 31: Overview of HQs for the General Public, Terrestrial Applications

Triclopyr

Scenario	Receptor (Form)	Hazard Quotients			RfD (mg/kg bw/day)
		Central	Lower	Upper	
Accidental Acute Exposures (dose in mg/kg/event)					
Direct spray, body	Child (TEA)	0.02	0.003	0.2	1.0
	Child (BEE)	0.05	0.01	0.7	1.0
Direct Spray, lower legs	Woman (TEA)	0.03	0.07	0.5	0.05
	Woman (BEE)	0.1	0.03	1.4	0.05
Accidental Spill	Child	0.1	0.01	2	1.0
All others	Mixed	≤0.001	≤0.0001	≤0.01	Mixed
Non-Accidental Acute Exposures (dose in mg/kg/event)					
Contact with Vegetation	Woman (TEA)	0.07	0.02	0.3	0.05
	Woman (BEE)	0.2	0.1	0.4	0.05
Contaminated Fruit	Woman	0.2	0.1	4	0.05
Contaminated Vegetation	Woman	3	0.2	27	0.05
All other scenarios	Mixed	≤0.007	≤0.00005	≤0.05	Mixed
Longer-term Exposures (dose in mg/kg/day)					
Contaminated Fruit	Woman	0.09	0.03	3	0.05
Contaminated Vegetation	Woman	0.2	0.004	6	0.05
All others	Mixed	≤0.02	≤0.003	≤0.03	Mixed

3,5,6-Trichloro-2-Pyridinol (TCP)

Scenario	Receptor (Form)	Hazard Quotients			RfD (mg/kg bw/day)
		Central	Lower	Upper	
Non-Accidental Acute Exposures (dose in mg/kg/event)					
Contaminated Fruit	Woman	0.1	0.06	2	0.025
Contaminated Vegetation	Woman	1.8	0.1	15	0.025
Longer-term Exposures (dose in mg/kg/day)					
Contaminated Fruit	Woman	0.2	0.08	4	0.012
Contaminated Vegetation	Woman	1.0	0.03	19	0.012

Table 32: Summary of Allometric Relationships of Triclopyr in Mammals

Species ^[1]	Duration (Days)	Doses (mg a.e./kg bw/day)			≈BW ^[4]	Endpoint	Reference ^[5]
		NOAEL	LOAEL	Mean ^[3]			
Subchronic Studies^[2]							
Mice	28	60	120	85	25	Liver damage	Tsuda et al. 1987
Rat, ♂ ^[2]	91	28	50	37	230	Kidney and liver	Barna-Lloyd et al. 1992
Rat ♀ ^[2]	91	70	250	130	168	Kidney and liver	Barna-Lloyd et al. 1992
Rat	91	5	20	10	199	Kidney	Landry et al. 1984
Dogs	10	0.5	10	2.2	2185	PSP excretion ^[6]	Quast et al. 1976
Dogs	183	0.5	3	1.2	2185	PSP excretion ^[6]	Quast et al. 1977
Dogs	228	10	20	14	2185	Kidney and liver	Quast et al. 1976
Chronic Studies^[2]							
Mice ♂ ^[7]	665	28.6	143	64	32	Kidney	Tsuda et al. 1987
Rats	760	12	36	21	388	Kidney	Eisenbrandt et al. 1987
Dogs	365	2.5	5	3.5	10,000	Kidney	Quast et al. 1988
Statistical Analyses							
Duration	Allometric Relationship for Dose (mg/kg) and Body Weight (g)		Adjusted r ²	p-value	Comments		
Subchronic	Dose = 2065 W ^{-0.82}		0.56	0.032	Relationship not significant (p=0.28) if PSP endpoint in dogs is excluded.		
Chronic	Dose = 394 W ^{-0.51}		0.99	0.037	None.		
All	Dose = 451 W ^{-0.50}		0.61	0.013	Excludes subchronic data on PSP in dogs.		

^[1] Both sexes unless otherwise specified.

^[2] All assays used triclopyr except for Barna-Lloyd et al. 1992, which used triclopyr BEE.

^[3] The geometric mean of the range from the NOAEL to the LOAEL were used in the allometric analyses.

^[4] Reference body weights (in grams) for subchronic studies from U.S. EPA/ORD (1988, pp. 1-7 to 1-9). Averages for strains when difference strains are given.

^[5] See Appendix 2, Table 8 for details of subchronic studies.

^[6] An inhibition of phenolsulfonphthalein (PSP) excretion may be of marginal toxicologic significance. See Section 4.1.2.1 for discussion.

^[7] Data on female mice is not included because the LOAEL of 135 mg/kg bw/day (NOAEL=26.5 mg/kg bw/day) is of questionable significance. Omission of the geometric mean of 60 mg/kg bw/day has no impact on the analysis.

See Figure 10 for illustration.
See Section 4.1.2.1.1 for discussion.

Table 33: Fish – 96-hour LC₅₀ for Triclopyr and TCP

Triclopyr Acid			
Species	Reference	N	96-hour EC ₅₀
Pink salmon	Wan et al. 1997	1	6.3
Chum Salmon	Wan et al. 1997	2	7.5
Sockeye salmon	Wan et al. 1997	3	7.5
Rainbow trout	Wan et al. 1997	4	7.5
Coho Salmon	Wan et al. 1997	5	9.6
Chinook salmon	Wan et al. 1997	6	9.7
Rainbow trout	Batchelder 1973	7	79.2
Bluegill sunfish	Batchelder 1973	8	155.4
Geometric mean:			15.3
Triclopyr TEA			
Species	Reference	N	96-hour EC ₅₀
Tidewater silverside	Ward and Boeri 1989	1	40.1
Bluegill sunfish	Abdelghani 1995	2	65.1
Catfish, juv	Abdelghani 1995	3	78.3
Fathead minnow	Mayes 1984 (flow-through)	4	85.8
Fathead minnow	Mayes 1990c	5	86.4
Chum Salmon	Wan et al. 1997	6	96.1
Chinook salmon	Wan et al. 1997	7	99
Sockeye salmon	Wan et al. 1997	8	112
Coho salmon, juv	Janz et al. 1991	9	127.2
Catfish, adult	Abdelghani 1995	10	141
Rainbow trout	Wan et al. 1997	11	151
Coho Salmon	Wan et al. 1997	12	167
Fathead minnow	Mayes 1990c	13	168.5
Bluegill sunfish	McCarty and Alexander 1978	14	233.1
Rainbow trout	McCarty and Alexander 1978	15	273.7
Rainbow trout	Morgan et al. 1984	16	286
Fathead minnow	McCarty and Alexander 1978	17	422.8
Geometric mean:			130.7
TCP			
Species	Reference	N	96-hour EC ₅₀
Rainbow trout	Wan et al. 1987	1	1.5
Coho salmon	Wan et al. 1987	2	1.8
Chum salmon	Wan et al. 1987	3	1.8
Chinook salmon	Wan et al. 1987	4	2.1
Sockeye salmon	Wan et al. 1987	5	2.5
Pink salmon	Wan et al. 1987	6	2.7
Bluegill sunfish	MRID 41829003	7	12.5
Rainbow trout	MRID 41829004	8	12.6
Geometric mean:			3.19
Triclopyr BEE			
Species	Reference	N	96-hour EC ₅₀
Bluegill sunfish	Woodbum et al. 1993c	1	0.25
Coho salmon	Mayes et al. 1986	2	0.26
Coho salmon	Barron et al. 1989b	3	0.47
Fathead minnow	McCarty and Alexander 1978	4	0.5
Bluegill sunfish	Gorzinski et al. 1991a,b	5	0.54
Bluegill sunfish	McCarty and Alexander 1978	6	0.58
Coho salmon	Janz et al. 1991	7	1
Fathead minnow	Milazzo and Batchelder 1981	8	1.5
Geometric mean:			0.539
Garlon 4			
Species	Reference	N	96-hour EC ₅₀
Bluegill sunfish	Weinberg et al. 1994a	1	0.2
Rainbow trout	Ross and Pell 1981	2	0.34
Rainbow trout	Weinberg et al. 1994b	3	0.47
Pink salmon	Wan et al. 1997	4	0.58
Sockeye salmon	Wan et al. 1997	5	0.67
Chum Salmon	Wan et al. 1997	6	0.81
Coho salmon	Johansen and Geen 1990	7	0.84
Pink salmon	Wan et al. 1991	8	0.93
Coho salmon	Servizi et al. 1987	9	0.97
Coho salmon	Wan et al. 1997	10	1
Rainbow trout	Morgan et al. 1991	11	1
Rainbow trout	Wan et al. 1997	12	1.3
Chinook salmon	Wan et al. 1997	13	1.3
Geometric mean:			0.716

See Section 4.1.3 for a discussion of frequency tabulation.
 See Figure 11 for illustration and Appendix 6 for details.
 See Section 4.1.3.1 for discussion of data.

Table 34: Amphibians - 96-Hour LC₅₀s for Triclopyr

Triclopyr TEA				
Species	Reference	N	96-h LC₅₀ (mg a.e./L)	Freq (n-.5)/Tot
African clawed frog	Perkins 1997	1	84	0.5
Triclopyr BEE Formulations Embryos				
Species	Reference	N	96-h LC₅₀ (mg a.e./L)	Freq (n-.5)/Tot
<i>Xenopus laevis</i>	Edington et al. 2005	1	13.7	0.1
<i>Xenopus laevis</i>	Perkins 1997	2	15.0	0.3
<i>Bufo americanus</i>	Edington et al. 2005	3	15.1	0.5
<i>Rana pipiens</i>	Edington et al. 2005	4	23.3	0.7
<i>Rana clarnitans</i>	Edington et al. 2005	5	24.6	0.9
Geometric mean:			17.78 mg/L	
Triclopyr BEE Formulations Tadpoles				
Species	Reference	N	96-h LC₅₀ (mg a.e./L)	Freq (n-.5)/Tot
<i>Rana pipiens</i>	Edington et al. 2005	1	0.79	0.07142857
<i>Bufo americanus</i>	Edington et al. 2005	2	0.88	0.21428571
<i>Xenopus laevis</i>	Edington et al. 2005	3	1.70	0.35714286
<i>Rana pipiens</i>	Wojtaszek et al. 2005	4	2.79	0.5
<i>Rana clamitans</i>	Wojtaszek et al. 2005	5	3.01	0.64285714
<i>Rana pipiens</i>	Wojtaszek et al. 2005	6	3.39	0.78571429
<i>Rana clarnitans</i>	Edington et al. 2005	7	11.50	0.92857143
Geometric mean:			2.34 mg/L	

See Section 4.1.3 for a discussion of frequency tabulation.

See Figure 12 for illustration and Appendix 7 for details.

See Section 4.1.3.2 for discussion of data.

Table 35: Aquatic Invertebrates - 48-Hour EC₅₀s

Triclopyr Acid and TEA (Non-bivalve)				
Species	Reference	N	48-h LC50 (mg a.e./L)	Freq (n-.5)/Tot
Grass shrimp	MRID 42646102	1	103.7	0.05555556
<i>Daphnia magna</i> (Acid)	Batchelder and McCarty, 1977	2	132.9	0.16666667
Pink shrimp	Heitmuller 1975	3	270.5	0.27777778
<i>Physella gyrina</i> (Acid)	Neuderfer 2009	4	293	0.38888889
<i>Daphnia magna</i>	McCarty and Alexander 1978	5	346	0.5
<i>Daphnia magna</i>	Gersich et al. 1985a	6	357	0.61111111
<i>Daphnia magna</i>	Gersich et al. 1982	7	376	0.72222222
<i>Daphnia magna</i>	Gersich et al. 1984	8	837	0.83333333
Red swamp crayfish	Abdelghani et al. 1995	9	6397.5	0.94444444
N=			Geometric mean:	401.58594 mg/L
Triclopyr TEA (Bivalves)				
Species	Reference	N	Shell Dep. 48-h EC50 (mg a.e./L)	Freq (n-.5)/Tot
Eastern oyster	MRID 42646101	1	18.4	0.25
Eastern oyster	Heitmuller 1975	2	21.1	0.75
N=			Geometric mean:	19.703807 mg/L
TCP				
Species	Reference	N	48-h LC50 (mg a.e./L)	Freq (n-.5)/Tot
<i>Daphnia magna</i>		1	10.9	0.5
N=			Geometric mean:	10.9 mg/L
TBEE and Garlon 4 (Arthropods)				
Species	Reference	N	48-h LC50 (mg a.e./L)	Freq (n-.5)/Tot
<i>Daphnia magna</i>	Weinberg et al. 1994c	1	0.25	0.03846154
<i>Daphnia pulex</i>	Servizi et al. 1987	2	0.54	0.11538462
Grass shrimp	Ward and Boeri 1991b	3	0.77	0.19230769
<i>Daphnia magna</i>	Milazzo and Batchelder 1981b	4	1.2	0.26923077
Grass shrimp	Ward and Boeri 1991e	5	1.8	0.34615385
Red swamp crayfish	Gorzinski and Barron 1996	6	3.1	0.42307692
Stonefly (<i>Calineuria californica</i>)	Peterson et al. 2001	7	3.6	0.5
Mayfly (<i>Ameletus</i> sp.)	Peterson et al. 2001	8	3.8	0.57692308
Caddisfly (<i>Brachycentrus americanus</i>)	Peterson et al. 2001	9	5	0.65384615
<i>Daphnia magna</i>	Milazzo and Batchelder 1981a	10	8.3	0.73076923
Mayfly (<i>Cinygma</i> sp.)	Peterson et al. 2001	11	8.95	0.80769231
Caddisfly (<i>Psychoglypha</i> sp.)	Peterson et al. 2001	12	12.5	0.88461538
Caddisfly (<i>Lepidostoma unicolor</i>)	Peterson et al. 2001	13	20	0.96153846
N=			Geometric mean:	2.8669884 mg/L
Triclopyr BEE and Garlon - (Bivalves)				
Species	Reference	N	Shell Dep. 48-h EC50 (mg a.e./L)	48-h LC50 (mg a.e./L)
Eastern Oyster (Garlon 4)	Ward and Boeri 1991c	1	0.14	0.25
Eastern Oyster	MRID 41971602	2	0.33	0.75
N=			Geometric mean:	0.2149419 mg/L

See Section 4.1.3 for a discussion of frequency tabulation.

See Figure 13 for illustration and Appendix 8 for details.

See Section 4.1.3.3 for discussion of data.

Table 36: Algae – 4 to 5-Day EC₅₀s

Algae: Acid and TEA			
Species	EC₅₀ (mg a.e./L)	Reference	Rank Order
<i>Ankistrodesmus spp.</i>	0.49	Gardner et al. 1997	1
<i>Anabaena flos-aquae</i>	4.1	MRID 41633706	2
<i>Skeletonema costatum</i>	4.6	MRID 41633707	3
<i>Chlorella vulgaris</i>	8	Baarschers et al. 1988	4
<i>Navicula pelliculosa</i>	10.6	MRID 41633708	5
<i>Chlorella vulgaris</i>	11	Baarschers et al. 1988	6
<i>Kirchneria subcapitata</i>	32.5	Cowgill and Milazzo 1989a	7
<i>Chlorella pyrenoidosa</i>	54	Baarschers et al. 1988	8
<i>Chlorella pyrenoidosa</i>	80	Baarschers et al. 1988	9
Geometric mean:	10.21	mg/L	
Algae: BEE			
Species	EC₅₀ (mg a.e./L)	Reference	Rank Order
<i>Navicula pelliculosa</i>	0.073	Hughes and Alexander 1993c	1
<i>Skeletonema costatum</i>	0.84	Hughes and Alexander 1993a	2
<i>Anabaena flos-aquae</i>	1.42	Hughes and Alexander 1993b	3
<i>Kirchneria subcapitata</i>	2.5	MRID 42090422	4
<i>Skeletonema costatum</i>	5.9	Cowgill et al. 1989b	5
Geometric mean:	1.03	mg/L	
Algae: TCP			
Species	EC₅₀ (mg a.e./L)	Reference	Rank Order
<i>Kirchneria subcapitata</i>	1.8	MRID 45312003	1
<i>Anabaena flos-aquae</i>	1.8	MRID 45312001	2
Geometric mean:	1.80	mg/L	

See Figure 15 for illustration and Appendix 8, Table A8-1 for details.

See Section 4.1.3.4.1 for discussion of data.

Table 37: Aquatic Macrophytes – 7 to 14-Day EC₅₀s

Monocots: Acid and TEA			
Species	EC₅₀ (mg a.e./L)	Reference	Rank Order
<i>Lemna gibba</i>	6.06	MRID 41633709	1
<i>Lemna gibba</i>	7.6	MRID 41736302	2
<i>Lemna gibba</i>	7.7	Cowgill et al. 1988	3
<i>Lemna gibba</i>	13.58	Perkins 1997	4
<i>Lemna minor</i>	15.8	Cowgill et al. 1988	5
Geometric mean:	9.47	mg/L	
Dicots: Acid and TEA			
Species	96-h EC₅₀ (mg a.e./L)	Reference	Rank Order
Watermilfoil (acid)	0.04	Perkins 1997	1
Watermilfoil (TEA)	0.04	Poovey et al. 2007	2
Milfoil Hybrid	0.08	Poovey et al. 2007	3
Watermilfoil (acid)	0.56	Roshon et al. 1999	4
Geometric mean:	0.09	mg/L	
Monocots: BEE			
Species	96-h EC₅₀ (mg a.e./L)	Reference	Rank Order
<i>Lemna gibba</i>	0.86	Milazzo et al. 1993	1
<i>Lemna gibba</i>	6.25	Perkins 1997	2
Geometric mean:	2.32	mg/L	
Dicots: BEE			
Species	96-h EC₅₀ (mg a.e./L)	Reference	Rank Order
Watermilfoil	1.49	Perkins 1997	1
Watermilfoil	4.62	Roshon et al. 1999	2
Geometric mean:	2.62	mg/L	

See Figure 16 for illustration and Appendix 8, Table A8-2 for details.

See Section 4.1.3.4.1 for discussion of data.

Table 38: Terrestrial Nontarget Animals Used in Ecological Risk Assessment

Animal	Representative Species	W ^[4]	Food Consumption ^[5]	Water Consumption	Other
MAMMALS^[1]					
Small mammal	Mice	20	2.514 W ^{0.507} [Eq 3-48]	0.099 W ^{0.9} [Eq 3-17]	
Larger mammal	Squirrels	400	2.514 W ^{0.507} [Eq 3-48]		
Canid	Fox	5,000	0.6167 W ^{0.862} [Eq 3-47]		
Large Herbivorous Mammal	Deer	70,000	1.518 W ^{0.73} [Eq 3-46]		
Large Carnivorous Mammal	Bear	70,000	0.6167 W ^{0.862} [Eq 3-47]		
BIRDS^[2]					
Small bird	Passerines	10	2.123 W ^{0.749} [Eq 3-36]	0.059 W ^{0.67} [Eq 3-17]	
Predatory bird	Owls	640	1.146 W ^{0.749} [Eq 3-37]		
Piscivorous bird	Hérons	2,400	1.916 W ^{0.704} [Eq 3-38]		
Large herbivorous bird	Geese	4,000	1.146 W ^{0.749} [Eq 3-37]		
INVERTEBRATES^[3]					
Honey bee	<i>Apis mellifera</i>	0.000116	≈2 (1.2 to 4) ^[6]	Not used	SA ^[7] : 1.42 cm ²
Herbivorous Insects	Various	Not used	1.3 (0.6 to 2.2)	Not used	

^[1] Sources: Reid 2006; U.S. EPA/ORD 1993.

^[2] Sources: Sibley 2000; Dunning 1993; U.S. EPA/ORD 1993.

^[3] Sources: Humphrey and Dykes 2008; Reichle et al. 1973; Winston 1987

^[4] Body weight in grams.

^[5] For vertebrates, based on allometric relationships estimating field metabolic rates in kcal/day for rodents (omnivores), herbivores, and non-herbivores. For mammals and birds, the estimates are based on Nagy (1987) as adapted by U.S. EPA/ORD (1993). The equation numbers refer to U.S. EPA/ORD (1993). See the following table for estimates of caloric content of food items. For herbivorous insects, consumption estimates are based on fractions of body weight (g food consumed/g bw) from the references in Note 3.

^[6] For honeybees, food consumption based on activity and caloric requirements. Used only when estimates of concentrations in nectar and/or pollen can be made, which is not the case in the current risk assessment.

^[7] Based on the algorithms suggested by Humphrey and Dykes (2008) for a bee with a body length of 1.44 cm.

See data on food commodities in following table.
See Sections 4.2.2.3. and 4.2.2.3 for discussion.

Table 39: Diets: Metabolizable Energy of Various Food Commodities

Food Item	Animal Group	Caloric Value ^[1] (kcal/g dw)	Water Content ^[2]	Comment/Source(s)
Fruit	Mammals	1.1	0.77	See Footnote 3
	Birds	1.1	0.77	See Footnote 4
Fish	Mammals	4.47	0.70	Water content from Ali et al. (2005)
	Birds	3.87	0.70	
Insects	Mammals	4.47	0.70	Water contents from Chapman (, p. 491). Typical ranges of 60-80%.
	Birds	4.30	0.70	
Vegetation (NOS)	Mammals	2.26	0.85	
	Birds	2.0	0.85	See Footnote 5

^[1] Metabolizable energy. Unless otherwise specified, the values are taken from U.S. EPA/ORD (1993), Table 3-1, p. 3-5 as adopted from Nagy 1987.

^[2] From U.S. EPA/ORD (1993), Table 4-2, p. 4-14 unless otherwise specified.

^[3] Based on a gross caloric value of 2.2 kcal/g dw (U.S. EPA/ORD 1993, Table 4-2). An assimilation factor for mammals eating fruit not identified. Use estimate for birds (see below).

^[4] Based on a gross caloric value of 2.2 kcal/g dw (U.S. EPA/ORD 1993, Table 4-2) and an assimilation factor for the consumption of fruit by birds of 51% [2.2 kcal/g dw x 0.51 ≈ 1.1 kcal/g dw]

^[5] Based on a gross caloric value of 4.2 kcal/g dw for dicot leaves (U.S. EPA/ORD 1993, Table 4-2) and an assimilation factor for the consumption leaves by birds of 47% [4.2 kcal/g dw x 0.47 = 1.974 kcal/g dw]

Table 40: Triclopyr Acid, Toxicity Values for Terrestrial Organisms

Group/Duration	Organism	Endpoint	Toxicity Value ^[1]	Reference
Terrestrial Animals				
Acute				
	Small (20 g) Mammals ^[2]	Estimated acute NOAEL	440 mg/kg bw	Section 4.3.2.1.1
	Medium (400 g) Mammals	Acute NOAEL	100 mg/kg bw	
	Canines ^[2]	Estimated acute NOAEL	20 mg/kg bw	Section 4.3.2.1.1
	Large Herbivorous Mammals ^[2]	Estimated acute NOAEL	8 mg/kg bw	Section 4.3.2.1.1
	Birds	Gavage NOAEL	126 mg/kg bw	Section 4.3.2.2.1
	Honey Bee	Indefinite LD ₅₀	620 mg/kg bw	Section 4.3.2.4
	Herbivorous Insect	Indefinite LD ₅₀	620 mg/kg bw	Section 4.3.2.4
Longer-term				
	Small (20 g) Mammals	Estimated chronic NOAEL	22 mg/kg bw	Section 4.3.2.1.1
	Medium (400 g) Mammals	Chronic NOAEL	5 mg/kg bw	
	Canines ^[2]	Estimated chronic NOAEL	1 mg/kg bw	Section 4.3.2.1.1
	Large Herbivorous Mammals ^[2]	Estimated chronic NOAEL	0.4 mg/kg bw	Section 4.3.2.1.1
	Bird	Reproductive NOAEL	7.5 mg/kg bw/day	Section 4.3.2.2.1
Terrestrial Plants				
Soil	Sensitive	Seedling emergence NOAEC	0.0028 lb/acre	Section 4.3.2.5
	Tolerant	Seedling emergence NOAEC	0.23 lb/acre	Section 4.3.2.5
Foliar	Sensitive	Foliar spray NOAEC	0.0028 lb/acre	Section 4.3.2.5
	Tolerant	Foliar spray NOAEC	2.0 lb/acre	Section 4.3.2.5

^[1]All toxicity values for triclopyr expressed as mg a.e./kg bw for animals and lb a.e./acre for plants.

^[2] Acute and chronic toxicity values based on allometric relationships. See Section 4.3.2.1.1 for details.

Table 41: Triclopyr BEE, Toxicity Values for Terrestrial Organisms

Group/Duration	Organism	Endpoint	Toxicity Value ^[1]	Reference
Terrestrial Animals				
Acute				
	Small Mammals ^[2]	Estimated acute NOAEL	440 mg/kg bw	Section 4.3.2.1.1
	Medium (400 g) Mammals	Acute NOAEL	100 mg/kg bw	Section 4.3.2.1.1
	Canines ^[2]	Estimated acute NOAEL	20 mg/kg bw	Section 4.3.2.1.1
	Large Herbivorous Mammals ^[2]	Estimated acute NOAEL	8 mg/kg bw	Section 4.3.2.1.1
	Birds	Gavage NOAEL	126 mg/kg bw	Section 4.3.2.2.1
	Honey Bee (oral)	Indefinite LD ₅₀	620 mg/kg bw	Section 4.3.2.4
	Herbivorous Insect	Indefinite LD ₅₀	620 mg/kg bw	Section 4.3.2.4
Longer-term				
	Small (20 g) Mammals ^[2]	Estimated chronic NOAEL	22 mg/kg bw	Section 4.3.2.1.1
	Medium (400 g) Mammals	Chronic NOAEL	5 mg/kg bw	Section 4.3.2.1.1
	Canines ^[2]	Estimated chronic NOAEL	1 mg/kg bw	Section 4.3.2.1.1
	Large Herbivorous Mammals ^[2]	Estimated chronic NOAEL	0.4 mg/kg bw	Section 4.3.2.1.1
	Bird	Reproductive NOAEL	7.5 mg/kg bw/day	Section 4.3.2.2.1
Terrestrial Plants				
Soil	Sensitive	Seedling emergence NOAEC	0.022 lb/acre	Section 4.3.2.5
	Tolerant	Seedling emergence NOAEC	2.0 lb/acre	Section 4.3.2.5
Foliar	Sensitive	Foliar spray NOAEC	0.0028 lb/acre	Section 4.3.2.5
	Tolerant	Foliar spray NOAEC	2.0 lb/acre	Section 4.3.2.5

^[1]All toxicity values for triclopyr BEE expressed as mg a.e./kg bw for animals and lb a.e./acre for plants.

^[2] Acute and chronic toxicity values based on allometric relationships. See Section 4.3.2.1.1 for details.

Table 42: Trichloro-2-pyridinol (TCP), Toxicity Values for Terrestrial Organisms

Group/Duration	Organism	Endpoint	Toxicity Value^[1]	Reference
Terrestrial Animals				
Acute				
	Non-canine Mammals	Acute NOAEL	25 mg/kg bw	Section 4.3.2.1.2
	Canine Mammals	Acute NOAEL	25 mg/kg bw	Section 4.3.2.1.2
	Birds	Acute LOAEL	116	Section 4.3.2.2.2
	Honey Bee (oral)	No data.	N/A	Section 4.3.2.4
Longer-term				
	Small Mammal	Chronic NOAEL	12 mg/kg bw	Section 4.3.2.1.2
	Large Mammal	Chronic NOAEL	12 mg/kg bw	Section 4.3.2.1.2
	Bird	No data.	N/A	Section 4.3.2.2.2
Terrestrial Plants				
Soil	Sensitive	No data.	N/A	Section 4.3.2.5
	Tolerant	No data.	N/A	
Foliar	Sensitive	No data.	N/A	Section 4.3.2.5
	Tolerant	No data.	N/A	

^[1] All toxicity values for TCP are in mg TCP/kg bw. The lack of variability in the toxicity value for mammals reflects limitations in the available data.

Table 43: Triclopyr Acid, Toxicity Values for Aquatic Organisms

Group/Duration		Organism	Endpoint	Toxicity Value ^[1]	Reference
Aquatic Animals					
Acute					
Amphibians	Sensitive	Acute NOAEC (only one study)		125 mg/L	Section 4.3.3.2.1
	Tolerant	Acute NOAEC (only one study)		125 mg/L	
Fish	Sensitive	Estimated acute NOAEC		20 mg/L	Section 4.3.3.1.1
	Tolerant	Estimated acute NOAEC		210 mg/L	Section 4.3.3.1.1
Invertebrates	Sensitive	Adjusted NOAEC		25 mg/L	Section 4.3.3.3
	Tolerant	Estimated NOAEC		320 mg/L	Section 4.3.3.3
Longer-term					
Amphibians	Sensitive	No data.		N/A	Section 4.3.3.2.1
	Tolerant	No data.		N/A	Section 4.3.3.2.1
Fish	Sensitive	Estimated chronic NOAEC		7.4 mg/L	Section 4.3.3.1
	Tolerant	Estimated chronic NOAEC		78 mg/L	Section 4.3.3.1.1
Invertebrates	Sensitive	Chronic NOAEC		25 mg/L	Section 4.3.3.3
	Tolerant	Chronic NOAEC		25 mg/L	Section 4.3.3.3
Aquatic Plants					
Algae	Sensitive	5-Day NOAEC		0.23 mg a.e./L	Section 4.3.3.4.1
	Tolerant	4-Day NOAEC		4.0 mg a.e./L	Section 4.3.3.4.1
Macrophytes	Sensitive	NOAEC (overt) ^[2]		0.0005 mg/L	Section 4.3.3.4.2
	Tolerant	NOAEC		5.6 mg/L	Section 4.3.3.4.2

^[1]All toxicity values for triclopyr expressed as mg a.e./L.

^[2] Inhibition of photosynthesis and chlorophyll but not impact on growth in watermilfoil. See Section 4.3.3.4.2.1 for discussion.

Table 44: Triclopyr BEE, Toxicity Values for Aquatic Organisms

Group/Duration		Organism	Endpoint	Toxicity Values	Reference
Aquatic Animals					
Acute					
Amphibians	Sensitive		Acute sublethal EC ₁₀	0.1 mg/L	Section 4.3.3.2.2.
	Tolerant		Estimated acute NOAEL	4.2 mg/L	Section 4.3.3.2.2.
Fish	Sensitive		Acute NOAEC	0.091 mg/L	Section 4.3.3.1
	Tolerant		Adjusted acute NOAEC	0.75 mg/L	
Invertebrates	Sensitive		Estimated NOAEC	0.045 mg/L	Section 4.3.3.3
	Tolerant		Estimated NOAEC	3.6 mg/L	Section 4.3.3.3
Longer-term					
Amphibians	Sensitive		No data	N/A	Section 4.3.3.2.2.
	Tolerant		No data	N/A	Section 4.3.3.2.2.
Fish	Sensitive		Chronic NOAEC	0.019 mg/L	Section 4.3.3.1
	Tolerant		Chronic NOAEC	0.019 mg/L	Section 4.3.3.1
Invertebrates	Sensitive		Chronic LOAEC	0.25 mg/L	Section 4.3.3.3.2
	Tolerant		Estimated chronic LOAEC	20 mg/L	Section 4.3.3.3.2
Aquatic Plants					
Algae	Sensitive		NOAEC	0.0014 mg/L	Section 4.3.3.4
	Tolerant		NOAEC	1.0 mg/L	Section 4.3.3.4
Macrophytes	Sensitive		Estimated NOAEC	0.043 mg/L	Section 4.3.3.4
	Tolerant		Estimated NOAEC	0.31 mg/L	Section 4.3.3.4

¹¹All toxicity values for triclopyr BEE expressed as mg a.e./L.

Table 45: Trichloro-2-pyridinol (TCP) Toxicity Values for Aquatic Organisms

Group/Duration		Organism	Endpoint	Toxicity Value ^[1]	Reference
Aquatic Animals					
Acute					
Amphibians	Sensitive	No data		N/A	Section 4.3.3.2
	Tolerant	No data		N/A	
Fish	Sensitive	Adjusted acute NOAEC		0.18 mg/L	Section 4.3.3.1
	Tolerant	Estimated acute NOAEC		0.63 mg/L	
Invertebrates	Sensitive	Estimated acute NOAEC		0.55 mg/L	Section 4.3.3.3
	Tolerant	Estimated acute NOAEC		0.55 mg/L	Section 4.3.3.3
Longer-term					
Amphibians	Sensitive	No data		N/A	Section 4.3.3.2
	Tolerant	No data		N/A	
Fish	Sensitive	Chronic NOAEC		0.18 mg/L	Section 4.3.3.1
	Tolerant	Chronic NOAEC		0.18 mg/L	Section 4.3.3.1
Invertebrates	Sensitive	Chronic NOAEC		0.058 mg/L	Section 4.3.3.3
	Tolerant	Chronic NOAEC		0.058 mg/L	Section 4.3.3.3
Aquatic Plants					
Algae	Sensitive	5-day NOAEC		0.36 mg/L	Section 4.3.3.4
	Tolerant	5-day NOAEC		0.65 mg/L	Section 4.3.3.4
Macrophytes	Sensitive	No data		N/A	Section 4.3.3.4
	Tolerant	No data		N/A	Section 4.3.3.4

^[1] All toxicity values for TCP are in mg TC/L. The limited variability in the toxicity values for aquatic organisms appears to reflect the limited number of bioassays that are available. It is likely that lower and higher values would be used if more bioassays were available.

Table 46: Upper Bound HQs Following Terrestrial Applications of Triclopyr

Receptor	Upper Bound HQs at an Application Rate of 1 lb a.e./acre ^[1]			
	TEA		BEE	
	Acute	Chronic	Acute	Chronic
Large Mammal fruit to grass	0.9 to 11	13 to 53	0.9 to 11	13 to 53
Small Mammal, fruit to grass	0.1 to 1.6	1.8 to 7	0.1 to 1.6	1.8 to 7
Large Bird, fruit to grass	0.1 to 1.5	1.3 to 6	0.1 to 1.5	1.3 to 6
Small Bird, fruit to grass	1.0 to 14	11 to 54	1.0 to 14	11 to 54
Terrestrial Plants, runoff	4		2	
Terrestrial Plants, drift ^[5]	0.9 to 35		0.9 to 35	
Fish	0.01	0.003	0.3	0.004
Aquatic Invertebrates	0.02	0.002	0.7	0.0004
Algae	1	0.3	21	0.05 ^[2]
Aquatic macrophytes	480	120	0.7	0.002 ^[2]

^[1] See summary worksheets in Attachments 1 and 2.

^[2] Longer-term risks to aquatic macrophytes and algae following applications of triclopyr BEE should be based on the HQs for triclopyr TEA. See Section 4.4.3.4.2 for rationale.

Table 47: Triclopyr – Selected HQs for Mammals and Birds

Non-Accidental Acute Exposures				
Contaminated Fruit (Lowest Residue Rate)				
	Small mammal (20g)	4E-02	5E-03	0.1
	Larger Mammal (400g)	4E-02	5E-03	0.1
	Large Mammal (70g)	0.3	4E-02	0.9
	Small bird (10g)	0.3	4E-02	1.0
	Large Bird (4 kg)	3E-02	4E-03	0.1
Contaminated Vegetation (Short Grass - Highest Residue Rate)				
	Small mammal (20g)	0.3	3E-02	1.6
	Larger Mammal (400g)	0.3	3E-02	1.6
	Large Mammal	2	0.2	11
	Small bird (10g)	3	0.3	14
	Large Bird (4 kg)	0.3	3E-02	1.5
Chronic/Longer Term Exposures				
Contaminated Fruit (Lowest Residue Rate)				
	Small mammal (20g)	0.3	3E-02	1.8
	Larger Mammal (400g)	0.3	3E-02	1.8
	Large Mammal (70g)	2	0.2	13
	Small bird (10g)	1.8	0.2	11
	Large Bird (4 kg)	0.2	2E-02	1.3
Contaminated Vegetation (Short Grass - Highest Residue Rate)				
	Small mammal (20g)	0.7	3E-02	7
	Larger Mammal (400g)	0.7	3E-02	7
	Large Mammal (70g)	5	0.2	53
	Small bird (10g)	5	0.2	54
	Large Bird (4 kg)	0.5	2E-02	6

Source: Attachment 1, Worksheet G02a
 See Sections 4.4.2.1.1 (mammals) and 4.4.2.2 (birds) for discussion.

Table 48: TCP: Selected HQs for Mammals

Non-Accidental Acute Exposures			
Contaminated Fruit (Lowest Residue Rate)			
Small mammal (20g)	0.2	2E-02	0.7
Larger Mammal (400g)	4E-02	6E-03	0.2
Large Mammal (70g)	2E-02	3E-03	9E-02
Contaminated Vegetation (Short Grass - Highest Residue Rate)			
Small mammal (20g)	1.6	0.2	8
Larger Mammal (400g)	0.4	4E-02	1.8
Large Mammal	0.2	2E-02	1.0
Chronic/Longer Term Exposures			
Contaminated Fruit (Lowest Residue Rate)			
Small mammal (20g)	0.3	3E-02	1.3
Larger Mammal (400g)	7E-02	8E-03	0.3
Large Mammal (70g)	4E-02	4E-03	0.2
Contaminated Vegetation (Short Grass - Highest Residue Rate)			
Small mammal (20g)	0.9	4E-02	10
Larger Mammal (400g)	0.2	9E-03	2

Source: Attachment 3, Worksheet G02
See Sections 4.4.2.1.1.2 for discussion.